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Preface

Andrew T. Parsa, MD, PhD
Guest Editor

The treatment of vestibular schwannoma is optimally a collaborative effort between neurosurgeons and otolaryngologists. A multidisciplinary approach is beneficial to the outcome of management decisions that can range from serial observations to surgical intervention. The advent of more precise radiosurgery, with lower marginal dosing, has ushered in a new era of treatment paradigm.

This issue presents classic articles on treatment for acoustic neuroma patients. The revelation in these articles is that, as state-of-the-art of scientific and clinical aspects of acoustic neuroma continue to advance, the concepts of basic knowledge and expert skill presented by our predecessors remain central to the treatment of this disease.

Presentations of William House and Clough Shelton's middle fossa approach, Neal Cohen's retrosigmoid approach, Dale Brown and Ugo Fisch's transotic approach are as relevant and useful to surgeons in 2008 as they were when discussed a decade ago. Conservative management of acoustic neuroma continues to develop and is discussed here by Julian Nedzelski, David Schessel, Andrew Pfleiderer, Edward Kassel, and David Rowed. The cochlear and brainstem presentation has been updated with current concepts by Elizabeth Toh and William Luxford. Selection of surgical approach continues to be the crucial to the outcome for the patient and is presented in this issue with a timeless discussion by Robert Jackler and Lawrence Pitts. More recent discussions on surgical approaches and complications, radiosurgery and radiotherapy, and stereotactic radiation techniques are presented by our colleagues in otolaryngology, Marc Bennett and David Haynes, Ilya Likhterov, Robert Allbright and Samuel Selesnick, and Steven Abram, Paul Rosenblatt, and Stephen Holcomb, respectively. Absolutely essential information on microsurgical anatomy, imaging, nerve monitoring, and guiding patients through surgery are presented by Albert Rhoton and Helder Tedeschi, Hugh Curtin and William Hirsch, Charles Yingling and John Gardi, and Douglas Backous and Huong Pham, respectively. Through the words of these masters, each of us is reminded of the depth and breadth of their work, which continues to spur us on to advance our skills and techniques and continue to collaborate with each other. Collectively, we strive to continually improve functional outcomes for our patients with acoustic neuroma.

What does the future hold? The concept that "the treatment of patients should be

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dictated by tumor biology" is a fundamental tenet of oncology. However, we have yet to embrace this completely in the realm of acoustic neuroma. For example, patients who present with small tumors or with minimal symptoms are often treated empirically with surgery or radiation as a pre-emptive measure, to prevent growth and improve long-term function. Ongoing studies into molecular attributes of tumors that correlate with growth rate, or response to radiation, should yield important insights into what may be most appropriate for a particular patient.

Once we gain insights into novel treatment paradigms that better tailor our management, how will we disseminate this information to patients and providers? As with many diseases, patient advocacy has played a critical role in this regard. In particular, the Acoustic Neuroma Association (ANA) serves as a model patient advocacy group that has provided important information for 25 years to its members.

The activities of the ANA include the following:

1) The newsletter NOTES is published quarterly. ANA is committed to keeping its members informed with professionally authored medical information, self-help aids, personal accounts from acoustic neuroma patients, and support group updates.

2) Patient booklets deal with all aspects of acoustic neuroma. Single copies are free to members; larger quantities may be purchased.

3) A national symposium is presented every other year for acoustic neuroma patients, family members, and health care professionals.

4) Local support groups provide opportunities for patients to communicate and network with others.

5) Research results are produced and published on the effects of acoustic neuroma.

6) The Web site (www.ANAUSA.org) is maintained with an interactive ANA discussion forum extending the goals of making information and networking opportunities available to patients.

The informed practitioner should be aware of the landmark contributions in the field, stay up-to-date on future directions for therapy, and be able to refer patients to resources that facilitate a better understanding of both.

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Microsurgical Anatomy of Acoustic Neuroma

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Acoustic neuromas, as they expand, may involve a majority of the cranial nerves, cerebellar arteries, and parts of the brain stem. On the lateral side, in the meatus, they commonly expand by enlarging the meatus and may infrequently erode into the vestibule and cochlea. On the medial side, they compress the pons, medulla, and cerebellum. An understanding of microsurgical anatomy is especially important in preserving the facial and adjacent cranial nerves, which are the neural structures at greatest risk during acoustic neuroma removal. A widely accepted operative precept is that a nerve involved by tumor should be identified proximal or distal to the tumor, where its displacement and distortion are the least, before the tumor is removed from the involved segment of nerve. Considerable attention has been directed to the early identification of the facial nerve distal to the tumor at the lateral part of the internal acoustic canal, whether the operative route be through the middle fossa, labyrinth, or posterior meatal lip. Less attention has been directed to identification at the brain stem on the medial side of the tumor. These anatomic considerations are divided into sections dealing with the relationships at the lateral end of the tumor in the meatus and those on the medial end of the tumor at the brain stem.

Meatal relationships

The nerves in the lateral part of the internal acoustic meatus are the facial, the cochlear, and the inferior and superior vestibular nerves (Fig. 1). The position of the nerves is most constant in the lateral portion of the meatus, which is divided into a superior and an inferior portion by a horizontal ridge, called either the *transverse* or *the falciform crest*. The facial and the superior vestibular nerves are superior to the crest. The facial nerve is anterior to the superior vestibular nerve and is separated from it at the lateral end of the meatus by a vertical ridge of bone, called the *vertical crest*. The vertical crest is also called *Bill's bar* in recognition of William House's role in focusing on the importance of this crest in identifying the facial nerve in the lateral end of the canal [6]. The cochlear and inferior vestibular nerves run below the transverse crest with the cochlear nerve being located anteriorly. Thus the lateral meatus can be considered to be divided into four portions, with the facial nerve being anterior-superior; the cochlear nerve, anterior-inferior; the superior vestibular nerve, posterior-superior; and the inferior vestibular nerve, posterior-inferior.

The anatomy of the region offers the opportunity for three basic approaches to the tumor in the meatus and cerebellopontine angle. One is directed through the middle cranial fossa and the roof of the meatus. Another is directed through the labyrinth and posterior surface of the temporal bone. The third is directed through the posterior cranial fossa and posterior meatal lip. The anatomy presented by all three approaches is reviewed here.

Middle fossa approach

In the middle fossa approach, the meatus is approached from above, through a temporal craniotomy located anterior to the ear and above the zygoma (Figs. 2 and 3) [2,11]. The dura under the temporal lobe is elevated from the floor of the

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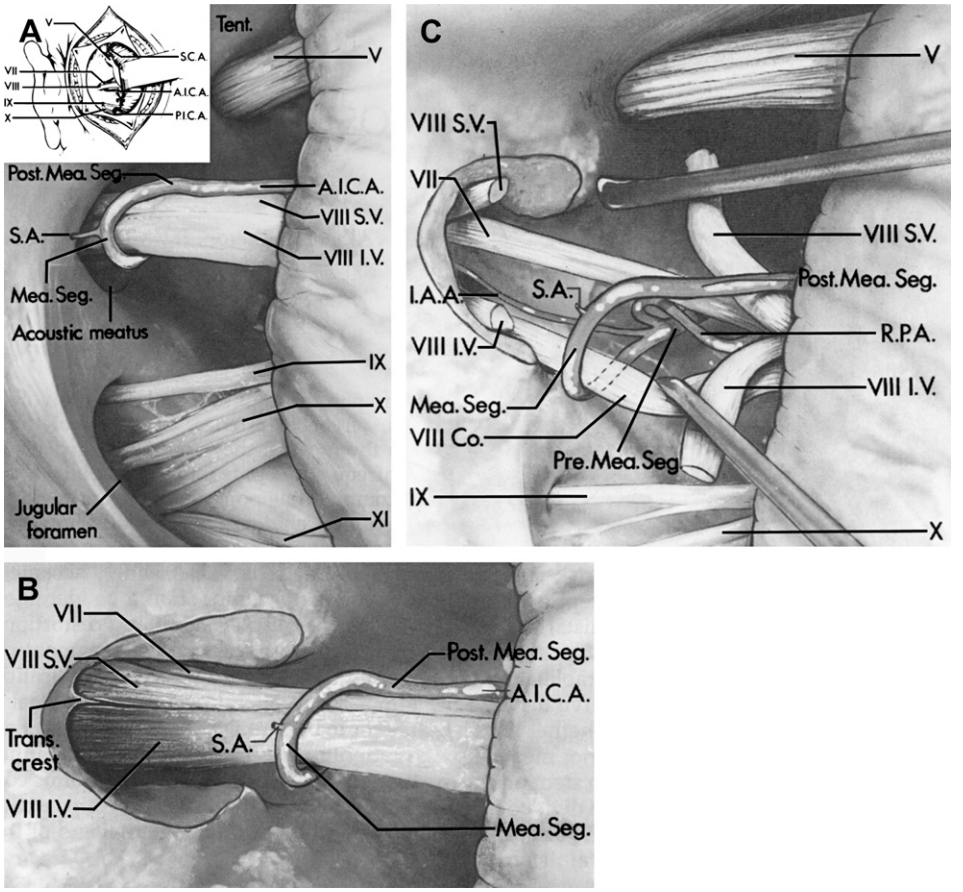


Fig. 1. Posterior view into the left cerebellopontine angle and internal acoustic meatus. Insert shows the orientation. (*A*) The tentorium (Tent.) is above the trigeminal nerve (V). The facial and vestibulocochlear nerves enter the internal acoustic meatus. The posterior surface of the vestibulocochlear nerve is formed by the inferior (VIII I.V.) and superior vestibular (VIII S.V.) nerves. The glossopharyngeal (IX), vagus (X), and spinal accessory nerves (XI) enter the jugular foramen. The premeatal segment of the anterior inferior cerebellar artery (A.I.C.A.) is not visible because it is anterior to the nerves. The meatal segment (Mea. Seg.) passes posterior to the nerves and gives rise to the subarcuate artery (S.A.). The postmeatal segment (Post. Mea. Seg.) passes above the nerves. The insert shows the superior cerebellar artery (S.C.A.) above the trigeminal nerve, and the posterior inferior cerebellar artery (P.I.C.A.) below the glossopharyngeal nerve. (*B*) The posterior wall of the internal acoustic canal has been removed. The facial nerve (VII) is anterior to the superior vestibular nerve. The subarcuate artery had to be divided to gain access to the posterior wall of the acoustic canal. The transverse crest (Trans. Crest) separates the superior and inferior vestibular nerves at the lateral end of the canal. (*C*) The superior and inferior vestibular nerves have been divided to expose the facial and cochlear nerves (VIII Co.). The premeatal segment (Pre. Mea. Seg.) gives origin to the internal auditory (I.A.A.) and recurrent perforating (R.P.A.) arteries. The initial segment of the recurrent perforating artery loops toward the meatus before turning medially to reach the side of the brainstem. (*From* Martin RG, Grant JL, Peace D, et al. Microsurgical relationships of the anterior inferior cerebellar artery and the facial-vestibuloco-chlear nerve complex. Neurosurgery 1980;6:483–507; with permission.)

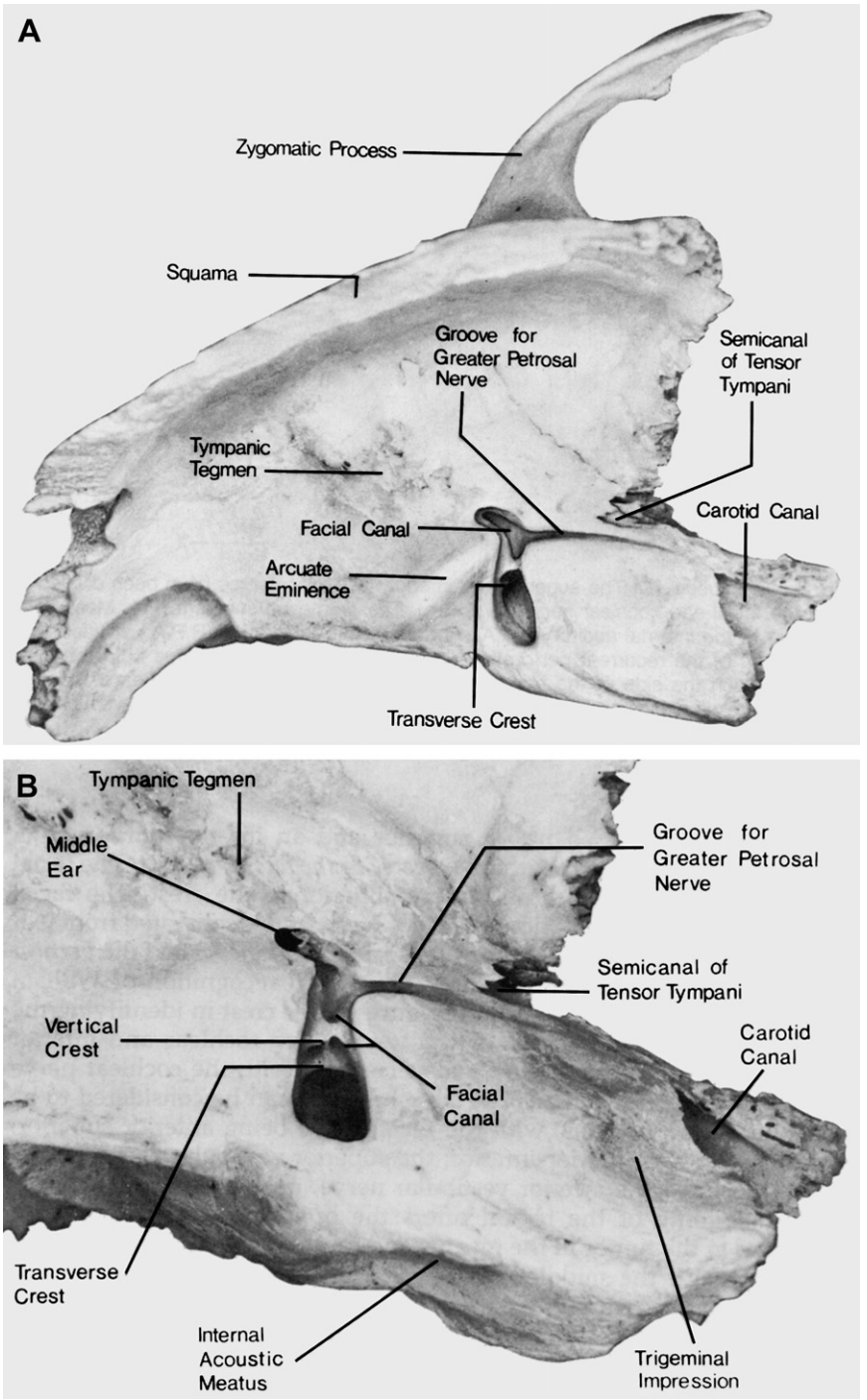
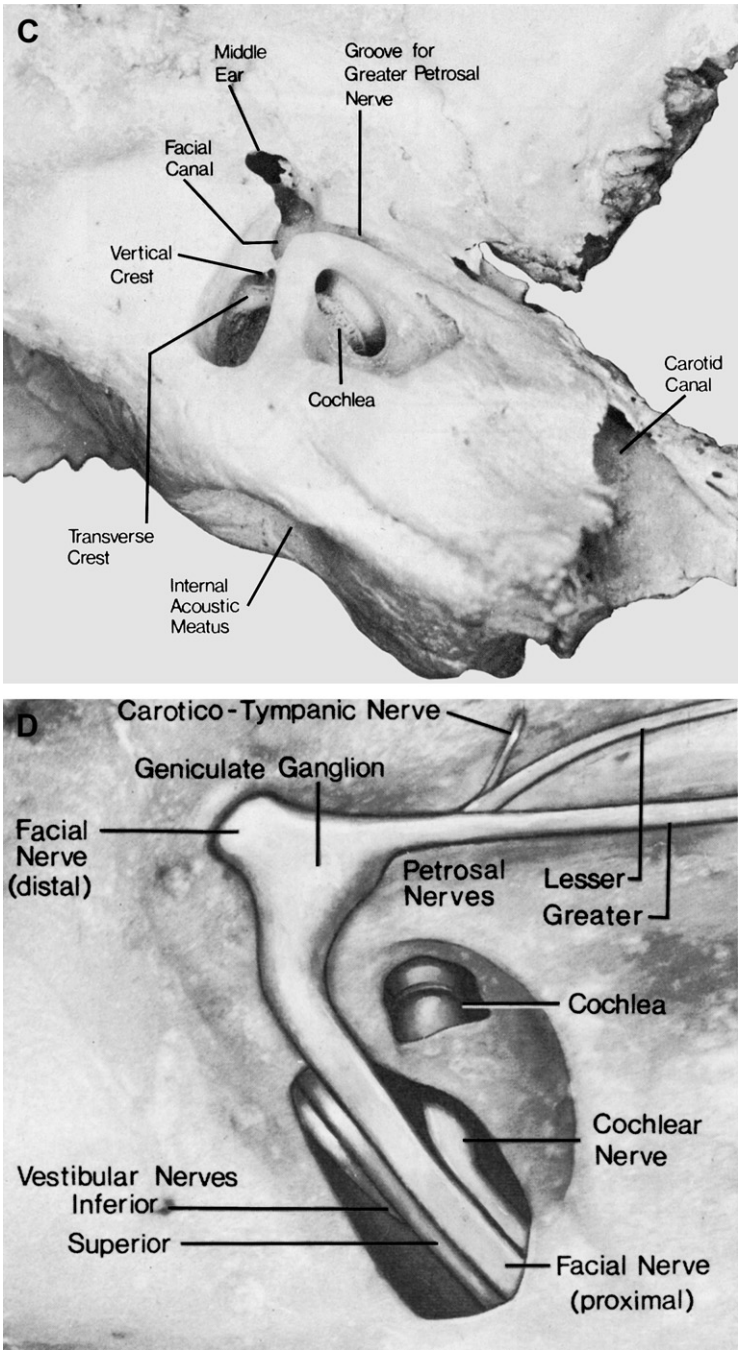


Fig. 2. Middle fossa approach to internal acoustic meatus. (*A*) Superior view of left temporal bone. Facial canal exposed proximal and distal to junction with canal of greater petrosal nerve. Internal auditory meatus unroofed. (*B*) Enlarged view. Vertical and transverse crests exposed at lateral end of internal auditory meatus. (*C*) Cochlea exposed in angle between the groove for the greater petrosal nerve and labyrinthine part of the facial canal. (*D*) Specimen with nerves intact. Dura and bone above facial canal and internal acoustic meatus removed. Cochlear nerve exposed medial to geniculate ganglion. (*E*) Three semicircular canals, nerves in meatus, and carotid artery exposed. (*From* Pait TG, Harris FS, Paullus WS, et al. Microsurgical anatomy and dissection of the temporal bone. Surg Neurol 1977;8:363–91; with permission.)



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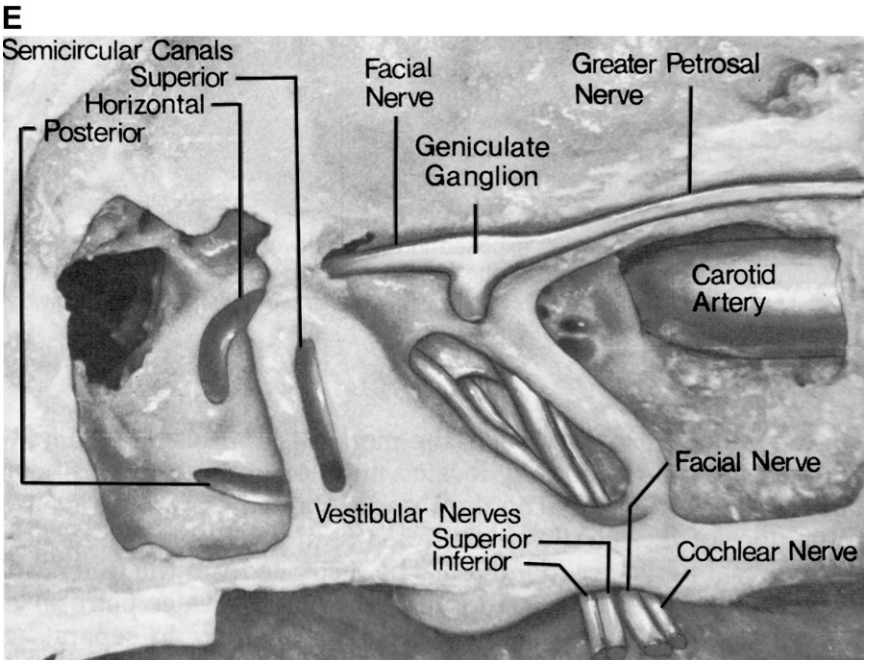


Fig. 2 (*continued*)

middle cranial fossa until the arcuate eminence and greater petrosal nerve are identified. The distance from the inner table of the skull to the facial hiatus, through which the greater petrosal nerve passes, ranges from 1.3 to 2.3 cm (average 1.7 cm) [17]. In separating the dura from the floor of the middle fossa, one should remember that bone may be absent over all or part of the geniculate ganglion. In a previous study of 100 temporal bones, we found that all or part of the geniculate ganglion and genu of the facial nerve was exposed in the floor of the middle fossa in 15 bones (15%) [19]. In 15 other specimens, the geniculate ganglion was completely covered, but there was no bone extending over the greater petrosal nerve. The greatest length of greater petrosal nerve covered by bone was 6 mm. More than 50% of the specimens had less than 2.5 mm of greater petrosal nerve covered.

It is also important to remember that the petrous segment of the carotid artery may be exposed without a covering of bone in the floor of the middle fossa deep to the greater petrosal nerve. In a previous study, we found that a 7-mm length of petrous carotid artery may be exposed without a bony covering in the area below where the greater petrosal nerve passes below the lateral margin of the trigeminal ganglion [5,12]. The foramen spinosum and middle meningeal artery and the foramen ovale and 3rd trigeminal division are situated at the anterior margin of the extradural exposure. The extradural exposure can usually be completed without obliterating the middle meningeal artery at the foramen spinosum. The tensor tympany muscle and eustachian tube, although not exposed in this approach, are located beneath the floor of the middle fossa roughly parallel to and in front of the horizontal portion of the petrous carotid (see Fig. 3).

In completing the middle fossa approach, bone is removed over the greater petrosal nerve to expose the geniculate ganglion and genu of the facial nerve. From here the labyrinthine portion of the facial nerve is followed to the lateral end of the internal auditory canal by removing bone. The lateral part of the bone removal is limited posteriorly by the superior semicircular canal, which is located a few millimeters behind and is oriented parallel to the labyrinthine segment of the facial nerve. The anterior edge of the exposure is limited by the cochlea, which sits only a few millimeters anterior to the site of bone removal in the angle between the labyrinthine portion of the facial nerve and the greater petrosal nerve. It is important that the cochlea and semicircular

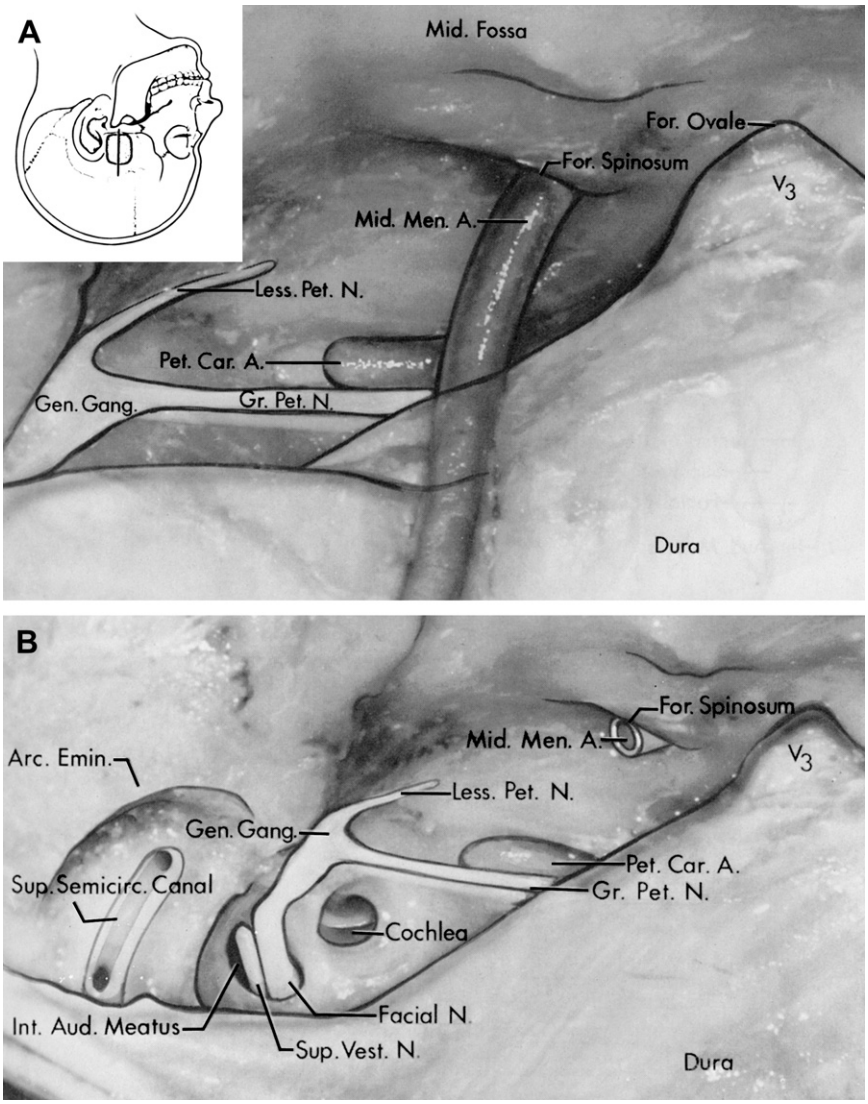


Fig. 3. Anatomy of middle fossa approach to the internal acoustic meatus. (*A*) The insert of the upper left shows the site of the scalp incision (*straight line*) and the site of the craniectomy (*stippled area*). The dura has been elevated on the floor of the middle cranial fossa (Mid. Fossa). In this case, the bone was absent over the geniculate ganglion (Gen. Gang.) and the petrous segment of the carotid artery (Pet. Car. A.) and both were exposed in the floor of the middle fossa. The dura has been elevated from the floor of the middle cranial fossa to expose the middle meningeal artery (Mid. Men. A.) at the foramen spinosum (For. Spinosum), and the third trigeminal division (V3) at the foramen ovale (For. Ovale). The geniculate ganglion is exposed by identifying the greater petrosal nerve and following it to the geniculate ganglion. The lesser petrosal nerve (Less Pet. N.) is also in the exposure. (*B*) The middle meningeal artery was divided at the foramen spinosum and bone was removed over the internal auditory meatus (Int. Aud. Meatus) to expose the superior vestibular (Sup. Vest. N.) and facial nerves (Facial N). Bone has been removed to expose the cochlea in the angle between the greater petrosal nerve and labyrinthine segment of the facial nerve. The superior semicircular canal (Sup. Semicirc. Canal) lies behind the superior vestibular nerve in the area deep to the arcuate eminence (Arc. Emin.). (*C*) The dura has been elevated to expose the gasserian ganglion (Gass. Gang.). The greater petrosal nerve courses above the petrous segment of the carotid artery. Bone has been removed in the floor of the middle fossa to expose the tensor tympany muscle (Tens. Tymp. M.) and eustachian tube (Eust. Tube). The superior vestibular and facial nerves are separated at the lateral end of the meatus by the vertical crest (Vert. Crest—Bill's Bar). (*D*) The facial and cochlear nerves (Cochlear N.) are in the anterior half and the superior and inferior vestibular nerves (Inf. Vest. N.) are in the posterior half of the meatus. The superior vestibular and facial nerves are separted from the inferior vestibular and cochlear nerves by the transverse crest (Trans. Crest). The cochlea sets in the angle between the facial and greater petrosal nerve.

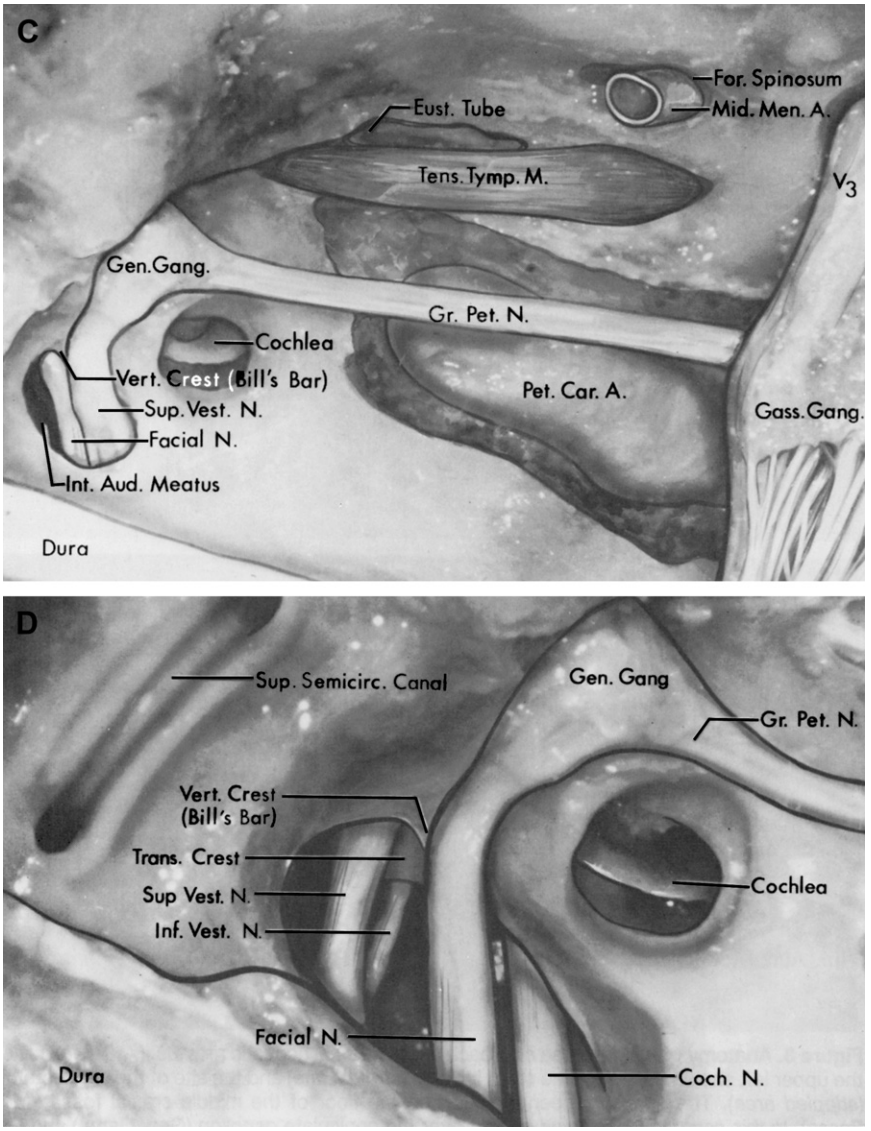


Fig. 3 (*continued*)

canals not be damaged in this approach because it is used only for removing small tumors in which there is an opportunity to preserve hearing. The vertical crest, which is identified at the upper edge of the lateral end of the internal acoustic canal, provides a valuable landmark for identifying the facial nerve. In the final stage of bone removal, the upper wall of the internal auditory canal is removed to expose the dura lining the entire superior surface of the internal auditory canal from the vertical crest to the porus acusticus.

Translabyrinthine approach

In the translabyrinthine approach, the meatus and cerebellopontine angle are approached through a mastoidectomy and labyrinthectomy (Figs. 4 and 5) [6,11]. There are two goals of bone removal in this approach: (1) to remove enough bone to be able to identify the nerves lateral to the tumor as they course through the internal auditory canal and by the transverse and vertical crests and (2) to expose the dura on the posterior

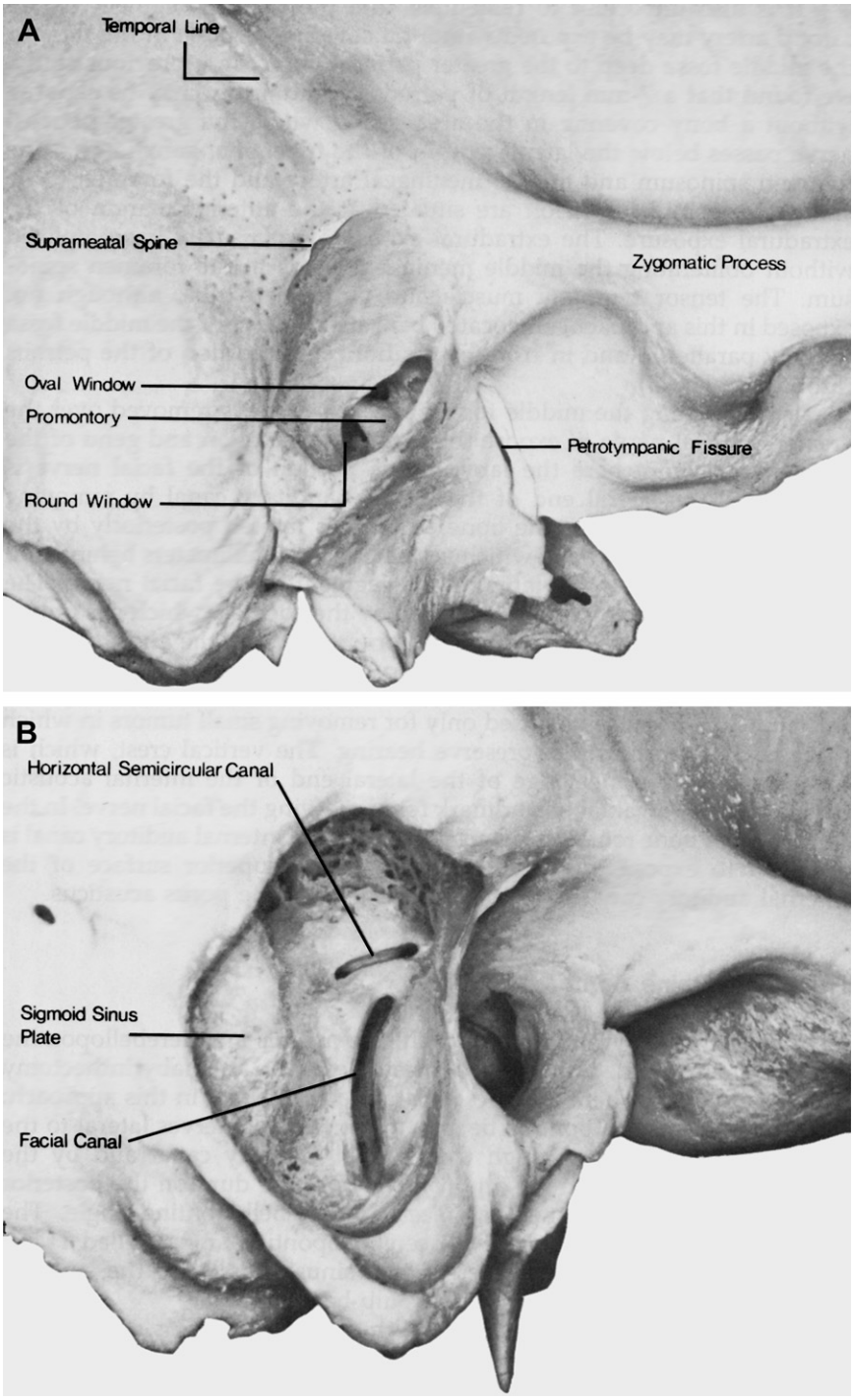
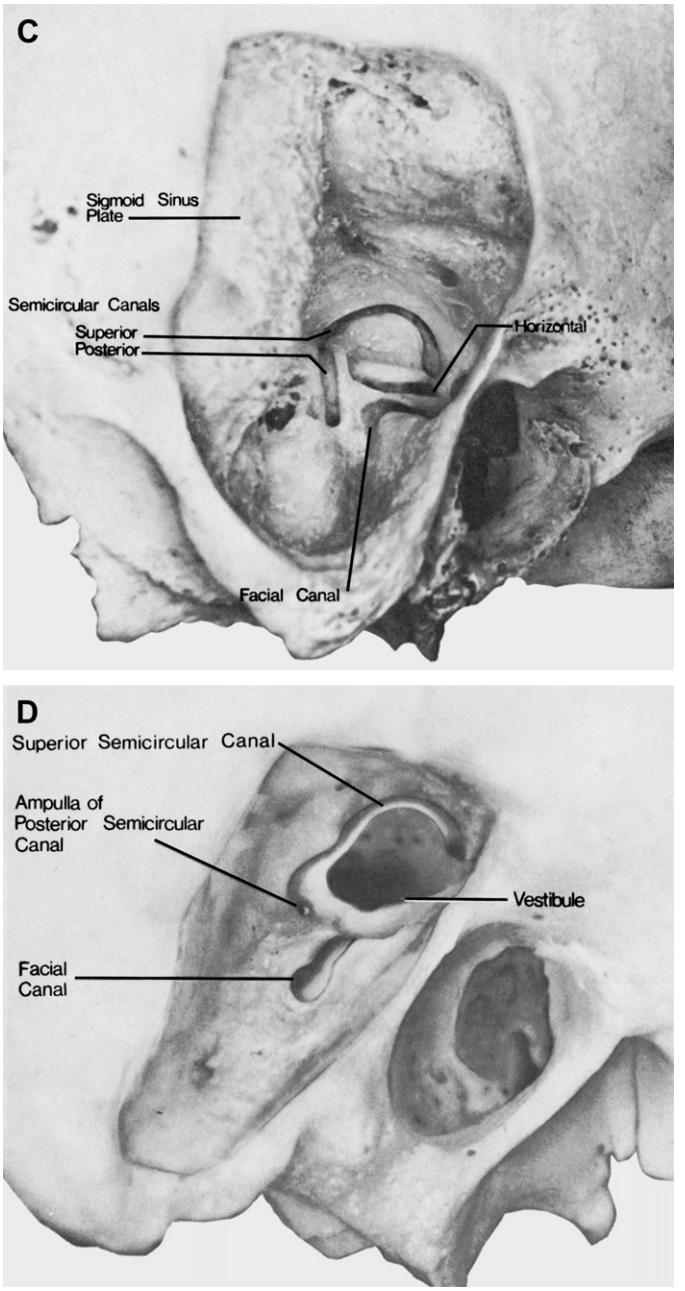


Fig. 4. (*A*) Translabyrinthine approach to the internal acoustic meatus. Lateral view of right temporal bone showing external acoustic meatus, oval and round windows and promontory. (*B*) Sigmoid sinus plate, facial canal and semicircular horizontal canal exposed. (*C*) Three semicircular canals and the facial canal exposed. (*D*) Canals removed to open into vestibule. (*E*) Specimen with nerves intact. Upper part of internal acoustic meatus exposed by drilling through the vestibule and ampulla of posterior canal. (*From* Pait TG, Harris FS, Paullus WS, et al. Microsurgical anatomy and dissection of the temporal bone. Surg Neurol 1977;8:363–91; with permission.)



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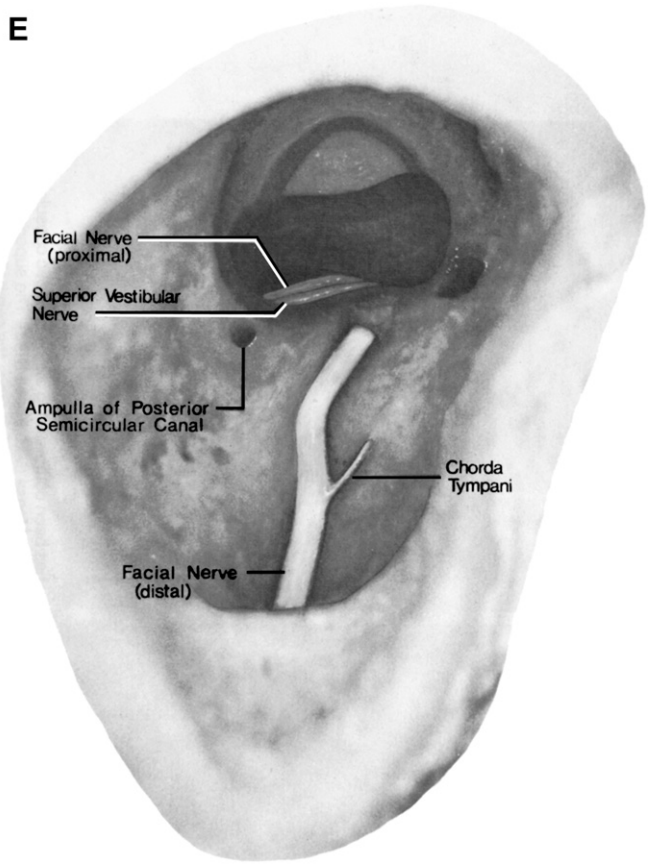


Fig. 4 (*continued*)

face of the temporal bone that faces the cerebellopontine angle. The triangular patch of dura facing the cerebellopontine angle, called *Trautman's triangle,* extends from the sigmoid sinus laterally to the superior petrosal sinus above and the jugular bulb below.

In the translabyrinthine exposure, the mastoid cortex is opened, and the exposure is directed through the triangular gateway between the facial nerve anteriorly, the sigmoid sinus posteriorly, and floor of the middle fossa above. Bone is removed to skeletonize the dura covering the sigmoid sinus; middle fossa; facial nerve; the angle between the sigmoid sinus and middle fossa dura, called the *sinodural angle;* and the upper surface of the jugular bulb. The mastoidectomy is carried down to the horizontal semicircular canal, which provides the landmark for identifying the other canals and the facial nerve.

The labyrinthectomy portion of the procedure involves removing the semicircular canals and vestibule to expose the dura lining the internal auditory canal. In the process of removing the semicircular canals, the dura of the middle fossa above the internal acoustic meatus is skeletonized, and the dura on the posterior fossa plate behind the canal is exposed. After opening and removing the canals, the vestibule is opened and removed, and the dura lining the posterior half of the internal auditory canal is exposed. Care is required to avoid injury to the facial nerve as it courses below the horizontal canal and the ampullae of the posterior canal and around the superolateral margin of the vestibule. Further bone removal at the lateral end of the meatus exposes the transverse and vertical and facial nerves. In removing bone behind the internal acoustic meatus, it is important to remember that the jugular bulb may bulge upward behind the posterior semicircular canal or internal auditory meatus. The vestibular aqueduct and endolymphatic sac are opened and removed as bone is

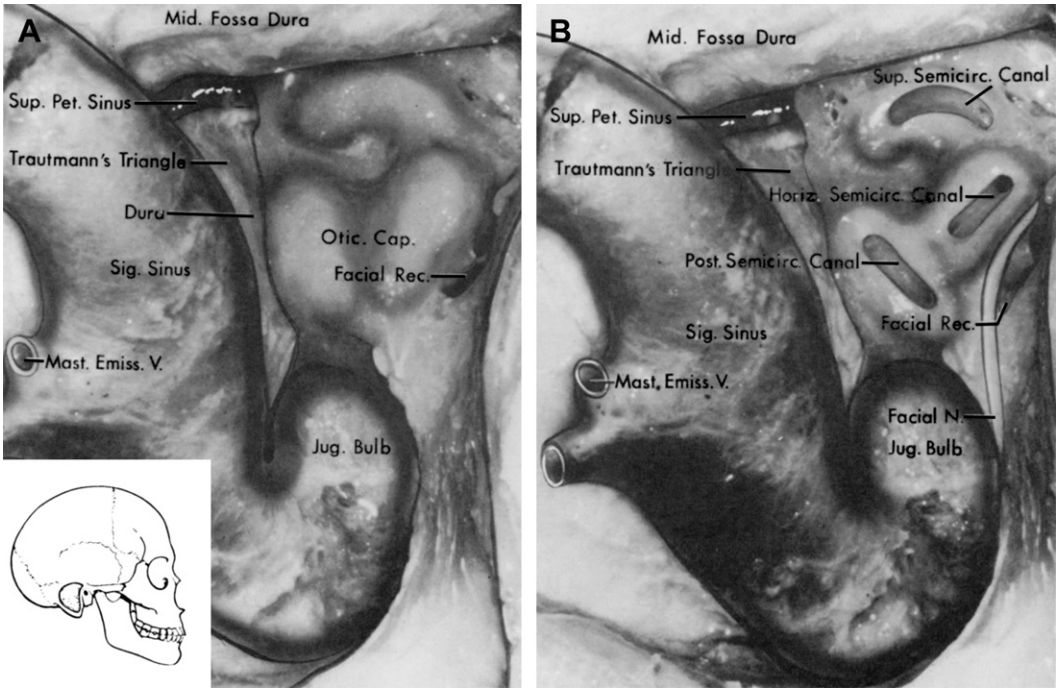
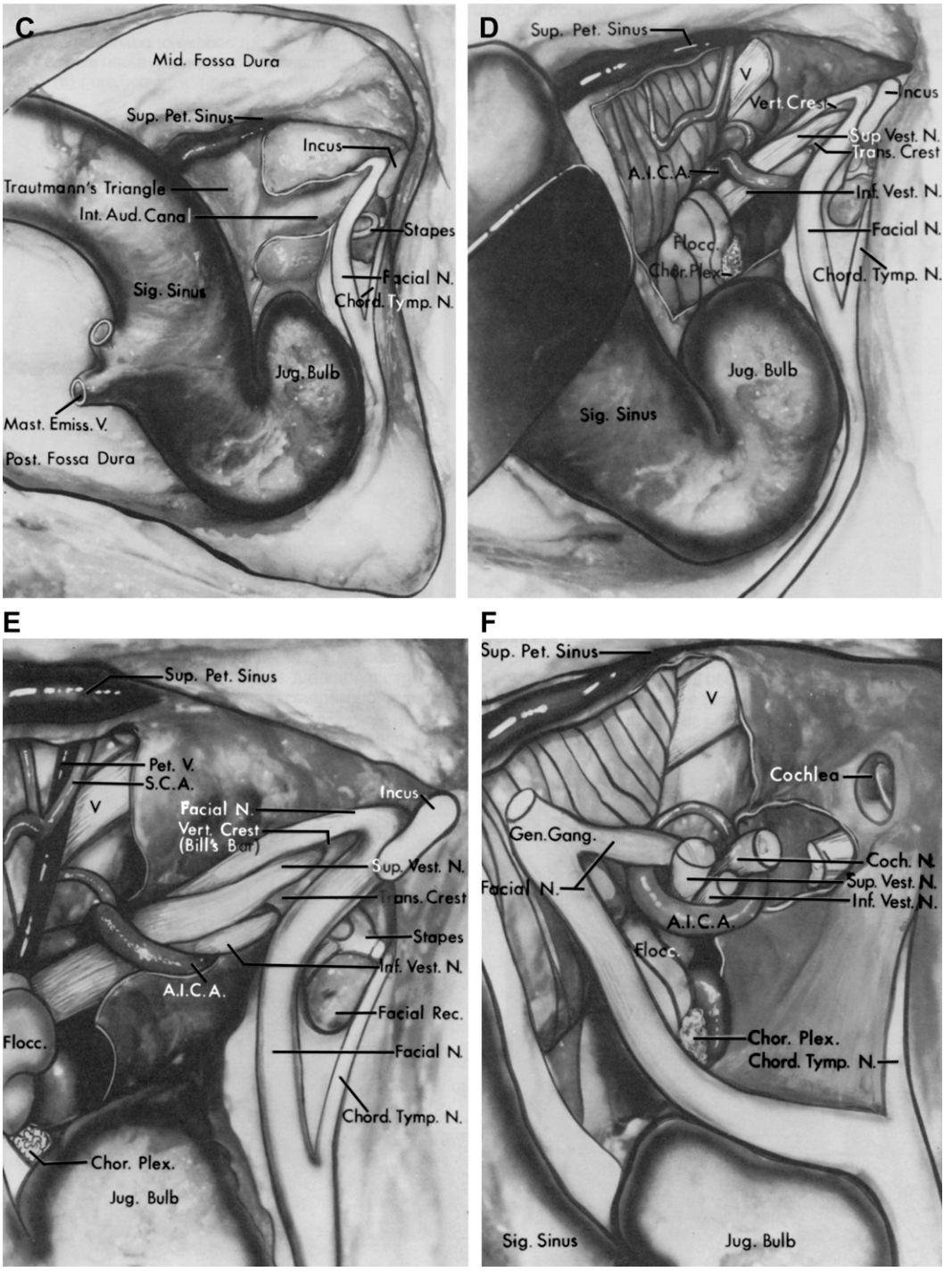


Fig. 5. (*A*) Translabyrinthine and transcochlear exposure. A mastoidectomy has been completed to expose the otic capsule (Otic Cap.), the sigmoid sinus (Sig. Sinus), jugular bulb (Jug. Bulb) and the dura lining the floor of the middle cranial fossa (Mid. Fossa Dura). The superior petrosal sinus (Sup. Pet. Sinus) extends medially from the sinodural angle. Trautman's triangle is the triangular portion of dura that faces the cerebellopontine angle. This triangle is located between the superior petrosal sinus above, the sigmoid sinus laterally, and the jugular bulb below. Mastoid emissary veins (Mast. Emiss. V.) join the sigmoid sinus. The facial recess (Facial Rec.) is located in front of the facial nerve. (*B*) Additional bone has been removed to expose the horizontal (Horiz. Semicirc. Canal), posterior (Post. Semicirc. Canal), and superior semicircular canals (Sup. Semicirc Canal). The facial nerve passes below the horizontal canal. (*C*) The semicircular canals and vestibule have been removed to expose the dura lining the internal auditory canal (Int. Aud. Canal). The chorda tympany (Chor. Tymp. N.) arises from the facial nerve. The posterior fossa dura (Post. Fossa Dura) is behind the sigmoid sinus. (*D*) The dura lining the internal auditory canal and facing the cerebellopontine angle has been removed to expose the trigeminal (V), facial, and vestibulocochlear nerves. The transverse crest (Trans. Crest) separates the superior (Sup. Vest. N.) and inferior vestibular nerves (Inf. Vest. N.). The vertical crest (Vert. Crest) separates the facial and superior vestibular nerves. The flocculus (Flocc.) is behind the vestibulocochlear nerve. Choroid plexus (Chor. Plex) protrudes from the foramen of Luschka. The anterior inferior cerebellar artery (A.I.C.A.) courses around the facial and vestibulocochlear nerves. The superior cerebellar artery (S.C.A.) courses above the trigeminal nerve. (*E*) Enlarged view. Petrosal veins (Pet. V.) join the superior petrosal sinus. (*F*) The facial nerve has been transposed posteriorly after dividing the greater petrosal nerve distal to the geniculate ganglion. The superior and inferior vestibular and cochlear nerves (Coch. N.) have been divided. Additional bone has been removed to expose the cochlea. The distal segment of the cochlear nerve penetrates the lateral end of the meatus to enter the cochlea. (*G*) The bone surrounding the cochlea has been removed to complete the transcochlear exposure. The bone removal extends to the lateral margin of the clivus and the inferior petrosal sinus (Inf. Pet. Sinus). The abducens nerve (VI) ascends beside the basilar artery (Bas. A.). The glossopharyngeal (IX) and vagus (X) nerves are in the lower margin of the exposure behind the vertebral artery (Vert. A.). (*H*) Another dissection. The jugular bulb has been removed to expose the glossopharyngeal, vagus, and accessory (XI) nerves as they course through the medial side of the jugular foramen. The internal carotid artery (Car. A.) and eustachian tube (Eust. Tube) are in the anterior part of the exposure.



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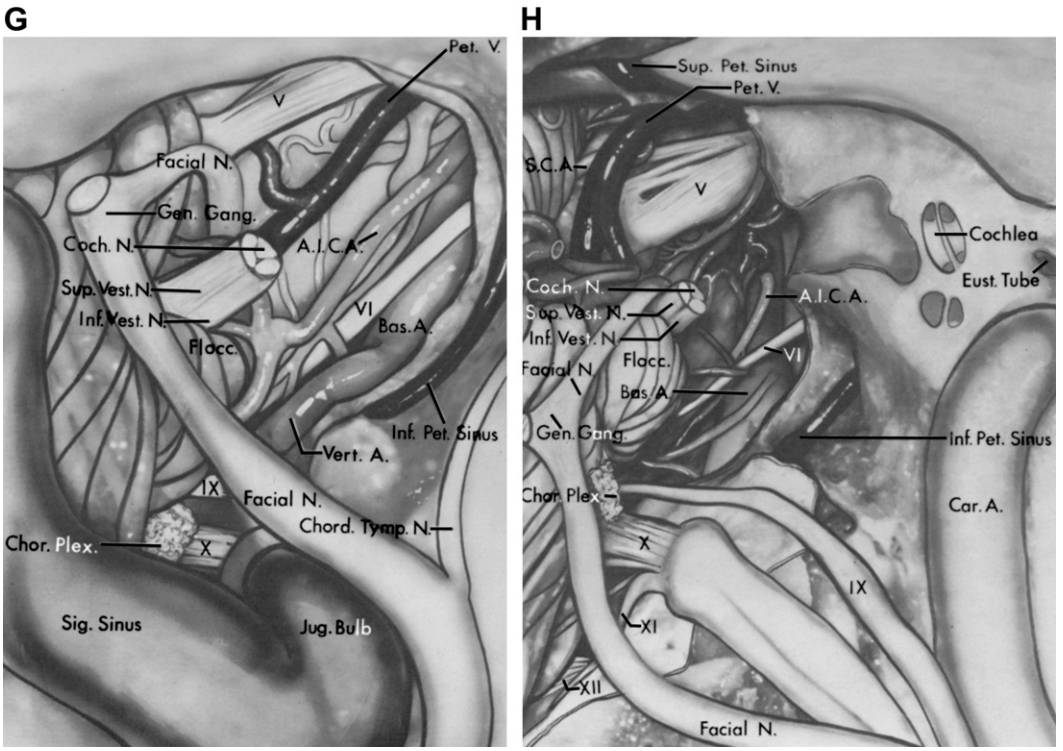


Fig. 5 (*continued*)

removed between the meatus and the jugular bulb. The cochlear cana-liculus is seen deep to the vestibular aqueduct as bone is removed in the area between the meatus and the jugular bulb. The lower end of the cochlear canaliculus is situated just above the area where the glosso-pha-ryngeal nerve enters the medial side of the jugular foramen.

The subarcuate artery, or the anterior-inferior cerebellar artery, may be encountered in the dura of Trautman's triangle. Commonly the subarcuate artery, which arises from the anterior-inferior cerebellar artery, passes through the dura on the upper posterior wall of the meatus as a fine stem, but, on occasion, the subarcuate artery along with its origin from the anterior-inferior cerebellar artery may be incorporated into the dura and dip into the subarcuate fossa, on the posterior face of the temporal bone.

Retrosigmoid approach

The retrosigmoid approach to the meatus is directed through a cranial opening situated just behind the sigmoid sinus and down the plane between the posterior face of the temporal bone and the anterior surface of the cerebellum (Fig. 6) [13,14,15]. In removing the posterior meatal wall, it is often necessary to sacrifice the subarcuate artery because it passes through the dura on the posterior meatal wall to reach the subarcuate fossa [8]. This artery usually has a sufficiently long stem that its obliteration does not risk damage to the anterior-inferior cerebellar artery from which it arises. In a few cases, however, the subarcuate artery and the segment of the anterior-inferior cerebellar arteries from which it arises are incorporated into the dura covering the subarcuate fossa. In this situation, the dura and artery have to be separated together from the posterior meatal lip in preparation for opening the meatus from posteriorly.

The posterior wall of the internal auditory canal may be removed before opening the arachnoid membrane around the tumor. The preservation of the arachnoid membrane that lies posterior to an acoustic neuroma during the removal of the posterior meatal wall with a drill prevents bone dust from entering the subarachnoid space. The posterior semicircular canal and its common crus

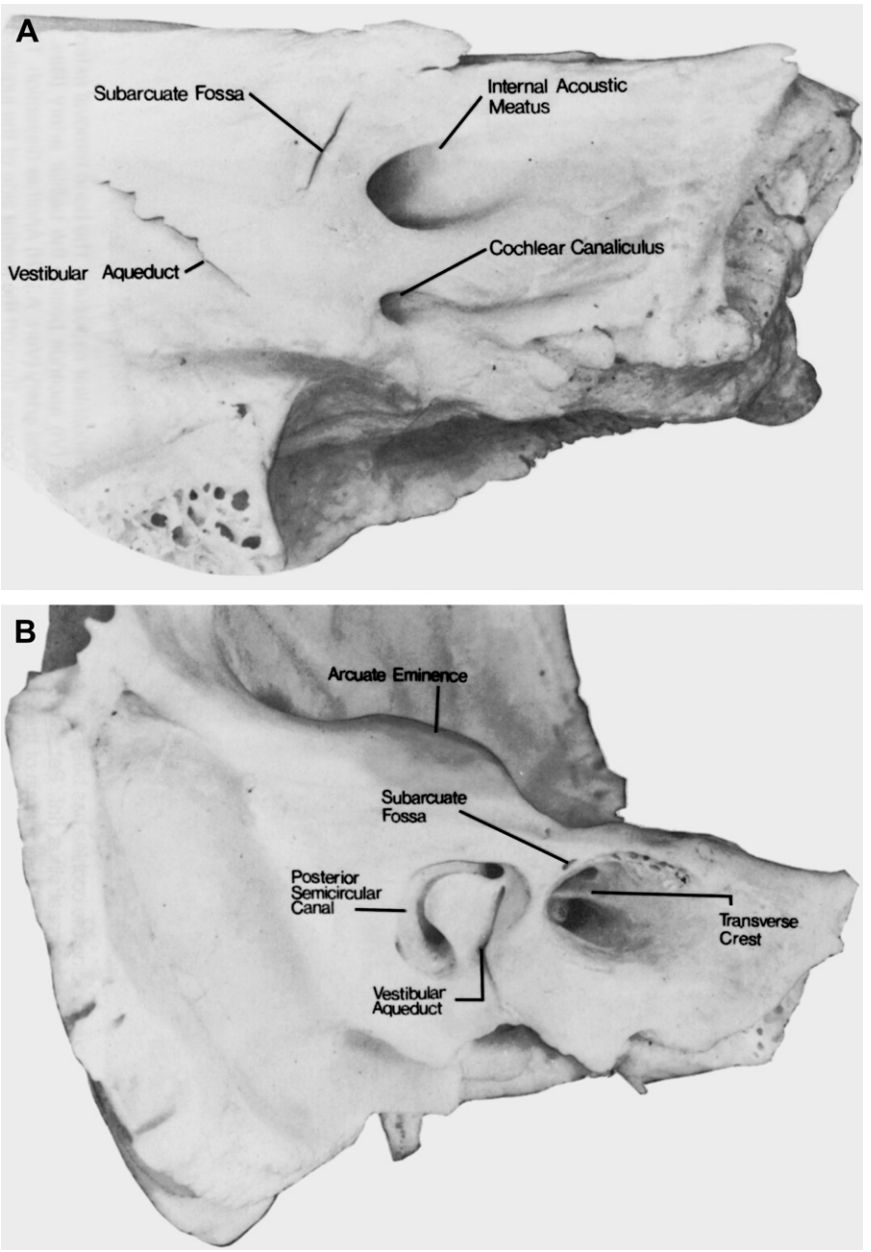


Fig. 6. Posterior fossa approach to internal acoustic meatus. (*A*) Posterior view of left temporal bone showing the subarcuate fossa, vestibular aqueduct, and cochlear canaliculus. (*B*) Bone removed to expose anteromedial part of transverse crest, vestibular aqueduct, and posterior semicircular canal. (*C*) Relationship of the three semicircular canals to the internal acoustic meatus. Horizontal canal exposed by removing bone anterior to posterior canal and lateral to superior canal. (*D*) Bone removed to show the lateral end of internal acoustic meatus. Transverse crest separates facial canal and superior vestibular area from cochlear and inferior vestibular areas. The vertical crest (Bill's bar) separates the facial canal and the superior vestibular area. (*E*) Enlarged view of the lateral end of canal. (*F*) Temporal bone with$_{v}$ nerves preserved. Posterior wall of internal auditory meatus removed to expose the nerves. (*G*) Bone removed lateral to the internal auditory canal to expose the posterior and superior semicircular canals. (*From* Pait TG, Harris FS, Paullus WS, et al. Microsurgical anatomy and dissection of the temporal bone. Surg Neurol 1977;8:363–91; with permission.)

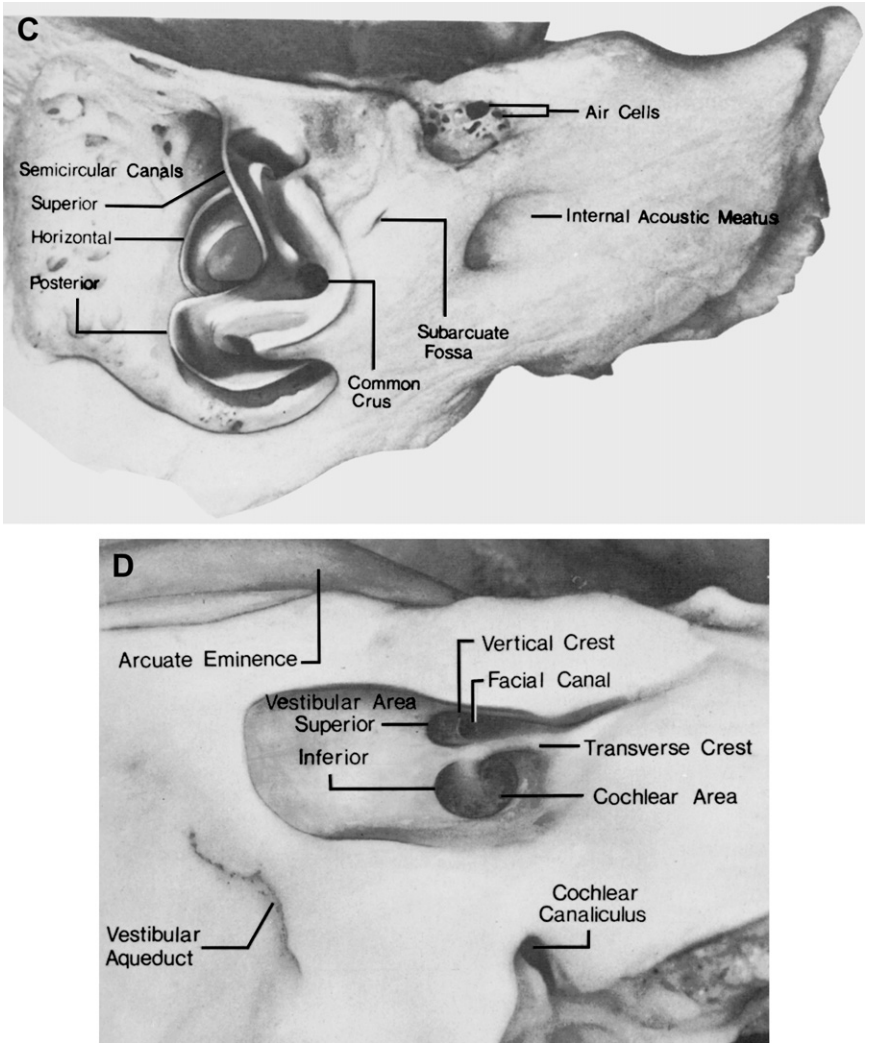


Fig. 6 (*continued*)

with the superior canal, both of which are situated just lateral to the posterior meatal lip, should be preserved in exposing the meatal contents if there is the possibility of preserving hearing because hearing will be lost if they are damaged. Care is also required to avoid injury to the vestibular aqueduct, which is situated inferolateral to the meatal lip and the endolymphatic sac, which expands under the dura on the posterior surface of the temporal bone inferolateral to the posterior meatal lip. The endolymphatic sac may be entered in removing the dura from the posterior meatal lip. There is little danger of encountering the cochlear canaliculus, which has a more anterior course below the internal auditory canal. An anomaly that may block access to the posterior meatal lip is an unusually high projection of the jugular bulb. Mastoid air cells are commonly encountered in the posterior meatal lip.

After removing the posterior wall of the meatus, the dura lining the meatus is opened to expose its contents. The facial nerve is identified near the origin of the facial canal at the anterior-superior quadrant of the meatus rather than in a more medial location, where the direction of displacement is variable. It is easy to expose the

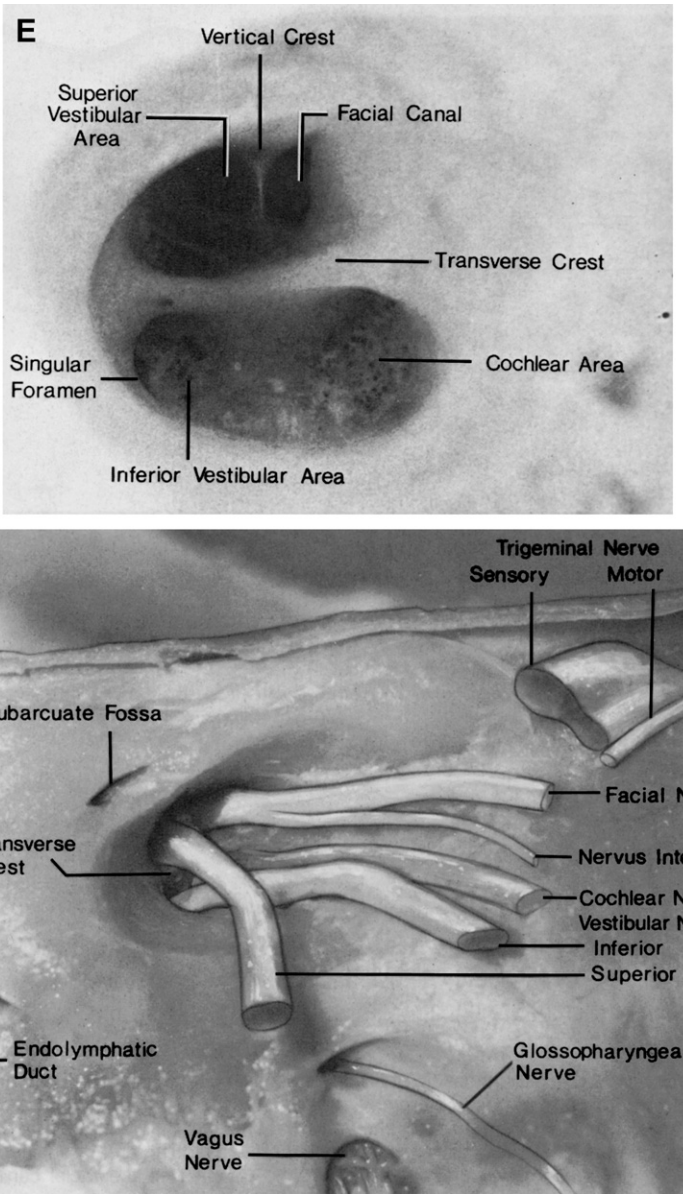


Fig. 6 (*continued*)

vestibule if the tumor extends into the vestibule by drilling along the posterior and superior semicircular canals.

Nervus intermedius

The filaments of the nervus intermedius are also stretched around an acoustic neuroma (Fig. 7). The nervus intermedius is usually described as a component of the facial nerve. Relatively little note has been taken of the fact that it may be closely bound to the vestibulocochlear nerve for a variable distance before it enters the brain stem and that in the cerebellopontine angle it may consist of as many as four rootlets. In previous studies, we found that the nervus intermedius is divisible into three parts: (1) a proximal segment that adheres closely to

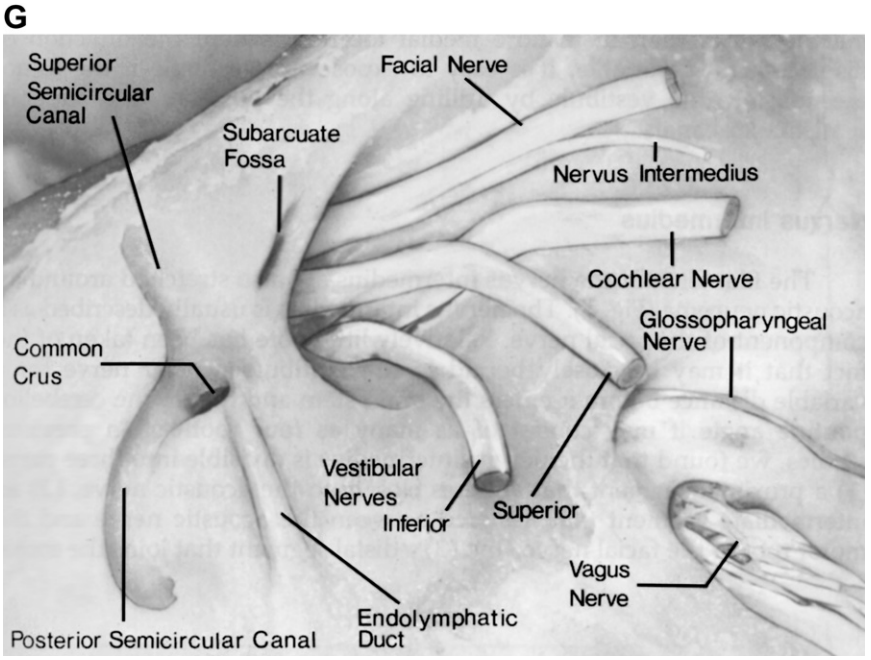


Fig. 6 (*continued*)

the acoustic nerve, (2) an intermediate segment that lies free between the acoustic nerve and the motor root of the facial nerve, and (3) a distal segment that joins the motor root to form the facial nerve [18]. Twenty-two percent of the nerves were adherent to the acoustic nerve for 14 mm or more (the entire course of the nerve in the posterior cranial fossa) and could be found as separate structures only after opening the internal acoustic meatus (see Fig. 7). In most instances, the nerve was a single trunk, but in some cases it was composed of as many as four rootlets. A single large root most frequently arose at the brain stem anterior to the superior vestibular nerve and, in the meatus, lay anterior to the superior vestibular nerve. When multiple rootlets are present, they may arise along the whole anterior surface of the acoustic nerve; however, they usually converge immediately proximal to the junction with the facial motor root to form a single bundle that lies anterior to the superior vestibular nerve.

Brain stem relationships

There is a consistent set of neural, arterial, and venous relationships at the brain stem that facilitate identification of the nerves on the medial side of the tumor [16].

Neural relationships

The neural structures most intimately related to the medial side of an acoustic neuroma are the pons, medulla, and cerebellum (Figs. 8 and 9). The landmarks on these structures that are helpful in guiding the surgeon to the junction of the facial nerve with the brain stem are the pontome-dullary sulcus; the junction of the glossopharyngeal, vagus, and accessory nerves with the medulla; the foramen of Luschka and its choroid plexus; and the flocculus.

Pontomedullary sulcus

The facial nerve arises from the brain stem near the lateral end of the pontomedullary sulcus. This sulcus extends along the junction of the pons and the medulla and ends immediately in front of the foramen of Luschka and the lateral recess of the 4th ventricle (see Fig. 8). The facial nerve arises in the pontomedullary sulcus 1 to 2 mm anterior to the point at which the vestibulocochlear nerve joins the brain stem at the lateral end of the sulcus. The interval between the vestibulocochlear and facial nerves is greatest at the level of

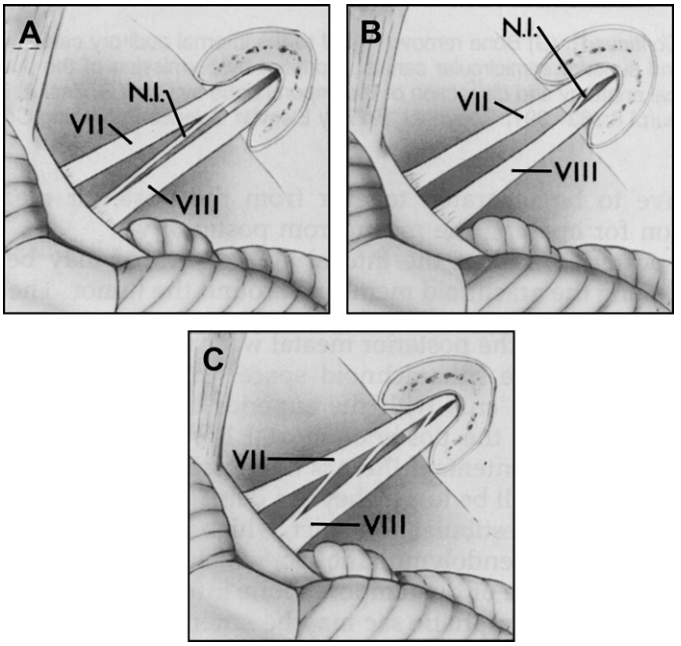
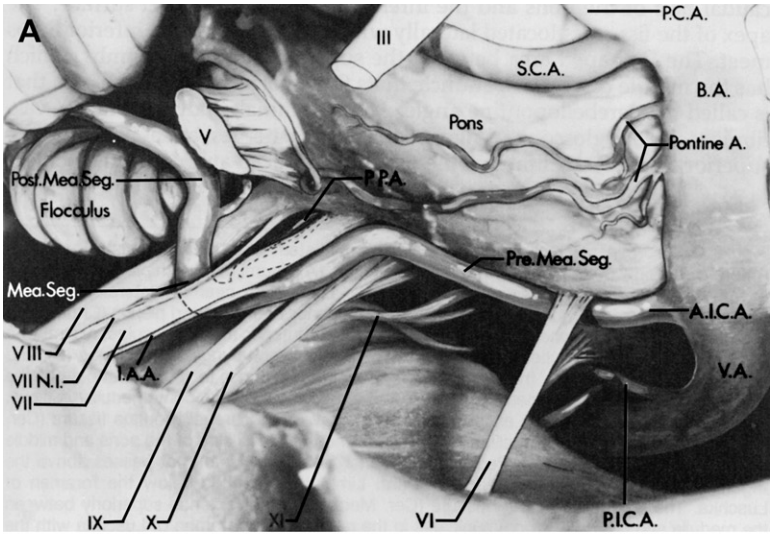
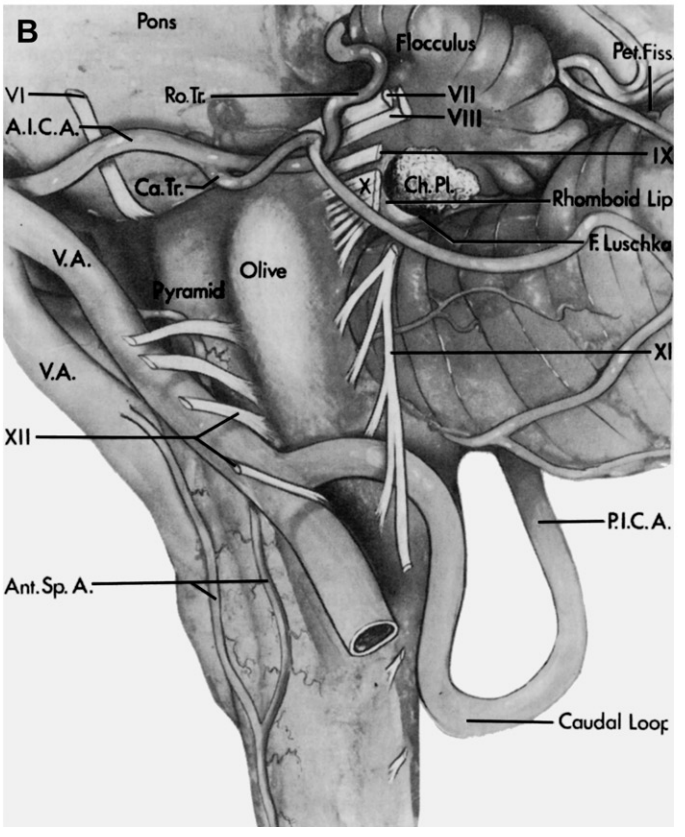
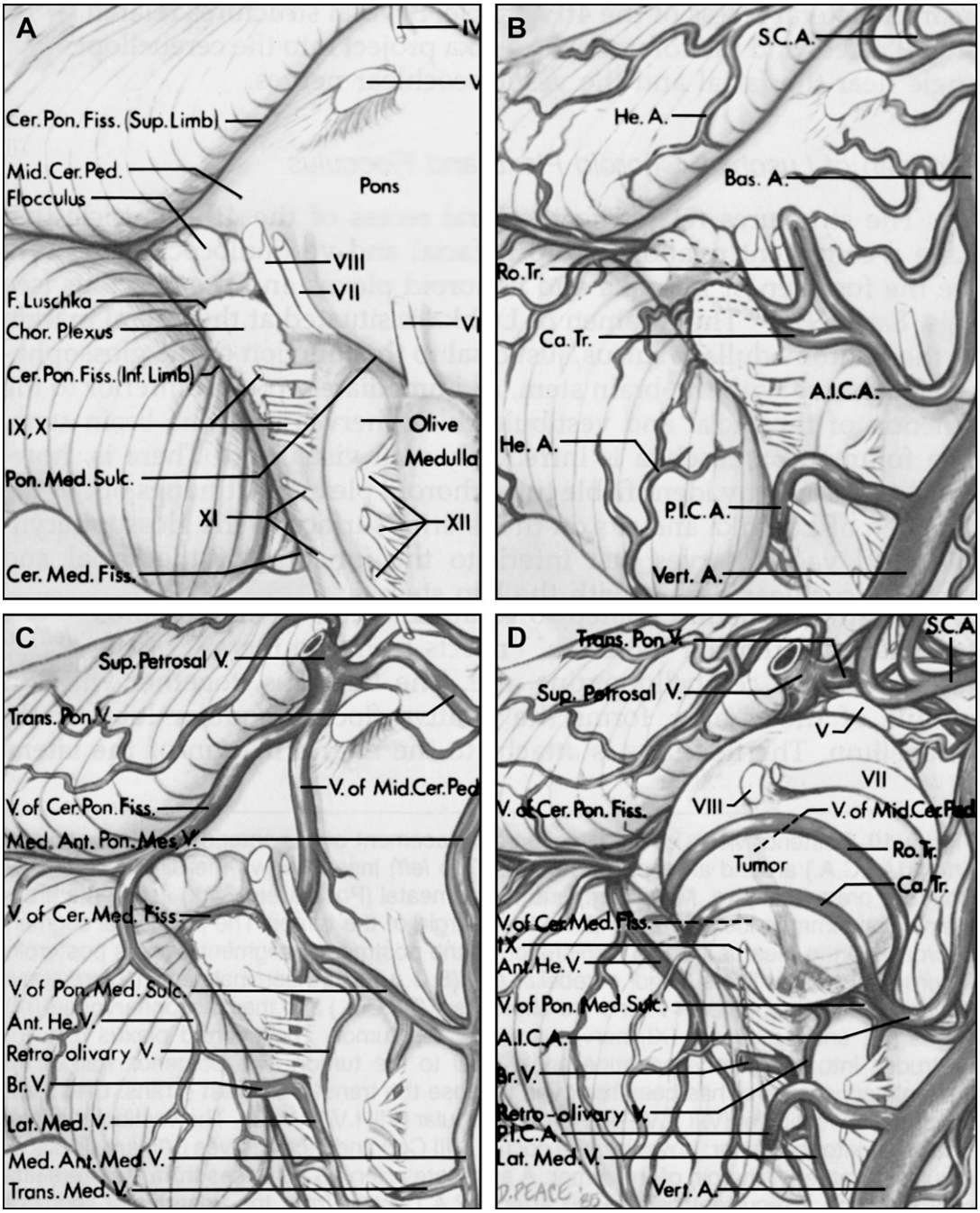


Fig. 7. View of cerebellopontine angle from above to show relationship of nervus intermedius to seventh (VII) and eighth (VIII) nerves. (*A*) Most common relationship. Nervus intermedius (N.I.) is joined to ventral surface of eighth nerve for a few millimeters adjacent to brain stem, then has a free segment in the cerebellopontine angle in its course to join the facial motor root. (*B*) Pattern present in 20% of nerves studied; free segment is entirely in meatus. (*C*) Nervus intermedius consists of three free segments. Two are in cerebellopontine angle, and one is in meatus. Nervus intermedius in A could be exposed in cerebellopontine angle without drilling off posterior lip of meatus. In B, it could not be found in the angle but only in the meatus.

Fig. 8. (*A*) Anterosuperior view of the right cerebellopontine angle. The right anterior inferior cerebellar artery (A.I.C.A.) arises from the basilar artery (B.A.), courses below the abducens nerve (VI), and passes between the nervus intermedius (VII N.I.) and the facial nerve (VII) anteriorly, and the vestibulocochlear nerve (VIII) posteriorly. The premeatal segment (Pre. Mea. Seg.) of the anterior inferior cerebellar artery passes below the abducens nerve. The meatal segment (Mea. Seg.) passes between the vestibulocochlear nerve and the nervus intermedius, and the postmeatal segment (Post. Mea. Seg.) passes above the flocculus. A recurrent perforating (R.P.A.) and an internal auditory artery (I.A.A.) arise from the meatal segment. Pontine arteries (Pontine A.) arise from the right side of the basilar artery. The oculomotor (III) and trochlear (IV) nerves are above and the trigeminal (V) nerve is below the superior cerebellar artery (S.C.A.). The right posterior inferior cerebellar artery (P.I.C.A.) arises from the right vertebral artery (V.A.) and courses by the glossopharyngeal (IX), vagus (X), accessory (XI) and hypoglossal (XII) nerves. The posterior cerebral artery (P.C.A.) is at the upper margin of the exposure. (*B*) Anterolateral view of the brain stem. A line drawn along the origin of the glossopharyngeal, vagus, and accessory nerves along the postolivary sulcus will pass through the site at which the facial nerve exits the brain stem. The facial nerve usually exits the brain stem 2 to 3 mm rostral to where the glossopharyngeal nerve enters the brain stem. The rootlets of the hypoglossal nerves arise along the preolivary sulcus. The glossopharyngeal and vagus nerves course anterior to the lateral recess of the fourth ventricle and the choroid plexus (Ch. PI.) protruding from the foramen of Luschka (F. Luschka). The facial vestibulocochlear nerves arise above the glossopharyngeal nerve near the peduncle of the flocculus. The anterior inferior cerebellar artery gives rise to a rostral (Ro. Tr.) and a caudal (Ca. Tr.) trunk. The rostral trunk courses above the flocculus to reach the petrosal fissure (Pet. Fiss.). The caudal trunk passes interiorly. The anterior spinal arteries (Ant. Sp. A) arise from the vertebral arteries. (*A from* Martin RG, Grant JL, Peace DA, et al. Microsurgical relationships of the anterior inferior cerebellar artery and the facial-vestibulocochlear nerve complex. Neurosurgery 1980;6:438–507; with permission. *B from* Lister JR, Rhoton AL Jr, Matsushima T, et al. Microsurgical anatomy of the posterior inferior cerebellar artery. Neurosurgery 1982;10:170–99; with permission.)


A
P.C.A.
III
S.C.A.
B.A.
Pons
V
Pontine A.
Post. Mea. Seg.
Flocculus
P.P.A.
Pre. Mea. Seg.
Mea. Seg.
A.I.C.A.
VIII
VII N.I.
VII
I.A.A.
V.A.
IX
X
XI
VI
P.I.C.A.


B
Pons
Flocculus
Pet. Fiss.
VI
Ro. Tr.
VII
VIII
A.I.C.A.
IX
Ca. Tr.
X
Ch. Pl.
Rhomboid Lip
F. Luschka
V.A.
Olive
Pyramid
XI
V.A.
XII
P.I.C.A.
Ant. Sp. A.
Caudal Loop


A
IV
V
Cer. Pon. Fiss. (Sup. Limb)
Mid. Cer. Ped.
Flocculus
Pons
VIII
VII
F. Luschka
Chor. Plexus
VI
Cer. Pon. Fiss. (Inf. Limb)
IX, X
Olive
Medulla
Pon. Med. Sulc.
XI
XII
Cer. Med. Fiss.
B
S.C.A.
He. A.
Bas. A.
Ro. Tr.
Ca. Tr.
A.I.C.A.
He. A.
P.I.C.A.
Vert. A.
C
Sup. Petrosal V.
Trans. Pon. V.
V. of Mid. Cer. Ped.
V. of Cer. Pon. Fiss.
Med. Ant. Pon. Mes. V.
V. of Cer. Med. Fiss.
V. of Pon. Med. Sulc.
Ant. He. V.
Retro-olivary V.
Br. V.
Lat. Med. V.
Med. Ant. Med. V.
Trans. Med. V.
D
Trans. Pon. V.
S.C.A.
Sup. Petrosal V.
V
VII
VIII
V. of Cer. Pon. Fiss.
V. of Mid. Cer. Ped.
Tumor
Ro. Tr.
Ca. Tr.
V. of Cer. Med. Fiss.
IX
Ant. He. V.
V. of Pon. Med. Sulc.
A.I.C.A.
Br. V.
Retro-olivary V.
P.I.C.A.
Lat. Med. V.
Vert. A.
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the pontomedullary sulcus and decreases as these nerves approach the meatus.

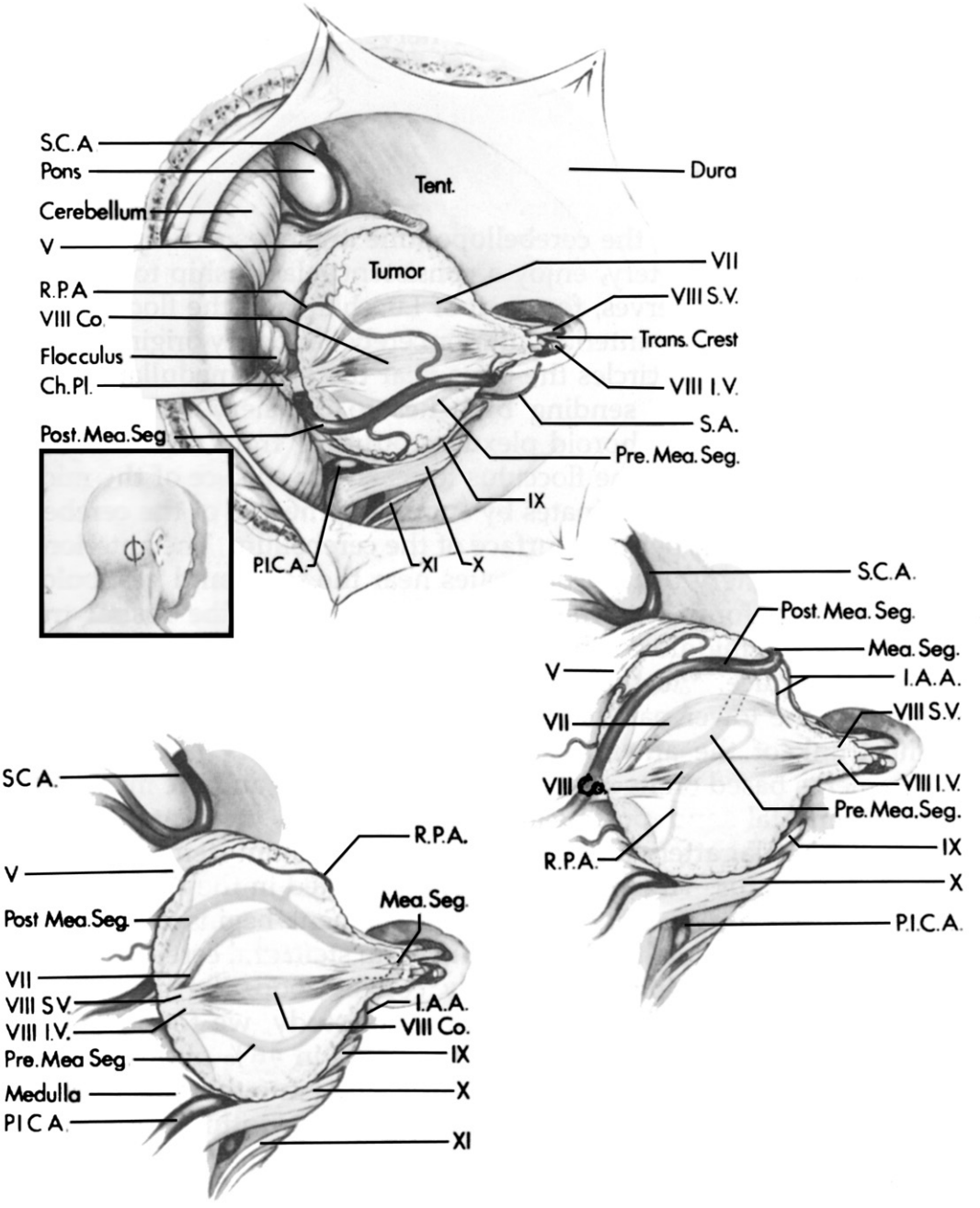
Glossopharyngeal, vagus, and accessory nerves

The facial nerve enjoys a consistent relationship to the junction of the glossopharyngeal, vagus, and accessory nerves with the lateral side of the medulla (see Figs. 8 and 9). The facial nerve arises 2 to 3 mm above the most rostral rootlet contributing to these nerves. A helpful way of visualizing the point where the facial nerve exits from the brain stem, even when displaced by tumor, is to project an imaginary line along the medullary junction of the rootlets forming the glossopharyngeal, vagal, and accessory nerves, upward through the pontomedullary junction. This line, at a point 2 to 3 mm above the junction of the glossopharyngeal nerve with the medulla, passes through the pontomedullary junction at the site where the facial nerve exits from the brain stem. The glossopharyngeal and vagus nerves are seen and should be carefully protected below the lower margin of the tumor in both the translabyrinthine and the retrosig-moid approaches.

Cerebellar–brain stem fissures

Acoustic neuromas are closely related to the cerebellopontine and cerebellomedullary fissures, the clefts formed by the folding of the cerebellum around the pons and medulla (see Fig. 9) [10].

Fig. 9. Neurovascular relationships on the brain stem side of an acoustic neuroma. Anterolateral view of the right cerebellopontine angle. (*A*) Neural relationships. The facial (VII) and vestibulocochlear (VIII) nerves arise from the brain stem near the lateral end of the pontomedullary sulcus (Pon. Med. Sulc), anterior-superior to the choroid plexus (Chor. Plex.) protruding from the foramen of Luschka (F. Luschka), anterior to the flocculus, rostral to a line drawn along the junction of the rootlets of the glossopharyngeal (IX), vagus (X), and accessory (XI) nerves with the brain stem, and slightly posterior to the rostral pole of the inferior olive. The abducent nerve (VI) arises in the medial part of the pontomedullary sulcus. The hypoglossal rootlets (XII) arise anterior to the olive. The cerebellopontine fissure (Cer. Pon. Fiss) formed by the cerebellum wrapping around the lateral side of the pons and middle cerebellar peduncle (Mid. Cer. Ped) has a superior limb (Sup. Limb) that passes above the trigeminal nerve (V), and an inferior limb (Inf. Limb) that extends below the foramen of Luschka. The cerebellomedullary fissure (Cer. Med. Fiss.) that extends superiorly between the medulla and cerebellum communicates in the region of the foramen of Luschka with the cerebellopontine fissure. The trochlear nerve (IV) is above the trigeminal nerve. (*B*) Arterial relationships. The anterior inferior cerebellar artery (A.I.C.A.) arises from the basilar artery (Bas. A.) and divides into rostral (Ro. Tr.) and caudal (Ca. Tr.) trunks. The rostral trunk, which is usually the larger of the two trunks, courses below the facial and vestibulocochlear nerves, and then above the flocculus to reach the surface of the middle cerebellar peduncle. The posterior inferior cerebellar artery (P.I.C.A.) arises from the vertebral artery (Vert. A.) and passes first between the hypoglossal rootlets, and then between the vagus and accessory nerves on its way to the cerebellar hemisphere. The superior cerebellar artery (S.C.A.) passes above the trigeminal nerve. The cerebellar arteries give rise to hemispheric branches (He. A.). (*C*) Venous relationships. The veins that converge on the junction of the facial and vestibulocochlear nerves with the brain stem are the veins of the pontomedullary sulcus (V. of Pon. Med. Sulc), cerebellomedullary fissure (V. of Cer. Med. Fiss), middle cerebellar peduncle (V. of Mid. Cer. Ped.) and the retro-olivary (Retro-olivary V.) and lateral medullary veins (Lat. Med. V.). The vein of the cerebellopontine fissure (V. of Cer. Pon. Fiss.) that passes above the flocculus on the middle cerebellar peduncle is formed by the anterior hemispheric veins (Ant. He. V.) that arise on the cerebellum. Transverse pontine (Trans. Pon. V.) and transverse medullary (Trans. Med. V.) veins cross the pons and medulla. The median anterior medullary (Med. Ant. Med. V.) and median anterior pontomesencephalic veins (Med. Ant. Pon. Mes. V.) ascend on the anterior surface of the medulla and pons. The veins of the middle cerebellar peduncle and the cerebellopontine fissure and a transverse pontine vein join to form a superior petrosal vein (Sup. Pet. V.) that empties into the superior petrosal sinus. A bridging vein (Br. V.) passes below the vagal rootlets toward the jugular foramen. (*D*) Neurovascular relationships of an acoustic neuroma. The tumor arises from the vestibulocochlear nerve and displaces the facial nerve anteriorly, the trigeminal nerve superiorly, and the vagus and glossopharyngeal nerves inferiorly. The facial nerve, although displaced by the tumor, enters the brain stem along the lateral margin of the pontomedullary sulcus, rostral to the glossopharyngeal and vagus nerves, anterior to the flocculus, and rostral to the choroid plexus protruding from the foramen of Luschka. The rostral trunk of the anterior inferior cerebellar artery, after passing below the tumor, returns to the surface of the middle cerebellar peduncle above the flocculus. The veins displaced around the medial side of the tumor are the veins of the middle cerebellar peduncle, cerebellomedullary fissure, cerebellopontine fissure, and pontomedullary sulcus and the retro-olivary and lateral medullary veins. (*From* Rhoton AL Jr. Microsurgical anatomy of the brain stem surface facing an acoustic neuroma. Surg Neurol 1986;25:326–39; with permission.)


S.C.A
Pons
Cerebellum
V
R.P.A
VIII Co.
Flocculus
Ch.Pl.
Post.Mea.Seg
P.I.C.A.
Tent.
Tumor
Dura
VII
VIII S.V.
Trans. Crest
VIII I.V.
S.A.
Pre. Mea. Seg.
IX
XI
X
S.C.A.
Post. Mea. Seg.
Mea. Seg.
I.A.A.
V
VII
VIII S.V.
VIII Co.
VIII I.V.
Pre. Mea. Seg.
R.P.A.
IX
X
P.I.C.A.
SC A.
R.P.A.
V
Mea. Seg.
Post Mea. Seg.
VII
VIII S.V.
I.A.A.
VIII I.V.
VIII Co.
Pre. Mea Seg
IX
Medulla
X
PI C A.
XI

The cerebellopontine fissure is a V-shaped fissure formed by the folding of the petrosal surface of the cerebellum around the lateral side of the pons and middle cerebellar peduncle. The petrosal surface is the cerebellar surface that faces the posterior surface of the petrous bone and is the cerebellar surface compressed by an acoustic neuroma. The cerebellopontine fissure has a superior limb situated between the rostral half of the pons and the superior part of the petrosal surface and an inferior limb located between the caudal half of the pons and the inferior part of the petrosal surface. The apex of the fissure is located laterally where the superior and inferior limbs meet. The V-shaped area between the superior and inferior limbs, which has the middle cerebellar peduncle in its floor, corresponds to the area that is called the cerebellopontine angle. The trigeminal, abducent, facial, ves-tibulocochlear, glossopharyngeal, and vagus nerves arise between the superior and inferior limbs of the fissure. The facial and vestibulocochlear nerves arise just anterior to the inferior limb of the fissure and just below the middle cerebellar peduncle. The trigeminal nerve arises near the superior limb of the fissure.

The cerebellomedullary fissure, the cleft between the cerebellum and medulla that extends upward between the cerebellar tonsil and the medulla, communicates with the inferior limb of the cerebellopontine fissure near the lateral recess of the 4th ventricle. Several structures related to the lateral recess and the foramen of Luschka project into the cerebellopontine angle near the facial and the vestibulocochlear nerves.

Foramen of luschka, choroid plexus, and flocculus

The structures related to the lateral recess of the 4th ventricle that have a consistent relationship to the facial and vestibulocochlear nerves are the foramen of Luschka and its choroid plexus and the flocculus (see Figs. 8 and 9) [3,10]. The foramen of Luschka is situated at the lateral margin of the pontomedullary sulcus, just dorsal to the junction of the glossopha-ryngeal nerve with the brain stem, and immediately posteroinferior to the junction of the facial and vestibulocochlear nerves with the brain stem. The foramen of Luschka is infrequently well visualized. There is, however, a consistently identifiable tuft of choroid plexus that hangs out of the foramen of Luschka and sits on the posterior surface of the glossopharyn-geal and vagus nerves just inferior to the junction of the facial and vestibulocochlear nerves with the brain stem.

Another structure related to the lateral recess is the flocculus. It is a fan-shaped cerebellar lobule that projects from the margin of the lateral recess into the cerebellopontine angle. The flocculus, together with the nodule of the vermis, forms the primitive flocculonodular lobe of the cerebellum. The flocculus is attached to the rostral margin of the lateral recess and foramen of Luschka. The flocculus is continuous medially with the inferior medullary velum, a butterfly-shaped sheet of neural tissue that forms on the surface of the nodule and sweeps laterally above the tonsil to form part of the inferior half of the roof of the 4th ventricle. The lateral part of the inferior medullary velum narrows to a smaller

Fig. 10. Posterior views of the direction of displacement of the anterior inferior cerebellar artery (A.I.C.A.) around an acoustic neuroma. (*Top left*) Insert shows the direction of view. Both the premeatal (Pre. Mea. Seg.) and the postmeatal (Post. Mea. Seg.) segments are in their most common location around the lower margin of the tumor. The premeatal segment approaches the meatus from anteroinferior, and the postmeatal segment passes posteroinferior to the tumor. The superior cerebellar artery (S.C.A.) and trigeminal nerve (V) are above the tumor, and the posterior inferior cerebellar artery (P.I.C.A.) and the glossopharyngeal (IX), vagus (X), and accessory (XI) nerves are below the tumor. The choroid plexus (Ch. PI.) protrudes into the cerebellopontine angle medial to the tumor. The posterior wall of the internal acoustic canal has been removed to expose the transverse crest (Trans. Crest) and the superior vestibular (VIII S.V.) and inferior vestibular (VIII I.V.) nerves. The vestibular nerves disappear into the tumor; however, the cochlear (VIII Co.) and facial nerves (VII) are displaced around the anterior margin of the tumor. A subarcuate artery (S.A.) arises from the premeatal segment, and a recurrent perforating artery (R.P.A.) arises from the postmeatal segment. (*Center right*) A less common pattern of displacement of the anterior inferior cerebellar artery in which the premeatal and postmeatal segments are above the tumor. The internal auditory arteries (I.A.A.) arise from the meatal segment (Mea. Seg.). (*Bottom left*) Both the premeatal and the postmeatal segments are displaced anterior to the tumor. This occurs if the anterior inferior cerebellar artery courses between the vestibulocochlear and facial nerves. The tumor arises in the vestibular nerves, and the tumor growth displaces both the premeatal and the postmeatal segments anteriorly. (*From* Martin RG, Grant JL, Peace D, et al. Microsurgical relationships of the anterior inferior cerebellar artery and the facial-vestibulocochlear nerve complex. Neurosurgery 1980;6:483–507; with permission.)

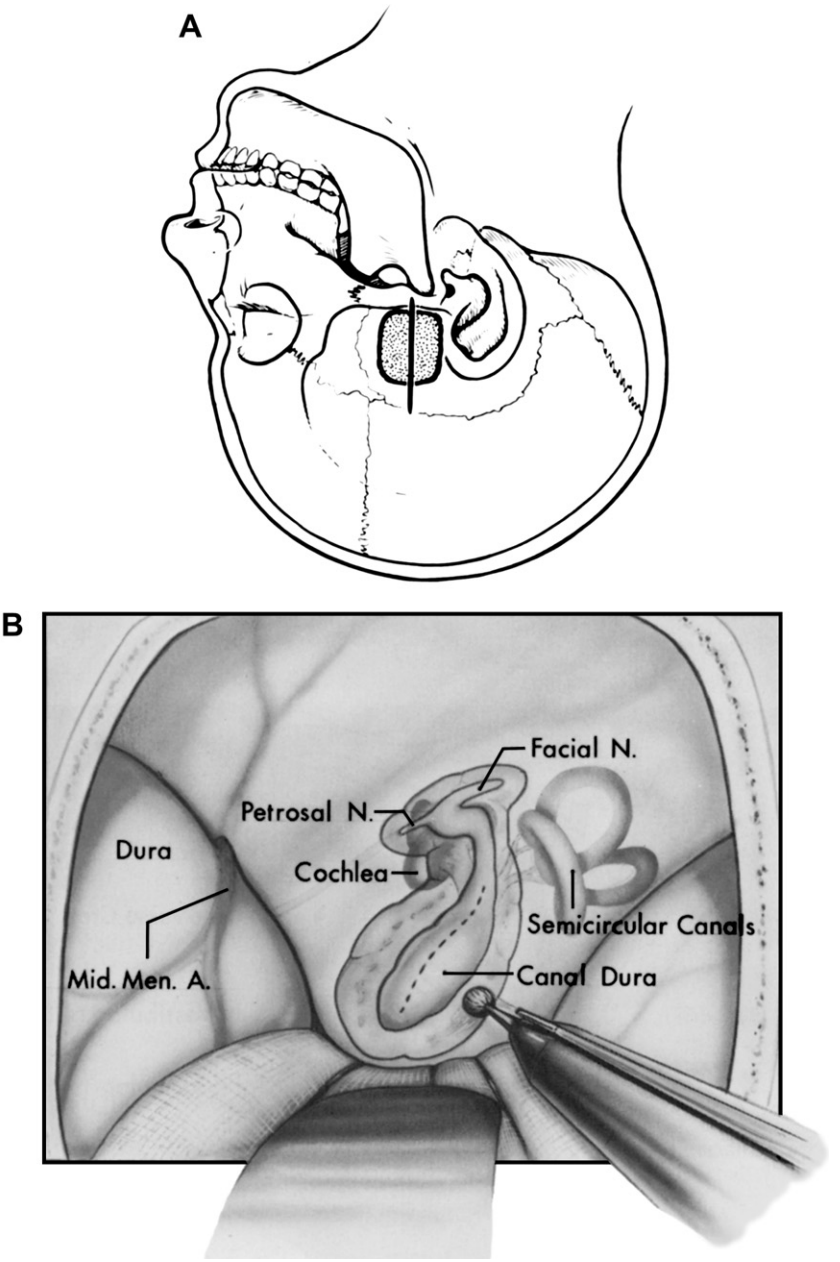


Fig. 11. Middle fossa approach for removing a small acoustic neuroma. (*A*) Left side. The vertical skin incision is located anterior to the ear and the craniotomy is situated with its base on the floor of the middle cranial fossa (*stippled area*). (*B*) The dura is elevated from the floor of the middle cranial fossa to identify the greater petrosal nerve (Petrosal N.). The middle meningeal artery (Mid. Men. A.) courses on the dura. Bone is removed over the greater petrosal nerve (Petrosal N.) to expose the facial nerve (Facial N.) which is followed proximally by removing bone to expose the superior wall of the internal auditory canal. Extreme care must be taken to avoid injuring the semicircular canals located in the bone at the posterior margin of the exposure and the cochlea situated in the bone just anterior and deep to the facial nerve. (*C*) Enlarged view of the area of bone removal. The dura has been opened to expose the tumor in the internal auditory canal. (Int. Auditory Canal). The tumor arises in the superior vestibular nerve (Sup. Vestibular N.) and displaces the facial nerve anteriorly. (*D*) The superior vestibular nerve has been divided above the transverse crest and elevated with the tumor. The superior vestibular nerve is being divided medial to the tumor. The facial, cochlear (Cochlear N.) and inferior vestibular (Inf. Vestibular N.) nerves are preserved.

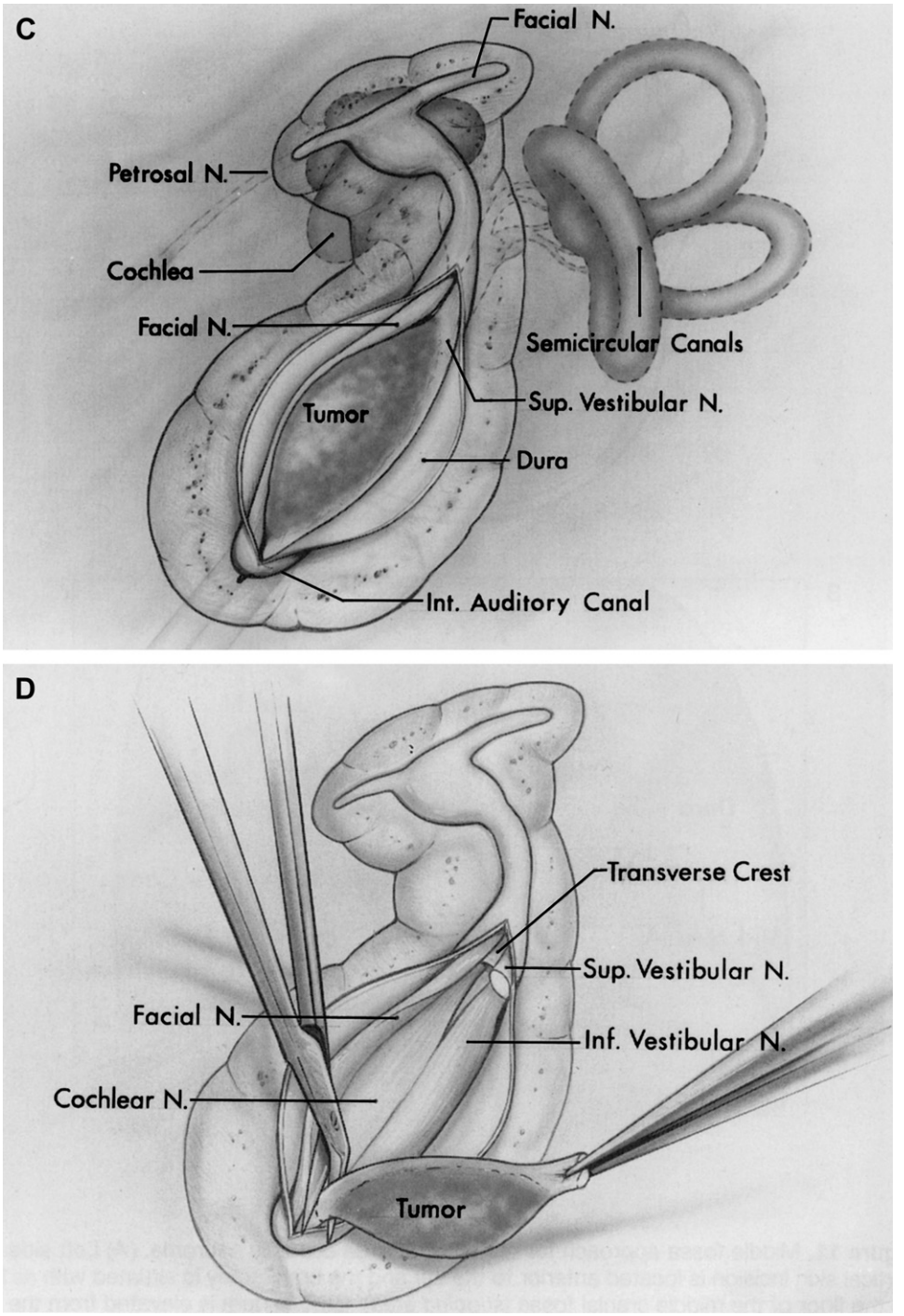
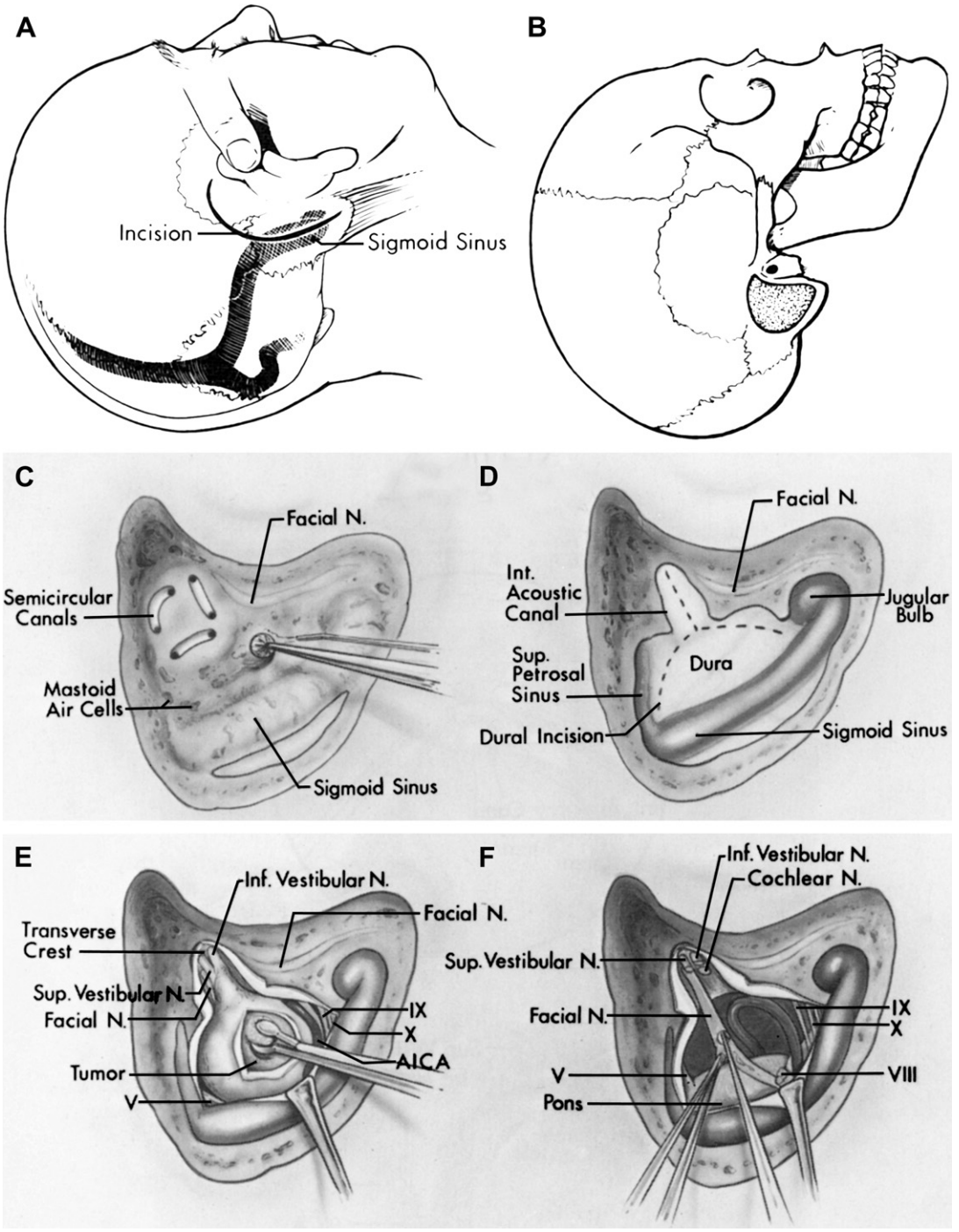


Fig. 11 (*continued*)


A
Incision
Sigmoid Sinus
B
C
Facial N.
Semicircular Canals
Mastoid Air Cells
Sigmoid Sinus
D
Facial N.
Int. Acoustic Canal
Jugular Bulb
Sup. Petrosal Sinus
Dura
Dural Incision
Sigmoid Sinus
E
Inf. Vestibular N.
Facial N.
Transverse Crest
Sup. Vestibular N.
Facial N.
IX
X
AICA
Tumor
V
F
Inf. Vestibular N.
Cochlear N.
Sup. Vestibular N.
Facial N.
IX
X
V
VIII
Pons

bundle, the peduncle of the flocculus, which fuses to the rostral margin of the lateral recess and foramen of Luschka. The flocculus projects from the peduncle of the flocculus into the cerebellopontine angle just posterior to where the facial and vestibulocochlear nerves join the pontomedullary sulcus.

Arterial relationships

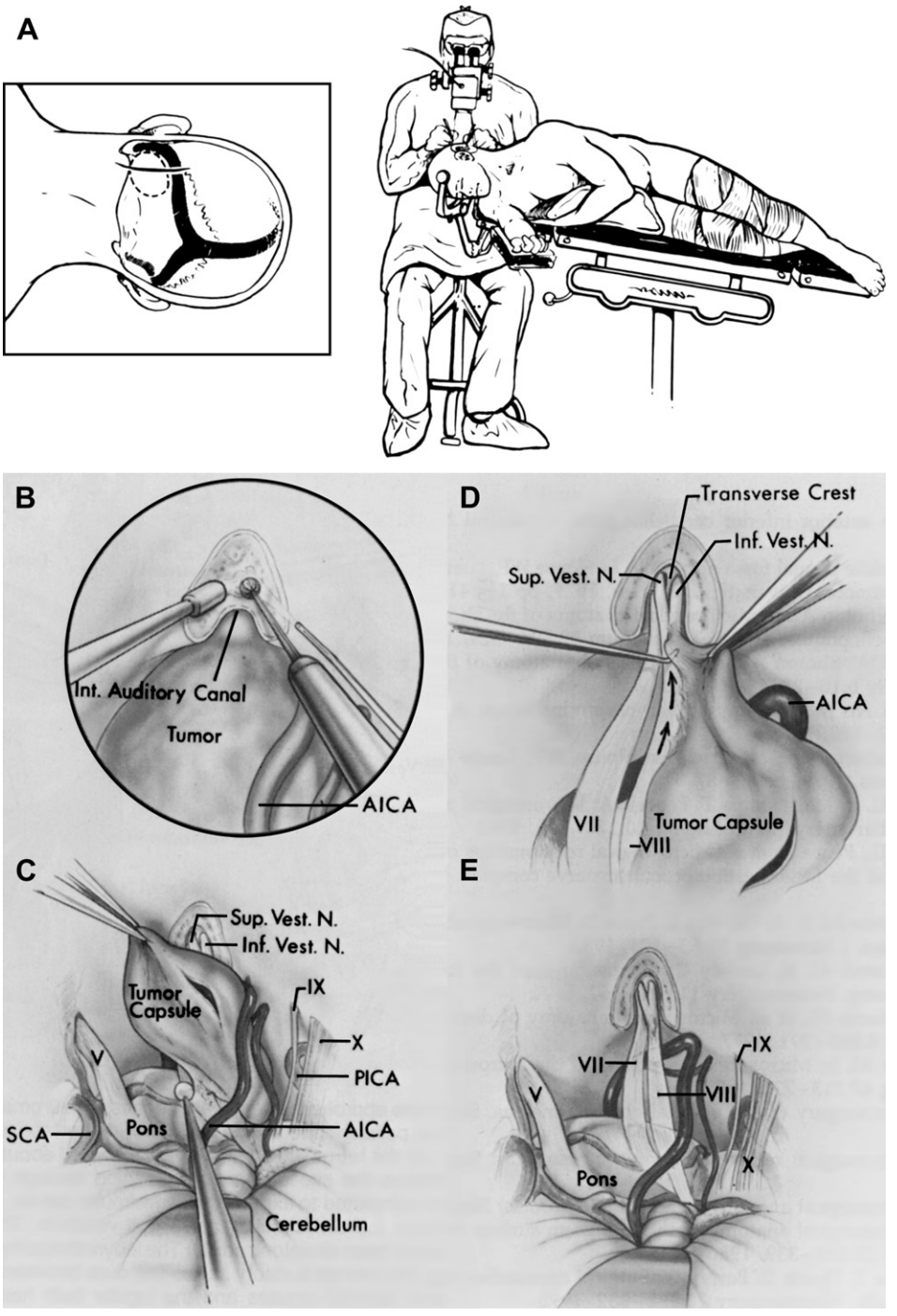
The arteries crossing the cerebellopontine angle, especially the anterior-inferior cerebellar artery, enjoy a consistent relationship to the facial and vestibulocochlear nerves, foramen of Luschka, and the flocculus (see Figs. 1, 8, 9,10) [1,4,7,8]. The anterior-inferior cerebellar artery originates from the basilar artery and encircles the pons near the pontomedullary sulcus. After coursing near and sending branches to the nerves entering the acoustic meatus and the choroid plexus protruding from the foramen of Luschka, it passes around the flocculus to reach the surface of the middle cerebellar peduncle and terminates by supplying the lips of the cerebellopontine fissure and the petrosal surface of the cerebellum. The anterior-inferior cerebellar artery usually bifurcates near the facial and vestibulocochlear nerves to form a rostral and a caudal trunk. The rostral trunk courses along the middle cerebellar peduncle to supply the upper part of the petrosal surface, and the caudal trunk passes near the lateral recess and supplies the lower part of the petrosal surface.

The trunk of the anterior-inferior cerebellar artery is divided into three segments based on its relationship to the nerves and the meatus: the premeatal, meatal, and postmeatal segments. The premeatal segment begins at the basilar artery and courses around the brain stem to reach the region of the meatus. The meatal segment is located in the vicinity of the internal acoustic meatus. The postmeatal segment begins distal to the nerves and courses medially to supply the brain stem and cerebellum. This meatal segment often forms a laterally convex loop, the meatal loop, directed toward or into the meatus. In a prior study, we found that the meatal segment was located medial to the porus in 46% of 50 cases and formed a loop that reached the porus or protruded into the canal in 54% [8]. In opening the meatus by the middle fossa, translabyrinthine, or posterior approach, care is needed to avoid injury to the meatal segment if it is located at or protrudes through the porus.

In most cases, the anterior-inferior cerebellar artery passes below the facial and vestibulocochlear nerves as it encircles the brain stem, but it may also pass above or between these nerves in its course around the brain stem (see Fig. 8). In the most common case, in which the artery passes below the nerves, the tumor would displace the artery inferiorly (see Fig. 10). If it courses between the facial and vestibulocochlear nerves, a tumor arising in the latter nerve displaces the artery forward. Tumor growth would displace the artery superiorly if it passes above the nerves.

The branches of the anterior-inferior cerebellar artery that arise near the facial and vestibulocochlear nerves are the labyrinthine (internal auditory) arteries, which supply the facial and vestibulocochlear nerves and adjacent structures; the recurrent perforating arteries, which may initially pass toward the meatus but subsequently

Fig. 12. Translabyrinthine approach to removal of acoustic neuromas. (*A*) Right side. The operation is done with the patient in the supine position with the face turned toward the side opposite the tumor. Site of the retromastoid skin incision located about 2 cm behind the ear. (*B*) The stippled area shows the site of the bony opening through the mastoid. (*C*) The mastoidectomy has been completed to expose the semicircular canals. The bone removal will be carried medially through the semicircular canals and vestibule. The sigmoid sinus and facial nerves (Facial N.) have been skeletonized. (*D*) The labyrinthectomy has been completed to expose the dura lining the internal auditory canal. The dura between the superior petrosai (Sup. Petrosai Sinus) and sigmoid sinuses and the jugular bulb has been exposed. The interrupted lines show the site of the dural opening. (*E*) The intracapsular contents of the tumor are being removed. The superior (Sup. Vestibular N.) and inferior vestibular (Inf. Vestibular N.) nerves are seen lateral to the tumor where they are separated by the transverse crest. The anterior inferior cerebellar artery (A.I.C.A.) courses around the lower margin of the tumor. The facial nerve is anterior to the tumor. The trigeminal nerve (V) is above and the glossopharyngeal (IX) and vagus nerves (X) are below the tumor. (*F*) The final fragments of tumor are being removed from the surface of the facial nerve. The superior and inferior vestibular and cochlear nerves (Cochlear N.) have been removed along with the tumor. The central stump of the eighth nerve (VIII) is exposed at the brain stem.


A
B
Int. Auditory Canal
Tumor
AICA
C
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Pons
AICA
Cerebellum
D
Transverse Crest
Inf. Vest. N.
Sup. Vest. N.
AICA
VII
Tumor Capsule
VIII
E
VII
IX
V
VIII
Pons
X

turn medially and supply the brain stem; and the subarcuate artery, which enters the subarcuate fossa. The subarcuate artery usually ends in the bone below the superior canal, but it may infrequently supply the distal territory of the labyrinthine arteries.

The superior cerebellar artery, which is separated from the tumor by the trigeminal nerve, is displaced rostrally by the tumor, and the posterior-inferior cerebellar artery is displaced caudally with the glossopharyngeal and vagus nerves by the tumor.

Venous relationships

The veins on the side of the brain stem that have a predictable relationship to the facial and vestibulocochlear nerves are those draining the petrosal surface of the cerebellum, the pons and medulla, and the cerebellopontine and cerebellomedullary fissures (see Fig. 9) [9,10]. The identification of any of these veins during removal of the tumor makes it easier to identify the site of the junction of the facial and vestibulocochlear nerves with the brain stem. These veins on the medial side of the tumor are the vein of the pontomedullary sulcus, which courses transversely in the pontomedullary sulcus; the lateral medullary vein, which courses longitudinally, along the line of origin of the rootlets of the glossopharyn-geal, vagus, and accessory nerves; the vein of the cerebellomedullary fissure, which passes dorsal or ventral to the flocculus before joining the other veins in the cerebellopontine angle; the vein of the middle cerebellar peduncle, which is formed by the union of the lateral medullary vein and the vein of the pontomedullary sulcus and ascends on the middle cerebellar peduncle to join the vein of the cerebellopontine fissure; and the vein of the cerebellopontine fissure, which is formed by the union of the veins that arise on the petrosal surface of the cerebellum and converge on the apex of the cerebellopontine fissure. All of these veins course near the lateral recess and the junction of the facial and vestibulocochlear nerves with the brain stem (see Fig. 9).

The veins surrounding an acoustic neuroma terminate by forming bridging veins, called *petrosal veins*, which empty into the superior petrosal sinus (see Figs. 5 and 9). These veins, which cross the cerebellopontine angle to reach the superior petrosal sinus, are the ones most frequently occluded in the course of operations in the cerebellopontine angle. Bridging veins are more frequently exposed and sacrificed in the rostral part of the cerebellopontine angle during operations near the trigeminal nerve than during operations near the nerves entering the internal acoustic meatus. The exposure of an acoustic neuroma in the central part of the cerebellopontine angle near the lateral recess can usually be completed without sacrificing a bridging vein. If a vein is obliterated during acoustic tumor removal, it is usually one of the superior petrosal veins, which is sacrificed near the superior pole of the tumor during the later stages of the removal of a large tumor. Small acoustic neuromas are usually removed without sacrificing a petrosal vein. The largest vein encountered around the superior pole of an acoustic neuroma is the vein of the cerebellopontine fissure, which passes from the petrosal surface of the cerebellum above the facial and vestibulocochlear nerves to join other tributaries of the superior petrosal sinus.

Fig. 13. Retrosigmoid approach for removal of an acoustic neuromas. (*A*) Right side. The patient is positioned in the three-quarter prone position with the surgeon behind the head. The insert (*left*) shows the site of the scalp incision (*continuous line*) and the bony opening (*interrupted line*). (*B*) The posterior wall of the internal auditory canal is removed. The anterior inferior cerebellar artery (A.I.C.A.) courses around the lower margin of the tumor. (*C*) The intracapsular contents of the tumor have been removed. The capsule of the tumor is being separated from the pons and the posterior surface of the facial (VII) and vestibulocochlear nerves. The superior (Sup. Vest. N.) and inferior vestibular nerves (Inf. Vest. N.) are seen at the lateral end of the internal auditory canal. The trigeminal nerve (V) and superior cerebellar artery (S.C.A.) are above the tumor and the glossopharyngeal (IX) and vagus (X) nerves and the posterior inferior cerebellar artery (PICA) are below the tumor. (*D*) The dissection along the eighth nerve (VIII) is done in a medial to lateral direction (*arrows*) in order to avoid tearing the tiny filaments of the nerve in the lateral end of the canal where they pass through the lamina cribrosa. The transverse crest separates the superior and inferior vestibular nerves in the lateral end of the canal. (*E*) Cerebellopontine angle and internal auditory canal after tumor removal with the facial and cochlear nerves preserved.

Summary

Because acoustic neuromas most frequently arise in the posteriorly placed vestibular nerves, they usually displace the facial and cochlear nerves anteriorly (Figs. 11, 12, and 13). The facial nerve is stretched around the anterior half of the tumor capsule. Variability in the direction of growth of the tumor arising from the vestibular nerves may result in the facial nerve being displaced, not only directly anteriorly, but also anterior-superiorly or anterior-inferiorly. The nerve is infrequently found on the posterior surface of the tumor. Because the facial nerve always enters the facial canal at the anterior-superior quadrant of the lateral margin of the meatus, it is usually easiest to locate it here, rather than at a more medial location where the degree of displacement of the nerve is more variable. The cochlear nerve also lies anterior to the vestibular nerve and is most frequently stretched around the anterior half of the tumor. The strokes of the fine dissecting instruments used in removing the tumor should be directed along the vestibulocochlear nerve from medial to lateral rather than from lateral to medial because traction medially may tear the tiny filaments of the cochlear nerve at the site where these filaments penetrate the lateral end of the meatus to enter the cochlea.

The landmarks that are helpful in identifying the facial and vestibulocochlear nerves at the brain stem on the medial side of the tumor have been reviewed. These nerves, although distorted by tumor, can usually be identified on the brain stem side of the tumor at the lateral end of the pontomedullary sulcus, just rostral to the glossopharyngeal nerve and just anterior-superior to the foramen of Luschka, flocculus, and choroid plexus protruding from the foramen of Luschka. After the facial and vestibulocochlear nerves are identified on the medial and lateral sides of the tumor, the final remnants of the tumor are separated from the intervening segment of the nerves.

In the three approaches to the meatus and cerebellopontine angle—retrosigmoid, translabyrinthine, and middle fossa—a communication may be established between the subarachnoid space and the mastoid air cells that requires careful closure to prevent a cerebrospinal fluid leak.

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Imaging of Acoustic Neuromas

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Imaging has become a sensitive method of evaluating patients with possible acoustic neuromas (ANs). Magnetic resonance imaging (MRI) continues to evolve but is already considered the preferred imaging study for the evaluation of a patient with suspected AN [1–13]. A negative gadolinium-enhanced MRI examination is considered a reliable indicator that the patient does not have an AN. Other imaging techniques remain as useful tools in certain clinical settings, but many techniques, considered state of the art less than a decade ago, have faded into almost complete obsolescence [3].

Imaging of the eighth cranial nerve sheath tumors has progressed from plain radiography to today's MRI [14]. In this evolution, the evaluation has progressed from attempts to show subtle findings that suggested the possibility of a lesion to actual visualization of the smallest of tumors deep within the internal auditory canal (IAC) itself. Because of the increasing acceptance of MRI as the imaging procedure of choice, much of the following discussion deals with MRI. Other modalities, such as computed tomography (CT), are discussed where appropriate. Imaging strategies in various clinical situations are discussed.

It should be remembered that either gadolinium-enhanced MRI or contrast-enhanced CT can demonstrate almost any AN [2]. The usual clinical situation, however, is that the clinician is trying to ensure that the patient does not have an AN, and so the most desirable test is the one that is the most sensitive. CT may not visualize the intracanalicular region well, and it is here that MRI establishes its advantage.

Anatomy and imaging

The bony anatomy is demonstrated in excellent detail by high-resolution CT when performed with a bone algorithm. The cortical edges of the IAC are sharply defined, and intricate internal anatomy of the labyrinth is routinely visualized. The bone algorithm allows limited visualization of the soft tissues, however, and the contents of the IAC are not seen.

The soft tissue or standard algorithm gives an improved visualization of the soft tissues, but the contrast of the nerves versus the cerebrospinal fluid (CSF) is still insufficient to allow demonstration of the fine soft tissue elements within the IAC. In addition, there is still a problem of artifact streaking obscuring the region of the porus acusticus and the cerebello-pontine angle (CPA) cistern.

MRI, on the other hand, gives excellent soft tissue visualization but does not show the bony detail nearly as well as CT. Cortical bone gives a lack of signal or signal void on MRI. Air also is seen as a lack of signal on the MRI scan. In a normal situation, therefore, the observer is not able to differentiate the otic capsule from the air-filled middle ear. Both appear black. The petrous apex often contains fat, which is seen as bright signal on the T_1-weighted (short TR/TE) image.

Fluid does give some signal on the T_1-weighted image. Fluid is not nearly as bright as fat but still can be seen quite easily, especially when contrasted against the signal void of the dense bone of the inner ear. Thus the perilymph/endolymph in the labyrinth and the CSF in the IAC can be seen on the image (see Fig. 1A).

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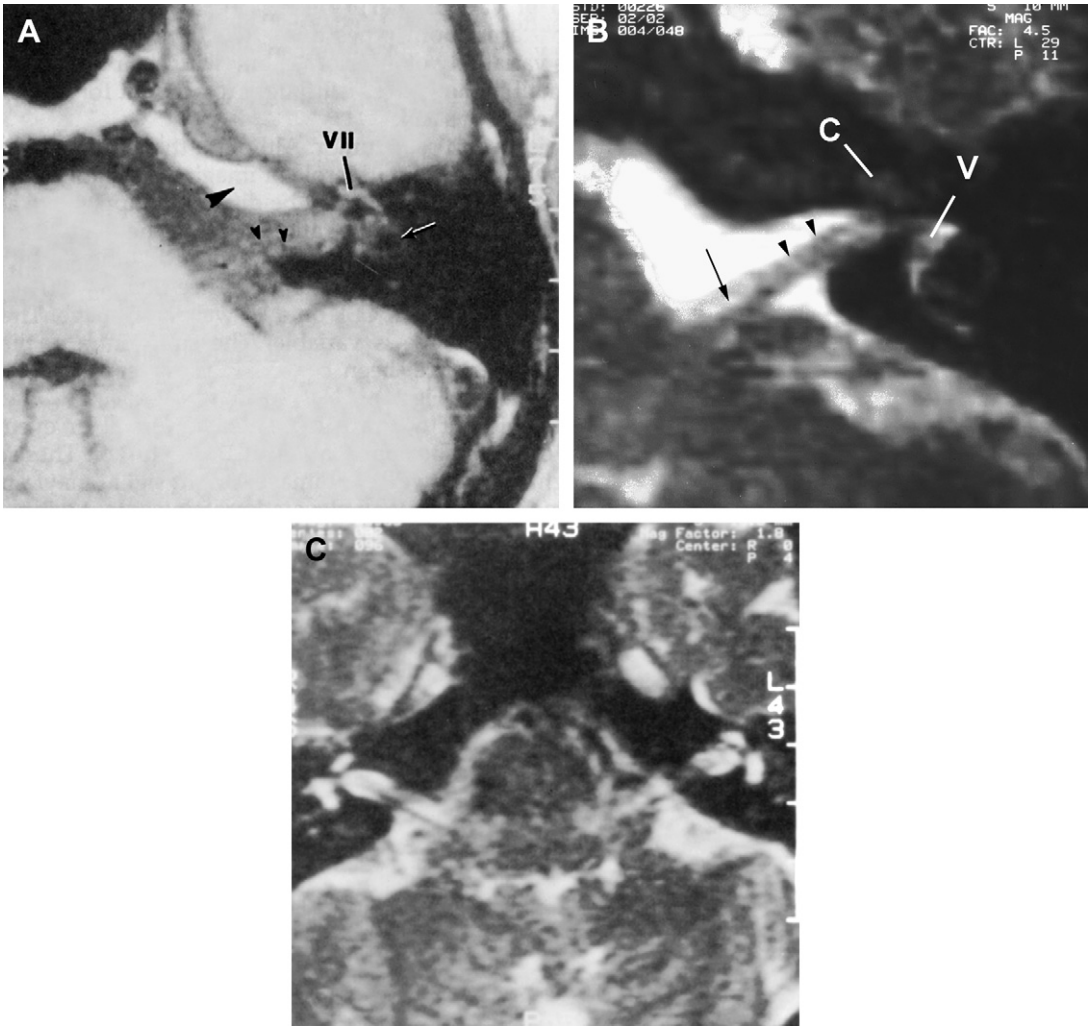

Fig. 1. (*A*) T_1-weighted (TR 600/TE 20) magnetic resonance image of the temporal bones. There is enough signal from the soft tissue of the facial nerve (VII) and the fluid in the internal auditory canal (IAC) as well as in the horizontal semicircular canal (*arrow*) to allow visualization contrasted against the mastoid and otic capsule. Note that the fluid and soft tissue signal is not nearly as bright as the fat (*large arrowhead*) in the petrous apex. *Small arrowheads* indicate neural elements. (*B*) T_2-weighted image (TR 2500/TE 90) turns the cerebrospinal fluid and the fluid in the vestibule (V) bright white. The intermediate signal from the cochlea (C) does not indicate that there is tissue within but rather that there is partial volume effect. The bright fluid in the cochlea is averaged with the contiguous bone to give an intermediate signal intensity. Nerve elements in the IAC (*arrowheads*) are extending from the root exit zone at the pontomedullary junction (*arrow*). (*C*) Extreme T_2-weighted image (TR 4000/TE 120) shows the bright signal in the cochlea, the vestibules, and IAC. On the right side a nerve can be seen from the pontomedullary junction to the fundus of the IAC.

On the T_1-weighted image, the soft tissue and the CSF have different signal intensities. Soft tissues such as the nerves (or brain) have somewhat greater signal than the CSF. Often the nerves can be seen crossing the CPA cistern and occasionally can be followed into the IAC (see Fig. 1A) [28,48]. The differences in signal intensity are not great enough on a T_1-weighted image to see the actual nerves consistently within the canal and to follow the nerves to their exit points at the lateral aspect of the canal [15].

The T_2-weighted (long TR/TE) image can be used to increase the signal difference between the nerves and the surrounding fluid (Fig. 1B, C). A

heavily T_2-weighted image makes fluid bright relative to the soft tissues. Thus the nerves can be seen more frequently if thin enough slices are obtained. The relative signal intensity from the labyrinth also increases, and so the inner ear structure may also be more conspicuous.

Acoustic neuroma

The appearance of an AN depends on the internal architecture of the tumor, its site of origin along the neural pathway, and the size of the lesion as well as the specifics of the imaging procedure being performed. The tumor can have a somewhat variable internal architecture. The tumor is made up of various concentrations of Antoni A and Antoni B type histologic patterns. Both types can often be identified within the same tumor. This variability of histology along with the presence of cystic areas and even small hemorrhages is thought to account for the wide spectrum of appearance of the AN.

An AN is a soft tissue tumor. Although the lesion can certainly come into contact with the brain, the role of imaging is usually to try to contrast the lesion against the CSF. It may be difficult to appreciate an AN on CT done without contrast administration because the density differences are insufficient for consistent visualization. On MRI without contrast administration, the tumor has a different appearance than CSF (Figs. 2 and 3). The tumor is brighter than CSF on a T_1-weighted image. The appearance on a T_2-weighted image is variable. The small intracanalicular tumors have, in our experience, been consistently darker than CSF on heavily T_2-weighted images (Fig. 4).

On MRI without contrast enhancement, small or even fairly large cystic areas can often be appreciated within the tumor. These cysts may be dark or bright on a T_1-weighted image. Bright signal may represent small areas of hemorrhage, [16]. but an elevated protein content within a cyst could result in a similar phenomenon (Fig. 5).

Virtually all ANs have an altered blood–brain barrier [17]. Thus on either MRI or CT, the tumors enhance after intravenous injection of a contrast agent that crosses such an abnormal blood–brain barrier. This phenomenon is responsible for the now classic appearance of the AN on both CT and MR [4,18] (Figs. 6–9).

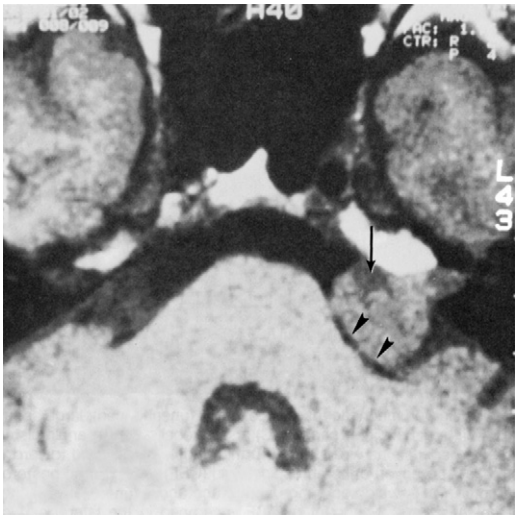

Fig. 2. T_1-weighted magnetic resonance image without contrast, acoustic neuroma left side. The tumor flattens against the cerebellar peduncle (*arrowheads*). A small area of lower signal intensity represents a cystic area (*arrow*). The lesion can be followed into the internal auditory canal.

The tumor shows as a bright white lesion protruding from the IAC on CT (see Figs. 6A and 7). On the MR image, the bright white of the high signal from the enhancing tumor is seen on the T_1-weighted image (see Figs. 8 and 9). There have been scattered reports of nonenhancing ANs, but if they exist, they are exceedingly rare. Some authors have recommended that, at least with CT, the contrast agent be injected 10 to 15 minutes before imaging to allow the contrast agent time to cross the blood–brain barrier into the lesion [19]. This has not seemed to be a problem on MRI.

Frequently the entire tumor enhances uniformly. If there are cystic regions within the lesion, they may not enhance, and the fluid contained within the cystic areas will have the same appearance after contrast administration as before (Fig. 10). These are avascular collections, so the contrast agent cannot reach the material within the cavity.

Calcifications are occasionally mentioned as rarely being present in ANs. They are extremely uncommon. In fact, if anything more than minimal calcification is present, an alternative diagnosis, such as meningioma, should be considered. Calcifications, when present, are expected to be tiny, and they are unlikely to be seen on

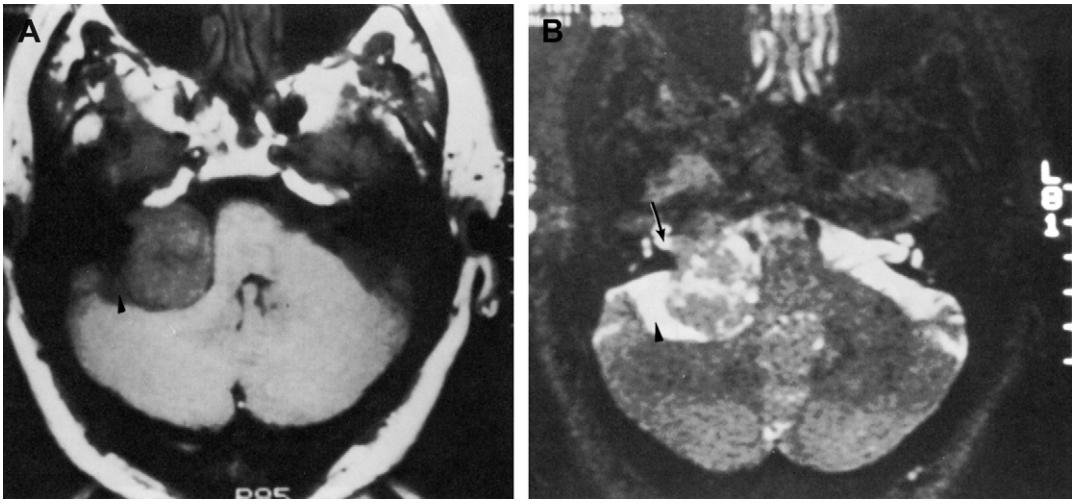

Fig. 3. (*A*) T_1-weighted magnetic resonance image (unenhanced); large acoustic neuroma is seen filling the internal auditory canal (IAC) and cerebellopontine angle cistern. The pons and the cerebellar peduncle as well as the fourth ventricle are pushed toward the contralateral side. There is a collection of cerebrospinal fluid (CSF) (*arrowhead*) posterolateral to the acoustic neuroma. (*B*) T_2-weighted image shows the acoustic neuroma in the cistern protruding into the IAC. A small amount of CSF is trapped in the lateral aspect of the IAC (*arrow*), thus the tumor is not completely filling the canal. The fluid collection posterolateral to the neuroma (*arrowhead*) shows the same brightening as the CSF.

MRI, where small calcifications are averaged together with contiguous soft tissue and become virtually invisible. CT is much more likely to show small flecks of calcium if they are present.

The shape of an AN is determined by its point of origin and its size. These lesions are thought to arise at or near the glial–Schwann cell junction. This junction point is usually found just inside the IAC, but the actual site is variable enough that

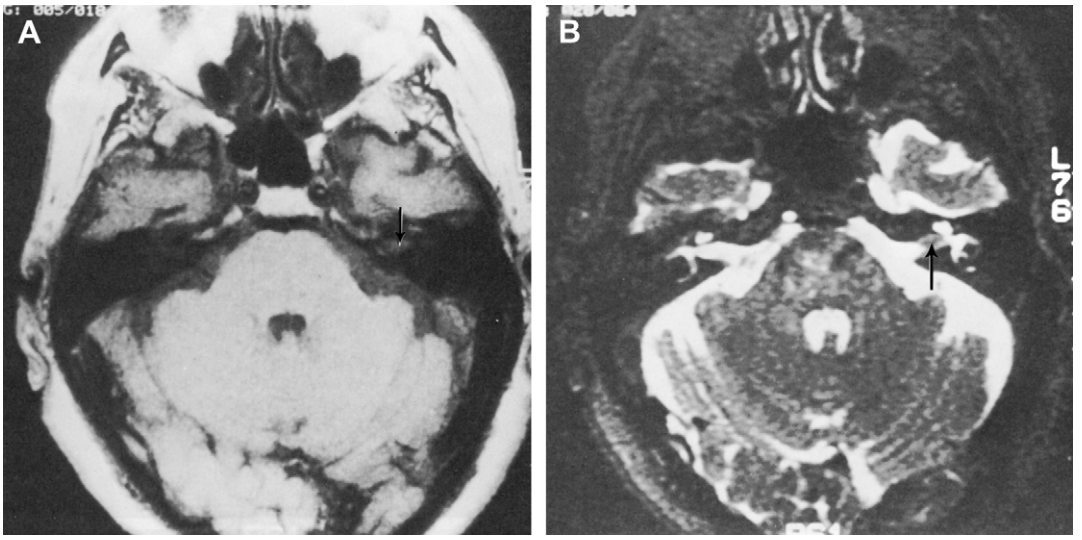

Fig. 4. (*A*) T_1-weighted magnetic resonance image (without gadolinium). There is a slight increase in signal on the side of the acoustic neuroma (*arrow*). This would be difficult to call positive with certainty. (*B*) On a T_2-weighted image the cerebrospinal fluid (CSF) is bright white. The tumor now shows as an area of low signal (*arrow*) contrasted against the CSF. Compare with the CSF-filled internal auditory canal on the normal side.

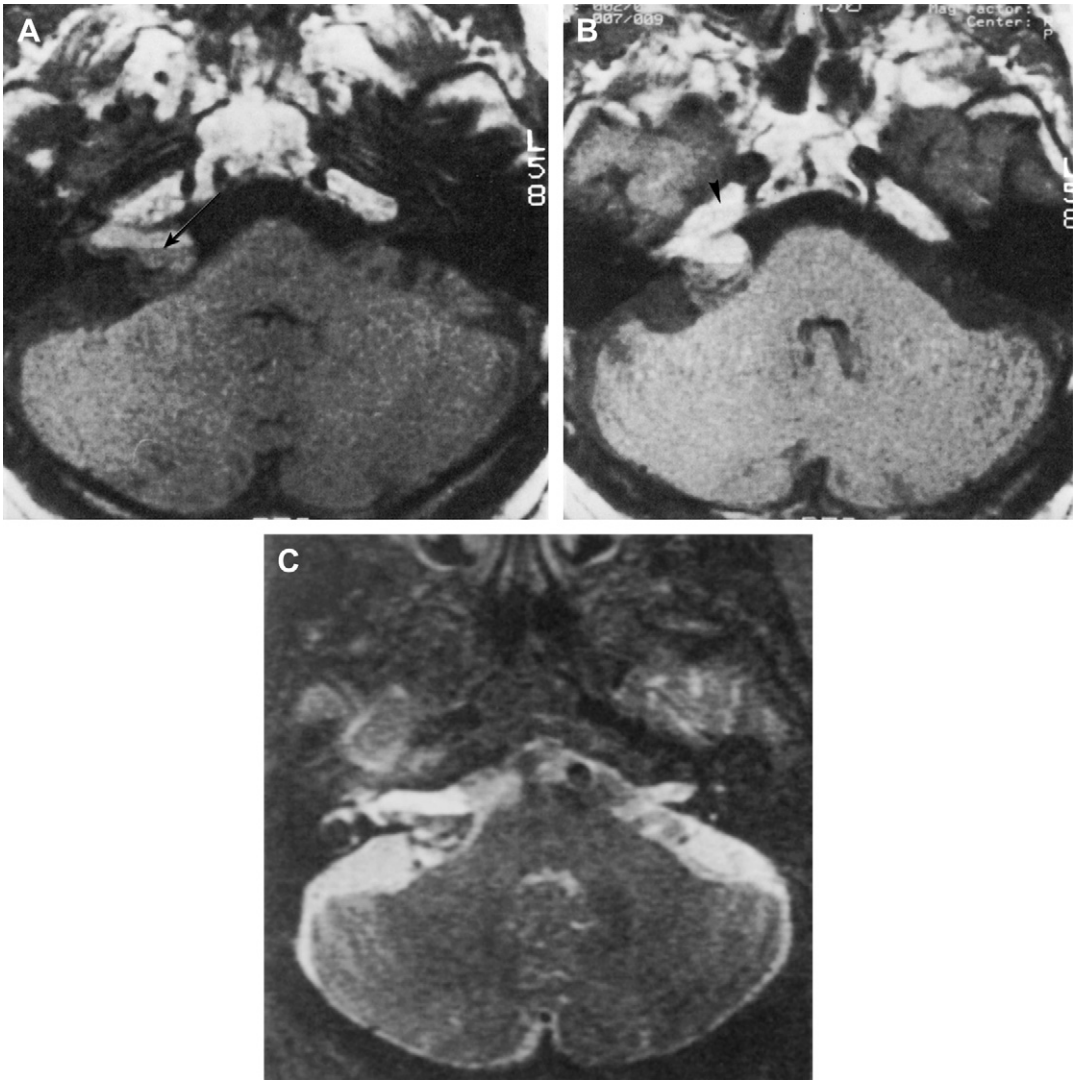

Fig. 5. Hemorrhage into an acoustic neuroma. This patient had an acoustic neuroma diagnosed previously. The patient had an acute hearing loss and underwent magnetic resonance imaging. At surgery the patient had a large hemorrhage into the acoustic neuroma. (*A*) T_1-weighted (TR 600/TE 20) image shows an acoustic neuroma on the right side with apparent fluid/fluid level (*arrow*). (*B*) T_1-weighted image slightly superior to that found in *A,* again shows fluid level representing layering of hemorrhage. Also note the bright signal (*arrowhead*) in the fat of the petrous apex. (*C*) T_2-weighted image showing even greater contrast between the fluid components of the hemorrhage.

the AN can develop completely within, completely outside, or partly inside, partly outside the canal. Most commonly, the lesion arises just inside the meatus and then grows out into the CPA cistern. Such a lesion is said to have both an intracanalicular and an extracanalicular component.

Intracanalicular tumor

As a tumor enlarges, several things happen that allow detection. First, the CSF, which usually is found within the canal, is replaced with soft tissue. This effect is difficult to see on CT but can be detected on MRI in many cases. Before the

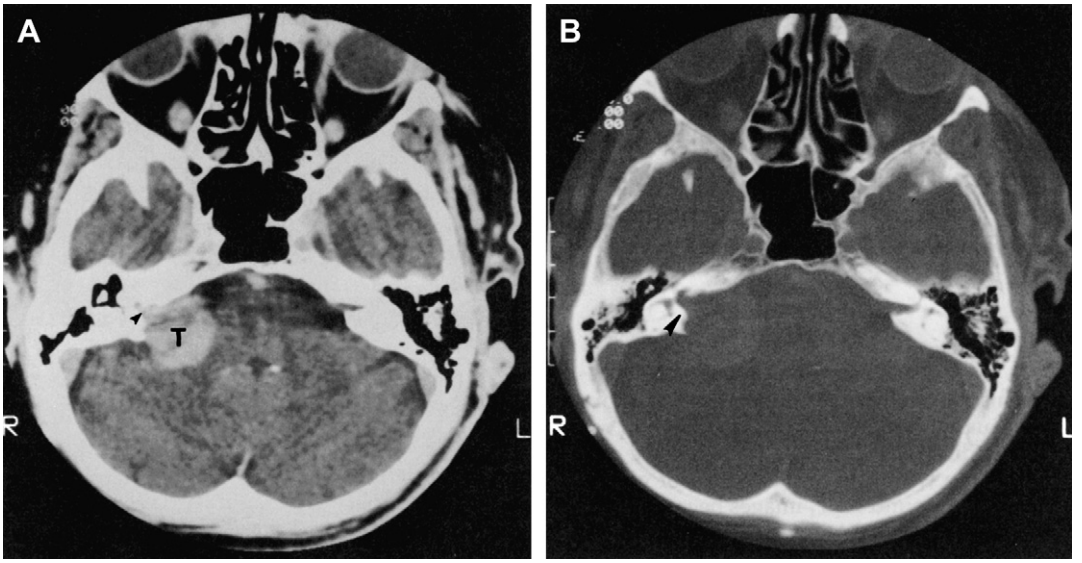

Fig. 6. (*A*) Acoustic neuroma with high resolution computed tomography with contrast. Soft tissue algorithms shows the tumor (T) protruding into the internal auditory canal (IAC) (*arrowhead*). (*B*) Bone algorithm shows the enlargement of the IAC (*arrowhead*).

widespread availability of gadolinium, many intracanalicular ANs were found on T_1 images because the tumor gave more signal than the CSF that should have been seen in the IAC if the patient were normal. Some radiologists preferred using extremely long TR sequences to make the CSF very bright so a small AN could be seen as a low-signal dark mass contrasted against the bright CSF (see Fig. 4B).

As the lesion grows within the canal, pressure is exerted on the walls of the canal, and a second

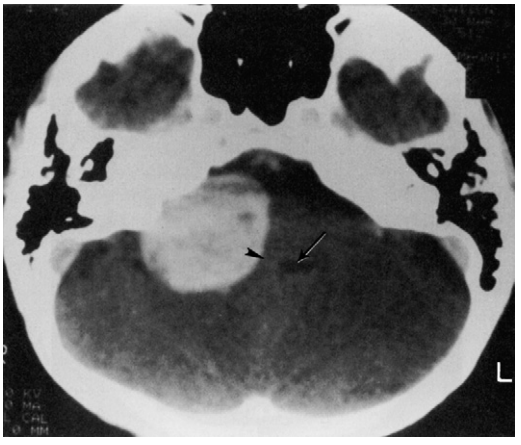

Fig. 7. Acoustic neuroma, enhanced computed tomography. Fairly homogeneous tumor pushing the pons, cerebellar peduncle, and fourth ventricle toward the contralateral side. Fourth ventricle (*arrow*), cerebellar peduncle (*arrowhead*).

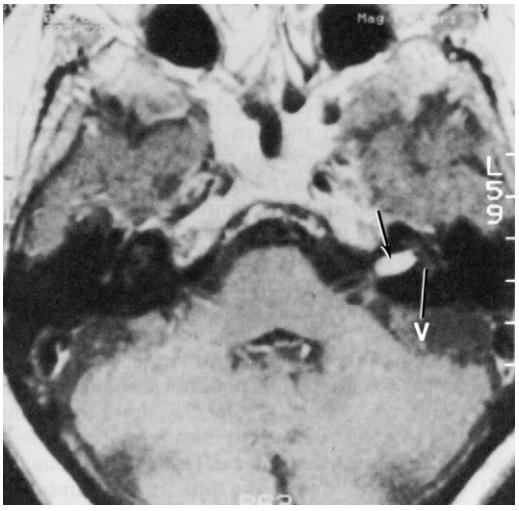

Fig. 8. Acoustic neuroma left side (T_1-weighted gadolinium-enhanced magnetic resonance image). Small enhancing neuroma (*arrow*) is seen filling the internal auditory canal but not protruding through the porus. Compare the enhancement of the neuroma to the nonenhancing fluid in the vestibule (V).

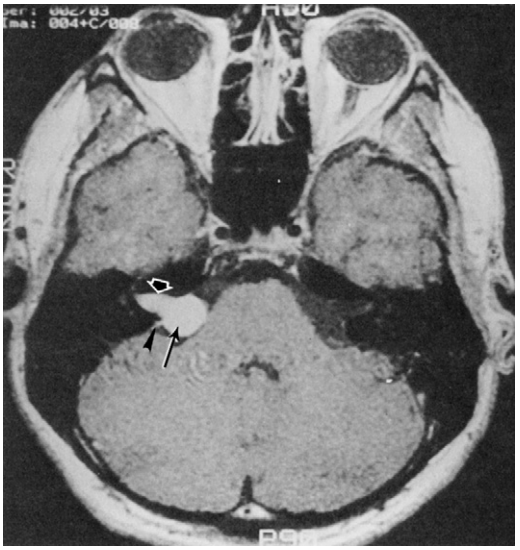

Fig. 9. Acoustic neuroma postgadolinium (T_1 weighted). The lesion (*arrow*) is seen protruding from the internal auditory canal. The intracanalicular portion (*open arrow*) is clearly identified contrasted against the extreme low signal of the petrous bone. Slight irregularity of the posterior edge of the tumor (*arrowhead*) is thought to represent slowly flowing blood in one of the branches of the petrosal vein.

effect can be detected. There is gradual enlargement of the canal. This secondary change was the basis of plain film and tomography screening of a decade ago. If significant asymmetry was seen, further, more invasive testing was done. This expansion of the canal can certainly be appreciated with modern imaging, especially on CT. The significance of the finding, however, is considerably diminished. Now the imaging shows the actual tumor margin rather than a secondary sign. Current imaging can confidently determine if an enlargement is due to a tumor or is simply a normal variation. The radiologist looks at the contents of the canal at the site of the enlargement (Fig. 11). If there is enhancement or soft tissue, the enlargement is the result of a tumor. But if the enlargement is filled with the typical density or signal intensity of CSF, the apparent enlargement can confidently be called a normal variation, and tumor as a cause of the enlargement is no longer a concern.

In our experience, essentially all small intracanalicular tumors exhibit enhancement on either CT or MRI. On CT, the enhancement may be obscured or difficult to detect because it is contiguous to the bone, which is very dense, and therefore the tumor is less conspicuous (see Fig. 11).

With MRI using gadolinium, the enhancing tumor is contrasted against the signal void of the cortical bone of the IAC. The bright signal is hard to miss (Fig. 12). The sensitivity of MRI in detecting intracanalicular tumors is therefore high. It is the resultant ability of MRI to exclude reliably small intracanalicular tumors that represents the real advantage of MRI over CT.

Extracanalicular tumor

As a lesion protrudes from the IAC into the CPA cistern, the tumor abuts the CSF and so becomes easily visible on CT as well as MRI. Even without contrast enhancement, most of these lesions are easily detectable on MRI. Once the lesion has grown beyond the plane of the porus, CT and MRI are about equal in the ability to detect the lesion. Either will detect almost every tumor.

The typical appearance of the mass growing into the CPA cistern is the mass tapering toward the porus (see Figs. 6A and 9). The angle made as the tumor meets the posterior surface of the petrous bone is acute rather than obtuse. These findings indicate that the site of origin is the IAC, and therefore the most likely diagnosis is AN.

Initially as the tumor grows into the CPA cistern, the advancing margin will be round until it encounters the brain stem. The level of the IAC is close to the great horizontal fissure of the cerebellum. Often the AN will push into the fissure. The tumor may appear to deform or flatten against the middle cerebellar peduncle (Fig. 13). This gives the appearance of a fairly rounded tumor with a flattened edge along the posteromedial aspect.

Further growth can push the pons and brain stem toward the contralateral side. As the shift of the brain stem occurs, the fourth ventricle can be deformed or compressed and obstructive hydrocephalus can occur (Fig. 14). Larger ANs can occasionally be associated with edema in the adjacent brain [20,21].

Extracanalicular tumor can also extend superiorly or interiorly. Superiorly the tumor can approach the fifth cranial nerve, which can easily be seen on axial or coronal MR images (Fig. 15) [22]. Further upward growth places the edge of the neuroma at the tentorial incisura. The superior extent is best appreciated on coronal

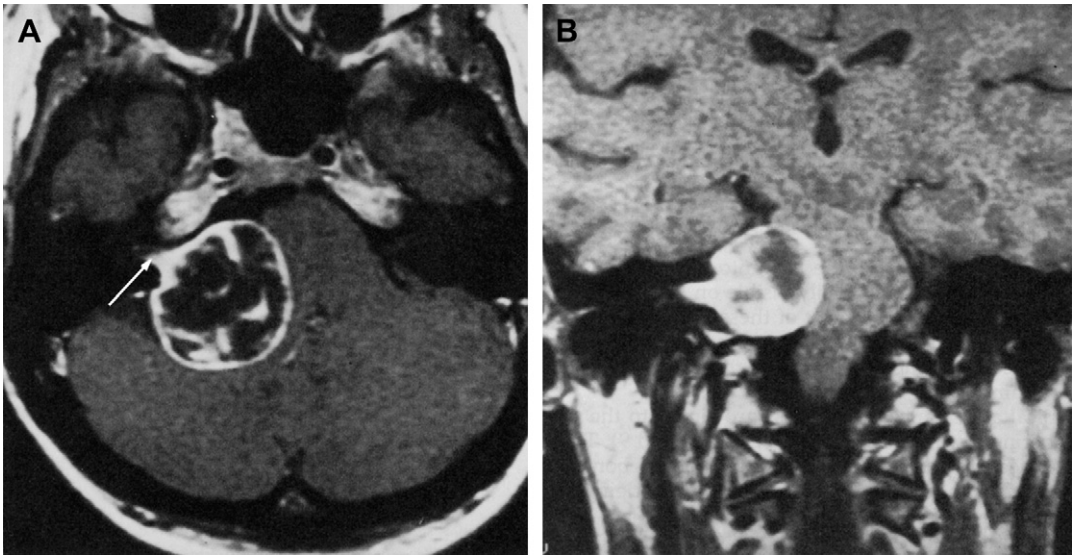

Fig. 10. (*A*) Partially enhancing acoustic neuroma in the CPA cistern. The edge of the tumor (*arrow*) protrudes into but does not completely fill the internal auditory canal. Note the many areas within the lesion that do not enhance and are of low signal. (*B*) Coronal image (same patient).

images but can be accurately estimated on axial images as well.

If the tumor has a component of growth in the caudal direction, the tumor is seen passing along the medulla and medial to the jugular foramen. The lesion should not obliterate the CSF signal (MRI) or density (CT) in the pars nervosa of the jugular foramen, nor does the tumor erode the mareins of the foramen (Fig. 16). If either of these findings is present, special care is warranted to be sure that the tumor is not actually arising in the jugular foramen rather than the IAC [9].

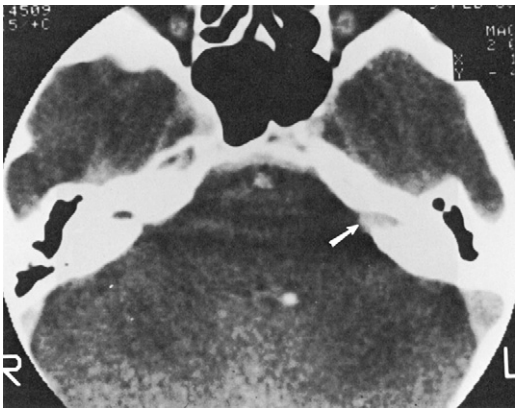

Fig. 11. Small acoustic neuroma on enhanced computed tomography. The edge of the tumor (*arrow*) is clearly seen. Compare the density within the canal with the cerebrospinal fluid density in the opposite canal.

Inferomedially directed growth follows the course of the seventh and eighth cranial nerves toward the root exit zone at the pontomedullary junction. The foramen of Luschka can be covered by the tumor. A small protrusion of choroid often passes through the foramen of Luschka into the lower CPA cistern. If this small bit of choroid is incorporated into the tumor, an arachnoid cyst can be formed in conjunction with the neuroma. Alternatively a portion of the CPA cistern can become isolated from the rest of the cisternal circulation, and a cyst or CSF collection can form posterolateral to the main part of the tumor. Cushing described such cysts or collections in a high percentage of the patients on whom he operated for AN (see Figs. 3A and 14). Currently these large cysts are less common probably because now lesions are confidently diagnosed and thus resected at a much earlier stage than they were in the early history of AN surgery.

Either CT or MRI can demonstrate these collections of CSF. CSF again has a very characteristic appearance. The fluid is dark on CT and homogeneous. There is little variation in the density within the fluid. On MRI, the CSF shows the characteristic low signal on T_1-weighted images and high signal on T_2-weighted images [10].

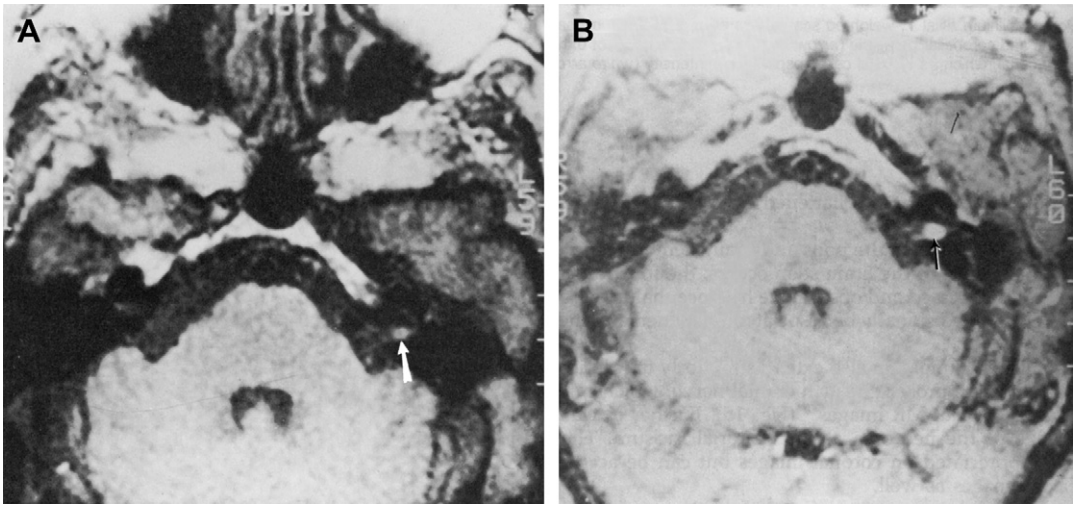

Fig. 12. (*A*) Pregadolinium T_1-weighted image shows a small acoustic neuroma (*arrow*) in the fundus of the left internal auditory canal (IAC). (*B*) Postgadolinium T_1-weighted image shows definite enhancement (*arrow*) of the rounded lesion in the IAC increasing the conspicuity of the lesion.

Most large ANs have both an intracanalicular and an extracanalicular component, but this is not always the case. Totally extracanalicular tumors can occur in two situations (see Fig. 13).

First, the Schwann cell–glial junction can actually be outside the meatus of the IAC. In this case, the tumor actually develops within the CPA cistern. The second postulated mechanism for occurrence of a totally extracanalicular AN is that a fairly firm or cellular tumor begins to develop in the medial part of the IAC. As it grows, the pressure begins to expand the canal. Eventually, however, the tumor develops enough leverage against the edge of the canal that the lesion actually pries or lifts itself out of the canal. In doing so, the tumor may actually avulse the nerve rootlets, leaving an "empty canal".

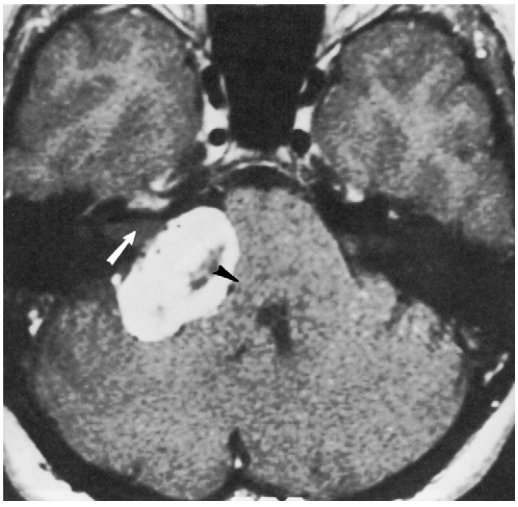

Fig. 13. Postgadolinium axial T_1-weighted scan shows an enhancing tumor at the porus pushing the pons and cerebellar peduncle (*arrowhead*). The lesion does not extend into the internal auditory canal, which has typical cerebrospinal fluid intensity (*white arrow*).

With either mechanism, the picture on CT or MRI is the same. There may be some erosion or expansion of the meatus of the IAC, but the intracanalicular region or especially the fundus is often relatively normal (see Fig. 13). The characteristic CSF density on CT or the characteristic CSF signal intensities on MRI can be seen in the depths of the canal. This is especially obvious on a long TR/long TE, heavily T_2-weighted MRI.

Rare occurrences

Most patients have a unilateral tumor and the tumor arises inside the IAC. There are rare exceptions. Bilateral ANs are seen in neurofibromatosis [23–27]. Because the diagnosis is not always known at the time of imaging, care should be taken to ensure that the opposite IAC is adequately visualized when an AN is first identified on one side (Fig. 17).

A much rarer occurrence is a neuroma originating within the otic Labyrinth. Only a few cases

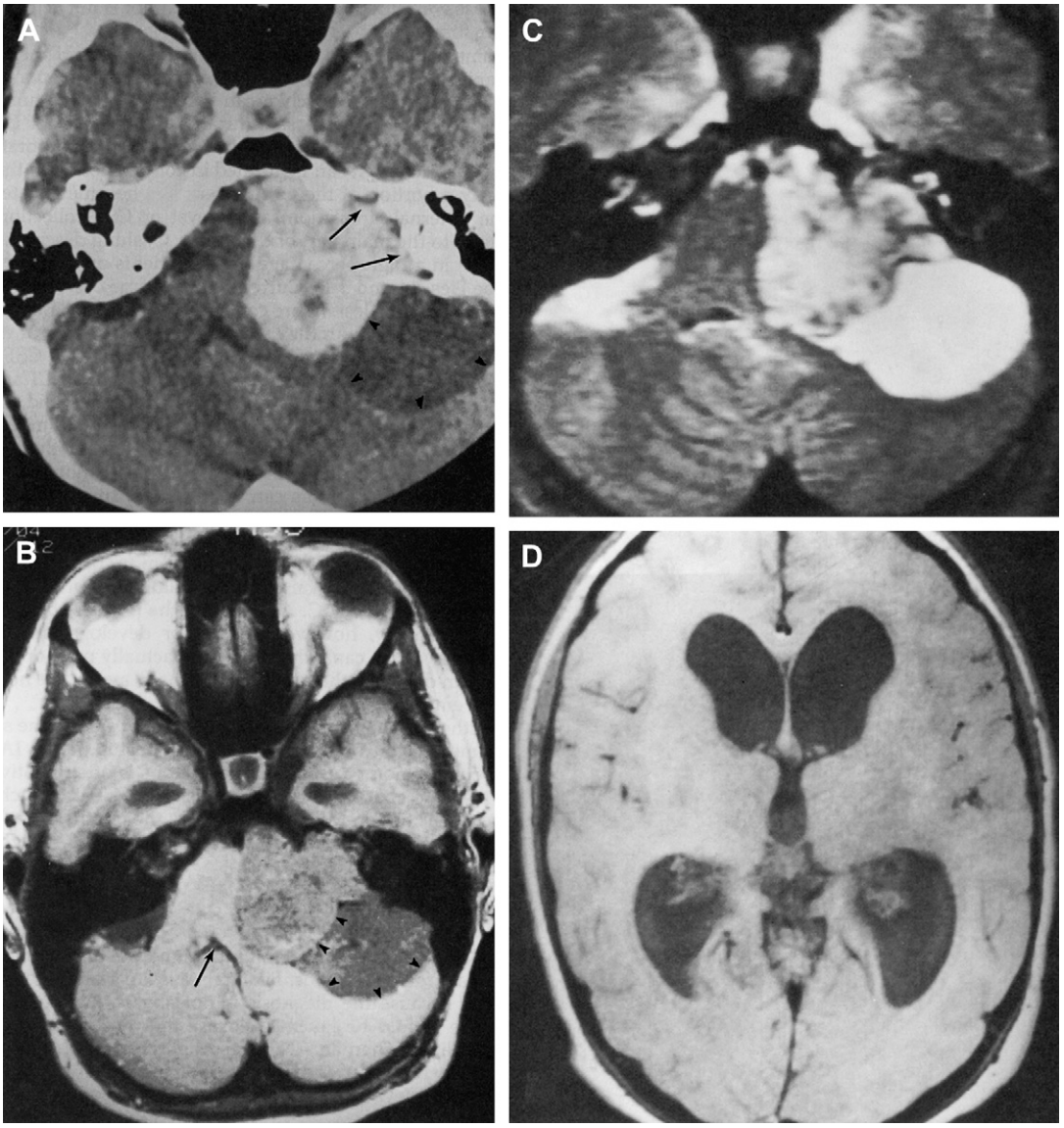

Fig. 14. Large acoustic neuroma with destructive enlargement of the internal auditory canal (IAC) and associated posterolateral arachnoid cyst. (*A*) There is destructive enlargement of the IAC (*arrows*). There is a large arachnoid cyst posterolaterally (*arrowheads*). (*B*) Axial T_1-weighted (spin echo 500/20) image. The trapped arachnoid cyst (*arrowheads*) is seen posterolateral to the tumor on this unenhanced image. The fourth ventricle (*arrow*) is deviated. (*C*) T_2-weighted image (SE 2500/75). The posterolateral component has the same brightening as cerebrospinal fluid. (*D*) Axial T_1-weighted slice through the upper brain shows hydrocephalus due to the compression of the fourth ventricle.

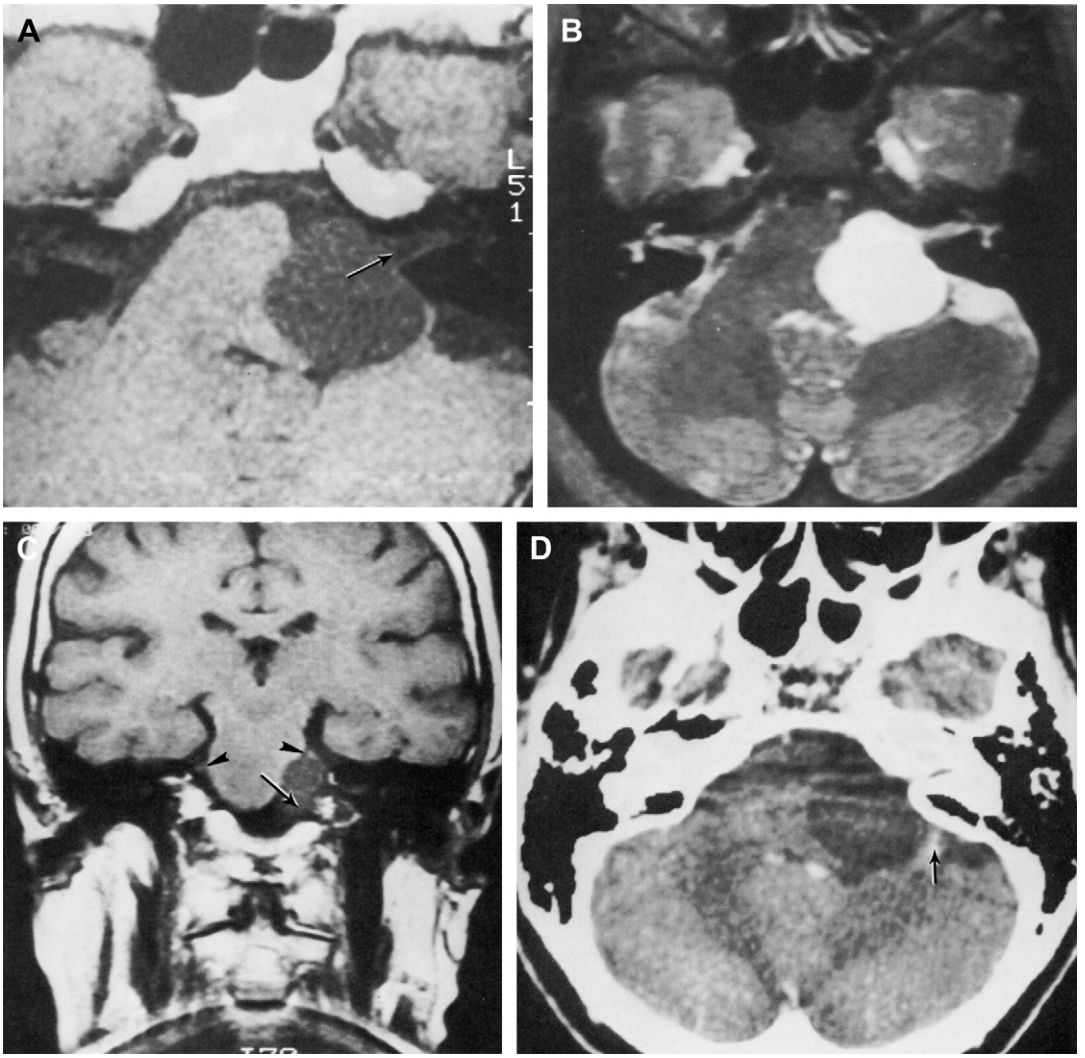

Fig. 15. Cystic neurilemoma of CPA cistern. Lesion probably arising from the glossopharyngeal nerve. (*A*) T_1-weighted image (no gadolinium). The lesion is seen in the CPA cistern pushing the brain stem and pons toward the right. The lesion protrudes into the porus but does not fill the internal auditory canal. Edge of lesion (arrow). (*B*) T_2-weighted image shows high signal in the apparently cystic abnormality. (*C*) Coronal T_1-weighted image. The lesion could be followed to the pars nervosa interiorly (*arrow*). The lesion pushed the trigeminal nerve (*arrowhead on right*) superiorly. Compare with trigeminal nerve on the opposite side (*arrowhead*). (*D*) Axial computed tomography scan post-contrast. Petrosal vein tributaries are pushed posteriorly (*arrow*) and the lesion does not enhance. This lesion was attached to the glossopharyngeal nerve at surgery.

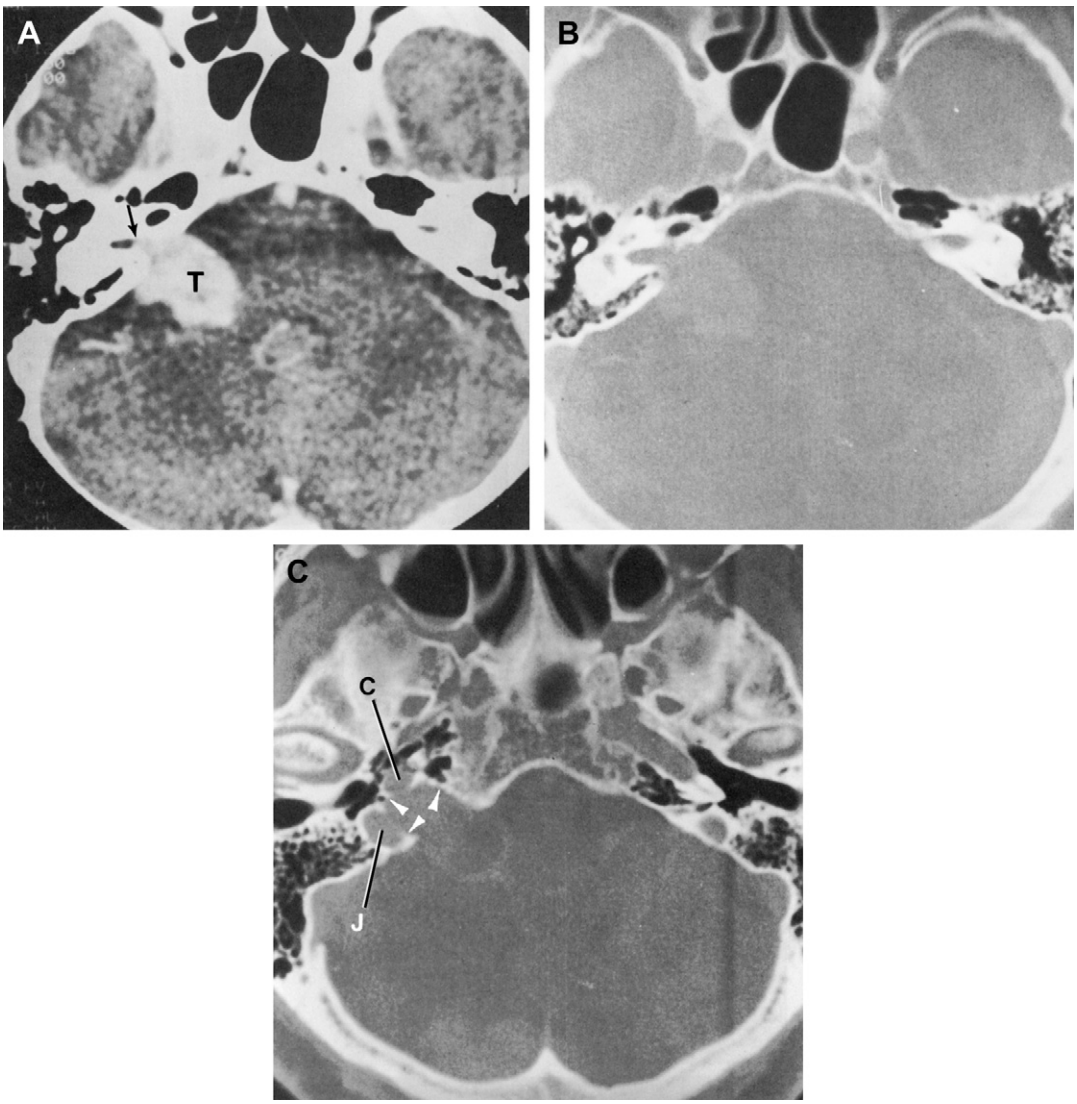

Fig. 16. Glossopharyngeal neurilemoma protruding into CPA cistern. (*A*) Tumor is seen at the level of the internal auditory canal (IAC) and protrudes into the porus (*arrow*). T, tumor. (*B*) On a bone algorithm there is no enlargement of the IAC. There is minimal erosion of the porus. (*C*) Inferior slice bone algorithm. There is enlargement of the pars nervosa (*arrowheads*) indicating the origin of the lesion. J, jugular fossa; C, carotid canal.

have been reported. These would be detected as enhancement within the confines of the labyrinth. On CT, there may be some bone expansion or erosion.

Differential diagnosis

The appearance of an AN is usually characteristic enough that the diagnosis can be made with confidence, and the possibility that the lesion is something else is remote. There are certainly other tumors, however, that occur in the region of the IAC and the CPA cistern that can be confused with ANs [11,49]. Probably more important are certain inflammatory conditions that can mimic tumors.

Nontumoral enhancement

The gadolinium-enhanced MRI scan has increased our ability to exclude an AN reliably.

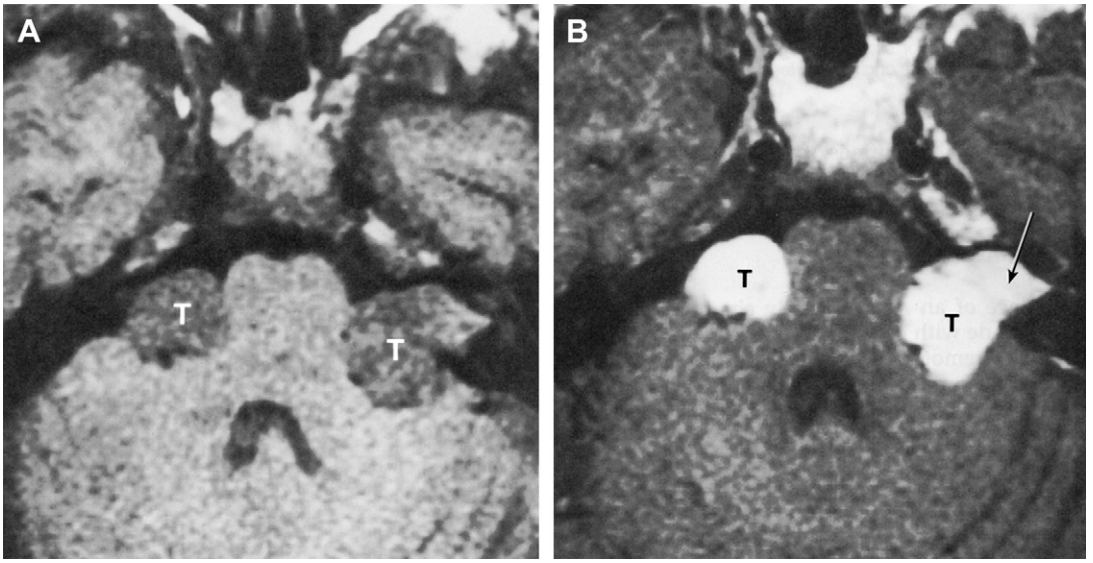

Fig. 17. (*A*) Bilateral acoustic neuromas. T_1-weighted image without contrast shows bilateral tumors (T). (*B*) Axial T_1-weighted image after gadolinium shows enhancement of the tumors bilaterally. Note the intracanalicular portion on the right side (*arrow*). The slice is too high to see the intracanalicular portion on the left.

Subtle enhancement in the IAC is dramatically obvious in even tiny tumors. Not everything that enhances, however, is a tumor. Indeed not everything that enhances is abnormal.

Depending on the time relationship between contrast injection and imaging, small vessels, especially those with slow flow, can appear to "light up." Even areas of vascular anastomosis can enhance. An example of this phenomenon is the region of the geniculate ganglion, where a small amount of enhancement is often seen in patients without any symptoms. Occasionally there is a small amount of enhancement within the IAC itself. This enhancement may represent a small vessel but can be of concern if seen on the side of symptomatology [29].

Recently attention has been focused on enhancing inflammatory nontumoral conditions (Fig. 18). This phenomenon was identified in Bell's palsy, in which definite enhancement was noted to occur in the facial nerve canal traversing the temporal bone [30,31]. The enhancement could involve the entire nerve or be more localized to the region of the geniculate ganglion. Because of the typical presentation of Bell's palsy, it was quickly realized that this enhancement did not represent a tumor. CT would show a normal-sized facial nerve canal, thus making a tumor unlikely. As the symptoms wane, the enhancement has been reported to decrease, although resolution of the radiologic finding may lag behind clinical resolution of symptoms [31].

Patients with acute inflammation of the labyrinth or the nerves within the IAC can exhibit similar enhancement but now in the region where the finding can be confused with an AN (see Fig. 18). Investigators have shown the increased signal to correlate with the rapid onset of symptoms, suggesting a viral infection [32,33].

It should also be remembered that not everything that is bright on a T_1-weighted image represents enhancement. Hemorrhage at various stages and even fluid with a high protein content can look exactly like enhancement (Figs. 19 and 20). One can easily determine if high signal represents enhancement by comparing the postgadolinium scan with a non–contrast enhanced image, but this is not always available [34]. In ambiguous cases, it may be helpful to repeat a scan after several days when the gadolinium has cleared.

Caution should therefore be exercised when a small amount of enhancement is seen inside the canal on MRI. This is especially true if there is no enlargement of the canal that correlates with the area that is enhancing or if the enhancement does not have a clearly defined edge to suggest a tumor. In questionable cases, following the clinical course and reimaging the patient after a short time interval is appropriate.

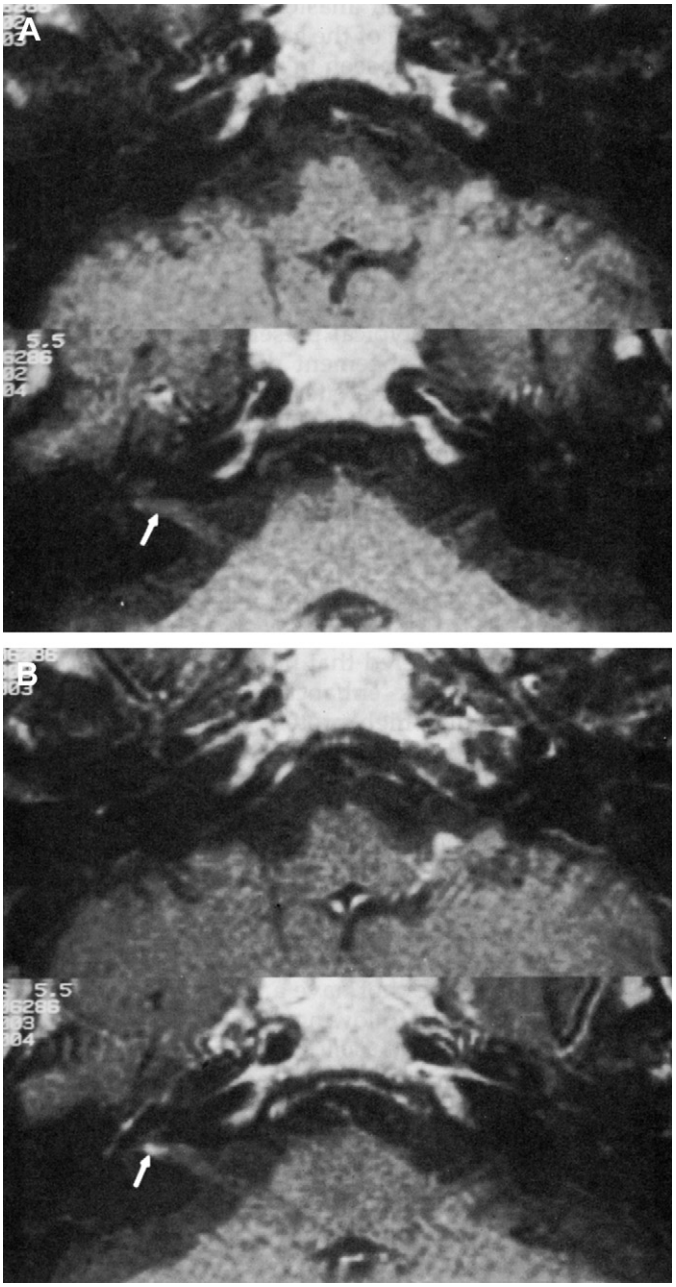

Fig. 18. Bell's palsy. (*A*) Axial T_1-weighted image pregadolinium shows slight increased signal in the internal auditory canal (IAC) (*white arrow*). (*B*) Postgadolinium image shows enhancement in the fundus of the IAC (*white arrow*). The enhancement is irregular and there is no obvious tumor edge. (*C*) Coronal postgadolinium image shows enhancement of the two dots representing the labyrinthine and tympanic segment of the facial nerve canal (*arrow*). Compare with opposite side (*open arrow*). (*D*) Slightly posterior slice shows some enhancement in the IAC (*open arrow*). Trigeminal nerve seen in cross section (*arrow*).

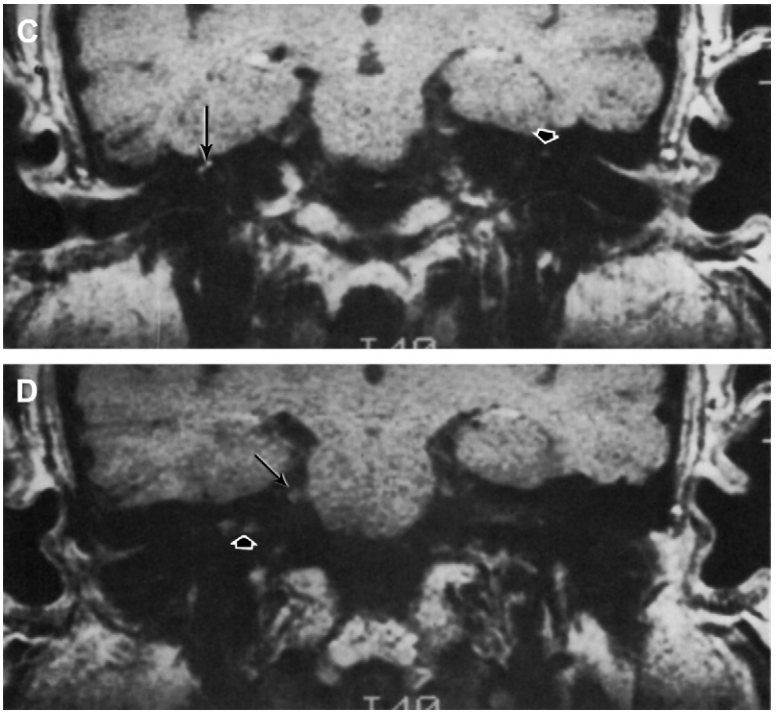

Fig. 18 (*continued*)

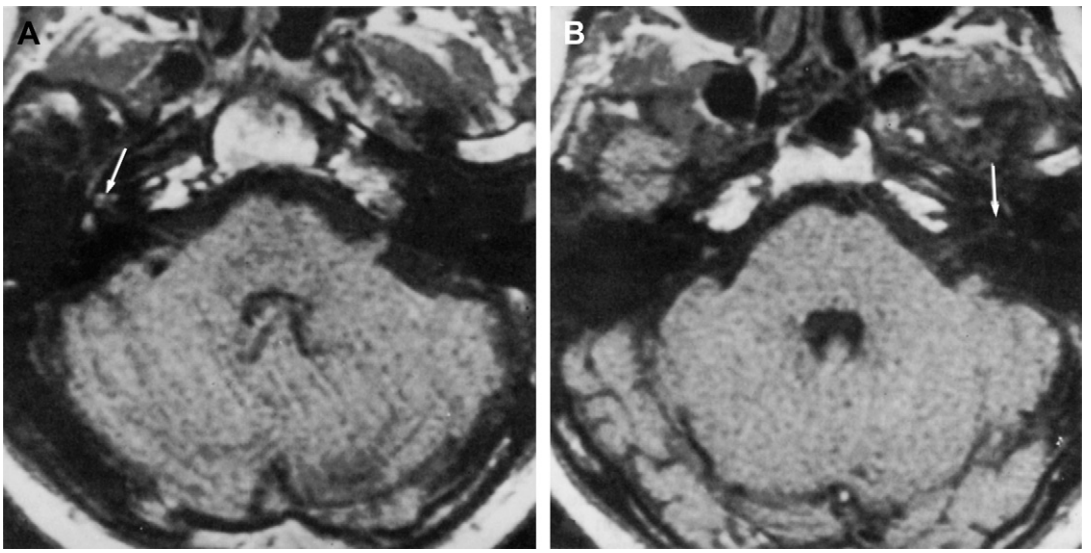

Fig. 19. Patient with sudden hearing loss. (*A*) Precontrast study shows increased signal in the cochlea (*arrow*) and vestibule. Compare with lower signal from the normal cochlea in *B*. (*B*) Image through the cochlea on the opposite side shows normal low signal in the labyrinth.

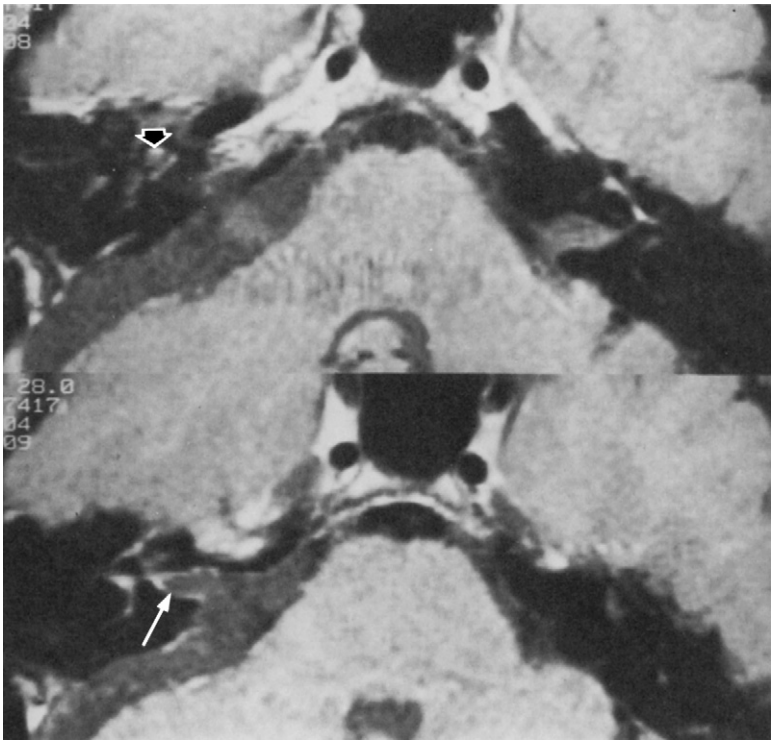

Fig. 20. Postresection postgadolinium T_1-weighted magnetic resonance imaging scan shows enhancement along the wall of the internal auditory canal (*arrow*) with some increased signal in the cochlea (*open arrow*). The increased signal in the cochlea could be a small amount of blood. This increased signal represented a change from the preoperative examination.

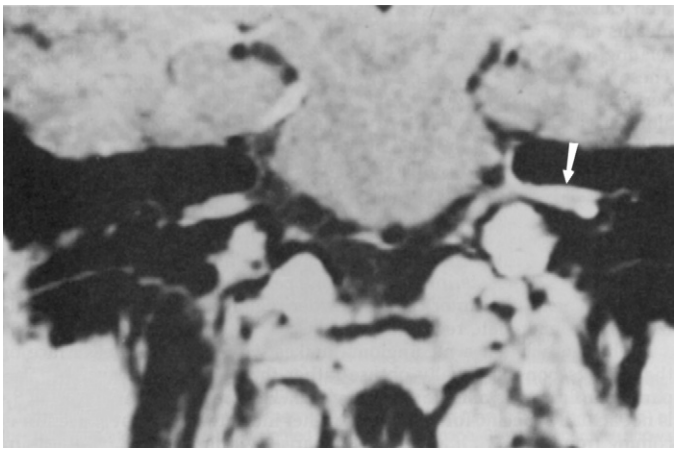

Fig. 21. Sarcoid involving the internal auditory canal (IAC). T_1-weighted image with gadolinium shows enhancement (*arrow*) in both IACs. There is no widening of the canal. (*Courtesy of* Dr. Alexander Marks.)

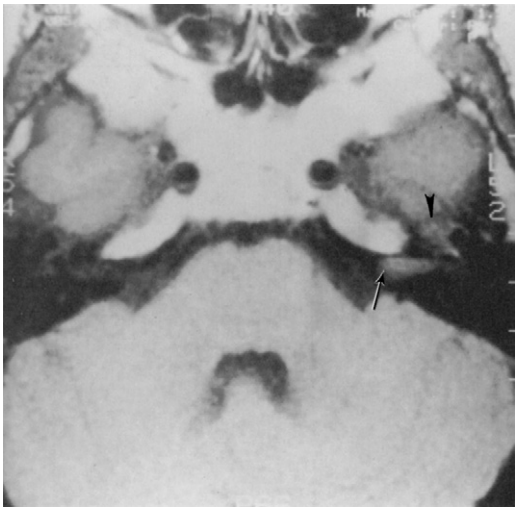

Fig. 22. Facial nerve neuroma shows an intracanalicular tumor (*arrow*) that is indistinguishable from an acoustic neuroma. There is a small lesion, however, in the geniculate (*arrowhead*) indicating the true nature of the lesion. Compare with opposite side.

Other types of inflammation have been reported to cause enhancement in the canal extending into the CPA cistern. Sarcoid and syphilis can cause enhancement that appears to follow the nerve and closely approximates the appearance of an AN (Fig. 21) [35]. Often the margin is unsharp rather than smooth, giving a clue that the lesion is not a nerve sheath tumor.

Tumors and cysts

AN is certainly the most common tumor of the IAC and CPA cistern. There are many other tumors that arise in this location and can occasionally be confused with AN. These include facial nerve sheath tumors, meningiomas, epidermoids, various intra-axial tumors, and a variety of extremely rare lesions [7,9,11,36,37]. Nerve sheath tumors developing from cranial nerve V or cranial nerves IX, X, or XI can grow into the region of the IAC. On CT, an aneurysm can look just like a seventh cranial nerve sheath tumor. Metastasis from distant primaries can involve the nerves in the IAC and thus mimic an AN on imaging. Leukemia has been reported to infiltrate along the nerves and look like an AN on MRI.

A facial nerve sheath tumor that arises totally inside the IAC would be impossible to differentiate from an AN based only on imaging. Like AN, the tumor arising from the sheath of the cranial nerve VII will enlarge the IAC and will enhance on either CT or MRI. Facial paralysis is usually but not always present. In most cases, the facial neurilemoma can be followed into the

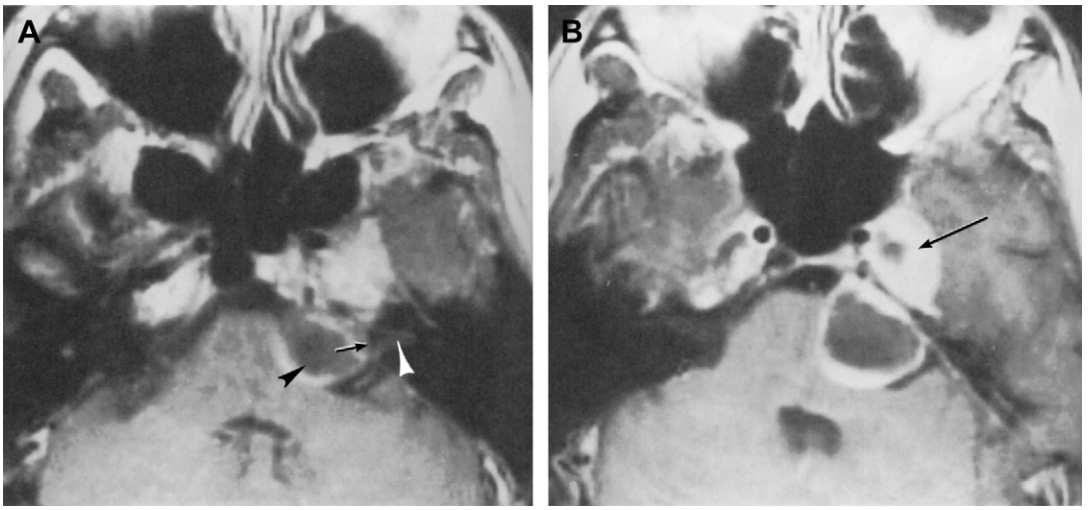

Fig. 23. (*A*) T_1-weighted postgadolinium magnetic resonance image. Trigeminal neuroma. Lesion in the CPA cistern shows a low density cystic center (*black arrowhead*). The lesion extends into the porus (*arrow*) but does not involve the deep fundus (*white arrowhead*) of the internal auditory canal. (*B*) T_1-weighted postgadolinium image. Slightly higher slice shows the lesion in the CPA cistern protruding into Meckel's cave (*arrow*) indicating the identity of the tumor.

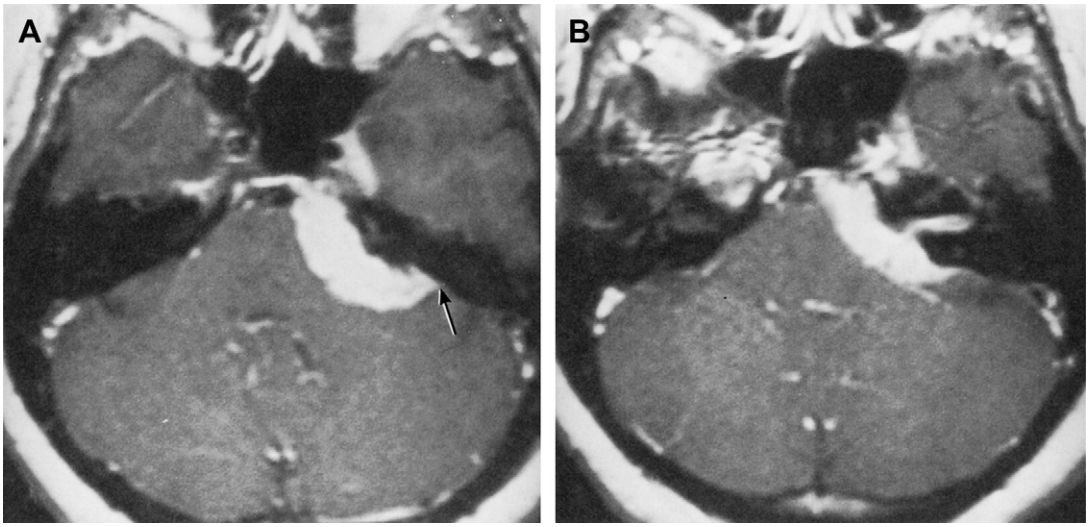

Fig. 24. (*A*) Meningioma of the posterior wall. An enhancing mass lesion is seen forming an obtuse angle (*arrow*) with the posterior wall of the petrous bone. (*B*) Enhancing lesion covers the internal auditory canal (IAC) but the tapering edge suggests the identity. There is enhancement indicating involvement of the IAC.

labyrinthine segment of the fallopian canal, and so the identity is determined (Fig. 22) [30].

Nerve sheath tumors developing from the nerves of the jugular foramen can grow up to the CPA cistern and can even seem to erode the meatus of the IAC (see Fig. 16). Indeed they can present with symptoms relating to the eighth cranial nerve rather than the actual nerve of origin. These lesions can be expected to erode the bony margin of the pars nervosa of the jugular foramen and can often be followed extracranially along a course just posterior to the internal carotid artery immediately below the skull base. For this

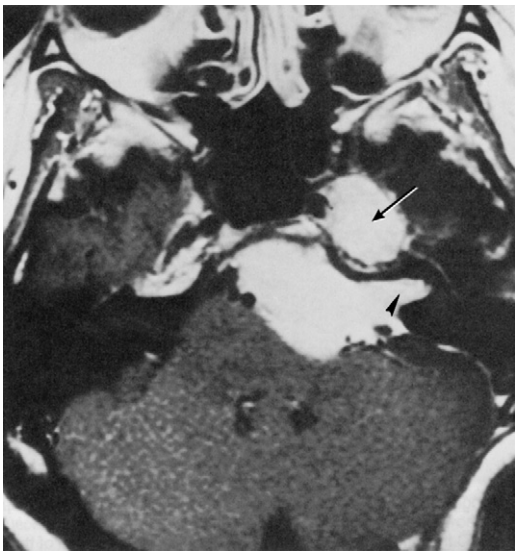

Fig. 25. Meningioma of the posterior and middle cranial fossa. T_1-weighted axial image postgadolinium shows the lesion in the posterior fossa protruding into the internal auditory canal (*arrowhead*). The lesion has a definite involvement in Meckel's cave (*arrow*) that should not occur with an acoustic neuroma.

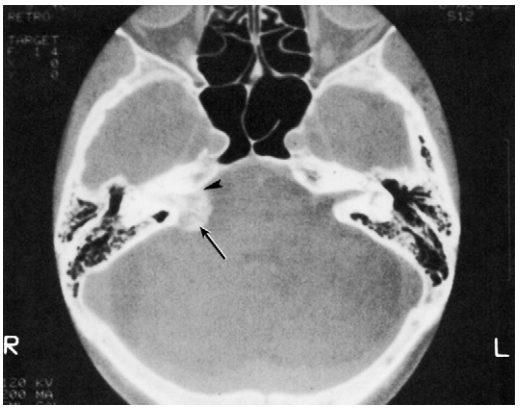

Fig. 26. Meningioma of the internal auditory canal (IAC). Computed tomography scan on a bone algorithm shows a calcified mass protruding from the IAC (arrow). Because this lesion arises from the IAC the junction between the lesion and the posterior wall of the petrous bone is more acute (*arrowhead*).

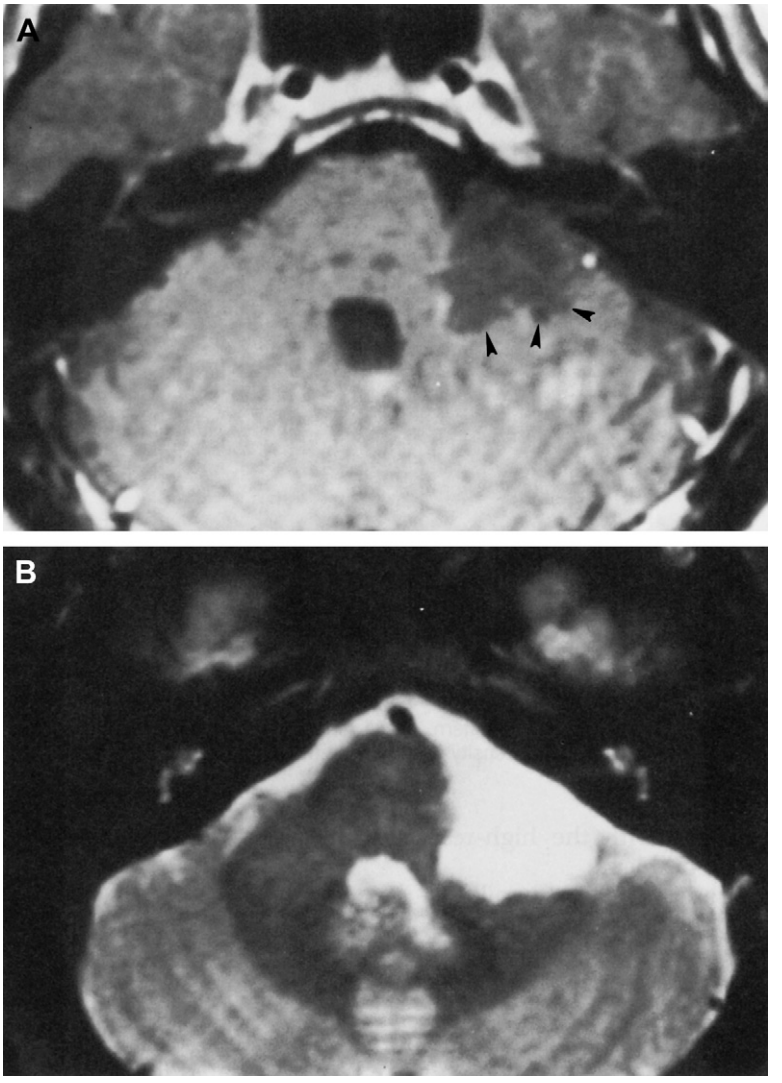

Fig. 27. (*A*) Epidermoid of the CPA cistern. T_1-weighted image. The lesion has slightly more signal than the cerebrospinal fluid. The lesion has an irregular margin as it extends into irregularities of the cerebellum (*arrowheads*). (*B*) T_2-weighted image shows the lesion to have very bright signal.

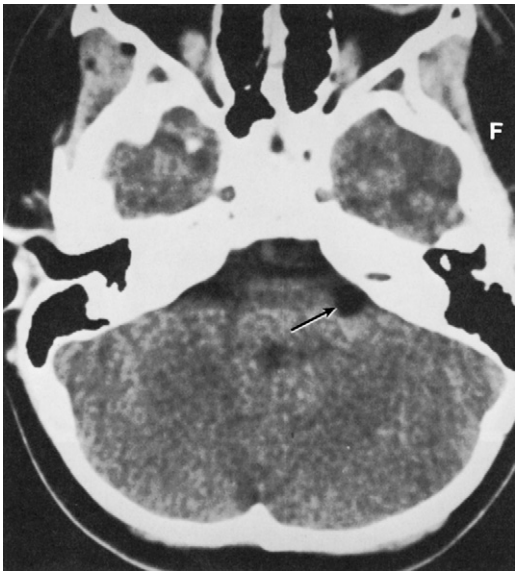

Fig. 28. Lipoma of the CPA cistern. Small lesion with extremely low density on an unenhanced scan (*arrow*). The density reading was the same as the subcutaneous fat (*F*).

reason, the imaging study should define the inferior extent of a presumed AN. For a similar reason, the superior margin should be found, thus making sure that the lesion is not arising from Meckel's cave and the fifth cranial nerve (Fig. 23).

Meningiomas can usually be differentiated from an AN [9,37a]. The classic description states that a meningioma has a flatter, more extensive attachment to the posterior surface of the petrous bone (Figs. 24 and 25) [38]. In other words, the tumor covers a wider region rather than being localized to the immediate region of the IAC [39].

The margin that a meningioma makes with the posterior surface of the petrous bone is usually obtuse rather than acute as in an AN. This particular finding is reliable when present. The presence of an acute angle is not actually specific for an AN but rather indicates only that the lesion is coming from the IAC. If a meningioma arises within the IAC, [39] this lesion can be expected to make an acute angle with the posterior surface of the petrous bone (Fig. 26).

The dura close to the margin of a meningioma can enhance for a variable distance away from the apparent edge of the tumor. This finding suggests the diagnosis of meningioma rather than AN.

Meningiomas can calcify and frequently cause hyperostosis of the contiguous bone. Either of these findings should be considered a strong indicator that the tumor is not an AN but rather a meningioma. Hyperostosis is said to be less common in those meningiomas arising along the

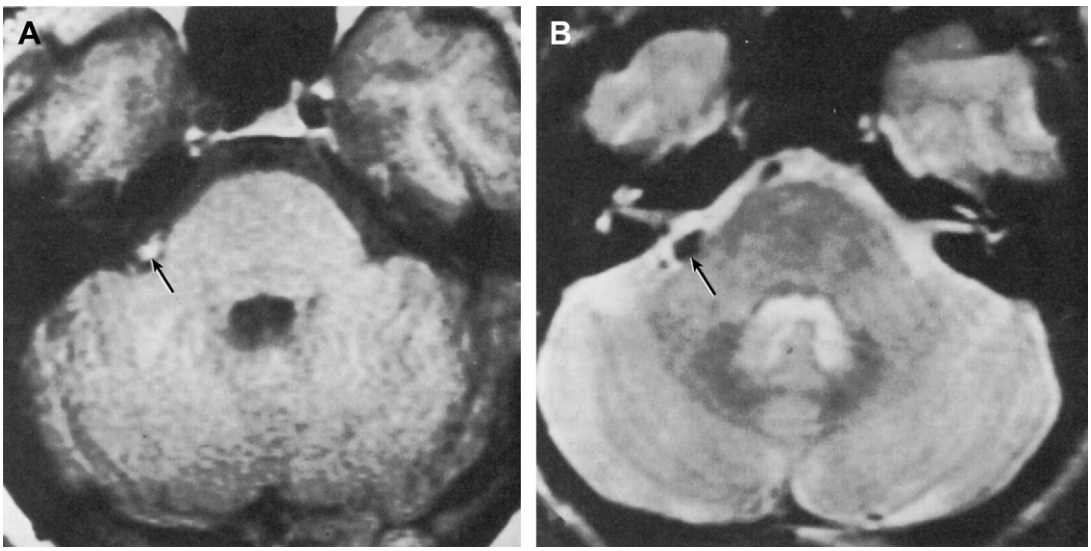

Fig. 29. Lipoma of the CPA cistern. (*A*) T_1-weighted image without contrast shows a small bright mass (*arrow*). (*B*) On the T_2-weighted image, the lesion becomes dark, which is characteristic of fat.

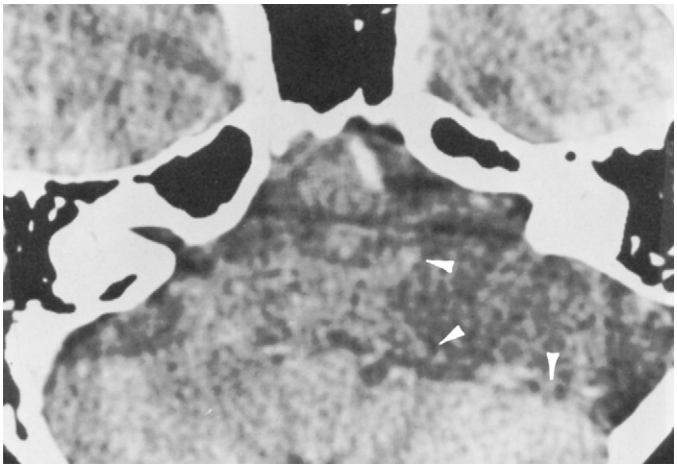

Fig. 30. Arachnoid cyst. Computed tomography with contrast. Cerebrospinal fluid density lesion shows mass effect by pushing the cerebellum and brain stem (*arrowheads*). (*From* Lo WWM: The temporal bone. In: Som PM, Bergron RT, editors. Head and Neck Imaging, edition 2. Mosby-Yearbook; 1991; with permission.)

posterior border of the petrous bone [38] than in other meningiomas but when present is suggestive of the diagnosis.

Hemangiomas arise in the IAC as well as in the region of the facial nerve canal [40,41]. These small lesions can look just like an AN, and this diagnosis should be considered when a small area of enhancement is seen in the IAC.

Lipomas and epidermoids have characteristic appearances on both CT and MRI and should not be confused with an eighth cranial nerve tumor. An epidermoid is expected to be low signal on T_1-weighted images and high signal on T_2-weighted images (Fig. 27). On CT, the lesion may have the same density as CSF. Rarely an epidermoid can be dense on CT [42]. An epidermoid will not

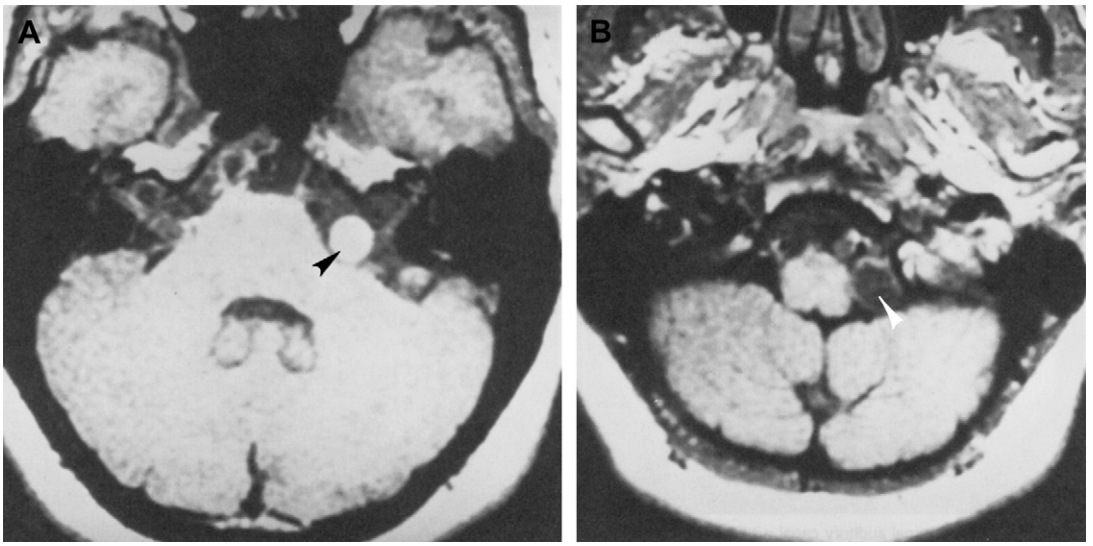

Fig. 31. Cysticercosis. (*A*) Noncontrast (SE 733/20) image shows high signal mass (*arrowhead*) in the CPA cistern. (*B*) Scan at the lower level shows another more typical cyst. Note that at this level a distinct wall can be identified. There is a small focus of signal from within the cyst (*arrowhead*). This may represent a scolex within the cyst.

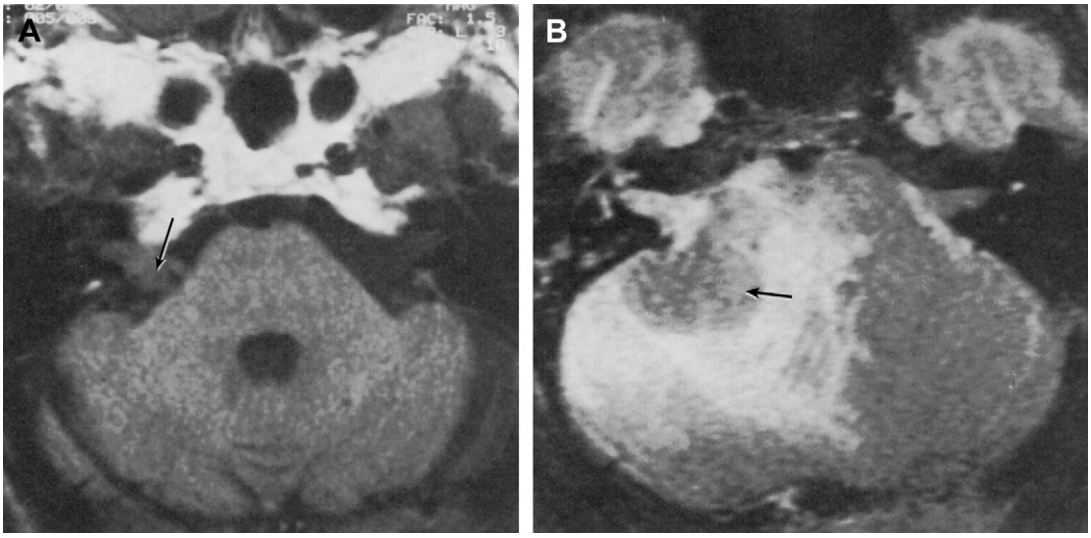

Fig. 32. Metastases to the internal auditory canal with facial nerve paralysis and hearing loss. (*A*) T_1-weighted image without gadolinium shows a soft tissue mass (*arrow*) filling the porus and protruding into the CPA cistern (*arrow*). Computed tomography scan showed slight enlargement of the facial nerve canal (not shown). (*B*) Two months later there is a considerable increase in symptoms. The mass (*arrow*) has increased in size. There is shift of the brain stem and considerable edema in the brain contiguous with the lesion.

enhance on either CT or MRI. A lipoma is high signal on T_1 without contrast enhancement and low signal on T_2, and on CT it will have a characteristic low (fat) density (Figs. 28 and 29).

Arachnoid cysts can occur in the posterior fossa either as an isolated finding or in association with an AN. An isolated arachnoid cyst may have an appearance much like that of an epidermoid. It will be the density of CSF on CT and behave like CSF on MRI (dark on T_1 and bright on T_2) (Fig. 30). Again there should be no enhancement on either CT or MRI.

Cysticercosis is a rare cause of a cystic abnormality in the cerebellopontine angle cistern (Fig. 31) [43,44]. The abnormality may be seen as a discrete cyst or may be irregular with the cysts forming conglomerations that cannot be clearly separated from the contiguous CSF spaces. The signal on MRI and the density on CT closely mimic CSF. This diagnosis should be considered in regions where this disease is common.

Metastasis affecting cranial nerves VII and VIII usually involves the bone first and extends into the region of the canal secondarily. Occasionally a metastasis can reach the IAC by direct hematogenous (Fig. 32) or even intrathecal spread (Fig. 33). This infrequent occurrence can be impossible to differentiate from AN, especially

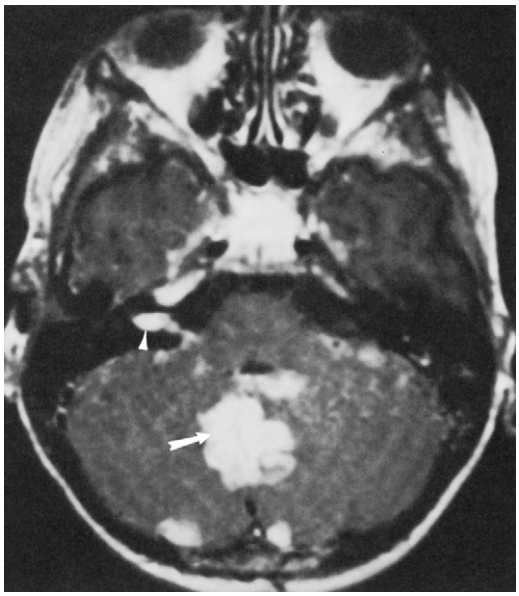

Fig. 33. Subarachnoid metastases from primitive neuroectodermal tumor. Axial postcontrast magnetic resonance images (SE 500/20). There is an enhancing mass in the cerebellar vermis (*arrow*). There are multiple subarachnoid metastases including one in the right internal auditory canal (*arrowhead*).

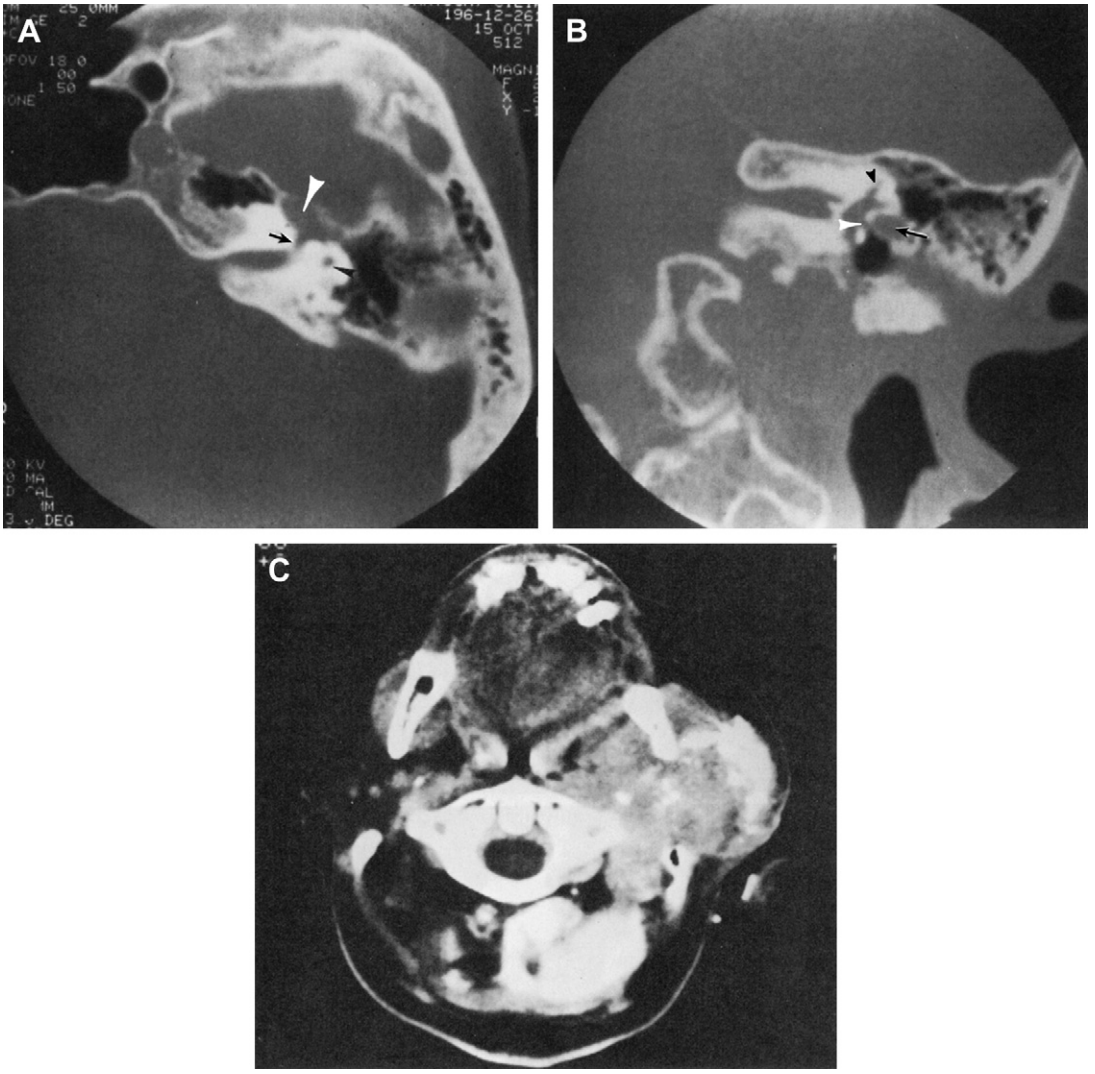

Fig. 34. Perineural extension along the facial nerve canal. Patient has parotid mass and facial paralysis. (*A*) There is enlargement of the geniculate ganglion (*white arrowhead*) with slight enlargement of the labyrinthine segment of the facial nerve canal (*arrow*). Superior semicircular canal (*black arrowhead*). (*B*) Coronal image shows enlargement of the tympanic segment of the facial nerve canal (*arrow*). Normal structures; oval window (*white arrowhead*), superior semicircular canal (*black arrowhead*). (*C*) Computed tomography sialogram. Inferior slice shows a large tumor in the parotid gland.

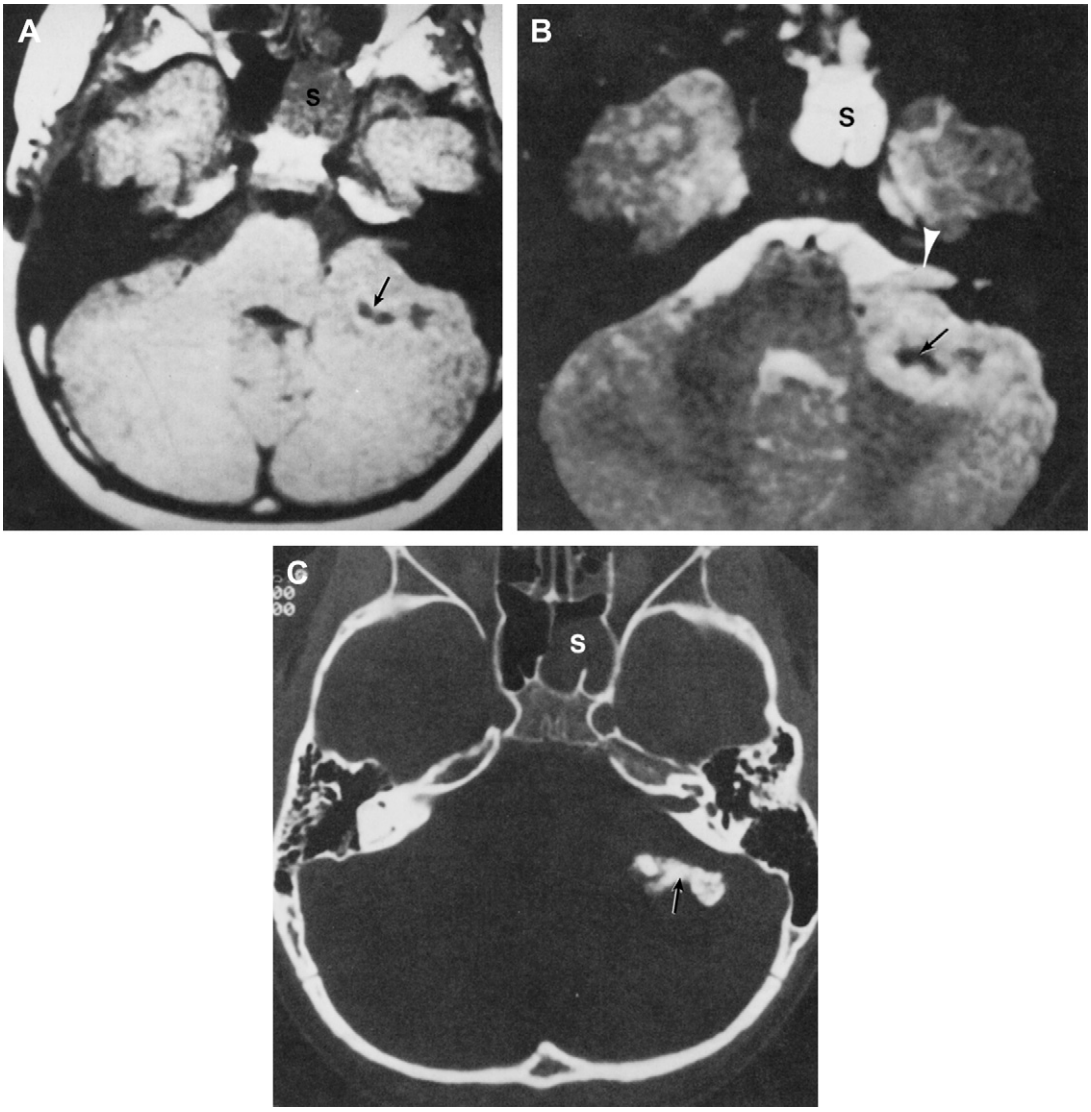

Fig. 35. Cerebellar astrocytoma with extension into the internal auditory canal in a 7-year-old boy. (*A*) Axial T_1-weighted image shows lesion in the CPA cistern. The low signal area (*arrow*) does not represent fluid but calcium. (*B*) T_2-weighted image again shows the low signal of the calcified region (*arrow*). The lesion protrudes into the internal auditory canal (*arrowhead*). (*C*) Computed tomography demonstrates calcification (*arrow*). Note the secretions (S) in the left sphenoid sinus.

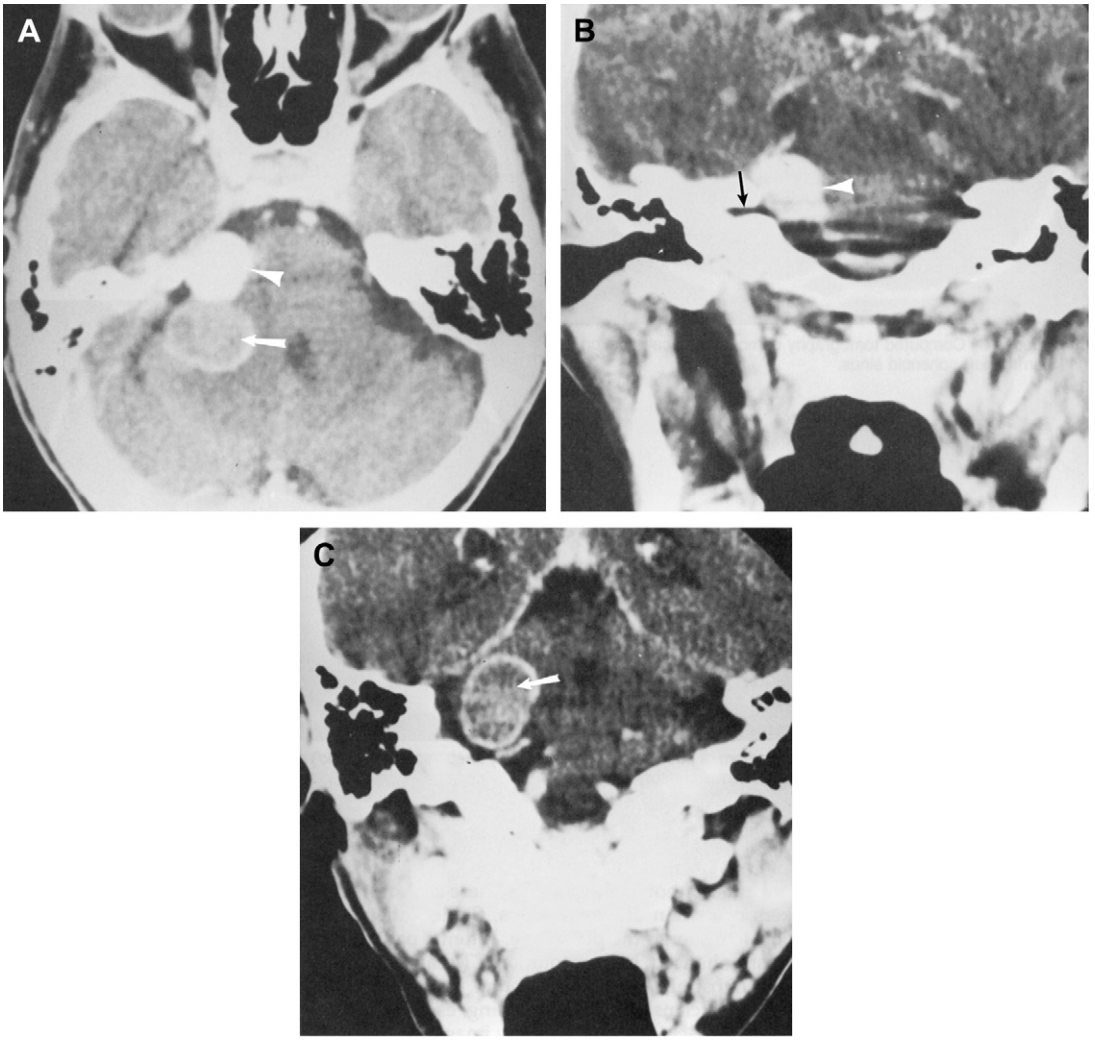

Fig. 36. (*A*) Partially thrombosed aneurysm of the vertebral artery. "Mass" in the CPA cistern is bilobed. The anterior portion (*arrowhead*) opacifies on venous injection of contrast. The posterior portion (*arrow*) does not opacify indicating thrombus. (*B*) Coronal image through the anterior portion of the mass shows enhancement (*arrowhead*). Note that the mass does not protrude into the internal auditory canal (*arrow*). (*C*) Posterior slice shows the thrombosed portion of the aneurysm (*arrow*).

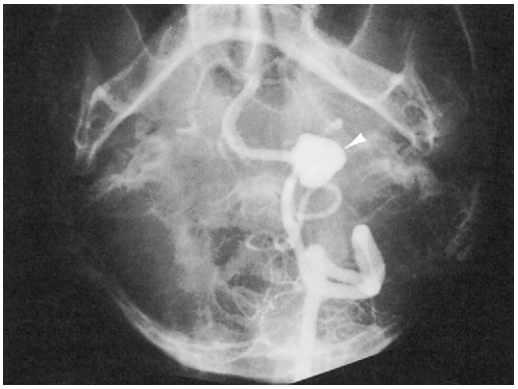

Fig. 37. Arteriogram of patient with vertebral artery aneurysm (*arrowhead*) at the approximate level of the internal auditory canal.

if the patient is not known to have a primary malignancy. Rapid growth may suggest this diagnosis.

Metastasis can also reach the IAC by spreading along the facial nerve. This so-called perineural spread is from malignancy of the parotid gland, usually an adenoid cystic carcinoma (Fig. 34). This should not be a diagnostic problem because the primary tumor is obvious either on the images or to palpation. The tumor can be followed along the nerve, enlarging the facial nerve canal.

Tumors arising in the brain stem or cerebellum can grow out into the CPA cistern (Fig. 35). In the past, differentiation from AN could be difficult but with the high-resolution imaging available today, this is seldom a problem.

A choroid plexus papilloma can arise in the fourth ventricle and extend through the foramen of Luschka into the CPA cistern. Alternatively a papilloma can arise from the small bit of choroid that has protruded through the foramen, and thus the lesion is totally within the cistern.

Vascular abnormalities can mimic ANs. Aneurysms cause a mass effect. They may be identified if there is an associated flow void or if clot within a partially thrombosed lumen is identified by typical MRI signal characteristics (Figs. 36 and 37). An artery may not have an actual aneurysm but can simply be tortuous or ectatic (dolichoectasia) and can cause symptoms by pressing on cranial nerves. The demonstration of a loop of the anterior inferior cerebellar artery close to a nerve or inside the canal is not uncommon but can be seen in a normal patient, and so the significance of this finding is not clear.

Lesions arising in the temporal bone such as a cholesterol cyst, cholesteatoma, or one of a variety of bone tumors are usually easily distinguished from AN. The origin outside the IAC is usually obvious. Occasionally a tumor, such as a paraganglioma, can have a substantial component protruding into the CPA cistern, but once again the finding of the characteristic erosion of the jugular fossa on CT or the characteristic black dots on MRI representing rapid flow in large tumor vessels (flow voids) indicate the true identity of the tumor (Fig. 38).

Finally a piece of polytef (Teflon) put in place to separate a vessel from a nerve can sometimes resemble a neuroma (Fig. 39). It can be dense on CT. There can be some enhancement of the contiguous tissues on CT or MRI. The site of the previous craniotomy is obvious, and the patient gives the appropriate clinical history.

Imaging in specific situations

Rule out acoustic neuroma

When the primary question is whether the patient has an AN, a gadolinium-enhanced MRI scan is the preferred imaging modality [3,18,45–47].

In the past one could not be completely sure that there was no AN unless the nerves could be visualized all the way through the IAC and no enlargement was present (Figs. 40 and 41). Now the contrast-enhanced MRI scan is considered sensitive enough that this is no longer necessary, and if no enhancement is seen, the examiner can confidently state that there is no AN present.

At our institution, we rely most strongly on high-resolution short TR/TE (T_1 weighted) axial images acquired after injection of gadolinium. Two interleaved sets of images are taken in one combined sequence, using 3-mm slices spaced every 4 mm. Because the slices are interleaved, the effective spacing is 2 mm. In other words, there is slight overlap and the entire canal is covered. Short TR/TE (T_2 weighted) sagittal and axial 5-mm slice thickness long TR/TE (T_2 weighted) sequences are also performed as part of the usual imaging protocol.

At many institutions, axial short TR/TE (T_1 weighted) images are performed before gadolinium is injected. Some radiologists perform T_1-weighted coronal sequences as well to have a second look at the canal. We do not find these necessary if the axial images are of excellent quality.

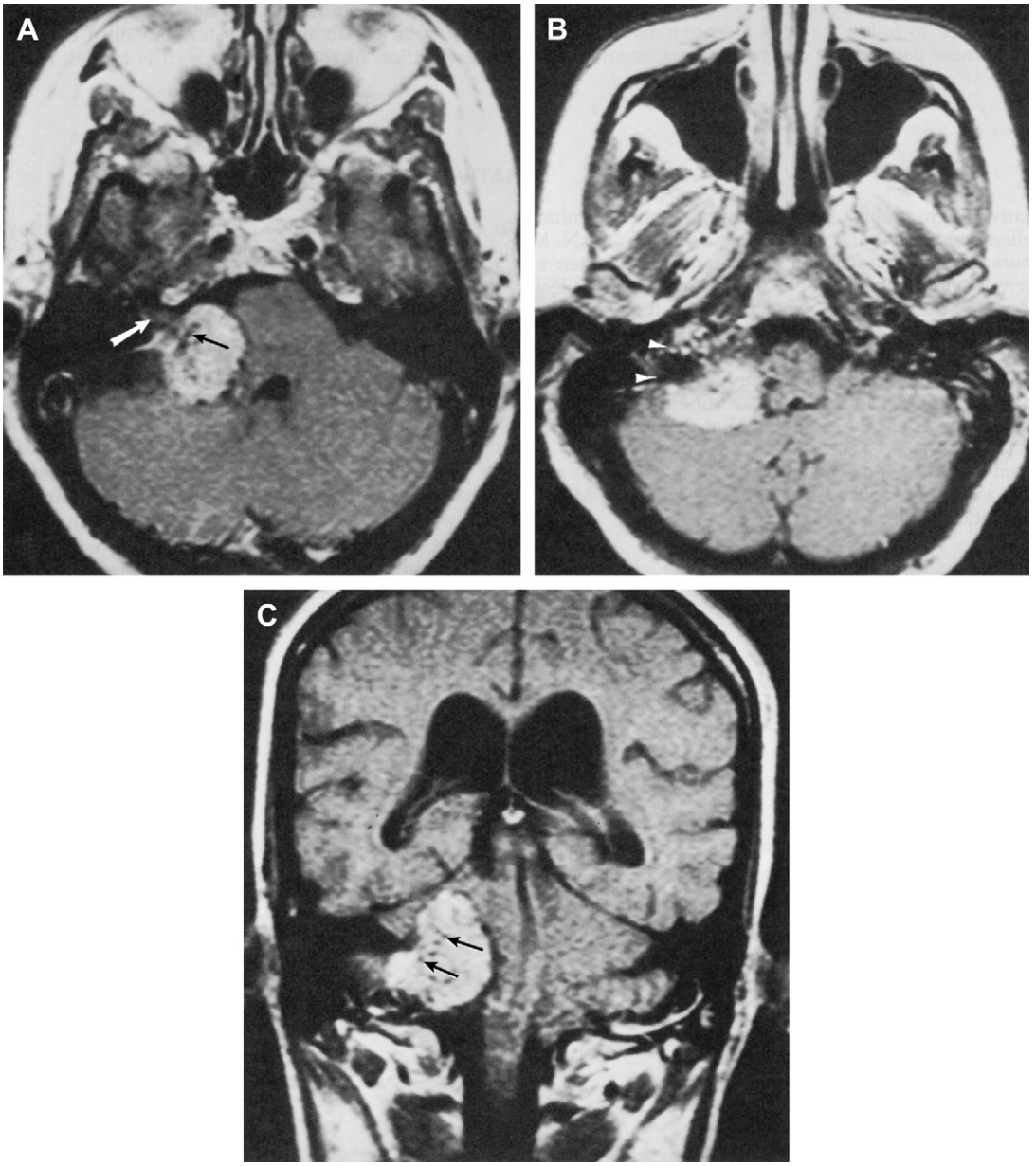

Fig. 38. (*A*) Paraganglioma protruding into CPA. Axial T_1-weighted postgadolinium image shows the lesion of the CPA cistern but it does not extend into the internal auditory canal (*white arrow*). There are small black dots that represent flow voids within the lesion (*black arrow*). (*B*) The lesion extends inferiorly and involves the bone along the lateral margin (*arrowheads*). (*C*) Coronal image shows the true origin of the lesion in the inferior temporal bone and the flow voids (*arrows*) characteristic of paraganglioma.

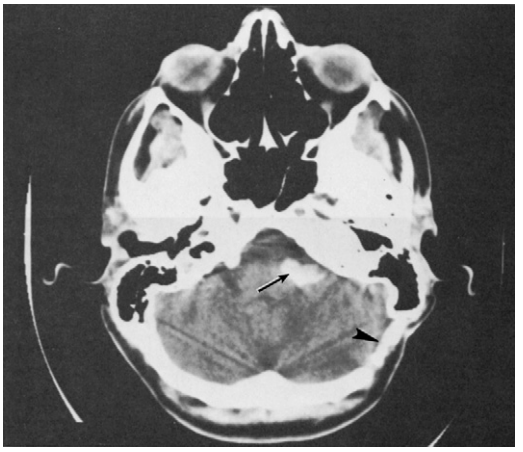

Fig. 39. Polytef (Teflon) in the CPA cistern. Increased radiodensity represents a piece of Teflon (*arrow*) used to separate nerve from vessel. Note the retromastoid craniotomy (*arrowhead*).

In some cases of acute symptoms, we have performed an axial T_1-weighted sequence before gadolinium injection to determine if there is abnormally high signal that is not due to enhancement. This is not likely to become part of our routine protocol. In cases in which there is higher than normal signal and there is a question of whether this represents enhancement or actually some other cause of increased signal (eg, hemorrhage), the scan can be repeated after several days when the gadolinium has disappeared from the system. If the signal is still present, it does not represent enhancement and some other explanation should be considered.

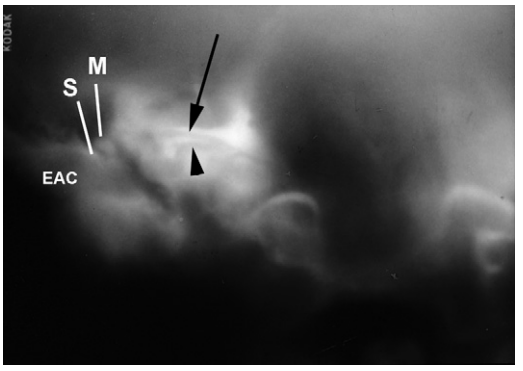

Fig. 40. Tomogram of the internal auditory canal after metrizimide instillation through a C1-2 puncture. The contrast can be followed into the canal outlining the facial nerve (*arrow*) and cochlear nerve (*arrowhead*). Normal size of the nerves indicates that there is no acoustic neuroma. EAC = external auditory canal; M = malleus; S = scutum.

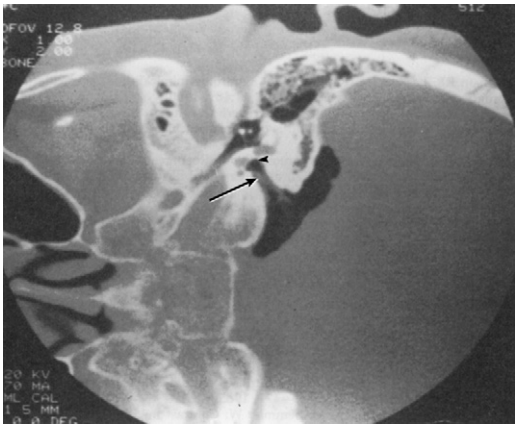

Fig. 41. Air cisternogram shows filling of the internal auditory canal (IAC) with air outlining the nerves (*arrow*). Note that the air passes all the way to the extreme lateral portion of the IAC (*arrowhead*).

Some patients cannot undergo MRI. There are contraindications such as pacemakers or certain aneurysm clips. Some patients are simply too claustrophobic to undergo the procedure. When a patient cannot undergo MRI, a contrast-enhanced, high-resolution CT scan is still a highly reliable examination. Almost all ANs will be found by high-quality modern CT scanning. Some clinicians prefer CT scanning, especially when the clinical picture is not as clear-cut and high-quality images of the otic labyrinth are desirable at the same time that the IAC is evaluated. Also CT remains more available in some locations and so becomes the initial study, with MRI reserved for the few cases in which the CT scan is considered equivocal.

Once an acoustic tumor is diagnosed by MRI, some surgeons request a CT scan for surgical planning if a posterior approach is contemplated in an attempt to preserve useful hearing (Fig. 42). A scan done for this purpose can be done without iodinated contrast material because the primary aim is not to see the tumor but to see the anatomy of the bone. On a high-resolution bone algorithm, the position of the vestibular aqueduct can be seen and the amount of bone separating the aqueduct from the lip of the IAC can be determined. The depth of the mastoid air cell system and the position of the sigmoid sinus are clearly shown, as is the relationship of the semicircular canals to the posterior wall of the petrous bone. Thus accidental violation of the labyrinth is, it is hoped, less likely.

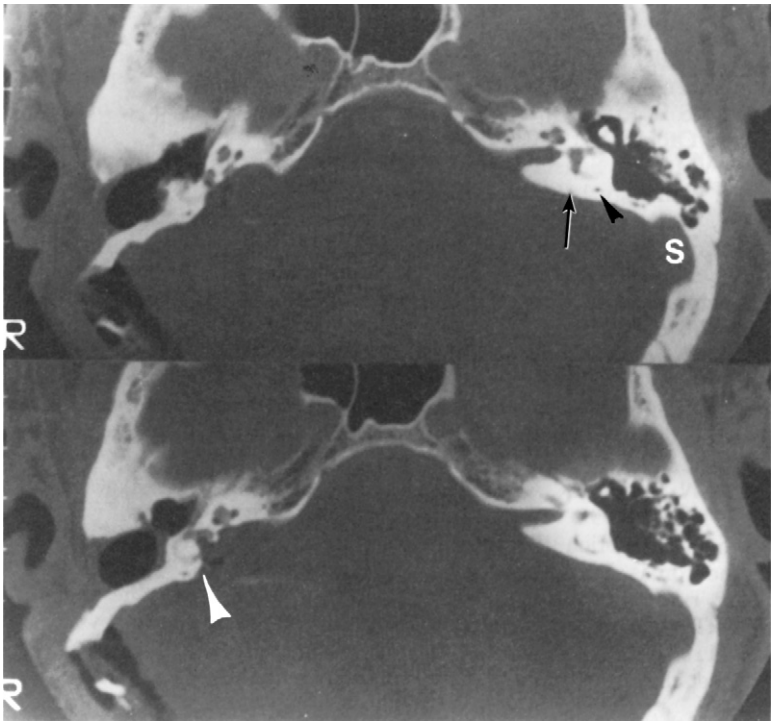

Fig. 42. Postsurgical resection acoustic neuroma right side. Bone algorithm shows the position of the vestibular aqueduct (*arrow*) on the right side. The surgical approach intersected the vestibular aqueduct on the left side (*white arrowhead*). This surgical defect extends into the vestibule. Other important structures include posterior semicircular canal (*black arrowhead*) and the sigmoid sinus close to the sinodural angle (S).

Postoperative evaluation

Once an AN has been removed, imaging may still play a role in following the patient to detect a recurrence. In hopes of retaining useful hearing, surgeons try meticulously to strip tumor away from the eighth cranial nerve. Then imaging is used to ensure that the tumor does not recur.

Either contrast-enhanced CT or contrast-enhanced MRI can be used. MRI is very sensitive. There is enhancement in the region of the resection in many cases, and this should not be considered to represent tumor (see Fig. 20). The dura can enhance and so can the reparative process in the region of the surgery. The initial examination can be considered as a baseline study. Future examinations are compared to find progressive changes that would indicate tumor regrowth. CT is again slightly less sensitive but, as with MRI, an enlarging area of enhancement on serial examinations suggests recurrence. Further experience is needed in the field of postoperative imaging before definitive statements regarding the significance of findings can be made.

Imaging is used to follow patients after gamma knife radiosurgery. The findings in these patients are covered in another article in this issue.

Summary

A negative high-quality, high-resolution, contrast-enhanced MRI scan is excellent evidence that a patient does not have an AN. Most nerve sheath tumors have a characteristic appearance, and when a tumor is detected there is seldom any doubt as to the identity of the lesion. There are other causes of enhancement, however, or of high signal that can be mistaken for an AN, and these must be kept in mind when a case is considered positive. In some cases, it may be appropriate to defer surgery to clarify a questionable finding by obtaining a follow-up scan.

CT is still a reliable examination. In addition to evaluating the IAC, valuable information about the architecture of the petrous bone and labyrinth is provided.

Improvements in imaging technology are occurring at a rapid rate. Thinner slices and more

rapid scan techniques will make MRI even more useful in evaluation of the IAC.

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NEUROSURGERY
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Conservative Management of Acoustic Neuromas

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Improved diagnostic screening, most notably auditory evoked brain stem response testing and more sophisticated and generally available imaging as well as a better informed population, have resulted in increasing numbers of smaller and less symptomatic acoustic neuromas being diagnosed [1].

Advances in surgical technique, anesthesia, and perioperative care have dramatically reduced both operative mortality and morbidity. Although the mortality associated with acoustic neuroma excision was on the order of 80% in the early 1900s, this figure has been reduced to approximately 1% in most current series. Lower cranial nerve palsy and significant residual trigeminal hypesthesia is now a rare and generally isolated event [2,3].

As a result, several authors have suggested that all tumors, with few exceptions, should be removed following diagnosis [4,5]. Others have proposed that a more expectant attitude is an acceptable alternative to immediate surgical excision [6–9].

A conservative (non–tumor excision) strategy is based on the premise that surgical removal may pose a greater risk. It assumes that given the nature of acoustic neuroma clinical behavior, ie, pattern of growth, these tumors will not result in either mortality or significant morbidity within the expected life span of a given individual.

The ability to predict a tumor's potential for growth would clearly be of benefit in the assessment of such a treatment strategy. A number of studies have been undertaken to answer this question. Studies of individuals with acoustic neuromas have been carried out to define their rate and pattern of growth. Overall tumor growth is generally considered slow [10]. Annual rates of growth of 0.2 cm per year or less have been noted in the majority of cases followed [11,12]. Importantly 40% or more of tumors studied had no growth, and in several cases, demonstrable tumor shrinkage occurred [13–15]. Very importantly, however, several studies have unequivocally identified a percentage of patients in whom tumor growth is relatively rapid, i.e., exceeding 0.2 cm per year [16,17].

A number of patients have been successfully followed for up to 10 years without appreciable change in symptoms [7]. Such studies confirm the feasibility of adopting a conservative course of management in selected patients. If a strategy could be developed that reliably predicts the future growth of acoustic neuromas, a more rational approach in the management of these patients would evolve. A conservative policy that ultimately results in a large tumor, causing significant disability in an older patient who has been followed for years, is undesirable.

Most clinical studies have used serial imaging as a method of determining tumor growth. Annual tumor growth rate has in many instances become the predominant factor in considering future tumor enlargement [6]. No statistically significant correlation between tumor size and patient age at the time of presentation has been found [7,15].

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Other parameters have been used to predict tumor growth. Attempts have been made to correlate the percentage of tumor cells undergoing mitosis to the clinical course of affected individuals. The use of 5-bromo-deoxyuridine followed by immunolocalization has supported the clinical impression of the overall slow growth of acoustic tumors [18]. Flow cytometric study has defined a variable mitotic rate, suggesting a variable growth pattern [19]. Immunohistochemical study, using the monoclonal antibody KI-67, has revealed a high mitotic rate associated with a faster growing tumor [20]. In the clinical studies published to date, however, no correlation was found between presenting tumor size and the rate of cell proliferation. In one study, a number of tumors studied with flow cytometry were found to have proliferative potential equivalent to some malignant tumors. The usefulness of such a comparison is unclear, given the inability to correlate this laboratory finding to actual tumor behavior. Clearly factors other than actual cellular turnover rates influence changing tumor size. Hemorrhage, cystic degeneration, and scarring also play a role.

If a nontreatment management strategy is to be a serious alternative in the management of patients with acoustic neuromas, a definition of selection criteria, guidelines as to follow-up, and clinical assessment and imaging parameters are crucial. In an attempt to answer some of these concerns, a prospective study was initiated at Sunnybrook Health Science Centre, University of Toronto, in 1978, to follow a select group of mostly older patients with unilateral, previously untreated acoustic neuromas.

Study group

Four hundred seventy-four patients with acoustic neuromas were seen at Sunnybrook Health Science Centre, between the years 1976 and 1991. A conservative approach (non–tumor excision) was adopted in 56 individuals with unilateral acoustic neuromas. All patients with recurrent or persistent tumors following previous surgery as well as those with bilateral tumors have been excluded. Of the 56 patients in whom conservative management was embarked on, six were excluded for lack of good quality computed tomography (CT) images.

Our study group therefore consisted of 50 patients, 34 of whom were female and 16 male, with an age range of 50 to 83 years (mean = 68.1). Thirty-nine were 65 or over. Table 1 details the reasons for selecting a conservative course of management. Note that of the patients under age 65, extenuating circumstances and refusal were responsible for the decision. Of particular interest are two individuals, one age 50 with an acoustic tumor in the only-hearing ear (the other ear having been deafened following mastoid surgery), and one age 60, who had severe Meniere's disease in the opposite ear.

Table 1
Rationale for conservative management (n = 50)

Rationale	Age ≥ 65 years	60–64	50–59
Age	23	—	—
Age and medical problems	13	—	—
Medical problems	—	2	—
Refusal	3	4	3
Tumor in only or better hearing ear	—	1	1

In patients selected or who themselves have opted for conservative management of their tumor, a follow-up policy has been adopted that includes regular full neurotologic examination together with serial CT scanning at 6-month intervals. In those patients followed for a number of years in whom there has been no clinical or radiologic evidence of tumor growth, the follow-up interval has been extended to 1 year. Conventional contrast-enhanced CT examination of the posterior cranial fossa using either 5-mm or 1.5-mm slices through the internal auditory canal has been the most frequently used method to monitor tumor growth during the follow-up period. In two individuals with lesions confined to the internal auditory canal, either air/CT meatography or more recently magnetic resonance imaging (MRI) has been used to monitor tumor growth. MRI was used to monitor one such patient in this series.

All scans were assessed, and all measurements related to tumor size were carried out by one neuroradiologist (EEK). The radiologist was unaware of the patients' clinical status and future management plans. All measurements were made more than once to validate their accuracy. In this study, the reliability coefficient obtained was 0.998, for simple replication. A value of 1 represents 100% repeatability/accuracy.

Tumor size would be best expressed by an exact measurement of tumor volume [17]. Because this has not been possible, a conservative method has been adopted whereby the mean of the maximum anteroposterior and mediolateral

dimensions of the cerebellopontine angle mass is used to represent tumor size. As described, internal auditory canal tumor content was not included in the measurement. The method and rationale for use of this technique are described in detail in a previous publication [7]. All patients included in this study had at least two CT scans (range 2 to 18), of sufficient quality to allow accurate measurements to be made. The period of follow-up in each case includes only the interval from the first to the last scan from which accurate measurement was possible. Clinical follow-up outside these limits during which scans were judged to be of poor quality has been excluded. Although this has inevitably led to a shortened follow-up interval (mean follow-up 41.7 months; range 7 to 152 months), a more accurate assessment of tumor growth has been ensured. For instance, the individual followed for the longest time in our study had a CT scan over 2 years earlier, which because of its poor quality rendered it inadmissible. This reduced the follow-up period in her instance from 181 to 152 months. Twenty-eight patients have been followed for 3 or more years, with 10 for more than 5 years.

Data Analysis

Annual tumor growth rate was calculated in two ways (Fig. 1). Method 1 uses the initial and final tumor size divided by the number of years of follow-up. Thus it does not take variability of growth within the total follow-up time into account. Method 2 accounts for growth variability. It is the mean of the individual annual growth rates obtained between each interval of assessment during the follow-up period.

Tumor size was measured to 0.001 cm using a micrometer. This value was rounded off to the nearest 0.01 cm (0.1 mm). Although the reproducibility of these measurements was demonstrated, for reasons of clinical significance, a tumor was judged to have changed in size only if the total growth was 0.1 cm or more. The calculated growth rate, however, was the total growth divided by the number of years of follow-up and as such could be less than 0.1 cm per year.

In an effort to determine whether tumor growth is constant or significantly variable in the same individual, a measure of change in growth rate between consecutive follow-up intervals was

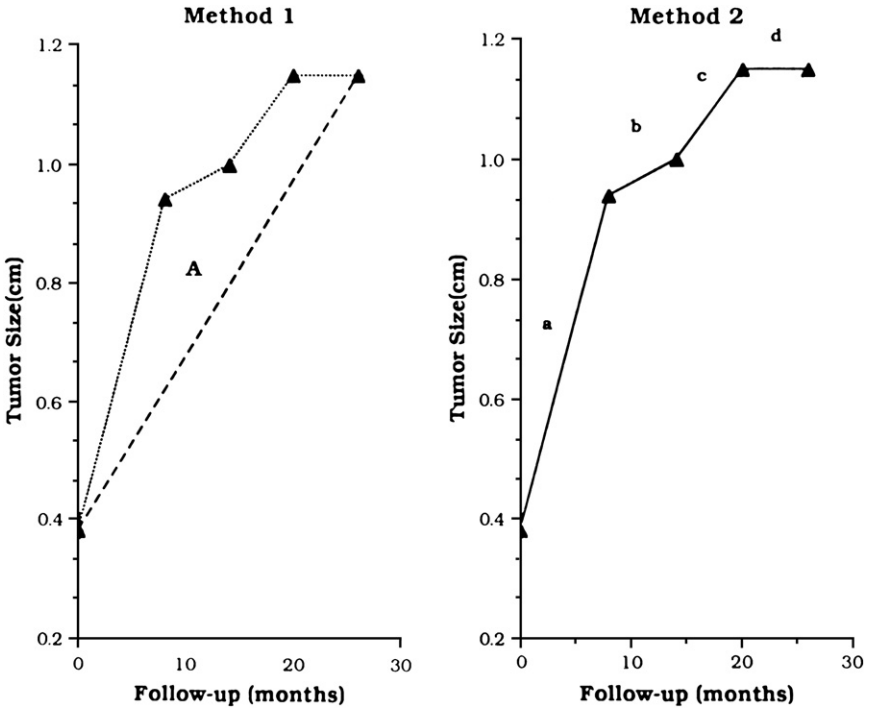

Fig. 1. Calculation of tumor growth rate. (Method 1) Growth rate is calculated as the difference between the first and last measurement and expressed in cm/yr; (Method 2) growth rate is calculated as the mean of individual measurements taken between the first and last study.

also calculated (growth variability). This was determined in 30 of the 50 patients. Small growth variability values represent constant growth, whereas large changes indicate significant variability, whether it be an increase or decrease in size (Fig. 2). Multiple regression analysis was also used to determine whether age or initial tumor size was related to subsequent growth.

Results

Growth rate

Using method 1, the mean annual growth rate for the study group was determined to be 0.11 cm per year, whereas method 2, which took into account variations in yearly growth, was 0.11 cm per year. All growth rates subsequently referred to are those obtained by method 1, which is easier to calculate. These rates range between an actual reduction in tumor size of 0.51 cm per year to an increase of 0.98 cm per year. Fig. 3 illustrates the distribution of growth rates of tumors for all patients. Note that the majority of patients demonstrate little, if any, growth. These findings are summarized in Table 2. Fully 78% of individuals had a growth rate of less than 0.2 cm per year. In nine patients, the tumor was noted to decrease in size (range of –0.51 to –0.01 cm per year). The CT images in Fig. 4 illustrate a reduction in tumor size from 2.2 cm to 1.05 cm, a 1.15 cm decrease over a 5.5 year follow-up period. Seventeen patients had no measurable change in tumor size. The interval CT scans of one such individual, followed for nearly 13 years (presently age 85), is shown in Fig. 5. Measurable tumor growth occurred in 24 patients (range 0.01 to 0.98 cm per year). The others are as listed in Table 2.

The maximum growth measured was 1.15 cm over a 14-month interval in a 66-year-old woman. Tumor size increased from 1.45 cm to 2.6 cm (Fig. 6). Although less dramatic, in terms of absolute size, another patient, age 65, realized a threefold increase over a 9-month period (0.38 to 1.0 cm) (Fig. 7). In the patients followed for more than 3 years and more than 5 years, the growth rates were +0.04 cm per year and –0.02 cm per year, respectively.

Growth variability

The mean variation in growth rate for the 30 tumors studied is 0.13. The distribution, illustrated in Fig. 8, indicates that the majority

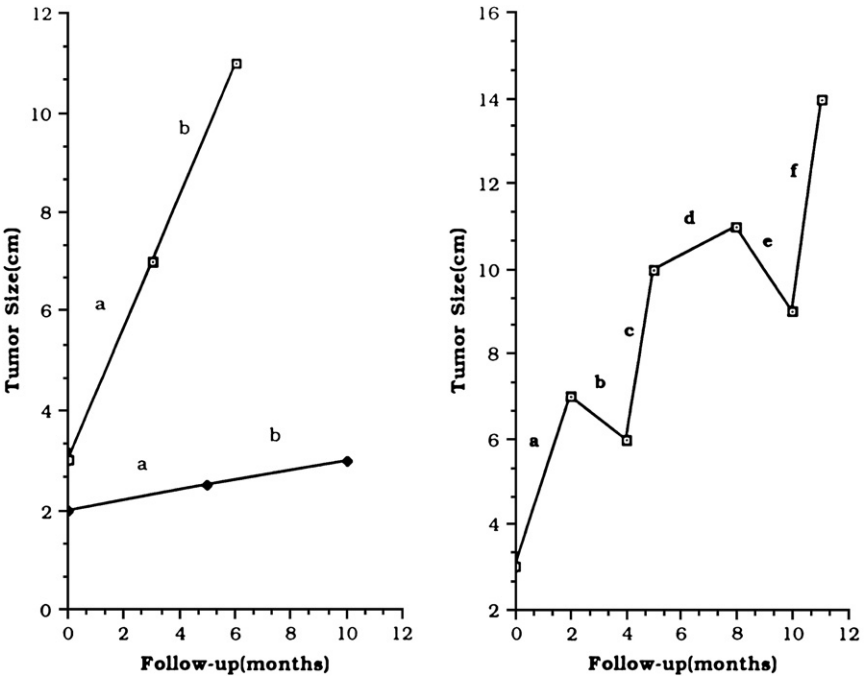

Fig. 2. Examples of growth rate patterns illustrating significant differences in the variability of growth. (*Left*) Constant rate of growth in a fast and slow (*bottom line*) growing tumor; (*Right*) a highly variable rate of growth in a single tumor.

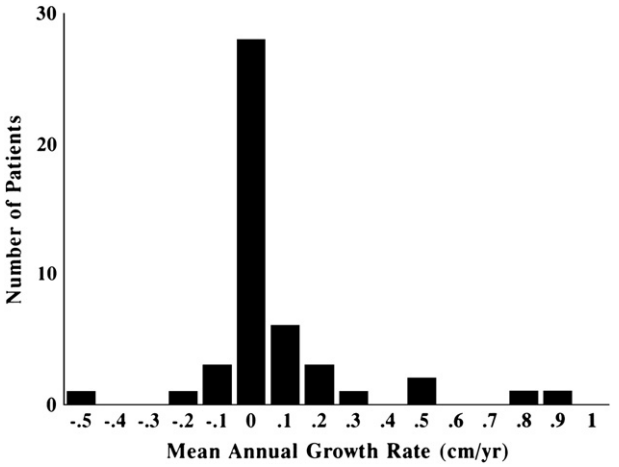

Fig. 3. Distribution of annual growth rates of study group (N = 50). Note that 0 growth includes rate changes between −0.099 and +0.099 cm/y. (See Table 2 for summary.)

of tumors demonstrate little or no variation in their growth rate, that is to say, a constant pattern of growth occurs regardless of the absolute rate.

Other factors affecting growth

Multiple regression analysis showed that there was no relationship between either the age of the patient or the initial tumor size with the annual growth rate. Of interest, a significant correlation was noted between patient age and tumor size. That is, the older the patient, the larger the tumor at presentation.

Outcome of follow-up

Patients with tumor growth less than 0.2 cm per year (n = 39)

Table 3 lists the outcome of the 39 patients in whom tumor growth has been less than 0.2 cm per year.

No surgical intervention (n = 37). Of the 39 patients, 37 have not required any form of operative intervention. The mean follow-up for this group is 48.2 months. Thirty-four are known to be alive and well, of whom 29 remain currently under review. The other five have been contacted by phone but decline to attend further clinical and CT evaluation. Apart from a progression of their unilateral hearing loss, all patients remain asymptomatic.

One patient has died during the follow-up period. Death was from causes unrelated to the acoustic tumor.

Surgical intervention (n = 2). Two patients have had a ventriculoperitoneal shunt inserted because of the development of associated significant hydrocephalus (mean tumor size 2.6 cm). In one patient, this was performed shortly after presentation. The shunt required revision 6 years later because of recurrent ataxia associated with increasing hydrocephalus. Clinical symptoms with associated hydrocephalus necessitated a shunt in the second patient 2 years after diagnosis, in spite of no obvious tumor enlargement.

Table 2
Annual growth rate of tumors (n = 50)

Measurable reduction with ≥ 0.1 cm net change	
≤ 0.1 cm/y	4 (8%)
> 0.1 cm/y	5 (10%)
No growth	17 (34%)
Growth with ≥ 0.1 cm net change	
≤ 0.1 cm/y	7 (14%)
> 0.1 cm/y	6 (12%)
≥ 0.2 cm/y	11 (22%)

Patients with growth rate greater than 0.2 cm per year (n = 11)

The outcome of patients with tumor growth exceeding 0.2 cm per year is listed in Table 4.

No surgical intervention (n = 2). Despite a documented tumor growth of more than 0.2 cm per year, no surgery has been carried out to date on two patients. One has refused surgery (age 68, tumor presently 2.7 cm, growth rate 0.26 cm per year); the other underwent stereotactic irradiation.

Surgical intervention (n = 9). Nine patients have undergone planned translabyrinthine removal of

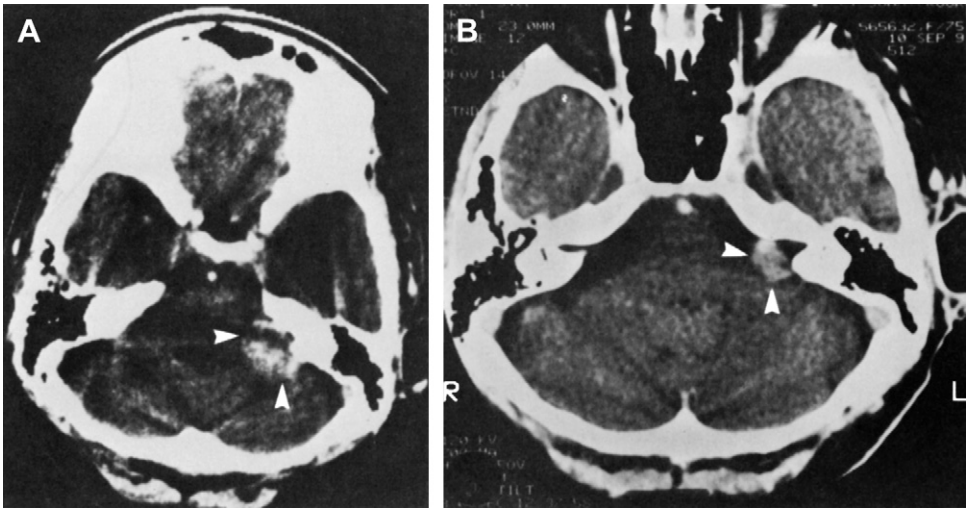

Fig. 4. Enhanced axial computed tomography images. Tumor demonstrating reduction in size over 7 years of follow-up. (*A*) Tumor measures 2.2 cm in May, 1984; (*B*) tumor measures 1.05 cm in September, 1991.

their tumors because of significant growth documented by repeat CT imaging. Complete removal was undertaken in seven, whereas deliberate subtotal excision was carried out in the other two (tumors measuring 3.63 and 2.6 cm). Five of the 11 patients with growth rates exceeding 0.2 cm per year developed either symptoms or signs referrable to their tumors. Three experienced increasing ataxia, one in combination with bidirectional gaze paretic nystagmus, whereas the other two developed varying degrees of trigeminal hypesthesia. Based on the onset of advancing symptoms, one would have expected these five patients to demonstrate enlargement of their tumors radiologically. Interestingly tumor enlargement was noted only in three individuals. Symptoms were in these cases due to developing hydrocephalus.

The mean tumor size in patients experiencing an increase in neurotologic signs was 2.64 cm, compared with 1.77 cm for those who did not.

Long term follow-up

A group of 28 elderly individuals have now been followed with good quality CT imaging for longer than 3 years. Ten of these patients have been followed for longer than 5 years. The mean

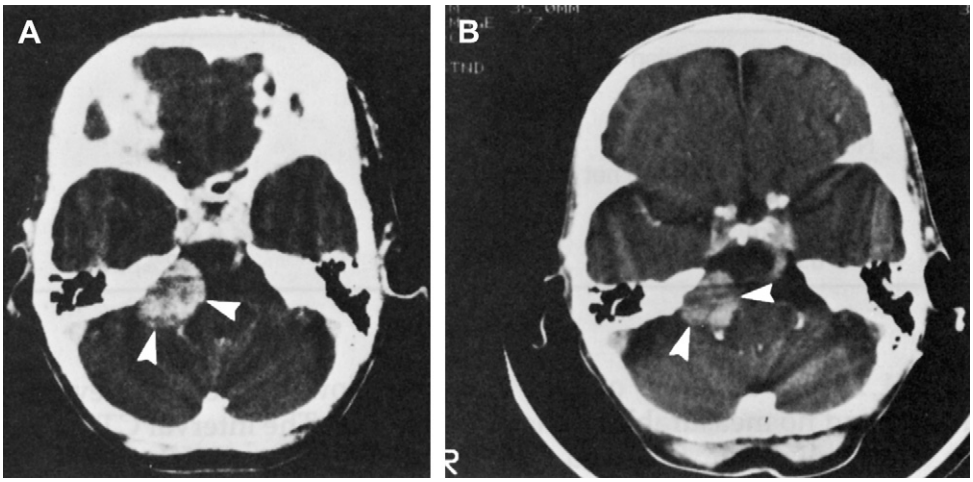

Fig. 5. Enhanced axial computed tomography images. Tumor demonstrating no change in size over a 13-year follow-up. (*A*) Scan from October, 1978; (*B*) scan from May 1991.

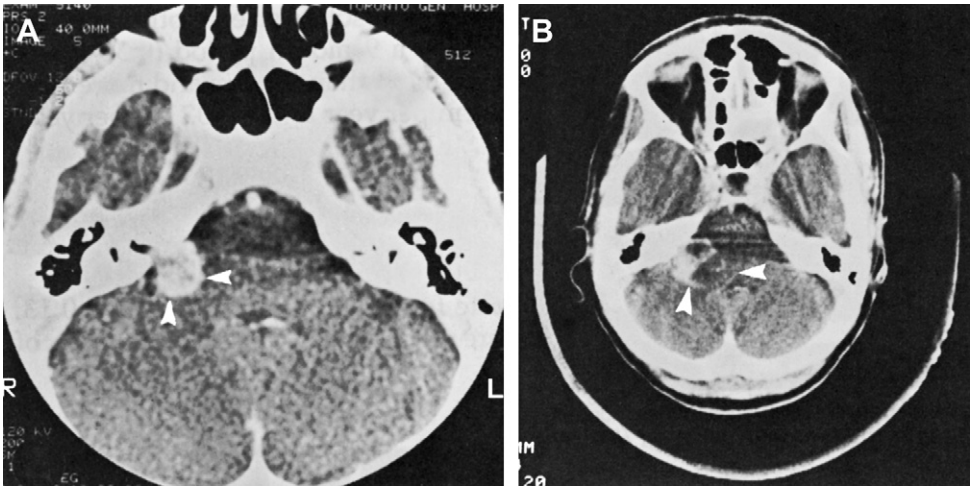

Fig. 6. Enhanced axial computed tomography images. Tumor demonstrating rapid growth rate of 0.98 cm/yr, (1.15 cm increase in 14 months). (*A*) 1.45 cm in November, 1985; (*B*) 2.6 cm in January, 1987.

growth rates of +0.04 and –0.02 cm per year, respectively, reflects little growth occurring in the tumors of these particular patients. No tumor followed for longer than 3 years and with an established pattern of slow growth has ever in our experience begun to grow rapidly and assume a more aggressive behavior, necessitating a change from a policy of observation to that of surgical intervention.

Discussion

In light of these findings, is an expectant attitude (ie, non–tumor removal) reasonable in the management of the selected individual with an acoustic neuroma? Clearly a non interventional management policy is dependent on (1) patient compliance; (2) a predictable growth pattern; (3) a non invasive, easily obtainable method of assessing tumor size; and (4) reassurance that overall surgical treatment outcome has not been compromised, should it become necessary.

The results of this study confirm the results of others, in that the majority of acoustic neuromas grow slowly. The mean growth rate of tumors in this population (0.11 cm per year) is not significantly different from that of others. Our current findings, however, as well as those of others confirm a wide variation in individual tumor growth. We emphasize that the use of a single

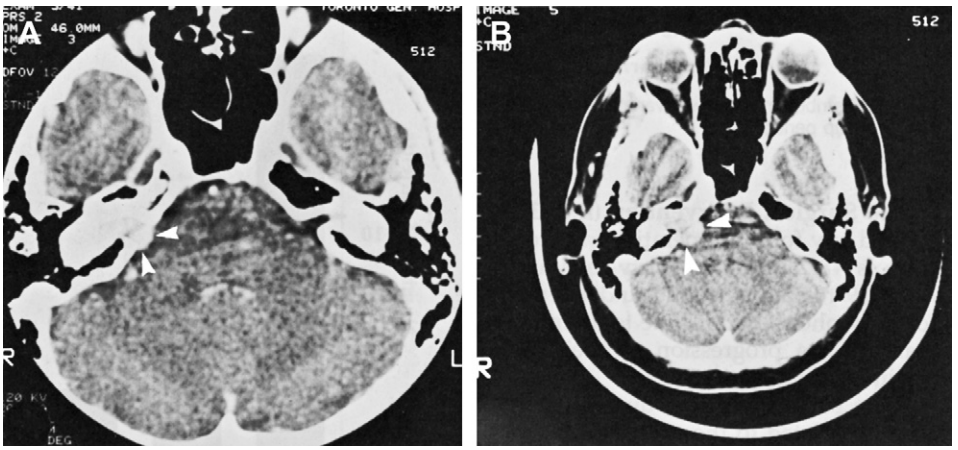

Fig. 7. Enhanced axial computed tomography images. Rapidly growing tumor; note threefold increase in size in 9 months. (*A*) 0.38 cm in August 1985; (*B*) 1 cm in May 1986.

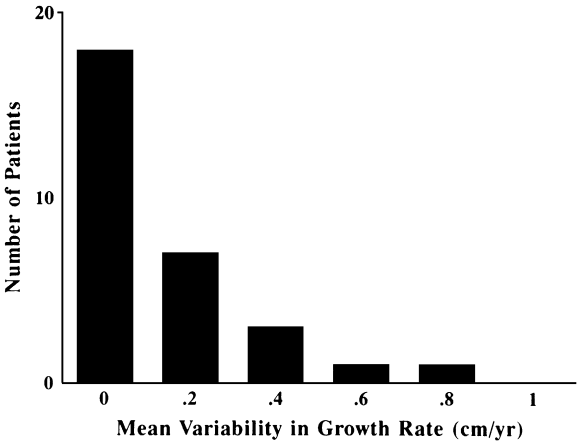

Fig. 8. Distribution of growth variability (n = 30). Lower values = constant rate of growth over follow-up period.

figure to estimate future tumor size is inappropriate. Our data indicate that a growth rate of 0.2 cm or greater was realized in only 22% of our patients. Excluding patients with growth rates greater than 0.2 cm per year, the mean annual growth rate for tumors in those individuals with measurable enlargement is 0.07 cm per year. Projected over 20 years, a time frame that exceeds the mean expected survival of 16 years in our elderly study population, this would equal a further growth of 1.4 cm [21].

In patients who have been followed for more than 3 years and more than 5 years, growth rates in our current study are +0.04 cm per year and −0.02 cm per year, respectively. This suggests that even allowing for an extended life span, few patients with small tumors would require surgery.

Results of analysis of growth variability indicate that the rate of growth of the individual tumor is relatively constant. The rate of growth at the start of follow-up predicted the future growth rate. No significant tumor enlargement occurred once a pattern of slow or absent growth had been established during the period of 3 or more years. This is corroborated by other authors. Consequently it seems reasonable to suggest that tumor behavior will become apparent within a relatively short period of observation subsequent to diagnosis, often within the first 18 months. Two of our patients underwent tumor excision after a longer interval. This was the result of their initial deference of surgery, however.

Aside from tumor growth rate, no other factor, including the size of the tumor at presentation, was found to be useful in predicting tumor growth. In isolated instances, large tumors were observed to become smaller, whereas small tumors were observed to grow rapidly. Explanations for such growth in addition to cell multiplication include hemorrhage into the tumor as well as cystic degeneration. The latter finding is by no means a feature peculiar to large or fast-growing tumors and is found in small tumors as well as those that have demonstrated little tendency to grow.

Factors that should be considered before adopting a nontreatment strategy

Age

Advanced age, in itself, is not a contraindication to successful surgical removal of acoustic neuromas. A small tumor in an otherwise fit and

Table 3
Patients with growth rates < 0.2 cm/y (n = 39)

Mean rate of growth—<0.01 cm/y (no growth)	
Mean age—69.7 y	
Mean follow-up—46.7 mo	
Disposition	
Alive and well	34
Decreased (unrelated cause)	1
Lost to follow-up	2
Surgery (ventriculoperitoneal shunt)	2

Table 4
patients with growth rates >0.2 cm/y n = 11)

Mean rate of growth—0.52 cm/y (range 0.26–0.9)	
Mean age—68.8 y	
Mean follow-up—14 mo	
Disposition	
Tumor excision	9
Refused surgery	1
Gamma knife	1

active elderly individual is as easily removed as one in a young patient, with the subsequent recovery profile frequently indistinguishable from that of a young patient. In those individuals who are elderly and are reluctant to proceed to tumor removal, however, a reasonable alternative is to adopt an expectant attitude with the understanding that close, ongoing monitoring is initially required.

To date, no similar study of the natural history of acoustic neuromas in younger individuals has been published. Consequently young patients who opt for a nontreatment course should be advised of the need for even closer scrutiny with the understanding that surgery will be recommended in the event of demonstrated growth.

Tumor size at presentation

To date, tumor size at the time of diagnosis and subsequent rate of growth are statistically unrelated. In those instances, however, in which tumor size is responsible for ataxia, obstructive hydrocephalus, and significant trigeminal hypesthesia, it would seem prudent to recommend excision.

Special extenuating circumstances

In those individuals with an acoustic neuroma affecting the only-hearing ear or a tumor located on the side of the only-seeing eye, concern with respect to deafness and facial weakness is advised. Such individuals at outset are candidates for an initial conservative course of management.

General health

Individuals who are in generally poor health and in whom tumor surgery poses a much greater risk than normal, should be considered for noninterventional management.

Incidental tumors

The cited incidence of acoustic neuroma in the general population, as determined by cadaveric temporal bone studies, is estimated to be approximately 1% [22,23]. A generally accepted study cites the incidence of clinically apparent acoustic neuromas as eight tumors per million population [24]. Given recent advances in contrast-enhanced MRI, it seems reasonable to assume that heretofore unsuspected tumors will be identified. Removal of such clinically silent tumors without hearing loss is highly unlikely. In such an instance, documented growth or onset of hearing loss before advocating removal would seem advisable.

Summary

The results of this study and others document the biologic behavior of acoustic neuromas. In view of the evidence presented, which describes both variable rates of individual tumor growth and spontaneous regression in size, it would seem prudent that before selecting a nonsurgical treatment modality, the growth rate for the particular tumor in question should be established. To date, none of the literature that addresses the use of focused irradiation has attempted to do so.

Our study as well as those of others suggests that the growth rate of acoustic neuromas becomes predictable over time. Based on this observation, a conservative (nontumor excision) management strategy is proposed for selected individuals.

Patients to whom this management philosophy has been recommended or who themselves have chosen this option are seen twice yearly. Each visit consists of a thorough neurotologic examination as well as high-definition CT or MRI. Careful comparison of the clinical course as well as calculation of the tumor size is carried out in each instance. If the clinical course and rate of tumor growth remain unchanged over a 3-year follow-up, annual assessments are recommended. In the event of tumor enlargement, surgery may or may not be recommended, depending on the rate of growth and the age of the patient. Our experience suggests that a rate of growth equal to or exceeding 0.2 cm per year constitutes an indication for tumor removal.

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Selection of Surgical Approach to Acoustic Neuroma

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Centers with special expertise in the management of acoustic neuromas fall into three broad categories in terms of their preference for operative approaches: (1) suboccipital preferred, (2) translabyrinthine preferred, and (3) eclectic. The number of teams in the last group has steadily grown in recent years as cooperating neurotologists and neurosurgeons have become aware of the relative advantages and disadvantages of the various techniques [1–4]. At the University of California, San Francisco (UCSF), we use a mixture of the suboccipital, translabyrinthine, and middle fossa approaches according to the characteristics of the particular tumor undergoing treatment. In this article, we review the anatomic and clinical factors affecting the choice of operative approach, examine the differences in postoperative outcome among the surgical techniques, and present a protocol for the selection of approach based on tumor size and clinical manifestations.

Factors affecting the choice of surgical technique

Tumor size

For almost all acoustic neuromas removed at UCSF, we select either the translabyrinthine or suboccipital approach (Fig. 1). Tumor size, in and of itself, is not a criterion in choosing between the translabyrinthine and suboccipital techniques when removing an acoustic neuroma. Either approach provides a sufficient exposure of the cerebellopontine angle and brain stem to permit atraumatic and complete tumor removal of even very large acoustic neuromas. A few authors, who limit their practice to the suboccipital approach, have maintained that the translabyrinthine approach affords insufficient exposure for larger tumors. This point of view was well articulated by DiTullio, Malkasian, and Rand, who maintained that "because of the limited operative field, this approach precludes adequate visualization not only of the medial aspect of the tumor but also its anatomical relationship to the vital brain stem and vascular structures" [5]. In contrast to the opinion expressed by these authors, we have found that the exposure of the brain stem surface facing an acoustic neuroma provided by the two approaches is essentially identical. Much of the criticism of the exposure provided by the translabyrinthine approach has undoubtedly arisen when neurosurgeons collaborated with inexperienced temporal bone surgeons who provided an insufficient transtemporal exposure of the posterior fossa. It should be emphasized that adequate exposure of the cerebellopontine angle for large tumors by the translabyrinthine approach requires that the surgeon perform a wide retrosigmoid decompression of the posterior fossa dura, remove bone from the jugular bulb and horizontal course of the sigmoid sinus, and excavate well anterior to the porus acusticus. We also disagree that there are neurotologic and neurosurgical approaches to acoustic neuroma. Both the translabyrinthine and suboccipital approaches are posterior fossa craniotomies that differ primarily in the way the head is opened. The essential issue at hand, the removal of the intracranial tumor, properly resides within the armamentarium of both specialties, depending on the surgeon's training, experience, and microsurgical skills.

On first reflection, it may seem that the angle of view of the internal auditory canal,

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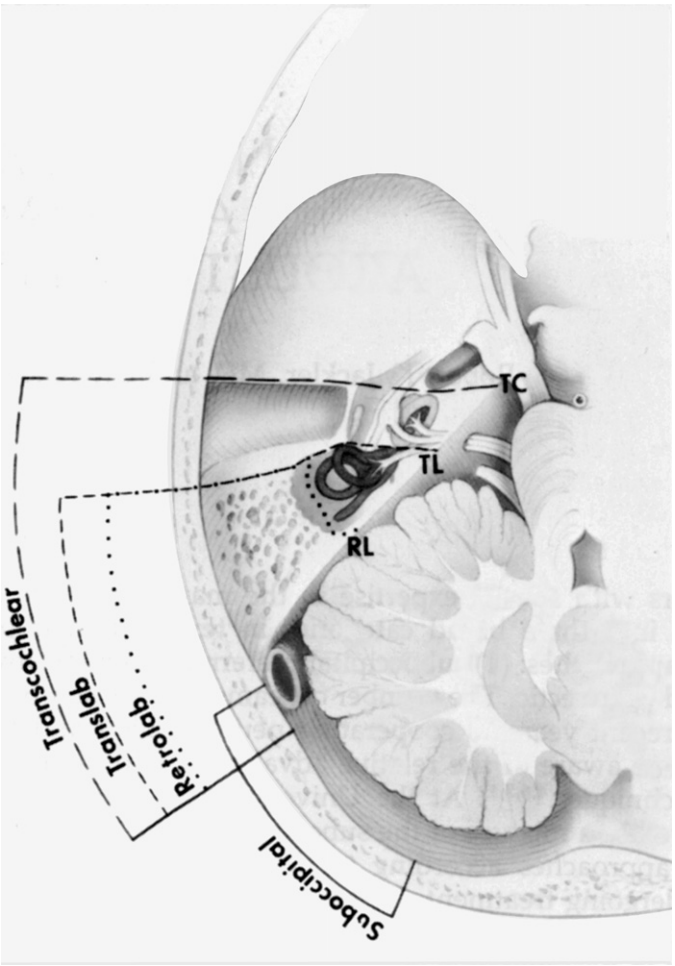

Fig. 1. Schematic view of suboccipital, translabyrinthine, retrolabyrinthine, and transcochlear approaches to the cerebellopontine angle as visualized in the axial plane.

cerebellopontine angle, and brain stem provided by the translabyrinthine and suboccipital approaches are quite different. After all, the areas of skull removed are quite separate, having only 1 to 2 cm of overlap behind the sigmoid sinus. Nevertheless, the exposure afforded by the two techniques is remarkably similar. The explanation for this lies in the fact that the angle of view employed is actually almost identical. In the translabyrinthine approach, the surgeon retrodisplaces the sigmoid sinus and looks along this posterior aspect of the craniotomy opening (Figs. 2 and 3). In the suboccipital approach, the surgeon removes bone up to the sigmoid sinus and then views along the most anterior edge of the craniotomy (Figs. 4, 5, and 6). In the average exposure, the angle of view of a translabyrinthine craniotomy is slightly more lateral and that of the Suboccipital more posterior, but this difference is usually less than a 10 degree angle.

Although the translabyrinthine approach provides excellent visualization of the internal auditory canal and cerebellopontine angle, it does not create as panoramic a view of the posterior fossa as the suboccipital approach. The translabyrinthine exposure, especially when the jugular bulb is high, is limited inferiorly. This may restrict access to the inferior-most portion of the cerebellopontine angle, the neural compartment of the jugular foramen, and to the foramen magnum region. This limitation is seldom problematic during surgery on acoustic neuromas because these

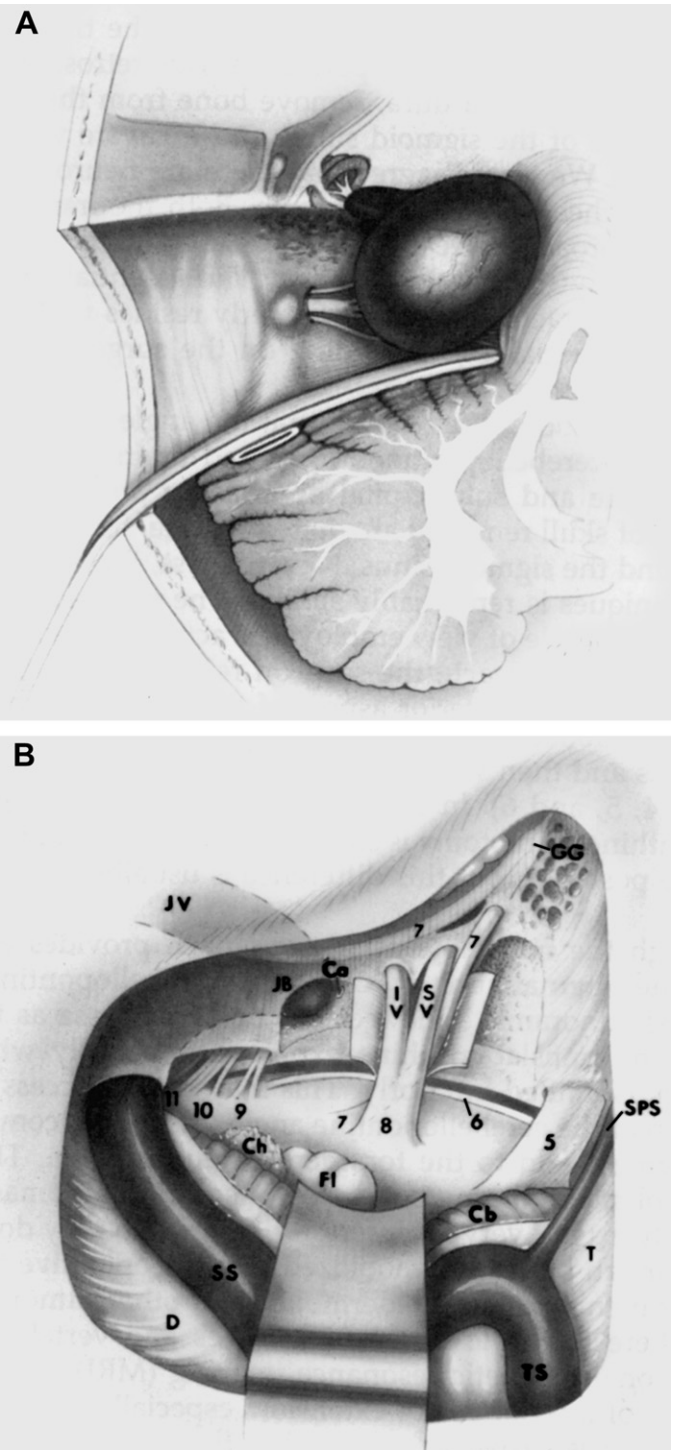

Fig. 2. Translabyrinthine approach viewed schematically in axial section (*A*) and from the surgeon's viewpoint (*B*) demonstrating key points of anatomy. JV, jugular veinz; JB, jugular bulb; SS, sigmoid sinus; TS, transverse sinus; SPS, superior petrosal sinus; Cb, cerebellum; D, retrosigmoid dura; Ca, cochlear aqueduct; IV, inferior vestibular nerve; SV, superior vestibular nerve; 5, trigeminal nerve; 6, abducens nerve; 7, facial nerve; 8, audiovestibular nerve; 9, glossopharyngeal nerve; 10, vagus nerve; 11, accessory nerve; GG, geniculate ganglion; Ch, choroid, Fl, flocculus.

Fig. 3. Translabyrinthine approach to a medium-sized acoustic neuroma. Inspection of the lateral end of the internal auditory canal reveals that the tumor originated from the superior vestibular nerve. Note deformation of the facial nerve over the ventral surface of the tumor.

tumors rarely extend very far inferiorly, and when they do, the lower pole of the tumor can be readily mobilized into the operative field. This is not the case for meningiomas, epidermoids, and other tumors, however, that tend to adhere to the lower cranial nerves and vertebrobasilar system. When a coronal magnetic resonance imaging (MRI) scan reveals an unusual degree of inferior tumor extension, especially when a non–acoustic neuroma tumor is suspected, we choose the suboccipital approach.

The middle fossa approach is generally considered to be suitable only for wholly intracanalicular lesions (Figs. 7 and 8). A few authors have advocated an extended middle fossa approach in the management of tumors with large cerebellopontine angle components [7–8]. They point out that through extensive removal of the temporal floor, accompanied by division of the superior petrosal sinus and a portion of the tentorium, a limited exposure to the cerebellopontine angle may be obtained via the middle fossa approach. In our opinion, this variation affords insufficient exposure of the inferior aspect of the cerebellopontine angle to assure control of vessels arising beneath the tumor. Also, the extended middle fossa approach to the cerebellopontine angle requires rather vigorous and prolonged retraction of the temporal lobe, a maneuver less forgiving than comparable displacement of the cerebellum. For these reasons, the extended middle fossa technique has not gained widespread acceptance.

Depth of internal auditory canal penetration

Each of the three major approaches to acoustic neuroma is capable of completely exposing the contents of the internal auditory canal for removal of the intracanalicular portion of the tumor. Only the middle fossa technique permits complete canal opening without violation of the inner ear. In the translabyrinthine approach, the

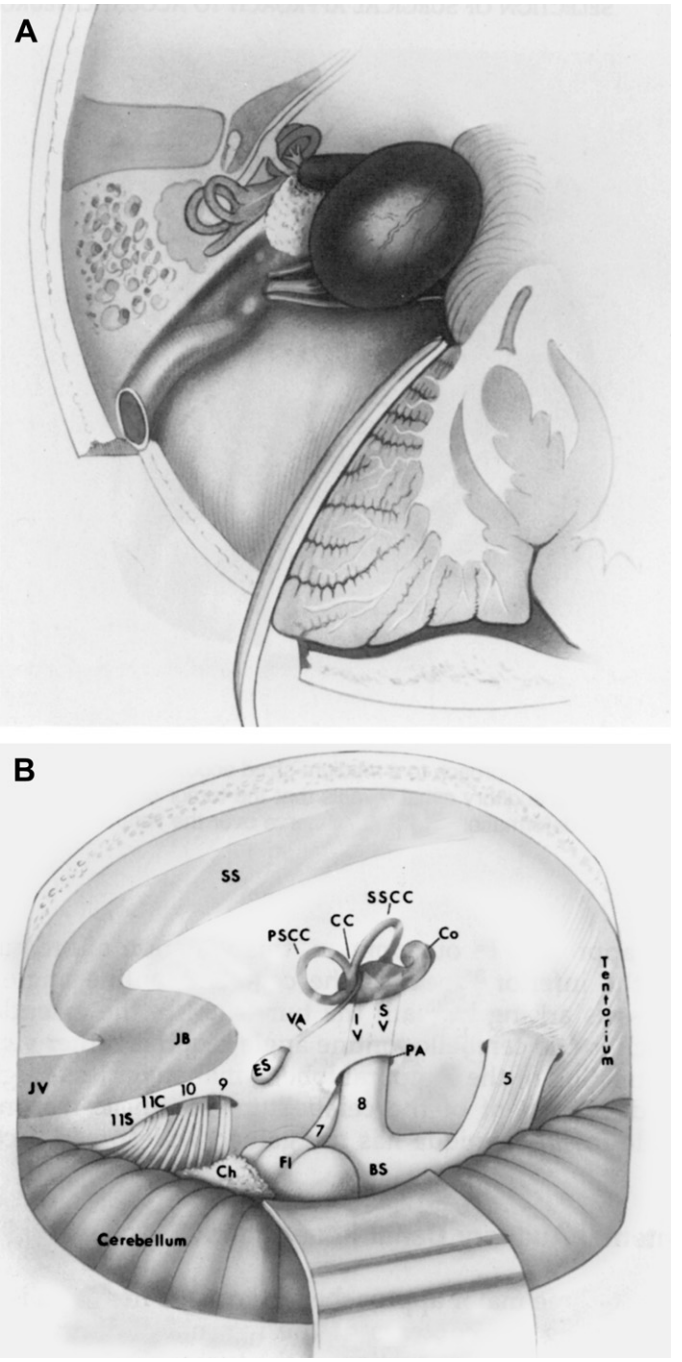

Fig. 4. Suboccipital approach viewed schematically in axial section (*A*) and from the surgeon's viewpoint demonstrating key points of anatomy (*B*). Note that the inner ear overlaps the lateral one third of the distal internal auditory canal. JV, jugular vein; JB, jugular bulb; SS, sigmoid sinus; 11S, spinal division of the accessory nerve; 11C, cranial division of the accessory nerve; 10, vagus nerve; 9, glossopharyngeal nerve; ES, endolymphatic sac; VA, vestibular aqueduct; PSCC, posterior semicircular canal; CC, common crus; SSCC, superior semicircular canal; Co, cochlea; IV, inferior vestibular nerve; SV, superior vestibular nerve; PA, poms acusticus; 7, facial nerve; 8, audiovestibular nerve; Ch, choroid; Fl, flocculus; BS, brain stem; 5, trigeminal nerve.

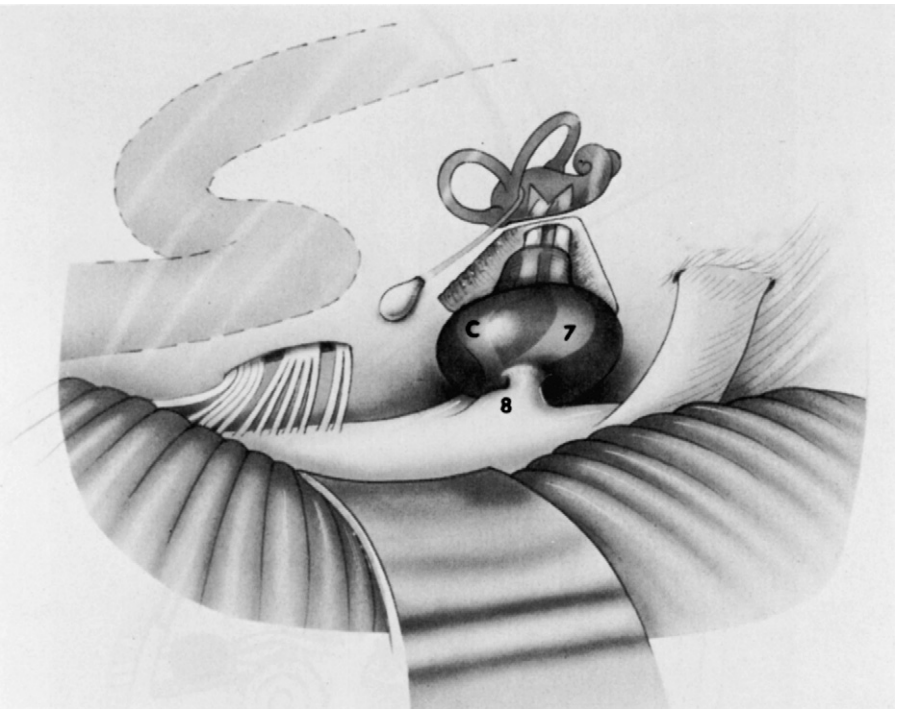

Fig. 5. Suboccipital approach to a small acoustic neuroma that does not deeply penetrate the internal auditory canal. It is usually possible to expose the tumor without entering the inner ear when the tumor is confined to the medial two thirds of the canal. Note that the surgeon has used the endolymphatic sac and aqueduct as guides to avoid entry into the common crus and vestibule. C, cochlear nerve; 7, facial nerve; 8, audiovestibular nerve.

internal auditory canal contents are exposed through exenteration of the semicircular canals.

In the suboccipital approach, only the proximal two thirds of the internal auditory canal can be exposed without traversing inner ear structures (see Fig. 4B). Optimally the internal auditory canal should be opened only as far laterally as required to visualize the deepest penetration of the tumor. Creating unneeded additional exposure may unnecessarily open petrous apex air cell tracts and thereby increase the risk of postoperative cerebrospinal fluid otorhinorrhea. Also, excessive exposure of the lateral end of the internal auditory canal may injure the inner ear and jeopardize efforts at sparing hearing. During an attempt to preserve hearing, the endolymphatic sac and vestibular aqueduct are useful landmarks to avoid violating the inner ear. After identifying the sac operculum, the aqueduct may be traced lateral to the common crus and is useful in demarcating the posterior and lateral extent of bone removal [9]. When using the suboccipital approach to tumors that deeply penetrate the internal auditory canal, complete exposure to the fundus can be achieved by removing portions of the vestibule, posterior semicircular canal, and common crus (see Fig. 6). To avoid entering the inner ear during a suboccipital approach to a tumor that deeply penetrates the internal auditory canal, some surgeons address the most lateral intracanalicular portion indirectly, through use of angulated instruments and a mirror. We do not favor this maneuver because of the considerable risk of leaving residual tumor at the terminus of the internal auditory canal. The laterally placed junction between tumor and the vestibular nerve of origin often tapers to become quite thin and fragile. During blind dissection, this tongue of tumor is easily torn from the main intracanalicular portion, giving the false impression of complete removal even though a nubbin of residual tumor remains in the fundus. These tumor cells, unlike those in a small bit of tumor capsule left on an attenuated facial nerve in the mid–cerebellopontine angle, are well vascularized and have considerable potential for generating a recurrence.

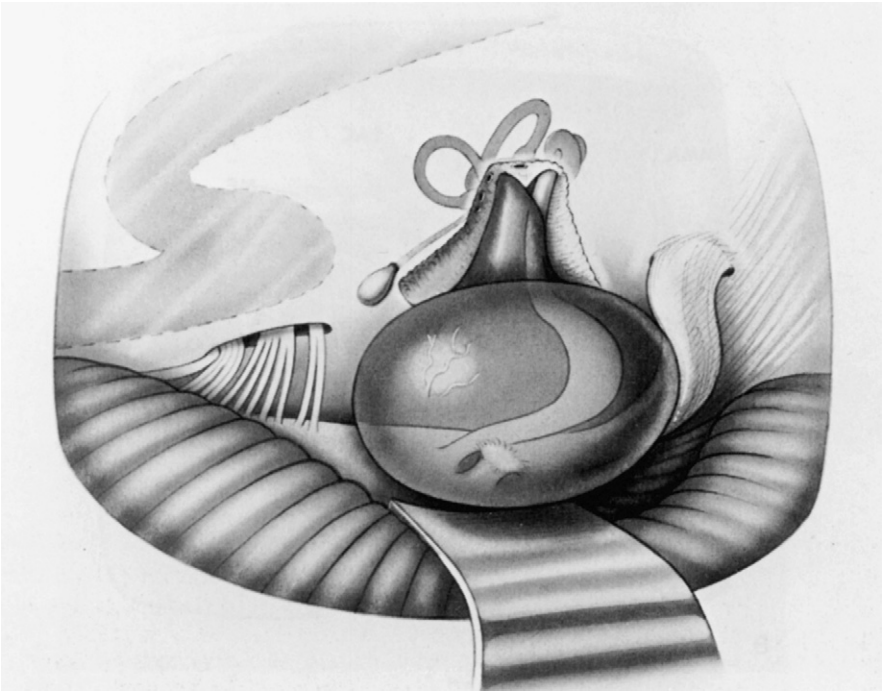

Fig. 6. Suboccipital approach to a large acoustic neuroma that deeply penetrates the internal auditory canal. In tumors that extend into the distal one third of the internal auditory canal, portions of the inner ear must be removed in order to expose the lateral extremity of the tumor. This eliminates any possibility of preserving residual hearing.

An accurate preoperative assessment of the depth of internal auditory canal penetration is important in selecting the optimal approach to an acoustic neuroma. We have found T_2-weighted MRI scans to be most helpful in making this determination. Although gadolinium-enhanced, T_1-weighted images most clearly delineate the tumor, T_2 images best elucidate the structures of the inner ear. The surgeon is less concerned with the absolute depth of the tumor in the internal auditory canal than the relationship between the tumor's deepest penetration to the posterior semicircular canal and vestibule. By drawing a line from a point on the suboccipital convexity approximately 2 cm behind the posterior edge of the sigmoid sinus to the lateral terminus of the tumor, the surgeon can predict whether a portion of the inner ear must be removed to visualize directly the lateral-most extension of the tumor via the suboccipital approach.

Hearing status

Hearing conservation is possible, in certain cases, using either the middle fossa or the suboccipital approach. With intracanalicular tumors, either technique may be employed, whereas with larger tumors, only the suboccipital approach is appropriate. The criteria we use for choosing among operative approaches to intracanalicular tumors is described in detail later. Selecting candidates for a hearing conservation approach involves a number of factors, including pure tone threshold, speech discrimination score, auditory evoked responses, tumor size, depth of tumor penetration in the internal auditory canal, status of the contralateral ear, and patient age. Hearing conservation would be attempted in every patient with significant residual hearing if there were not occasional adverse consequences from undertaking the effort. In our hands, the suboccipital approach has a slightly higher morbidity than the translabyrinthine approach, particularly with regard to cerebrospinal fluid leakage and persistent postoperative headache. As is discussed later, our recommendation to the patient depends on the probability of maintaining useful levels of hearing balanced against the somewhat increased likelihood of these bothersome (but seldom serious) complications.

Removal of an acoustic neuroma from an only hearing (or better hearing) ear represents a therapeutic dilemma. We avoid this unless the

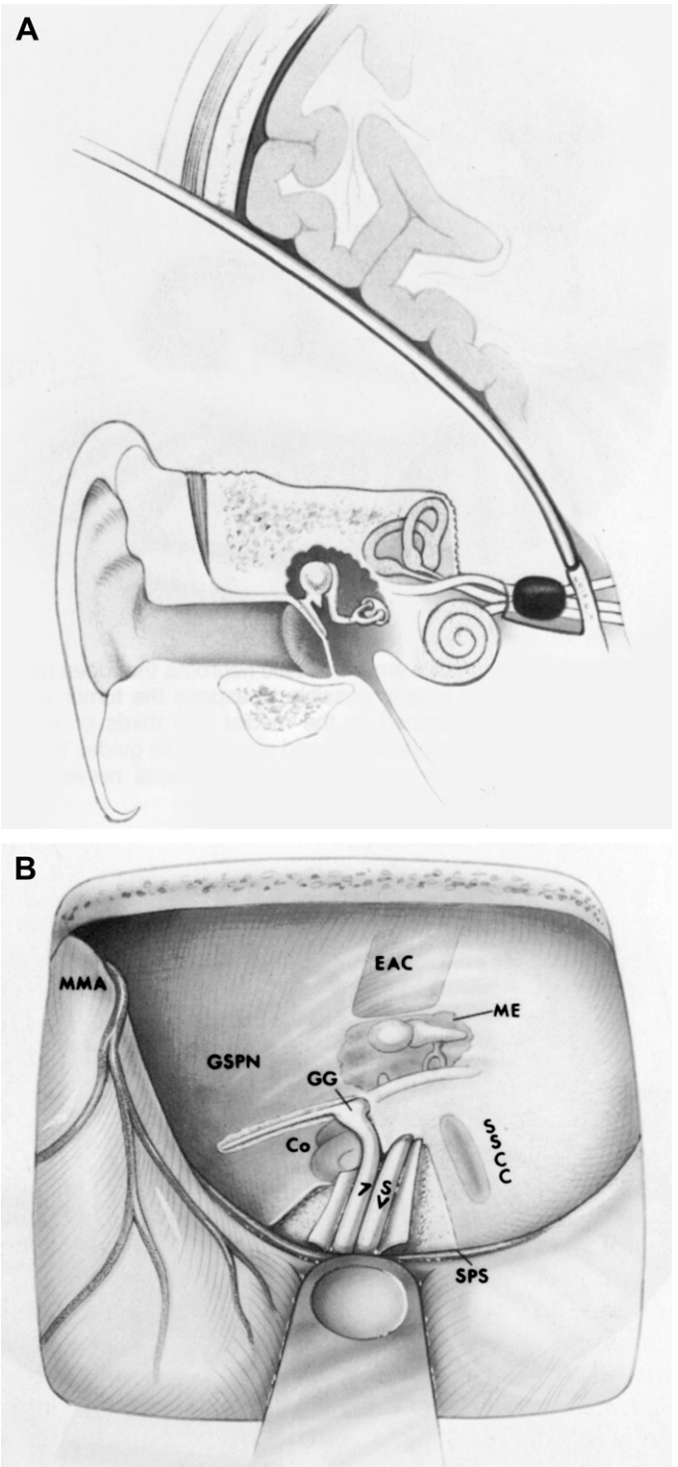

Fig. 7. Middle fossa approach viewed schematically in coronal section (*A*) and from the surgeon's viewpoint, demonstrating key points of anatomy (*B*). MMA, middle meningeal artery; EAC, external auditory canal; ME, middle ear; GSPN, greater superficial petrosal nerve; GG, geniculate ganglion; Co, cochlea; 7, facial nerve; SV, superior vestibular nerve; SSCC, superior semicircular canal; SPS, superior petrosal sinus.

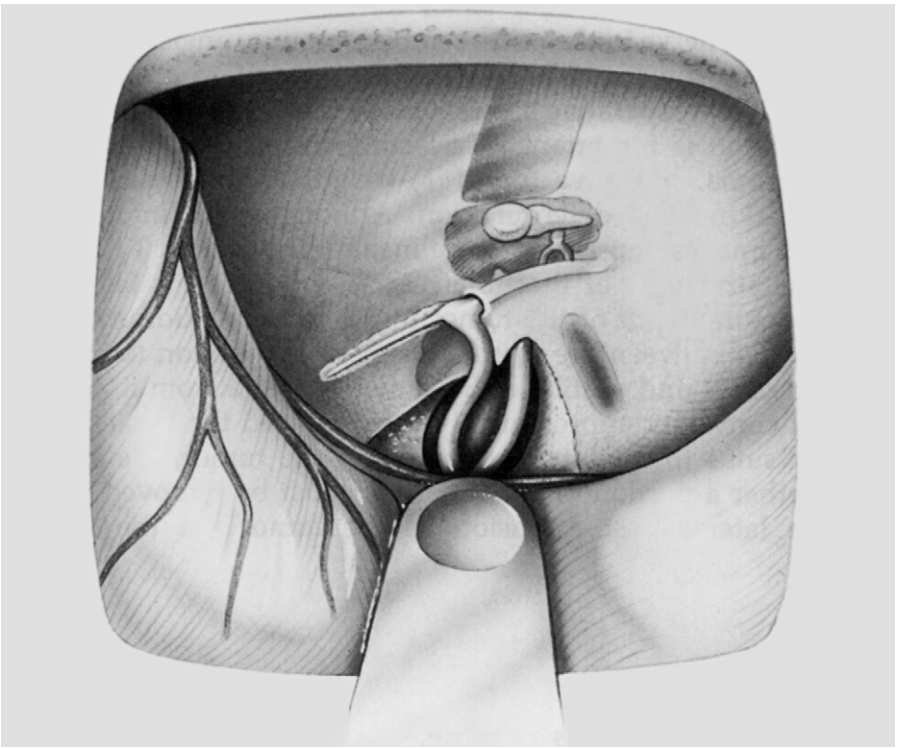

Fig. 8. Middle fossa approach to an intracanalicular tumor arising from the inferior vestibular nerve. Note that the facial nerve is draped on the superior surface of the tumor and must be manipulated during tumor resection. This increases the risk of postoperative neurapraxia.

tumor has been shown to grow rapidly or produce substantial brain stem compression. In such cases, we advocate debulking the cisternal component via the suboccipital route, taking great pains to avoid manipulating the cochlear nerve. During this procedure, the posterior margin of the porus acusticus is removed with a drill to help decompress the internal auditory canal. It is unwise to attempt removal of tumor from the internal auditory canal in large tumors because this usually results in loss of residual auditory function. Slitting the internal auditory canal dura, particularly the ring at the poms acusticus, may help to improve the decom-pressive effect further.

The translabyrinthine technique is the only approach to acoustic neuroma that inherently sacrifices hearing in the course of the procedure. Recently a modification of the translabyrinthine approach intended to afford a slight hope of maintaining hearing has been proposed [10]. The authors reported that atraumatic removal of the membranous labyrinth, sealing of the vestibule with bone wax, and replenishment of lost perilymph with lactated Ringer's solution maintained hearing in one case. Nevertheless, it is doubtful that this fortunate outcome will be realized with enough regularity to consider the translabyrinthine approach suitable for attempts to preserve hearing.

Exposure of the facial nerve

The challenge of preserving the facial nerve in acoustic neuroma surgery can be divided into three steps in ascending order of difficulty: (1) identification of the nerve in the distal internal auditory canal beyond the most lateral extension of the tumor, (2) identification of the nerve at its exit from the brain stem, and (3) separation of the nerve from its region of greatest splaying and adherence.

We have seldom found it difficult to identify a distal interface between the tumor and the facial nerve at the lateral end of the internal auditory canal, regardless of the surgical technique employed. With the translabyrinthine, suboccipital,

or middle fossa technique, it is possible to drill open the internal auditory canal sufficiently to identify the facial nerve in the first segment of the fallopian canal before it becomes involved with tumor. In both the middle fossa and translabyrinthine approaches, exposure of the far lateral segment of the internal auditory canal is an inherent part of the technique. In the suboccipital approach, exposure of the distal portion of the internal auditory canal is optional and is usually tailored to the depth of penetration of the tumor within the canal. Some surgeons who primarily employ the translabyrinthine approach have argued that a facial nerve plane in the distal internal auditory canal is more easily and atraumatically established using this technique [11]. Having performed a substantial number of each of these procedures, we believe that identification of the facial nerve plane distally is accomplished equally well via either the translabyrinthine or the suboccipital approach.

Within the internal auditory canal, the facial nerve is virtually always located on the anterior surface of the tumor. It may lie relatively anterosuperior or anteroinferior depending on whether the tumor arose from the superior or inferior division of the vestibular nerve. Both the suboccipital and the translabyrinthine approaches view the internal auditory canal contents from a posterior perspective, where the facial nerve lies on the deep surface of the tumor (see Figs. 3, 5, 6, 9, 10). This is favorable for removing the tumor from the nerve with minimal disturbance to it. With the middle fossa approach, however, the internal auditory canal is viewed from above. When the tumor arises from the inferior vestibular

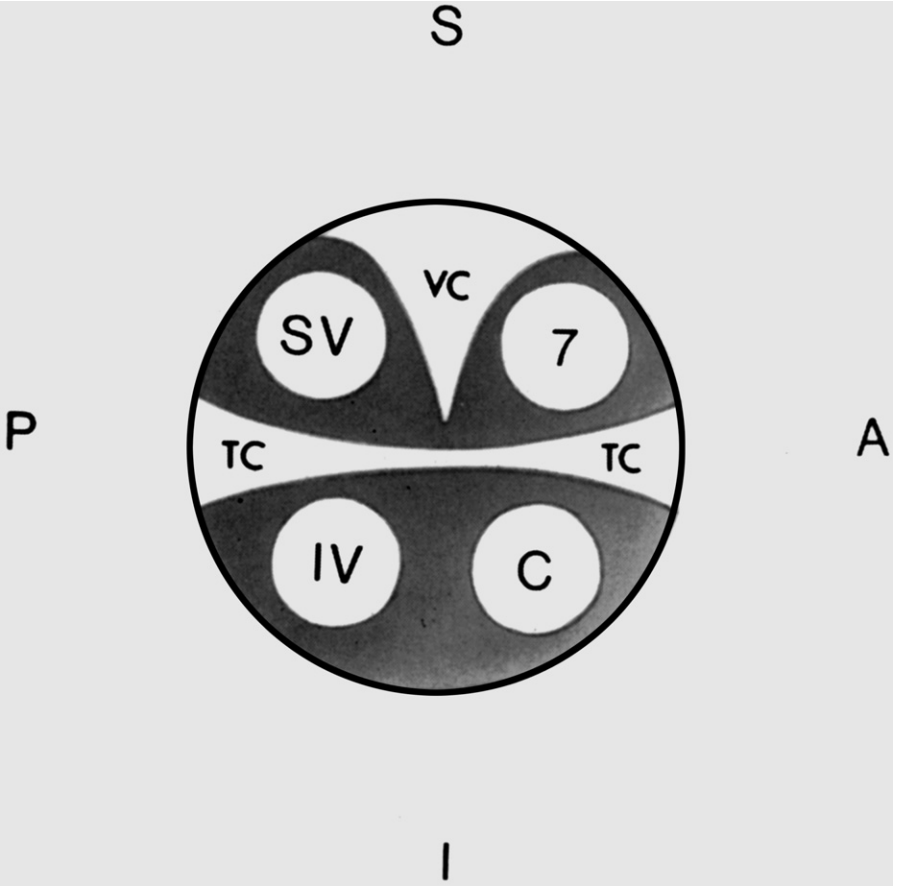

Fig. 9. Relationships at the lateral extremity of the internal auditory canal. The superior compartment is partitioned by the vertical crest (VC), also known as Bill's bar, which separates the superior vestibular nerve from the facial nerve. The canal is completely divided in the horizontal plane by the transverse crest (TC). SV, superior vestibular nerve; IV, inferior vestibular nerve; 7, facial nerve; C, cochlear nerve.

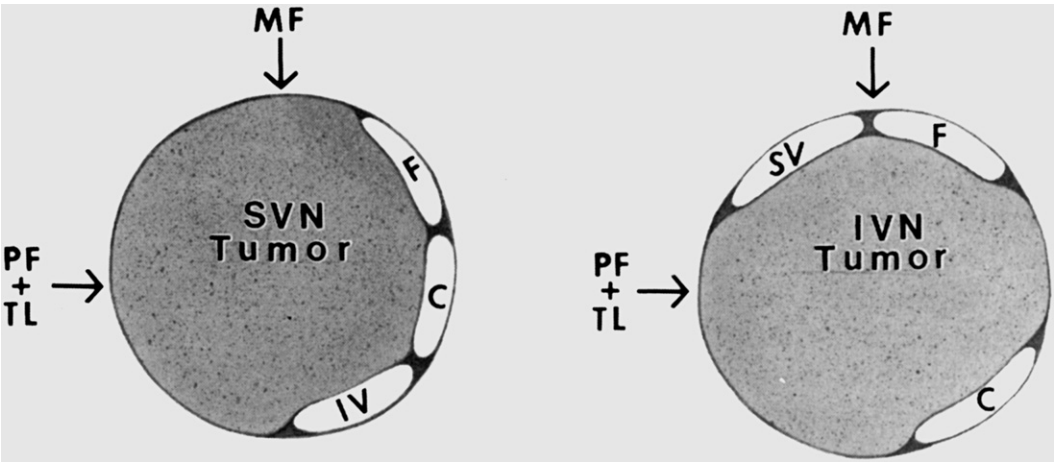

Fig. 10. Schematic representation of a superior vestibular nerve (SVN) tumor and an inferior vestibular nerve tumor (IVN) viewed in cross section through the mid–internal auditory canal (IAC). The surgeon's perspective of the contents of the IAC from the middle fossa (MF) approach is compared with that of the suboccipital (PF) and translabyrinthine (TL) approaches.

nerve, the facial nerve lies in the surgeon's view and must be manipulated from the tumor (see Figs. 8, 10). This increases the chances of transient postoperative facial weakness but is unlikely to result in lasting cosmetic deficit.

Identification of the facial nerve at its exit from the brain stem is straightforward in tumors with little or no brain stem interface. In large tumors, finding the facial nerve proximally may be quite challenging, although it was achieved in virtually every case in our series and is seldom the limiting factor in facial nerve preservation. Routine use of a flexible tip stimulating electrode for facial nerve identification has proved very helpful in locating the root entry zone when it is visually obscure. We have found no difference in exposure of the facial nerve at the brain stem between the translabyrinthine and suboccipital approaches.

The critical portion of the facial nerve dissection is at its region of greatest splaying and adherence, which is almost always located on the tumor surface in the cerebellopontine angle just medial to the porus acusticus. At this point, the nerve makes an acute angulation over the anterior surface of the tumor. In the vast majority of cases, dissection of the facial nerve from the tumor is done equally well with either the translabyrinthine or suboccipital approach. In a few instances, when the facial nerve takes an extreme anterior course, there is a slight advantage to the somewhat more posterior angle of view provided by the suboccipital approach. As a general rule, we prefer not lifting the facial nerve from its diverted course while dissecting the tumor capsule from it. Leaving the facial nerve in situ minimizes potentially injurious traction and torsion on delicate neural fibers. Although dissection of such an anteriorly angulated facial nerve can be accomplished by the translabyrinthine approach, it may require pulling the tumor capsule and attached nerve out of their native position into a more favorable line of sight.

In the few cases in which anatomic continuity of the facial nerve cannot be preserved, the translabyrinthine approach has certain advantages. When the facial nerve deficiency is short, it may be possible to reroute the facial nerve out of the mastoid and gain sufficient length to permit a primary anastomosis. This is desirable because the success of a nerve repair is, in part, dependent on the number of anastomoses. It may also be easier to accomplish an interposition graft via the translabyrinthine approach. Anastomoses of interposition grafts are better done to a nerve of normal diameter than they are to a nerve end that has been flattened out by tumor. With the suboccipital approach, it may be difficult to find and manipulate a normal segment of nerve lateral to the tumor, particularly when it has invaginated the internal auditory canal deeply.

Anatomic variations

An anteriorly placed sigmoid sinus reduces the size of the mastoid and makes the translabyrinthine approach technically more difficult (Fig. 11). In our experience, it has been possible to

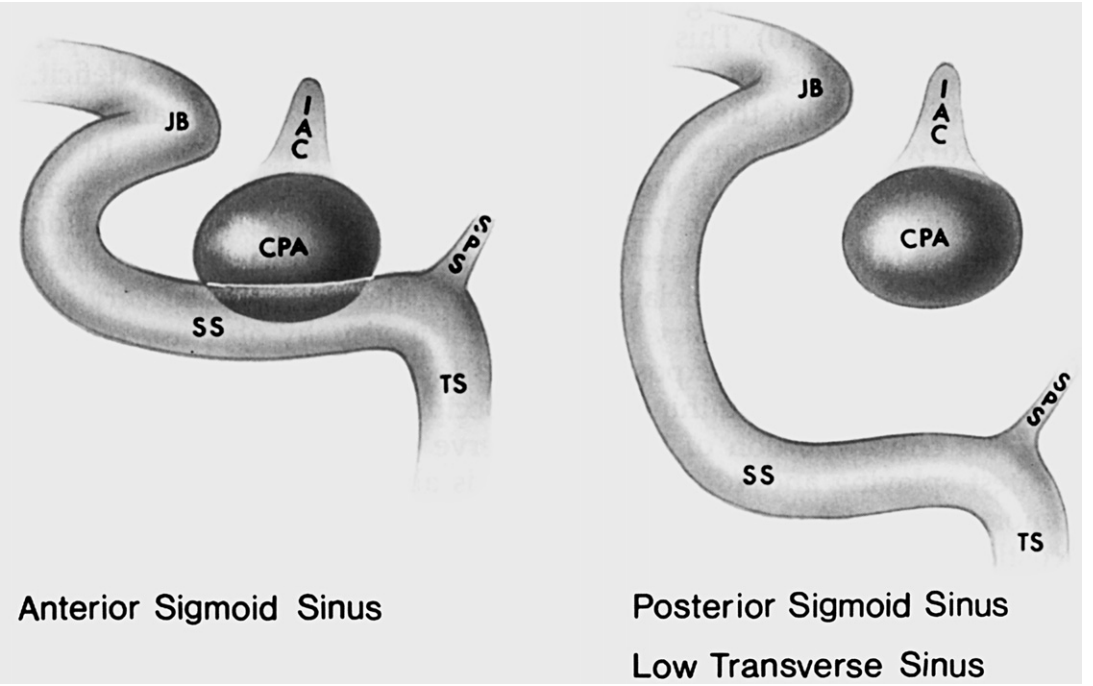

Fig. 11. Anatomic variations of the sigmoid and transverse sinus. When the sigmoid sinus is anteriorly located, a greater degree of retraction is required to obtain adequate exposure during translabyrinthine craniotomy. When the sigmoid sinus lies relatively posterior, especially when the transverse sinus is also low, exposure of the cerebellopontine angle (CPA) and internal auditory canal (IAC) through the suboccipital approach is more difficult.

compensate for this anatomic variation, in every instance that it has been encountered, by increasing the amount of retrosigmoid bone removal and retracting the sinus somewhat further posteriorly.

In the suboccipital approach, access to the cerebellopontine angle may be hindered somewhat when the sigmoid sinus takes an unusually posterior course (see Fig. 11). This mandates that the anterior edge of the craniectomy lie more posteriorly, thus necessitating a greater degree of cerebellar retraction. When a posteriorly placed sigmoid sinus is accompanied by a low-lying transverse sinus, the location of the craniotomy may force an awkward angle of operation. This is particularly true for patients with a short neck and a prominent shoulder.

A high jugular bulb restricts access to the internal auditory canal in both the suboccipital and the translabyrinthine approaches (Fig. 12). When the dome of the bulb lies adjacent to the inferior aspect of the internal auditory canal, it becomes difficult, by either operative route, to create an optimal trough around the canal. This usually constitutes a nuisance only that slows tumor exposure and dissection within the internal auditory canal. In rare cases, the bulb extends superiorly to overlap part or all of the internal auditory canal. When we have encountered this in the past, retraction of the tentorium has permitted creation of a sufficient bony trough superiorly to obtain complete removal of the internal auditory canal portion of the tumor. In extreme cases, however, atraumatic removal of the tumor from the facial nerve in the obscured midportion of the internal auditory canal may be rendered impossible, necessitating incomplete removal. In such cases, a middle fossa approach may allow complete removal of residual tumor within the internal auditory canal, regardless of how high the jugular bulb lies.

A high jugular bulb does not, in and of itself, impair exposure of the cerebellopontine angle. This anatomic variation, however, is occasionally associated with a relatively superior sigmoid sinus course that may restrict access to the inferior portion of the cerebellopontine angle in the region of the neural compartment of the jugular foramen when using the translabyrinthine approach (see Fig. 12). This anomaly is not problematic with

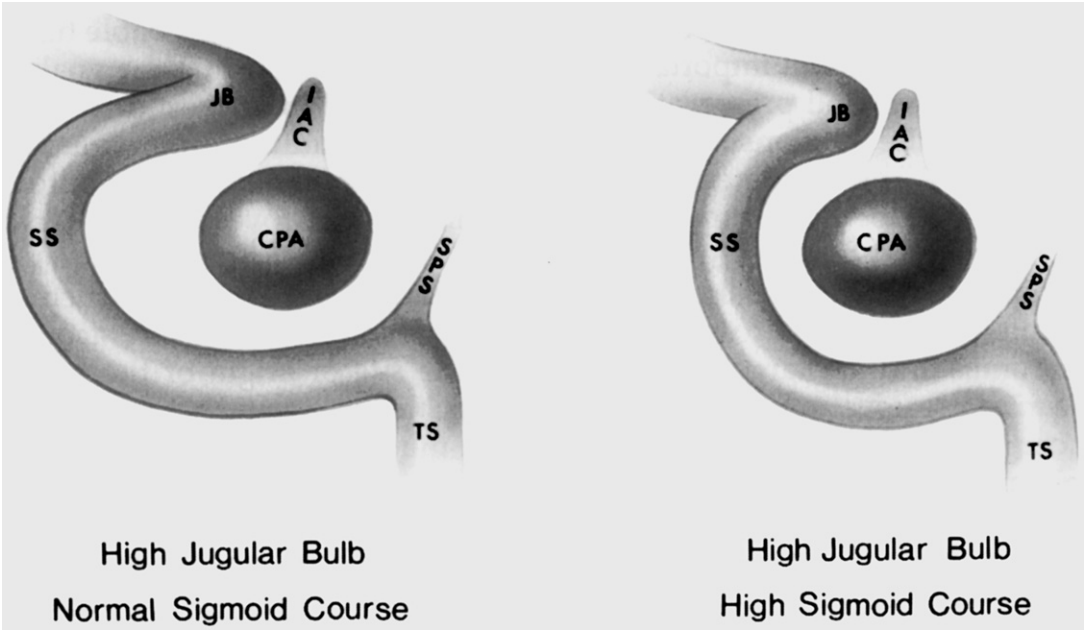

Fig. 12. A high jugular bulb impairs access to the internal auditory canal (IAC) during both the translabyrinthine and suboccipital approaches. When the bulb approaches the IAC it is difficult to create a trough wide and deep enough for unhindered exposure of the inferior compartment of the IAC. Creating additional superior exposure usually compensates well for this limitation. When the course of the sigmoid sinus is unusually superior, it may restrict access to the lower portion of the cerebellopontine angle (CPA) during the translabyrinthine approach.

acoustic neuromas because they typically are not adherent to the nerves of the jugular foramen and, once debulked, are readily mobilized into the operative field. With nonacoustic lesions of the posterior fossa that protrude into the medial aspect of the jugular foramen (neural compartment) or toward the foramen magnum region, this poor visualization of the lower cerebellopontine angle may be limiting. This is particularly true for meningiomas, which do not easily mobilize owing to their broad and tenacious dural attachment.

Suspicion that the lesion may not be an acoustic neuroma

Tumors other than acoustic neuromas constitute between 5% and 15% of lesions involving the cerebellopontine angle. The majority of these are meningiomas followed in incidence by epidermoids and a variety of other uncommon lesions. A preoperative awareness of the probable tumor type is of substantial importance to the surgeon in achieving the goals of tumor removal and preservation of adjacent neural structures. In acoustic neuromas, the relationships of cranial nerves around the tumor are quite predictable. The facial nerve is typically located on the anterior or anterosuperior surface of the tumor, the Vth cranial nerve above, and the nerves of the jugular foramen (IXth, Xth, and XIth cranial nerves) below. In meningiomas, the relationships of critical neural and vascular structures within the posterior fossa are much less predictable than in acoustic neuromas and are largely dependent on the site of origin of the tumor. Hearing preservation, which is seldom possible in acoustic neuromas over 2 cm in size, is occasionally achieved with much larger meningiomas and epidermoids. Removal of these lesions leads to an improvement of hearing in some cases, a result rarely seen with acoustic neuromas. When a patient has significant residual hearing and preoperative imaging studies suggest the presence of a non–acoustic neuroma cerebellopontine angle tumor, a suboccipital craniotomy should be selected regardless of tumor size. An exception to this rule is a meningioma that deeply penetrates the internal auditory canal, a situation in which we recommend translabyrinthine removal to

minimize risk of later recurrence within the temporal bone [12].

Until recently, it was often not possible to distinguish between a cerebellopontine angle meningioma and an acoustic neuroma preoperatively. With gadolinium-enhanced MRI, we are able to differentiate between these lesions in nearly every case. Unlike acoustic neuromas, meningiomas usually (1) do not penetrate the internal auditory canal; (2) have their main bulk eccentrically positioned with respect to the long axis of the internal auditory canal; (3) are sessile, possessing a broad base against the petrous face rather than being globular; (4) demonstrate hyperostosis of the subjacent bone; (5) possess intratumoral calcification; and (6) are characterized by a distinctive dural "tail" extending away from the tumor surface [13]. Epidermoids, unlike acoustic neuromas, are low intensity on T_1-weighted scans and high intensity on T_2-weighted scans and do not enhance with gadolinium.

Operating time

We have not found duration of the operation to be a significant criterion in selecting among approaches. The length of time it takes to expose the cerebellopontine angle and internal auditory canal components of the tumor completely is roughly the same, in our hands, with either the translabyrinthine or the suboccipital approach. For the tumor to be entirely exposed, the suboccipital technique requires a craniectomy, retraction of the cerebellum, and removal of the posterior petrous face overlying the internal auditory canal. In the translabyrinthine approach, the same goal is entirely accomplished by the transtemporal craniotomy. The relative operating time between approaches may vary significantly, however, for surgical teams that emphasize one method and are therefore more facile with it. When this is the case, duration of surgery may be a criterion in selecting the method of operation in patients with medical conditions that may be adversely affected by a prolonged anesthetic.

Operation for recurrent tumor

In reoperations for recurrent acoustic neuromas, we try to avoid using the same approach as was used in the primary procedure [14]. By revising suboccipital procedures translabyrinthine, and translabyrinthine procedures suboccipital, we are able to avoid areas of dural scarring. This permits more rapid and atraumatic establishment of favorable arachnoid planes.

Otologic considerations

Although uncommon, the presence of a perforation in the tympanic membrane contraindicates the translabyrinthine approach. Although the normal middle ear and mastoid mucosa are sterile, with an eardrum perforation the pneumatic spaces of the temporal bone are bacterially colonized, even in the absence of chronic otitis media. Although the translabyrinthine approach more extensively traverses the mucosa within the temporal bone, the suboccipital approach may also enter contaminated air cells surrounding the internal auditory canal. Cerebrospinal fluid leakage through a bacterially contaminated middle ear space carries a high risk of meningitis. Before surgery for acoustic neuromas, we favor grafting tympanic membrane perforations and controlling any active infection present in the temporal bone.

Patient age

Although the patient's age is a major influence over whether surgery is performed and the extent to which tumor is resected, age is only a minor factor in choosing among operative approaches. We avoid the middle fossa approach in patients over the age of 60 because of the thinness and fragility of the temporal floor dura. In older individuals, the dura is difficult to elevate and frequently tears, exposing the temporal lobe. This increases the chances of temporal lobe injury and cerebrospinal fluid leak. We prefer to approach intracanalicular lesions in the aged with either the suboccipital or the translabyrinthine approaches.

Most older individuals with small or medium-sized tumors are operated on only when sequential MRI scans document progressive growth that threatens to compress the brain stem within the patient's predicted life span. The most common reason for removing a relatively small tumor in the aged is disabling imbalance. Elderly patients tolerate perverted vestibular input poorly, and compensation is often improved by surgically ablating vestibular function on the affected side. When dysequilibrium is the primary indication for surgery, we prefer the translabyrinthine approach, which, by removing the semicircular canals, completely eliminates afferent input in the vestibular system on the side of the tumor. The translabyrinthine technique maintains the option of

performing subtotal tumor removal without fear of leaving residual vestibular fibers. If an elderly patient with a significant balance disorder is operated on via the suboccipital approach, it is important to perform a thorough vestibular neurectomy at the time of tumor resection.

Medical considerations

The medical condition of the patient is of paramount importance in deciding whether to operate and the extent of resection but seldom dictates the choice of operative method. Medical factors that have impact on the selection of surgical approach are rare and highly specific. For illustrative purposes, we provide a few examples. In a patient with a history of temporal lobe epilepsy, it is best to avoid the middle fossa route because the required brain retraction may aggravate the condition. One of our patients had a history of jugular vein thrombophlebitis associated with recurrent pulmonary embolism. We recommended the suboccipital over the translabyrinthine route to avoid retraction on the sigmoid sinus, which, by slowing flow in the jugular vein, may have precipitated further thrombosis. When a patient has an only seeing eye ipsilateral to the tumor, we favor conservative tumor removal to avoid facial palsy with the risk of exposure keratopathy. In such cases, we prefer a subtotal removal, which is usually accomplished via the translabyrinthine approach. In one patient, who was severely deficient in abdominal fat (owing to anorexia nervosa), the suboccipital approach was chosen because it permits primary dural closure without the need for a tissue graft. In the great majority of thin patients, sufficient adipose tissue can be obtained to close a translabyrinthine craniotomy satisfactorily. When there is a paucity of fat in the anterior abdominal wall, the fat graft may be harvested from the hip via an incision placed just beneath the iliac crest. In extremely obese individuals, access to the operative site may be difficult, especially when the neck is short and the shoulder is large. This limitation of exposure equally affects the translabyrinthine and suboccipital approaches. In one massively obese individual, we elected to use a sitting position because the traditional lateral or supine positions provided insufficient access to the suboccipital region. In this patient, the translabyrinthine approach was not a viable option. Undoubtedly there are other uncommon medical factors that may influence selection of operative approach, each of which should be analyzed by the clinician according to its unique attributes.

Incidence of postoperative complications

The incidence of serious complications, such as brain stem stroke, postoperative hemorrhage into the cerebellopontine angle, and death, is remarkably infrequent in acoustic neuroma surgery. Such rare events are largely attributable to the size and complexity of the tumor as well as the patient's age and medical condition rather than to which particular surgical approach was employed. In a few, less serious complications, there are some differences in the rate of occurrence among the various approaches.

In comparing approaches to medium-sized and large tumors, there are some differences in the incidence of complications with suboccipital and translabyrinthine approaches. Most importantly, we have found cerebrospinal fluid otorhinorrhea to be both more common and more difficult to manage following suboccipital craniectomy. Although most cerebrospinal fluid leaks respond to conservative management (lumbar drain, head elevation, fluid restriction, and acetazolamide administration), some require a secondary procedure (usually transtemporal obliteration of the eustachian tube) to stop the flow occurring through pneumatic tracts surrounding the internal auditory canal. This problem persists despite diligent efforts to seal the transected air cells with bone wax, fascia, muscle plugs, and fibrin glue applied to the drilled surface of the internal auditory canal at the time of suboccipital surgery. Fortunately, once cerebrospinal fluid leakage has been controlled, the patient suffers no lasting consequences other than the psychological and financial burden of a somewhat prolonged recovery period. Not all surgical teams report a higher incidence of cerebrospinal fluid leakage with the suboccipital as compared with the translabyrinthine approach. An equal incidence has been reported or even a higher rate with the translabyrinthine technique [15,16].

Another difference between the suboccipital and translabyrinthine approaches is the incidence of persistent headaches lasting from several months to a year postoperatively. This is more frequent following the suboccipital approach, presumably as a consequence of aseptic meningitis. In the suboccipital approach, the internal auditory canal is drilled open intradurally,

whereas in the translabyrinthine approach, all bone removal is accomplished extradurally. During the suboccipital approach, some contamination of the subarachnoid space by bone dust is inevitable despite the liberal use of Gelfoam and a rubber dam to contain the debris. This is particularly true with small tumors, in which the cisternal space is not blockaded by tumor. In our experience, most patients with persistent headache have had small tumors operated on by a suboccipital hearing conservation approach.

The translabyrinthine technique has a few complications not shared with the suboccipital approach. Hematoma may form in the site of harvesting the abdominal fat graft. Meticulous hemostasis and the use of drains have rendered this uncommon, but a rare patient may require reoperation for persistent bleeding. The cosmetic effects of an abdominal scar may be significant, especially when aesthetic considerations are important to the patient. In these cases, we have avoided an abdominal scar by obtaining the fat graft from the hip via an incision just beneath the iliac crest. This technique is particularly suitable for women, who tend to have a fat deposit in this area. Finally the translabyrinthine approach is more likely to cause injury to the sigmoid sinus than the suboccipital approach. Although this injury is almost always well tolerated, we are aware of two potential complications. One patient succumbed to a massive pulmonary embolus that apparently derived from a thrombosis of the sigmoid sinus, which propagated into the jugular vein (Althaus S, personal communication, 1990). In another case, a partial visual loss resulted from venous congestion in the optic nerve as a result of occlusion of a dominant sigmoid sinus (S. Seiff, personal communication, 1991).

The middle fossa route is accompanied by a few risks not shared with the translabyrinthine and suboccipital approaches. Many patients suffer a degree of postoperative trismus as a result of manipulation of the temporalis muscle, but this is seldom prolonged. Retraction of the temporal lobe, particularly on the dominant side, may rarely lead to transient dysphasia or episodes of auditory hallucinations. Finally epidural hematoma has been reported and may have serious consequences. The risk of this may be minimized by meticulous hemostasis, tacking the dura up to the bone flap at the end of the procedure, and judicious use of a subtemporal drain.

Patient choice

A patient occasionally has a preference for a particular operative approach for personal reasons that may be well founded or arbitrary. For example, an individual may reject the middle fossa approach because it involves a more visible head shaving than the translabyrinthine or suboccipital approach. A musician may prefer an incomplete suboccipital removal to maximize chances of hearing conservation. In familial cases, a patient may prefer to have the same approach a relative had. In general, we are willing to accommodate patients' preferences if they have carefully considered their options and have strongly felt reasons for their decision, and the choice is medically reasonable.

Protocol for the selection of operative approach according to tumor size, location, and clinical presentation

Intracanalicular tumors

At first reckoning, therapeutic decision making in intracanalicular tumors would seem to be relatively straightforward. Actually it is quite complex (Fig. 13). Anatomically, intracanalicular tumors may be considered to occur in three varieties: (1) involving the entire internal auditory canal, (2) limited to the proximal canal adjacent to the porus acusticus, and (3) limited to the distal canal in proximity to the transverse crest and vestibule (Fig. 14). Any intracanalicular tumor with poor hearing (roughly $< 30\%$ speech discrimination score, or 70-dB speech reception threshold) is addressed via a translabyrinthine approach. When significant residual hearing persists, the size and location of the tumor become important.

Medially placed tumors that do not involve the distal one third of the internal auditory canal are approached suboccipitally in an attempt to preserve hearing. This permits exposure of the tumor under direct vision, without violating the inner ear, at an angle favorable for atraumatic dissection of the facial nerve.

Small intracanalicular tumors that are limited to the lateral portion of the internal auditory canal in ears with good hearing are usually approached via the middle fossa route because this is the only method that provides direct access to this region without violating the inner ear. When the tumor has arisen from the inferior vestibular nerve, the facial nerve may lie draped on the tumor surface between the surgeon and the

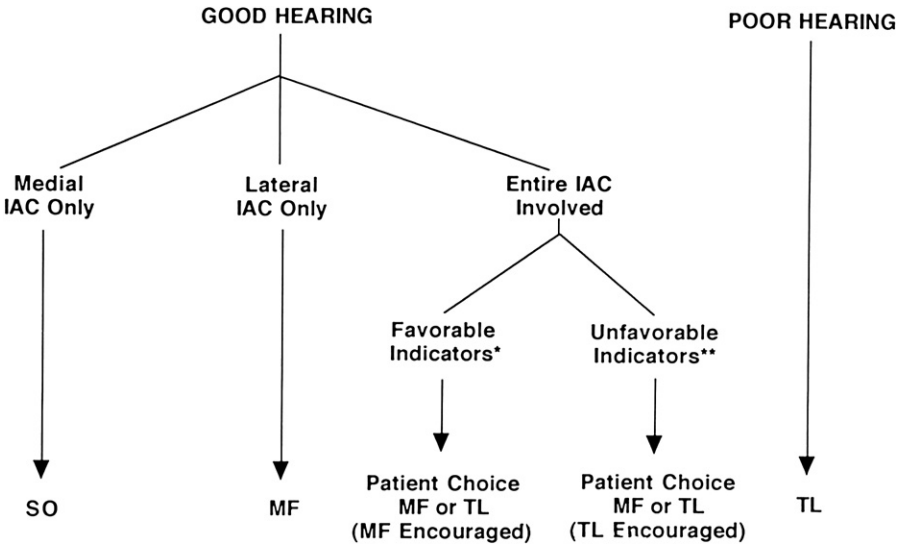

Fig. 13. Management of intracanalicular acoustic neuromas.

tumor (see Fig. 8). This requires a greater degree of facial nerve manipulation than with the suboccipital or translabyrinthine approaches, making postoperative facial neurapraxia more common following the middle fossa procedure. Fortunately this weakness is usually transient, and serious neural injury is unlikely to occur. We explain to the patient who has a small, laterally placed intracanalicular tumor that an attempt to preserve hearing comes at the price of a higher incidence of temporary facial weakness, but that a permanent, cosmetically significant deformity is improbable. The risk that the facial nerve will have an unfavorable relationship with the tumor via the middle fossa approach may be estimated, to some degree, by measurement of the preoperative caloric response. The caloric response is generated by the lateral semicircular canal that is innervated by the superior vestibular nerve. When the caloric response is normal (or nearly so), this suggests the

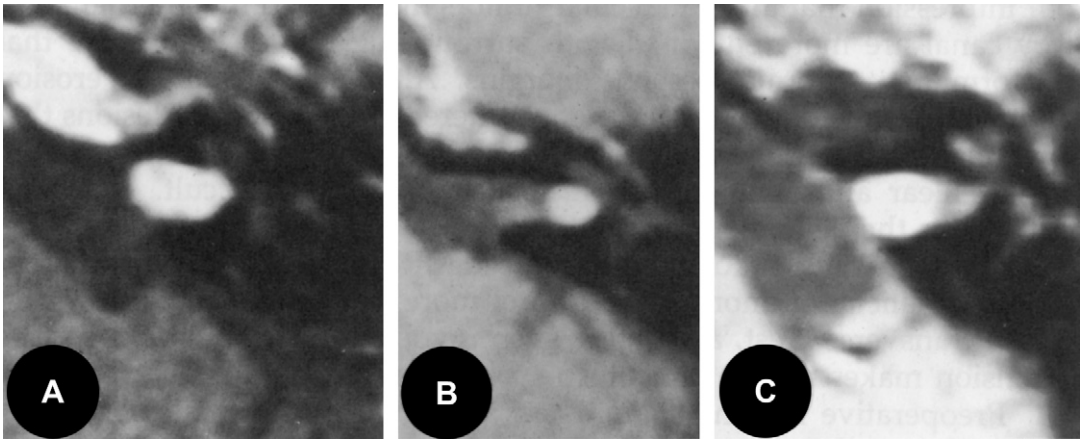

Fig. 14. Intracanalicular acoustic neuromas on gadolinium-enhanced magnetic resonance imaging scans in the axial plane. Examples include a tumor involving the entire internal auditory canal (*A*), only the lateral extremity (*B*), and only the proximal portion adjacent to the porus acusticus (*C*). The management implications of these lesions are addressed in the text.

presence of an inferior vestibular nerve tumor and predicts an unfavorable facial nerve relationship. A substantially impaired or absent caloric response does not necessarily imply favorable anatomy. Although the lesion may have originated from the superior vestibular nerve, inferior vestibular schwannomas can also impair both divisions of the nerve. When presented with these choices, most individuals with small, laterally placed intracanalicular tumors select a middle fossa hearing conservation attempt. The alternative is a translabyrinthine procedure with sacrifice of residual hearing.

The most common variety of intracanalicular tumor, in our experience, is one that involves the entire length of the internal auditory canal from the porus acusticus to its distal extremity. When good hearing persists, the middle fossa route is the only method that affords direct exposure to the entire tumor and the chance of preserving hearing. As previously discussed, however, this technique carries a higher risk of facial neurapraxia. In advising the patient as to the probability of hearing preservation and the relative degree of risk to the facial nerve, we take several factors into account in addition to those already mentioned. It has been our impression that intracanalicular rumors that expand the internal auditory canal are more entwined with surrounding nerves than those that conform to its normal osseous margins. Although most bony erosion occurs posteromedially, we have encountered intracanalicular lesions that expanded the anterior aspect of the canal. In such cases, dissection of both the cochlear and facial nerves has been unusually difficult. A second criterion is the degree of penetration of the lateral extremity of the canal by the tumor. With gadolinium-enhanced MRI scans, a surprising number of intracanalicular tumors penetrate 1 or more mm deep to the free margin of the transverse crest, and a few even involve the vestibule. Such deep extension makes it very difficult to spare hearing.

Preoperative hearing status is an important determinant of surgical approach to an intracanalicular tumor. As a general rule, the more normal the hearing, the higher the probability of success with hearing preservation. We place particular importance on the level of speech discrimination scores and the auditory brain stem response results. Selection of the optimal surgical approach in these cases is often a matter of judgment and is not easily codified into a set of rules. Of course, when hearing is normal, the canal is not expanded, and the far lateral end of the internal auditory canal is uninvolved, we strongly encourage either the suboccipital or the middle fossa approach. Similarly, when hearing is marginal, the osseous canal is substantially eroded, and deep penetration of the lateral extremity is detected, we encourage selection of the translabyrinthine technique. In essence, we present the relative degree of risk to hearing and facial function as well as the incidence of complications and the predicted postoperative recovery period and allow the patient to decide between a hearing conservation attempt (middle fossa or suboccipital) and a hearing sacrificing approach (translabyrinthine).

Medium-sized tumors (1 to 3 cm)

Tumors that are 1 to 3 cm in diameter in the cerebellopontine angle are approached by either the suboccipital or translabyrinthine approach (Fig. 15). Because all of these tumors possess a significant cisternal component, the middle fossa approach is not a viable option, in our opinion. Whenever hearing is poor, the translabyrinthine approach is employed. When there is significant residual hearing, a number of factors come into play. Tumor size influences decision making but is not a major criterion. Although it is true that the probability for success in hearing conservation drops off substantially in acoustic neuromas exceeding 2 cm in diameter in the cerebellopontine angle, hearing preservation may occasionally be achieved in larger tumors. When the hearing is especially good, we favor an attempt at saving hearing, even in tumors at the upper end of this size range. On the other hand, if the amount of residual hearing is marginal and the tumor is relatively large, we typically recommend a translabyrinthine approach.

The depth of tumor penetration within the internal auditory canal is a major determinant in selecting surgical approaches in patients with substantial residual hearing. When the tumor involves the lateral one third of the internal auditory canal, the suboccipital approach cannot expose the deepest portion of the tumor without exenterating a portion of the inner ear and thus sacrificing hearing (Figs. 16 and 17). Although it is possible to address this part of the tumor indirectly, without violating the inner ear, by using angled instruments and mirrors, this risks leaving viable tumor cells in the fundus of the internal auditory canal. The incidence of clinically significant

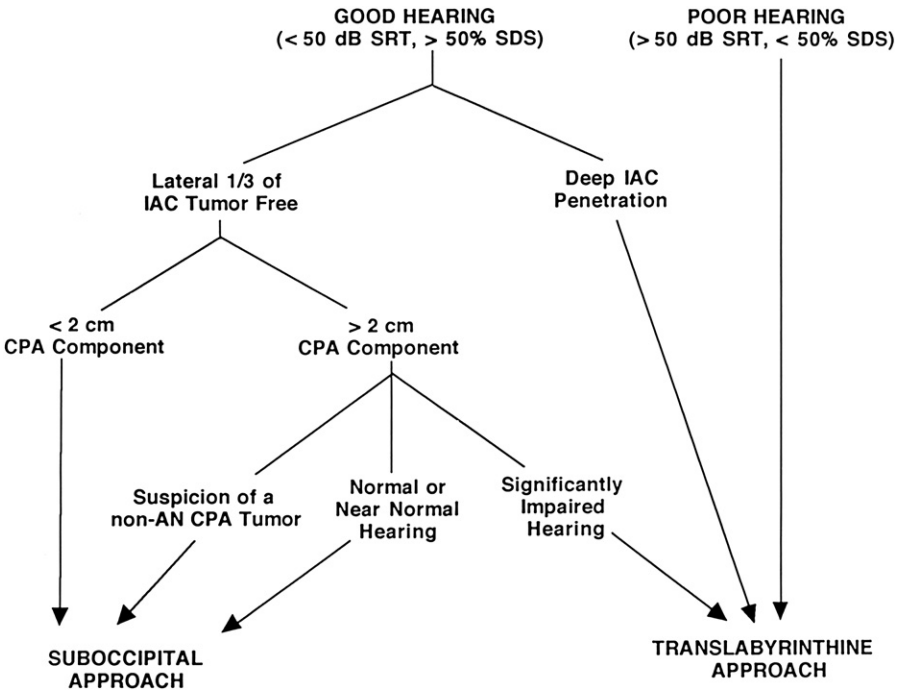

Fig. 15. Management of 1 to 3 cm acoustic neuromas.

recurrence as a result of persistent tumor in the distal internal auditory canal is unknown, although it is most likely quite small. Nevertheless, we have treated several patients in whom this situation appeared to cause a recurrence that necessitated reoperation. Under certain circumstances, assuming this risk may be warranted. In older patients with particularly good hearing, it is reasonable to perform a suboccipital approach and indirectly address the lateral extremity of the tumor. If the cochlear division is involved by tumor or the intraoperative auditory monitoring suggests a loss of hearing during dissection, further internal auditory canal exposure can be obtained to ensure complete removal under direct vision. As a general rule, we do not favor indirect dissection of tumor from the distal internal auditory canal in younger patients, for whom total tumor removal takes priority over attempts at hearing preservation.

Large tumors (>3 cm)

We remove the majority of large tumors via the translabyrinthine approach. It is chosen for its lower incidence of cerebrospinal fluid leakage, fewer prolonged headaches, and somewhat less postoperative morbidity. The suboccipital approach is used in a few special circumstances. When preoperative imaging studies produce significant doubt that the tumor is an acoustic neuroma, we favor the suboccipital approach in certain situations. The usual rules about tumor size and hearing preservation do not apply to non–acoustic cerebellopontine angle tumors. When the hearing level associated with a meningioma or epidermoid is relatively good, we employ a suboccipital approach, even when the lesion is large. The same exception applies for the rare large acoustic neuroma that is wholly extracanalicular, although in such circumstances there is usually significant doubt on preoperative studies as to the nature of the lesion (Fig. 18). We also use the suboccipital approach when a tumor extends into the region of the jugular foramen or foramen magnum, an area not readily exposed with a translabyrinthine craniotomy. This is seldom the case with acoustic neuromas but is frequent with larger, non–acoustic neuroma cerebellopontine angle lesions, especially meningiomas.

In cases of neurofibromatosis type 2, there are often multiple tumors in the posterior fossa. The more panoramic view of the lower portion of the cerebellopontine angle afforded by the

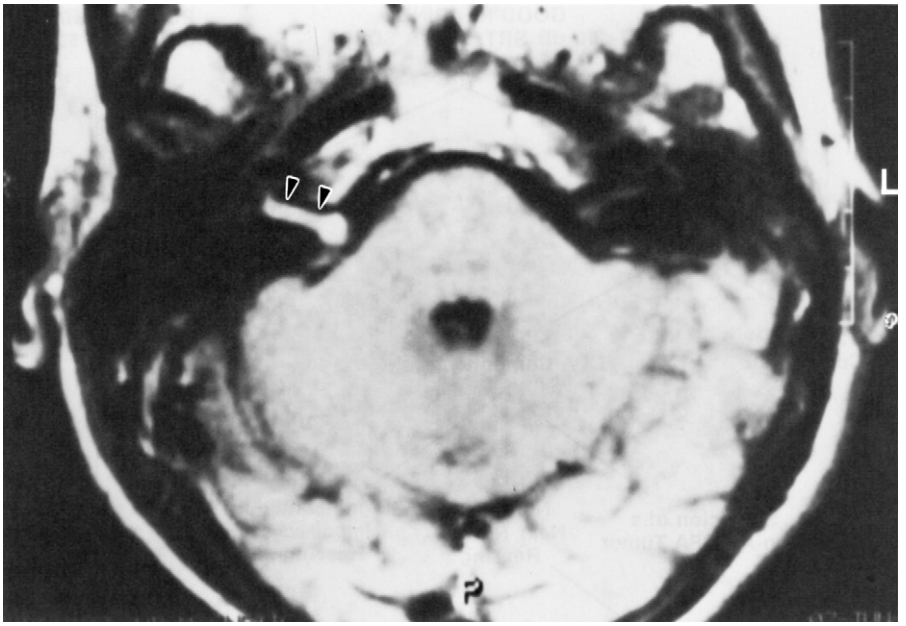

Fig. 16. Deep involvement of the internal auditory canal (*arrowheads*) by a small acoustic neuroma but only a small cisternal component as seen on an axial gadolinium-enhanced T_1 magnetic resonance imaging scan. It is difficult to save hearing with such a lesion with the suboccipital approach because exposure of the tumor requires drilling away a portion of the inner ear.

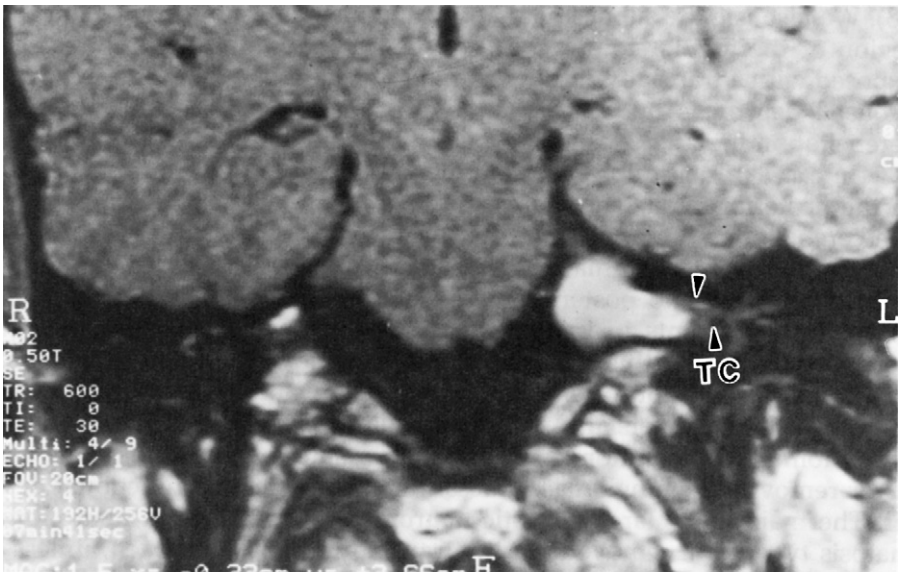

Fig. 17. Coronal gadolinium-enhanced MRI scan of an acoustic neuroma that penetrates approximately two thirds of the depth of the internal auditory canal. The cerebrospinal fluid in the distal one third of the canal can be visualized (*arrowheads*) as well as the transverse crest (TC). This tumor illustrates the maximum degree of IAC penetration that may be exposed through the suboccipital-transmeatal route.

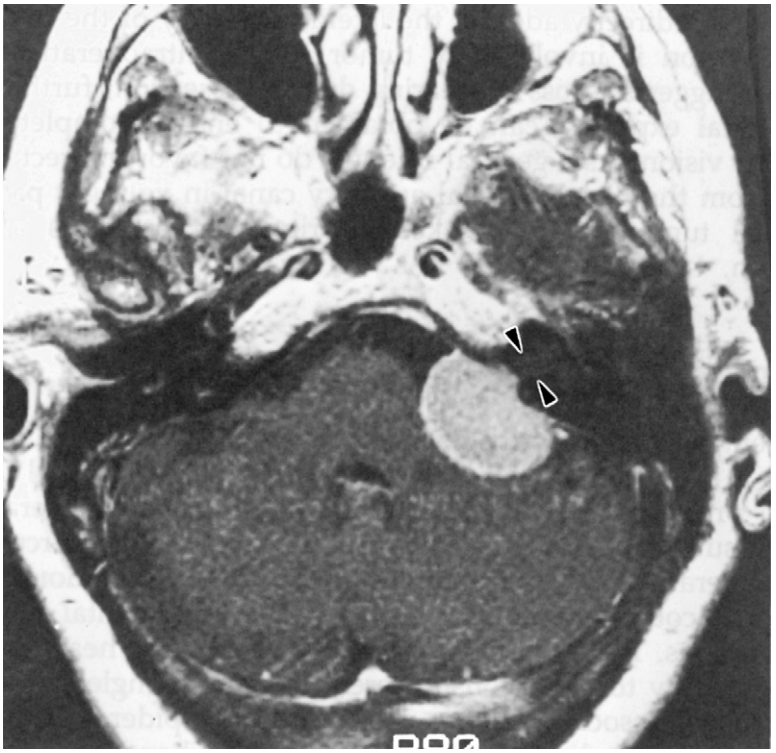

Fig. 18. An atypical medium-sized acoustic neuroma on a gadolinium-enhanced MRI scan demonstrating no extension into the internal auditory canal (*arrowheads*). When useful hearing remains in a wholly extracanalicular tumor, even though it is relatively large, the authors recommend a suboccipital hearing conservation approach.

suboccipital route allows inspection of the lower cranial nerves (IX through XII) for tiny schwannomas and the dura of the posterior fossa floor for early meningiomas. With recent improvements in imaging technology, especially gadolinium-enhanced MRI scans, this advantage has become less important. In any event, whether or not to remove miniscule schwannomas from functioning nerves is controversial. As a general rule, we leave undisturbed small, asymptomatic schwannomas in neurofibromatosis type 2 patients but favor removing meningiomas, unless this would create a functional deficit. The majority of large acoustic neuromas associated with neurofibromatosis type 2 are removed via the translabyrinthine approach, unless a second tumor in the jugular foramen or foramen magnum is visualized on MRI scan.

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Retrosigmoid Approach for Acoustic Tumor Removal

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COMMENTARY

This is an update to the approach and technique as described in the original article that follows.

Noel L. Cohen, MD, J J Thomas Roland, Jr, MD, John S. Golfinos, MD

The technique for this approach to the posterior cranial fossa continues to change and evolve, partially due to changing technology such as sophisticated monitoring techniques, and partially the result of on-going critical evaluation of complications and results of the surgery.

The main indication for the retrosigmoid approach remains the patient with useful hearing and an acoustic neuroma less than 15 mm extra canalicular. Occasionally, it is used in the case of a tumor, which is entirely intracanalicular, in which it is deemed wiser (eg, the older patient) not to perform a middle fossa approach. The translabyrinthine approach is used in virtually all patients with poor hearing and/or larger tumors, in which hearing preservation is not a reasonable goal.

The surgery is performed as follows:

Monitoring: EMG based VII, BAER, SSEP, transcranial facial MEP. Position, usually supine with head rotated away and semi-fixed with tape. Occasionally, lateral with head turned and rigidly fixed (for the ''fullback'' patient). The incision currently used is a ''gentle hockey-stick.'' An anteriorly-based skin and soft-tissue flap is turned, and then a pericranial anteriorly-based (Palva) flap is raised. Barbless fishhooks are used for retraction. Mannitol is administered intravenously. The mastoid is drilled and the sigmoid sinus skeletonized from the level of the transverse sinus to close to the jugular bulb, exposing the dura behind the sigmoid. A crescentic bone flap (>/=2.5 cm) is removed and preserved and the dura opened, creating an anterior-based C flap, and the CSF is decompressed. Retractors are not necessary but the cerebellum and any visible tumor are protected.

Dural flaps are raised on the posterior face of the petrous bone over the IAC, which is then drilled approximately 300 degrees around at the porus and less so laterally, out to +/-9 mm from the porus. The internal dura of the IAC is opened horizontally, the various nerves identified, a monitoring electrode is placed on the cochlear nerve and the tumor removed.

Closure: perimeatal cells, if any, are waxed, fat placed and glued in the bony defect, the petrous dural flaps are closed, as is the posterior fossa dura with the help of a temporalis fascia graft. The bone flap is replaced with titanium mini plates. The perilabyrinthine and retrofacial mastoid cells are waxed, and abdominal fat is glued in the mastoid so as not to inhibit incus motion. The Palva flap is closed and the scalp closed in layers.

The retrosigmoid approach to the posterior fossa is a modification of the traditional neurosurgical suboccipital craniotomy. The suboccipital craniotomy gives a wide view of the posterior fossa and has been the mainstay of access to this area by neurosurgeons since it was first described by Fraenkel and colleagues [1] in 1904 (Fig. 1). In fact, Woolsey had performed the first operation in 1903, to be followed very shortly by Krause in 1905 [2].

Cushing [3] then described his operation, consisting of a large, ''crossbow'' bilateral exposure of the posterior fossa, operating on the patient in the prone position. He advocated a subtotal removal. Dandy [4] then brought the operation

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ANNALS OF SURGERY

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ORIGINAL MEMORIS

CONTRIBUTION TO THE SURGERY OF NEUROFIBROMA OF THE ACOUSTIC NERVE.[1]

BY JOSEPH FRAENKAL, M.D., AND J. RAMSAY HUNT, M.D.,
OF NEW YORK

WITH REMARKS ON THE SURGICAL PROCEDURE

BY GEORGE WOOLSEY, M.D., AND CHARLES A. ELSBERG, M.D.,
OF NEW YORK

THE rapid advances made during recent years in our Knowledge of the location of funtions in the cerebal cortex and the function and course of various conduction paths within the brain have rendered possible the localization of a large number of brain tumors.

In some instances, especially when situated in the Rolandic area or at the base of the brain implicating cranial nerves, the location of the neoplasm may be indicated with great exactness; more often, however, only an approximate idea of its situation is possible.

An estimation of the size and character of the growth is nearly always a mere matter of speculation, based on tumor

[1] Read before the New York Neurological Society, February 2, 1904.

Fig. 1. Fraenkel's original article.

into the more modern period, using a unilateral approach in 46 cases, with a mortality of only 10.8% (Fig. 2). Considering that the operation was done with primitive inhalation anesthesia, without the use of the operating microscope, adequate lighting, microsurgical instrumentation, or intraoperative monitoring, this was quite a remarkably low mortality rate. He was an advocate of total removal, accepting the almost inevitable loss of facial nerve function (44 of 46 cases). Interestingly Dandy reported that there was "good hearing" in 34 of his cases preoperatively.

Traditionally the suboccipital approach was performed in the seated position and consisted of a long straight incision extending well into the neck; elevation of the nuchal muscles from the posterior aspect and undersurface of the occipital bone; and removal of a large segment of the bone, extending laterally to the sigmoid sinus, medially to the midline, superiorly to the transverse sinus,

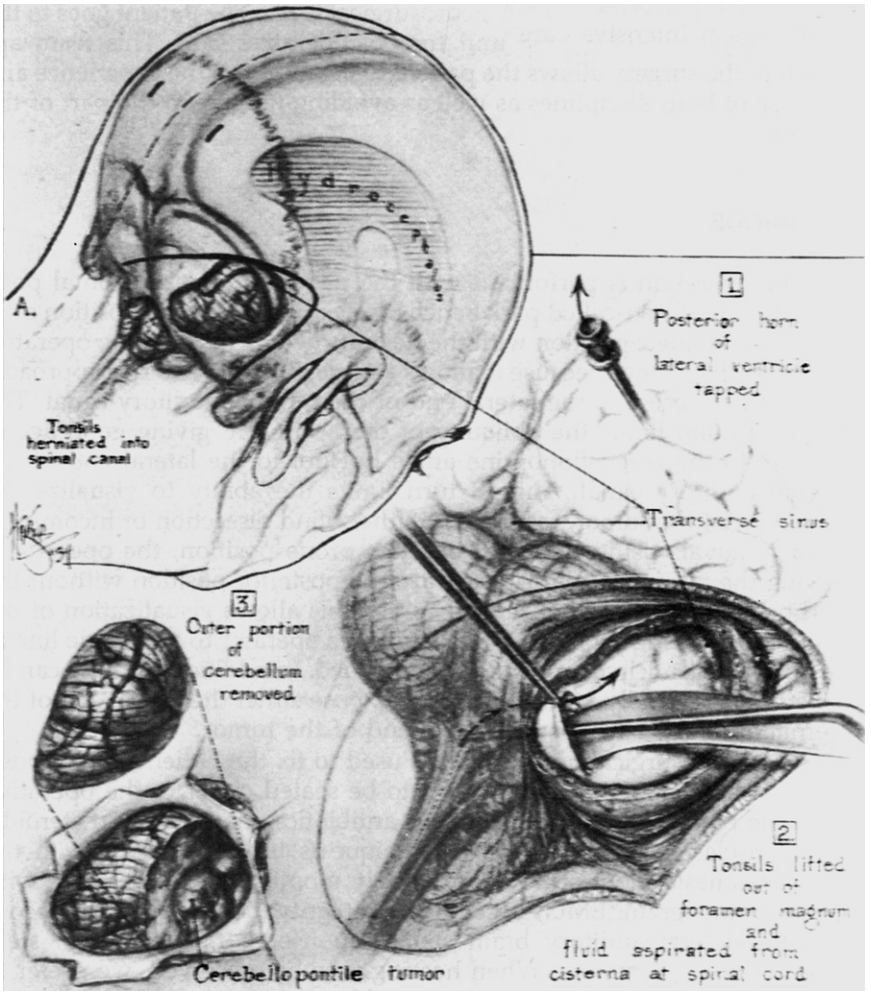

Fig. 2. Dandy's 1941 technique.

and inferiorly to the foramen magnum. Following opening of the dura, a segment of cerebellum was resected to gain access to the cerebellopontine angle, after which the tumor could be removed [5–8].

The suboccipital operation underwent many modifications, ultimately resulting in what has come to be called the *retrosigmoid-transmeatal operation* [9–13]. This approach involves a small curved or angulated incision, stopping well above the undersurface of the skull, no dissection of nuchal muscle from the bone, a limited removal of bone behind the sigmoid sinus, and no resection of the cerebellum. This is combined with exposure of the internal auditory canal by removing the posterior wall: hence the term *retrosigmoid-transmeatal*.

The surgical team consists of a neurosurgeon, neurotologist, anesthesiologist, and monitoring electrophysiologist, in addition to the usual nursing personnel. The neurosurgical team prepares the patient, makes the incision, performs the craniotomy, and exposes the tumor or the posterior face of the petrous bone as well as the VIIth and VIIIth cranial nerves in the cerebellopontine angle. If the tumor is large, the team debulks it and partially dissects it off the brain stem. Following this, the neurotologist opens the internal auditory canal and removes the remainder of the tumor.

Following tumor removal and plugging of the internal auditory canal, the closure is performed by the neurosurgical team. The patient goes to the neurosurgical intensive care unit for postoperative

care. This team approach to the surgery allows the patient to benefit from the experience and expertise of both disciplines as well as avoiding fatigue on the part of the surgeon.

Technique

The operation is performed with the patient in the horizontal position, either in the so-called park bench (three quarters prone) position or in the supine otologic position with the head turned away from the operator. We prefer the former because it allows a more oblique posterior approach, giving better access to the lateral end of the internal auditory canal. The supine position limits the obliquity of the approach, giving good access medially to the cerebellopontine angle but not to the lateral end of the internal auditory canal. This in turn limits the ability to visualize the lateral end of the tumor, resulting in either blind dissection or incomplete tumor removal. Using the three quarters prone position, the operator is viewing the internal auditory canal from a posterior position without the head being rotated on the cervical spine. This allows visualization of the bone as the drilling proceeds and enables the operator to see a blue line as the posterior semicircular canal is approached. In addition, drilling can be carried as far laterally as is necessary to expose either the lateral end of the internal auditory canal or the lateral end of the tumor.

The neurosurgical head holder is used to fix the patient's head position as well as allowing the surgeon to be seated closer to the operative field. The patient receives intravenous antibiotics, mannitol, and steroids; a nonmuscle-relaxant anesthesia technique is used. In addition to the usual anesthesia monitoring, we routinely monitor the facial nerve with the electromyogram (EMG)-based nerve integrity monitor. We also record the contralateral auditory brain stem response to monitor brain stem integrity in larger tumors. When hearing is to be preserved, we prefer to monitor the VIIIth cranial nerve potential by placing an electrode directly on the surface of the VIIIth cranial nerve. If this is not feasible, ipsilateral auditory evoked responses are recorded. The incision consists of an L-shaped or C-shaped flap based anteriorly and centered at the approximate location of the transverse sinus (Fig. 3). The flap is retracted forward with barbless fishhooks. Soft tissues are elevated off the bone, and a craniotomy measuring approximately 3 to 5 cm in diameter is performed. The anterior border is the sigmoid sinus, whereas the superior border is the transverse sinus (Fig. 4). Often retrosigmoid mastoid air cells are entered, and these are filled with acrylic to seal them.

An anteriorly based dural flap is then incised and sutured forward, exposing the cerebellum. With the patient in the three quarters prone position, the head slightly elevated, and mannitol given intravenously, the cerebellum generally tends to fall away from the tumor once the

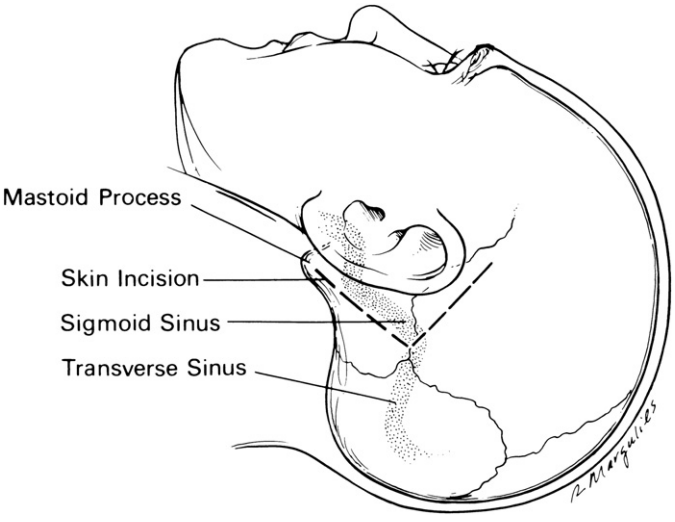

Fig. 3. Incision for retrosigmoid approach.

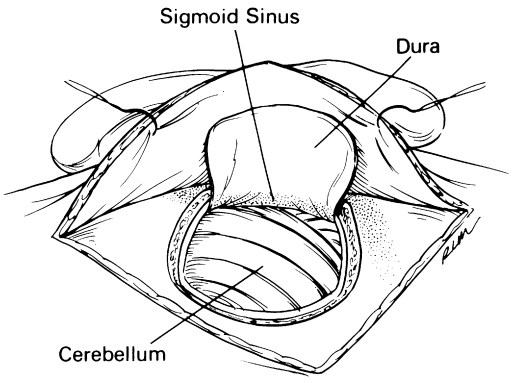

Fig. 4. Dural flap elevated.

subarachnoid space is opened. Usually the cerebellum need only be very lightly supported (Fig. 5A). This then allows opening into the cerebellopontine cistern, with release of the cerebrospinal fluid. The cerebellum is protected with Gelfoam (Upjohn, Kalamazoo, Michigan), a rubber dam, or Silastic sheeting (Fig. 5B). It need be retracted only for a larger tumor.

If the tumor is bulky, it may decompressed with the use of an ultrasonic aspirator at this point. We no longer use the carbon dioxide laser to debulk the tumor because the ultrasonic aspirator is much more rapid, causes no heating or charring of the tissue, does not require any bulky addition to the operating microscope, and avoids the potential danger of accidental burns inherent in all laser surgery. The rapidity of tumor removal can be varied by adjusting the power of the ultrasonic output as well as the suction. Care is taken to remain well within the capsule of the tumor to avoid injury to the facial nerve and brain stem. The ultrasonic aspirator need not be used when dealing with tumors that are less than 15 mm in diameter as measured from the porus. Obviously in the case of intracanalicular tumors, cerebellar retraction is minimal, and the tumor can usually be excised in one piece.

If necessary, the tumor is dissected off the cerebellum as well as the brain stem, depending on the size of the tumor. The facial nerve can be identified at or near the brain stem at this point, and if hearing preservation is the goal, an electrode can be placed on the cochlear nerve as well (Fig. 6A). When attempting hearing preservation, it is important to limit electrocautery as much as possible, depending on gentle blunt dissection to tease blood vessels away from the tumor and the VIIIth cranial nerve, while using Gelfoam to stop light venous oozing.

The dura over the posterior face of the petrous bone is coagulated using monopolar current and then excised after curettage. Alternately a laterally based dural flap can be raised [14]. Bleeding is controlled with the cautery. A cutting bur is used to outline the approximate location of the internal auditory canal, following which diamond burs are used for the final drill out. The magnetic resonance image (MRI) is used as a guide to the length of tumor extension into the internal auditory canal, and every attempt is made not to open into the posterior semicircular canal or vestibule, since this would probably result in the total loss of hearing. The endolymphatic sac and duct can also be used as landmarks: it is imperative

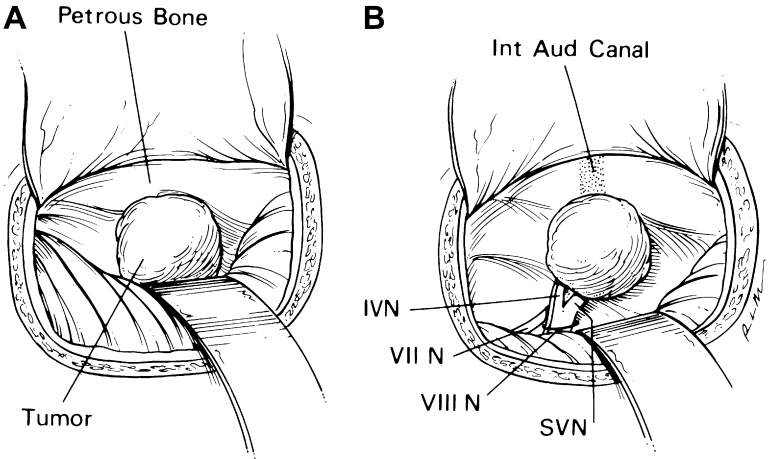

Fig. 5. (*A*) Tumor exposed; (*B*) cerebellum retracted.

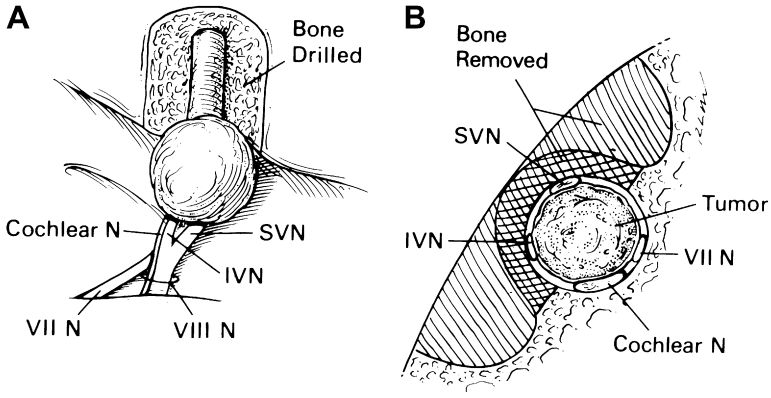

Fig. 6. (*A*) Bone drilled and nerves identified; (*B*) bone removed.

to remain medial and anterior to the duct to avoid entering the labyrinth [15,16]. When hearing is not to be preserved, either because of the low level of hearing or the size of tumor, as much bone as is required can be removed with impunity.

In attempting hearing preservation, up to 7 mm of the posterior internal auditory canal wall can generally be removed without endangering the posterior semicircular canal, but close observation is necessary to recognize a blue line as the labyrinth is approached. As drilling proceeds laterally, it is usually possible to recognize the lateral end of the tumor, even through the intact dura. The dura bulges over the tumor and transmits a fleshy red or yellow color. The internal auditory canal lateral to the tumor has flat dura, which generally appears blue or white. If the surgeon is unsure of whether the lateral end of tumor has been reached, the dura can be slit to inspect the canal. Further bone can then be removed if necessary.

It is important not only to thin the bone over the posterior aspect of the internal auditory canal, but also to drill well superior and inferior to the canal to expose least 180 degrees if not 270 degrees of the circumference of the canal, leaving only the anterior wall intact (Fig. 6B). This allows much easier and less traumatic dissection of the tumor off the nerves to be preserved. Care must be taken inferiorly if the jugular bulb is high-riding.

During this drill out, the subarachnoid space above and below the tumor should be protected with Gelfoam or Cottonoid to prevent bony debris from entering the subarachnoid space to an unnecessary extent [15]. If extensive, this material might lead to aseptic meningitis during the postoperative period or hydrocephalus at a later date. Suction irrigation also helps to limit the amount of bony debris and blood clot in the posterior fossa.

Once the bony removal has been completed, the dura of the internal auditory canal is slit with a fine sharp knife, ideally creating superior and inferior dural flaps by making a T-shaped incision in the dura (see Fig. 6). If the lateral end of the tumor cannot be visualized or readily delivered into the wound with an elevator, further bone should be removed. Dissection of the lateral end of the tumor should reveal the origin from the superior or inferior vestibular nerve, whereas retraction of these allows visualization of the facial and cochlear nerves (Fig. 7A).

After cutting of the vestibular nerves laterally, a plane is established between the tumor and the facial nerve superiorly and the cochlear nerve inferiorly (Fig. 7B). Dissection can then proceed either from lateral to medial or medial to lateral. There is a theoretical advantage to dissection from medial to lateral because it may not avulse fine blood vessels from the lateral end of the internal auditory canal and therefore the cochlea, but this has never been critically investigated. We have used both techniques and cannot perceive a difference in our hearing preservation results.

Attention is then paid to the medial end of the tumor, where the. superior and inferior vestibular nerves are very often splayed apart by the smaller tumor. These nerves may be cut at this point to allow dissection of the tumor from medial to lateral, but when attempting hearing preservation, it is safer to use blunt rather than sharp dissection because the blood supply to the inner ear may be inadvertently sacrificed by cutting the vestibular

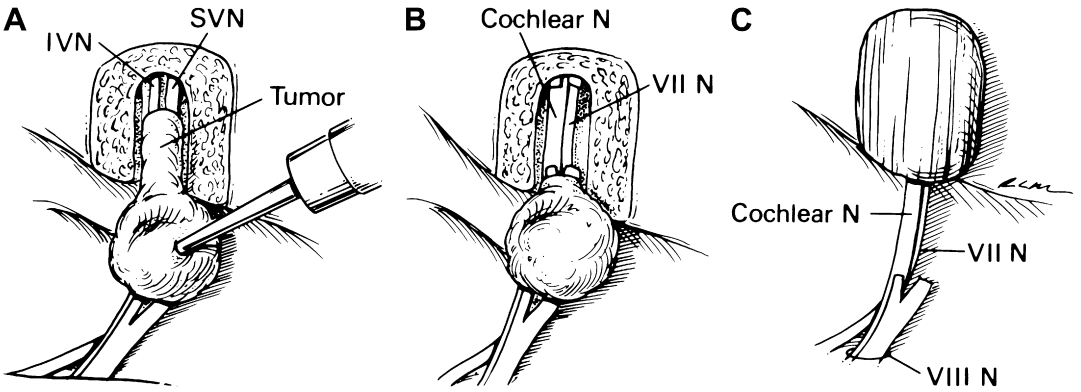

Fig. 7. (*A*) Cross section internal auditory canal (IAC); (*B*) SVN and IVN cut; (*C*) tumor out, IAC plugged.

nerves [7]. In large tumors, it is probably better to use sharp dissection rather than blunt to separate the tumor from both the facial and the cochlear nerves as long as no visible vessels are cut. This avoids further stretching of the already attenuated facial nerve [15].

A plane is developed between the tumor and the cochlear nerve, and dissection proceeds to remove the entire tumor with preservation of both the facial and the cochlear nerves or the facial nerve only (Fig. 7C).

When the operation is performed for hearing preservation, the primary goals are still total removal of the tumor with preservation of facial nerve function. Less than total removal should not be performed to preserve hearing but may be necessary occasionally to preserve facial nerve function. This is usually not the case because the facial nerve is seldom splayed out or distorted by tumors less than 2 cm in diameter. Occasionally a small scrap of tumor or capsule may be left attached to the facial nerve either just medial to the porus, where the nerve takes a sharp jog anteriorly, or at the brain stem, where the nerve is flattened by a large tumor.

In dealing with a larger neuroma, the ultrasonic aspirator is used to debulk the interior of the mass as it occupies the angle, then to remove the tumor as it is dissected off the brain stem, facial nerve, and out of the internal auditory canal (see Fig. 7A). With larger tumors or poor hearing, bipolar cautery is an important aid, but its use should be limited when hearing preservation is one of the goals of surgery. In general, it is safer to cauterize blood vessels rather than stretch them to the point where they might tear and retract. This is particularly important when dealing with a large petrosal vein, which is usually found at the superior aspect of the tumor and, if torn, can give rise to extremely troublesome bleeding as it retracts toward the tentorium.

Intraoperative auditory monitoring consists primarily of direct cochlear nerve recording. This is greatly preferable to using the auditory brain stem response because the latter requires extensive sampling and entails a very considerable time delay before any changes are noted [5,17,18]. It has been our experience that intraoperative auditory brain stem response monitoring has not increased our ability to preserve hearing [19]. Currently many surgeons use direct cochlear nerve monitoring, which is "real time" and allows the surgeon to stop dissection immediately when the amplitude or latency of the first wave diminishes [20]. At this point, there is not sufficient experience to be able to state definitely that this will permit us to preserve hearing in a higher percentage of cases than was previously possible, but data are being accumulated.

Following removal of tumor, the wound is copiously irrigated and bony debris and blood clot removed. Further hemostasis is obtained with bipolar cautery.

Any visible petrosal air cells that have been opened during the drill out of the internal auditory canal are then coated with bone wax, and a large piece of muscle taken from the edge of the wound is glued into the bony defect using fibrin glue made from the patient's own cryoprecipitate (see Fig. 7C).

The operation is then turned back over to the neurosurgical team, which removes the self-retaining retractor, controls any oozing from the cerebellum, performs a watertight closure of the

dura, inserts a metal screen or the original bone-plug into the craniectomy, and closes the incision in layers. The patient continues on perioperative antibiotics and steroids.

Pros and cons of the retrosigmoid approach

The retrosigmoid approach is the most versatile operation for acoustic neuroma surgery [10,11,13,14,21]. It can be used for any size tumor, from intracanalicular to 4 cm or more from the porus. In dealing with a larger tumor, the skin incision may be lengthened to allow a lower craniectomy, and the superior-inferior diameter of the craniectomy may be extended to 5 cm or more.

The wide exposure of the posterior fossa gives excellent visualization of the tumor bed and the cranial nerves ranging from V above to IX, X, and XI below. Posterior fossa vessels are also well visualized. The prone position as well as slight elevation of the head allow for the cerebellum to fall away from the field and they do away with the need for excision or even hard retraction. Intravenous mannitol and dexamethasone cause shrinkage of the brain and prevent postoperative swelling.

Criticism of the posterior fossa approach in the past has been aimed mainly at the seated position, the need for flexion of the head on the cervical spine, the long nuchal incision, and the alleged difficulty in identifying the facial nerve. The seated position is uncomfortable for the surgeon and exposes the patient to the danger of air embolism. For these reasons, we decided to abandon the use of the seated position in favor of the three quarters prone horizontal position, with the head tilted slightly up. We occasionally operate in the seated position when dealing with a heavyset patient with a short neck and large tumor. The most common site for intraoperative air embolus formation in the seated position is the transverse sinus. Because this forms the superior margin of the craniectomy, it is relatively easy to nick the sinus and allow air to enter the venous bloodstream. Other sites of potential leak are the sigmoid sinus, large veins in the neck, and venous lakes within the bone of the skull itself. To avoid these leaks, meticulous care is used in coagulating visible blood vessels, waxing bone, and using ointment-impregnated gauze around the sites of head-holding pins. As already noted, despite these precautions, air emboli sometimes occur, are detected by the Doppler monitor, and require prompt action to correct them. The advantages of the seated position are that blood, cerebrospinal fluid, and bony debris tend to flow out of the wound rather than remain in the subarachnoid space and that the surgeon has a panoramic view of the entire posterior fossa. It also allows visualization of both ends of the VIIth cranial nerve at approximately the same plane, as well as visualization of both the brain stem and the lateral end of the internal auditory canal by merely rotating the microscope about its long axis. Visualization of the lateral end of the internal auditory canal is easily accomplished, with less bony removal than in any other position. Surgeons have attempted to address these pros and cons of the traditional suboccipital approach in several ways, leading to the evolution of the retrosigmoid technique.

The shorter-angled incision eliminates trauma to the nuchal muscles, leads to less blood loss, and has virtually eliminated postoperative neck pain and headache. Postoperative nuchal pain and headache have variously been ascribed to the presence of bone debris in the subarachnoid space and the suboccipital incision itself. We find that the latter is more likely to be the case. There have been a small number of patients who complain of severe pain following their suboccipital surgery, some of whom required release of a trapped occipital nerve, others rehabilitation, and still others protracted use of analgesics or nonsteroidal anti-inflammatory drugs. With the shorter, curved incision, we have not seen any protracted postoperative pain.

The size of the craniectomy can be varied between 3 cm for intracanalicular or small tumors to more than 5 cm for a large tumor, without substantially altering the external incision.

Facial nerve monitoring has been shown to lessen significantly the risk of damage in posterior fossa monitoring and has become the standard among otologists. Both pressure transducer and EMG-based systems have been used, with the latter having gained wider acceptance [12,22].

Facial monitoring has significantly reduced the incidence of severe facial nerve weakness in our experience, from 14.5% to 3.6%, and is expected to yield a higher percentage of patients with normal facial function (currently 80%) postoperatively as our series grows [12]. In the case of a small tumor, with the facial nerve anatomy not significantly distorted, the monitor should not affect results at all, but in dealing with larger tumors, in which the facial nerve is distorted and displaced, the monitor is invaluable in identifying

the nerve, tracing its course, and preserving nerve integrity.

Although there has been some debate on the theoretical aspects of hearing preservation surgery, having to do with the possibility of incomplete tumor removal [23–25], the chance of tumor recurrence [26,27], and the fear of increasing complications as being the ''cost'' of hearing preservation surgery [28], to date these fears have not proved to be justified. The hearing goal should be to preserve hearing at or near the preoperative level. An occasional patient may note an improvement [9,29–31].

Although the retrosigmoid approach may be used for tumors of all sizes, regardless of hearing, it is not our custom to do so. There is a growing belief that the choice of approach should be dictated by size of tumor, level of hearing, patient's age and health, and experience and expertise of the surgical team. The middle fossa approach offers an equal likelihood of hearing preservation surgery for the intracanalicular and small extracanalicular tumor, with the advantages of a more limited craniotomy [21,27,32,33]. When it is not indicated to do the middle fossa approach in the older patient, or in the case of a larger tumor, the retrosigmoid approach is to be preferred.

When hearing is poor, the translabyrinthine approach is preferred, although we are more comfortable in using the retrosigmoid approach for tumors that are 3 cm or more from the porus. In a series of 445 cases, 277 were accomplished via the suboccipital-retrosigmoid approach and 173 via the translabyrinthine. Early in our series, six patients had two-stage operations (Table 1). In this series of 445 cases, 202 (45%) were tumors that were 2 cm or less from the porus, whereas 73 (16%) were intracanalicular tumors or protruded 1 cm or less from the porus, and 170 (39%) were larger than 2 cm (Table 2). Of these 445 patients, 183 (41%) had good hearing, as defined by a pure tone average of 50 dB or less, and speech discrimination of 50% or more, whereas 108 (24%) had excellent hearing, as defined by a pure tone average of 30 dB or less, and a speech discrimination score of 80% or more (Table 3).

Table 1
Approach in 445 cases

Suboccipital/retrosigmoid	277
Translabyrinthine	173
Middle fossa (2-Stage surgery)	1 (6)

Table 2
Tumor size

170 $\geq$ 2 cm EC	39%
202 $\leq$ 2 cm EC	45%
73 1C $<$ 1 cm EC	16%

EC, extracanalicular; IC, intracanalicular.

Because we were dealing with a relatively high percentage of patients with small tumors or good hearing, we chose the retrosigmoid or suboccipital approach more often than the translabyrinthine. In general, the mean tumor size was greater in the suboccipital group than the translabyrinthine.

Results

Normal facial nerve function should be the expectation in all but the largest tumors [34,35]. Facial nerve function was completely intact (House type I) in 80% of our group as a whole, whereas there was an incidence of facial paralysis of 3.6% in the monitored group, compared with 14.5% in the unmonitored. There were no fatalities in the translabyrinthine group, but three postoperative mortalities in the suboccipital group. All of these occurred before 1985, with none since then. Mortality from all acoustic neuroma surgery should not exceed 1% to 2%. Hearing preservation surgery was performed in the suboccipital-retrosigmoid approach in 96 cases, of which hearing was preserved in 43 cases (43.8%). Presently it seems not possible to preserve hearing more than 50% of the time, regardless of tumor size, although there are occasional reports of better results in small series of perhaps selected cases [14,15,17,19,20,26,27,30–32,36–42]. This failure to preserve hearing in more than half the cases, regardless of whether the surgery is performed via the posterior or middle cranial fossa approach, remains an enigma to the surgeon. A difficulty in comparing results of hearing preservation surgery is the variation in definitions of success, ranging from 50% speech discrimination down to 15% [36,39]. In theory, one would think that earlier diagnosis of small tumors, improved surgical

Table 3
Level of hearing

183 (41%)	PTA $\leq$ 50 dB, SDS $\geq$ 50%
108 (24%)	PTA $\leq$ 30 dB, SDS $\geq$ 80%

PTA, pure tone average; SDS, speech discrimination score.

techniques, greater experience, and ability to monitor hearing intraoperatively would all contribute to a greater yield of hearing preservation surgery. Unfortunately this has not been the experience. An example of this is the fact that in our series, we attempted to preserve hearing in 42 patients from 1974 to 1988 and were successful in 21 cases, for a yield of exactly 50%. In 1989 and 1990, with the advantages of gadolinium-enhanced MRI scanning and intraoperative monitoring, we were successful in only 22 of 54 cases, for a yield of 41%.

Multiple factors probably come into play to explain these disappointing data. Although, in general, smaller tumors will present with better hearing, this is not at all necessarily the case (Table 4). A small tumor may be densely adherent to the cochlear nerve (this may be particularly true of inferior vestibular nerve tumors), or the blood supply to the inner ear may traverse the body of a small tumor; intracanalicular tumors may extend sufficiently laterally to require opening into the inner ear and therefore destruction of hearing; and finally far field monitoring such as the auditory brain stem response is much too slow to reveal damage to the inner ear, blood supply, or the cochlear nerve in time for the surgeon to react. In addition, the fact that many surgeons are operating on patients with poorer hearing probably also is at least in part responsible for these discouraging results. Despite this, it is our strong belief that, in the absence of any increased risk to the patient, attempts at hearing preservation are justified if the hearing is potentially useful to the patient.

Cerebrospinal fluid leaks have been bothersome occurrences, following both retrosigmoid and translabyrinthine approaches [28]. Our overall incidence has been approximately 12%. Our routine is to use spinal drainage for 3 to 5 days at the first diagnosis of a leak and to obliterate the mastoid in the case of the leak that follows a retrosigmoid operation or to repack the mastoid following a translabyrinthine leak. This surgery has been required in 4% of our 455 cases.

Table 4
Hearing level by tumor size

Small tumor (<2 cm), poor hearing	61.9%
Larger tumor (>2 cm), good hearing	20.3%
Larger (>2 cm) tumor, excellent hearing	46.4%
Small (<2 cm) tumor, poor hearing	32.3%

Since we have been using fibrin glue to attach the muscle in the retrosigmoid transmeatal drill out as well as the fascia graft and fat in the translabyrinthine approach, our incidence has decreased to 5%.

The retrosigmoid-transmeatal approach to the posterior fossa is a very versatile one and can be used for tumors of every size regardless of hearing. The technique has evolved from the classic suboccipital approach, thereby lessening the potential danger to the patient while allowing good access to the tumor and possible hearing preservation and decreasing the incidence of postoperative pain.

We use this approach in the majority of cases but advocate both the translabyrinthine and the middle fossa approaches for patients whose level of hearing and tumor size make these approaches reasonable choices. We firmly believe that there is more than one approach to the posterior fossa and that the level of hearing, the tumor size, and the patient's general health, occupation, and age should dictate the choice [9,10,21].

Summary

The retrosigmoid technique has evolved from the traditional suboccipital operation and, when combined with removal of the posterior wall of the internal auditory canal, affords a wide exposure of the cerebellopontine angle. This approach may be used for acoustic neuromas of all sizes, from intracanalicular to more than 4 cm from the porus acusticus. Hearing preservation may be attempted and is generally successful in a substantial minority of cases. The facial nerve is readily visualized at the lateral end of the internal auditory canal and is at no greater risk than in the translabyrinthine operation. We use this approach for all hearing preservation surgery as well as for tumors of more than 3 cm, regardless of hearing.

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Translabyrinthine Approach for Acoustic Tumor Removal

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The translabyrinthine approach is the most direct route to the cerebellopontine angle. We believe that this approach offers many advantages for acoustic tumor removal. In this article, the indications and relative contraindications for the approach are outlined. The surgical technique is then detailed and illustrated.

Indications

Small tumors that extend no further than 5 mm into the cerebellopontine angle in patients with good hearing are usually approached via the middle fossa [1]. Larger tumors in which good hearing remains are approached via the retrosigmoid route. This route is ideal when the tumor arises more medially and is not impacted into the fundus of the internal auditory canal and does not expand the canal.

In general, the outlook for hearing preservation for acoustic tumors with greater than 2 cm extension into the cerebellopontine angle is very poor. These tumors and all tumors with poor hearing are removed via the translabyrinthine approach. There is no tumor too large to be approached via the translabyrinthine route. For large tumors, more bone removal is accomplished posterior to the sigmoid sinus to gain access.

Contraindications

The presence of chronic otitis media is a contraindication for the translabyrinthine approach. Perforations of the tympanic membrane should first be repaired, following which the translabyrinthine approach may be used when healing has occurred. In the case of a mastoid cavity, a total obliteration is first performed with blind sac closure of the external auditory canal. The translabyrinthine approach may then be performed when healing has occurred. A relative contraindication is a patient who has good hearing and a tumor amenable to a hearing conservation approach as already described.

Advantages

The translabyrinthine approach offers several advantages for acoustic tumor removal. It requires a minimum of cerebellar retraction. Exposure and dissection of the lateral end of the internal auditory canal ensures complete tumor removal from that area and allows positive identification of the facial nerve at a consistent anatomic location [2].

If the facial nerve is lost during acoustic tumor removal, the translabyrinthine approach offers the best opportunity for immediate repair by end-to-end anastomosis or interposition of a nerve graft [3].

There is a lower incidence of cerebrospinal fluid leaks with this approach compared with the retrosigmoid approach.

Finally and most importantly, this approach carries the lowest morbidity and mortality. The mortality rate for this approach is 0.4% for the last 2300 cases [4]. Experienced teams performing the retrosigmoid approach are reporting nearly equivalent mortality rates.

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Limitations

The obvious disadvantage of the translabyrinthine approach is the sacrifice of any residual hearing in the operated ear. The approach is therefore reserved for patients whose hearing is poor or for large tumors in which the possibility of hearing preservation is slight.

In the past, it has been said that the approach is limited to smaller tumors. As already stated, we have not found this to be true. There is no tumor too large to be approached translabyrinthine. In fact, we believe that there are advantages for large and giant tumors in that the approach puts one directly into the center of the tumor at its origin. Intracapsular removal of the tumor allows the capsule to be displaced toward the opening by surrounding brain structures.

Technique

Preparation for surgery

The patient is placed supine on the operating table with the head at the foot of the table. This allows the anesthesiologist, who is seated at the patient's feet, easy access to the controls for moving the table. The patient's head is turned toward the opposite side and maintained in a natural position without fixation. The surgeon is then seated at the patient's side. This position minimizes fatigue and allows stabilization of the arms and hands during the exacting microsurgical procedures (Fig. 1).

Instruments

Standard neurotologic instruments are used. One special instrument s used and is discussed later.

Anesthesia

General endotracheal anesthesia with inhalation agents is used. Muscle relaxants are used only for induction of anesthesia because intraoperative monitoring of facial nerve activity is routinely used. Prophylactic antibiotics or steroids are not routinely used. Occasionally with very large tumors these measures are employed. A nasogastric tube and Foley catheter are placed after the patient is asleep.

Operative technique

The suboccipital area, pinna, and ear canal are prepared with povidone-iodine (Betadine

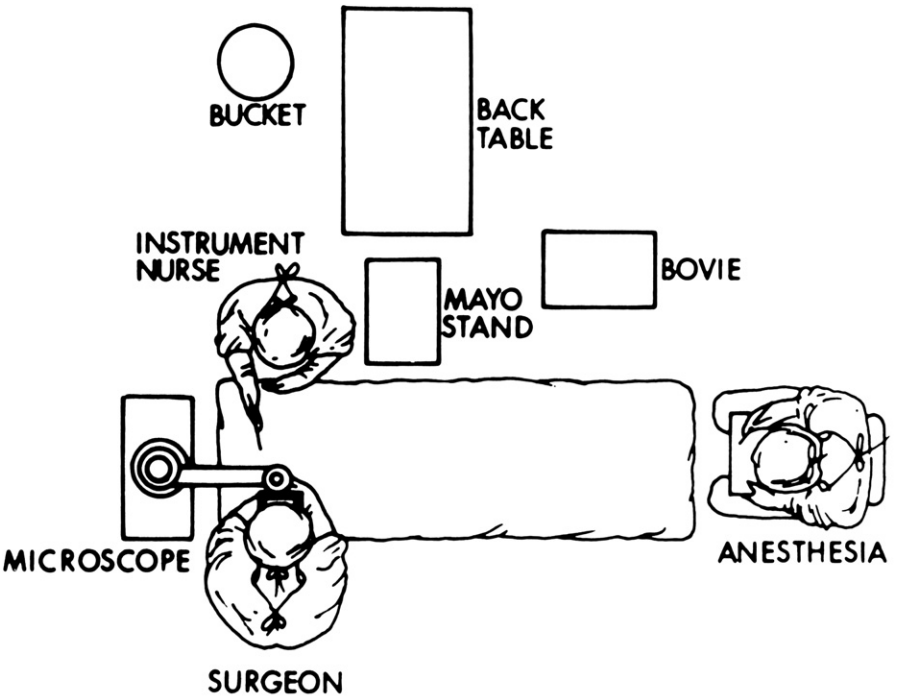

Fig. 1. Room arrangement for the translabyrinthine aproach. Note positions of surgeon, anesthesiologist, and nurse.

solution), and plastic drapes are applied. A postauricular incision is made approximately 2 cm behind the postauricular crease (Fig. 2). The incision is curved anteriorly to allow anterior retraction of the pinna. The posterior curve of the incision allows access to the area behind the sigmoid sinus. Because most of the surgical view of the cerebellopontine angle is along the plane of the posterior fossa dura, posterior access is important.

The incision first extends to the fascia temporalis, and the dissection is carried to the linea temporalis, lateral to the fascia temporalis. An incision is then made through the fascia and periosteum along the linea temporalis posteriorly to the sinodural angle and then inferiorly on the mastoid bone to the mastoid tip. The Lempert periosteal elevator is used to free the postauricular tissues from the underlying cortex, posterior to the sinodural angle and forward until the spine of Henle and the external auditory canal are identified. Care must be taken not to tear into the external auditory canal because this would introduce a possible route for infection. If this should occur, the patient is placed on prophylactic antibiotics, the defect into the external auditory canal is repaired, and the operation is continued.

Self-retaining retractors are placed to maintain the ear forward and to elevate the temporalis muscle superiorly. Suction on the posterior blade of the retractor removes excess irrigation fluid and blood from the wound.

Cortical mastoidectomy

After adequate exposure of the cortex has been obtained, bone removal is carried out with continuous suction-irrigation and a large cutting bur. Bone removal is started along the external auditory canal, and then a horizontal incision is made along the temporal line. The junction of these incisions lies over the mastoid antrum. Identification of the mastoid antrum and the lateral semicircular canal therein is the key to the beginning dissection of the temporal bone.

Bone removal continues with care taken not to undercut the mastoid cortex. The external opening must be as large as possible. The middle fossa plate is identified superiorly and the sigmoid sinus posteriorly. Removal of bone is then continued over the sigmoid sinus to the area of the posterior fossa dura. In large tumors, bone removal is carried out far behind the sigmoid sinus. In some cases, the bone is removed with a rongeur or drill as far as 2 or 3 cm posterior to the sigmoid sinus and inferiorly beneath the cerebellar hemisphere. This gives more decompression of the posterior fossa and allows room for retraction of the dura posteriorly. Care must be taken, however, not to injure the dura. Dural tears allow the cerebellum to herniate into the defect, which may result in infarction of that portion of the cerebellum.

Removal of bone over the sigmoid must be done carefully. If the cutting bur tears the sigmoid

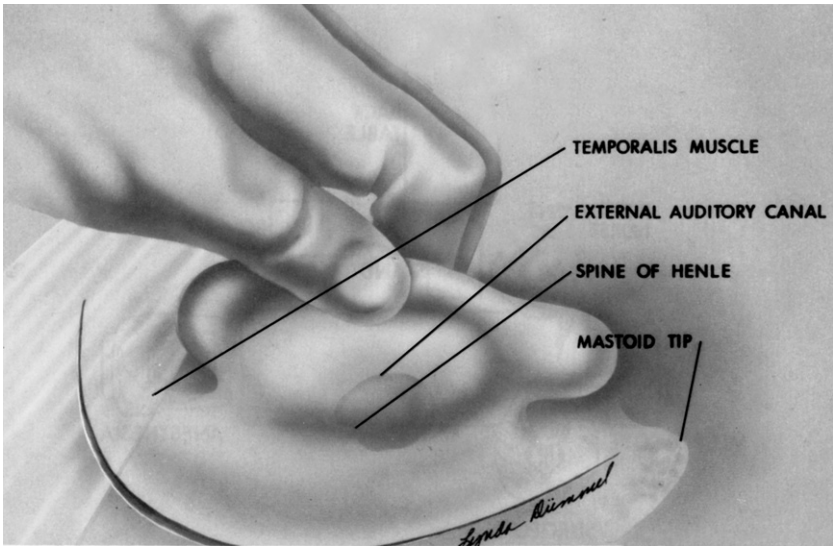

Fig. 2. Skin incision 2 cm behind the postauricular sarcous.

sinus, profuse bleeding ensues and requires packing with oxidized regenerated cellulose (Surgicel). Large emissary veins often arise from the posterior aspect of the sigmoid sinus. They can be identified through the bone as it is removed, since suction-irrigation keeps the bone clean. If the emissary vein is injured, bleeding must be controlled with bone wax, cautery, Surgicel packing, or in some cases suture of the emissary.

Complete, simple mastoidectomy

As soon as the mastoid cortex has been removed and the sigmoid sinus has been outlined, the operating microscope is brought into place. Magnification allows more accurate bone removal and exposure of all the structures of the temporal bone. A thin layer of bone is left over the sigmoid sinus and around the emissary veins, and a complete, simple mastoidectomy is performed down to the level of the horizontal semicircular canal (Fig. 3). It is important that the antrum be opened and the horizontal semicircular canal be identified. This canal is the basic landmark in temporal bone surgery. Once the position of this canal is known, the depth and three-dimensional relationship of the facial nerve and posterior and superior semicircular canals can be viewed. Expertise in temporal bone surgery depends on a thorough knowledge of the anatomy of the temporal bone and the ability to identify the structures as they are encountered. This appreciation of the anatomy comes only after many hours of diligent temporal bone dissection.

Labyrinthectomy

After the mastoid air cells have been removed to the level of the horizontal semicircular canal, labyrinthectomy is begun. Bone is removed in the sinodural angle along the superior petrosal sinus. This area, which is farthest from the facial nerve, is the key to this step in the dissection. The opening along the superior petrosal sinus is gradually deepened and widened until the labyrinthine bone is encountered. The lateral and posterior semicircular canals are then progressively removed, and the facial nerve, which lies anteriorly, is carefully approached (Fig. 4). The lateral semicircular canal is opened, and the common crus of the superior and posterior semicircular canals is identified deep in the dissection. The superior semicircular canal is followed to its ampulla. The vestibule is then opened, and the facial nerve is skeletonized from the genu inferiorly to near the stylomastoid foramen. It is not necessary to remove bone lateral to the facial nerve; rather, the facial nerve is skeletonized from a posterior direction, where access is needed to approach the cerebellopontine angle.

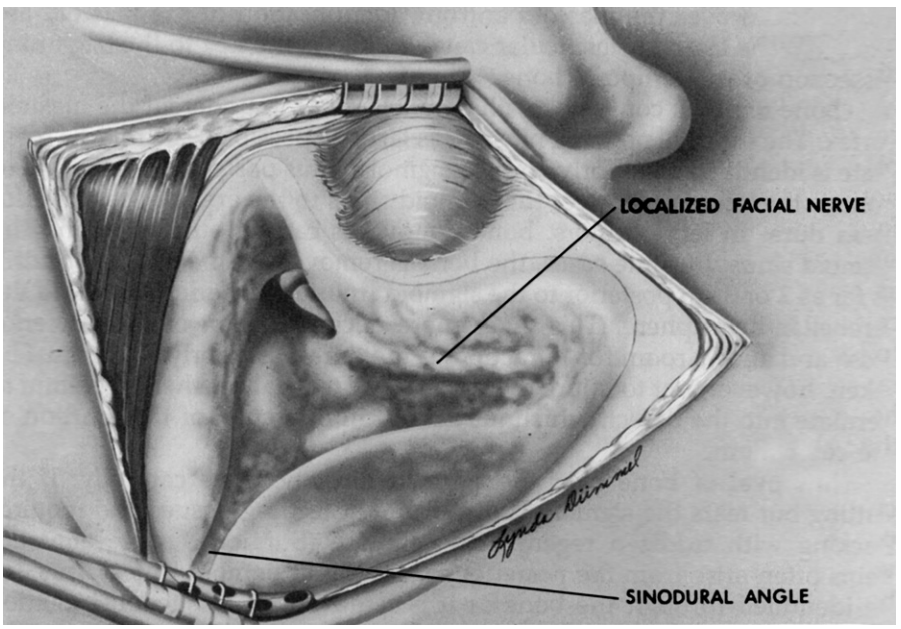

Fig. 3. Mastoidectomy is completed. Facial nerve is localized and sigmoid sinus skeletonized.

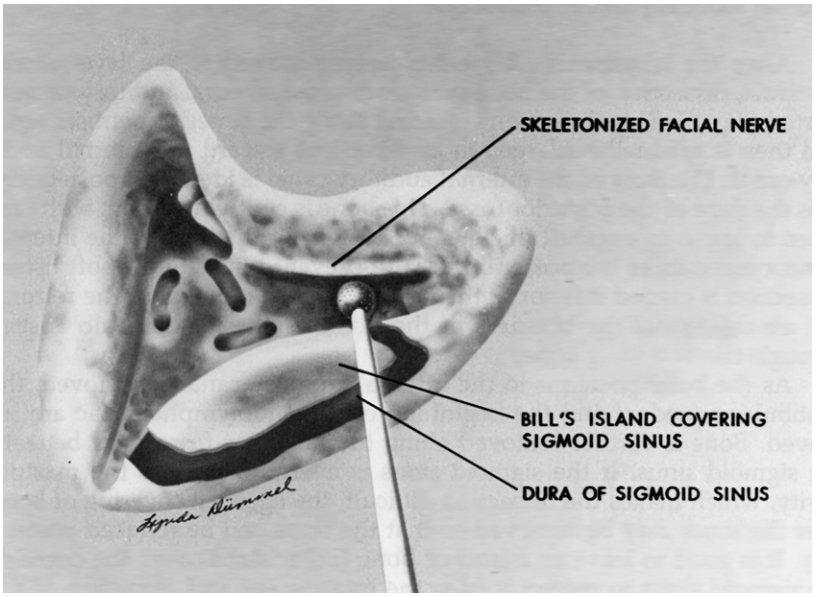

Fig. 4. Semicircular canals are opened. An island of bone over the sigmoid sinus is created.

The final removal of bone along the facial nerve is accomplished with a diamond bur. Having removed the labyrinthine bone from posterior to the nerve, the surgeon may then use the side of the diamond bur rather than the end and at all times view the plane between the side of the bur and the facial nerve. This reduces the hazard of injury to the facial nerve, which is very slight with this technique. As the facial nerve is skeletonized, the cribriform area of the superior vestibular nerve entering the vestibule is seen. It is important to skeletonize the facial nerve adequately so that the vestibule can be seen in this area (Fig. 5).

Internal auditory canal dissection

After the labyrinthine bone has been removed to the level of the vestibule, dissection of the bone surrounding the internal auditory canal is started (Fig. 6). This dissection is started along the superior petrosal sinus and then is gradually enlarged in all directions toward the internal auditory canal. The dura of the internal auditory canal is identified posteriorly, as is the dura of the posterior fossa. This bone is gently removed, with care taken to leave an eggshell thickness of bone over the dura of the internal auditory canal and the posterior fossa to prevent injury to the soft tissue. Dissection is carried inferior to the labyrinth, with removal of the retrofacial air cells, until the blueness of the dome of the jugular bulb is seen through the overlying bone.

As the bone posterior to the internal auditory canal is removed, the vestibular aqueduct and the beginning of the endolymphatic sac are removed. Bone is further removed along the posterior fossa dura beneath the sigmoid sinus. If the sigmoid sinus is overhanging into the mastoid cavity, which makes the dissection difficult, the eggshell covering of bone over the sinus may be removed so that the sinus can be retraced posteriorly. It is good to leave an island of bone (Bill's island) over the dome of the sigmoid sinus to protect it from the rotating bur and retraction of the suction-irrigation at this point.

We complete the dissection around the inferior portion of the internal auditory canal first. This is the area that is farthest from the facial nerve, and we find that completing the dissection here makes orientation to the superior portion of the internal auditory canal easier. Bone removal is continued medially and anteriorly between the dome of the jugular bulb and the internal auditory canal until the cochlear aqueduct is identified.

The cochlear aqueduct is not always readily identifiable. In large tumors, it is occluded at its

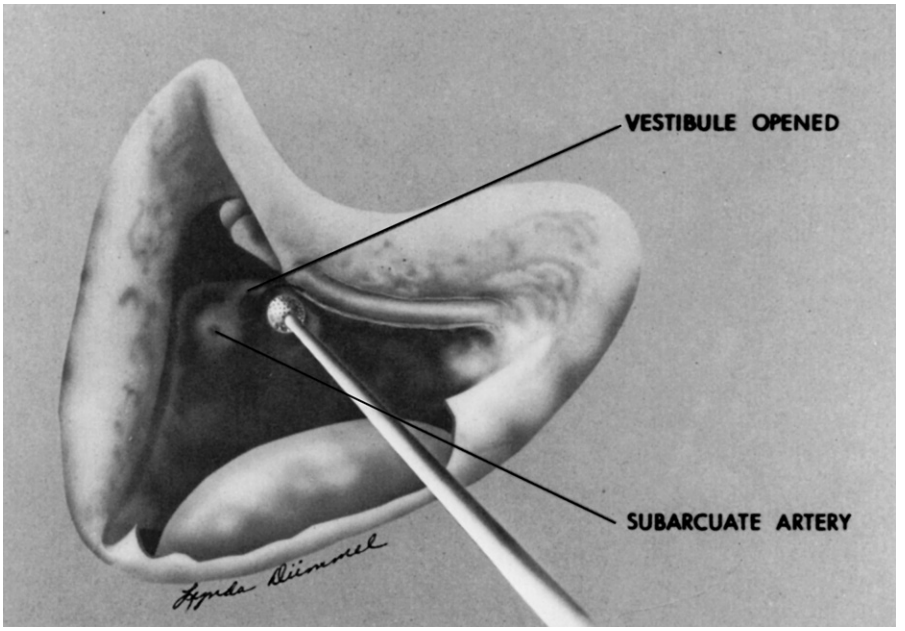

Fig. 5. Lateral and posterior semicircular canals are removed. The vestibule is opened and the facial nerve is skeletonized in its tympanic segment.

medial orifice, and spinal fluid is not likely to escape. The cochlear aqueduct enters the posterior fossa directly inferior to the midportion of the internal auditory canal above the jugular bulb. It is an important landmark because it identifies the location of the IXth, Xth, and XIth cranial nerves in the neural compartment of the jugular foramen anterior to the jugular bulb. If the dissection is

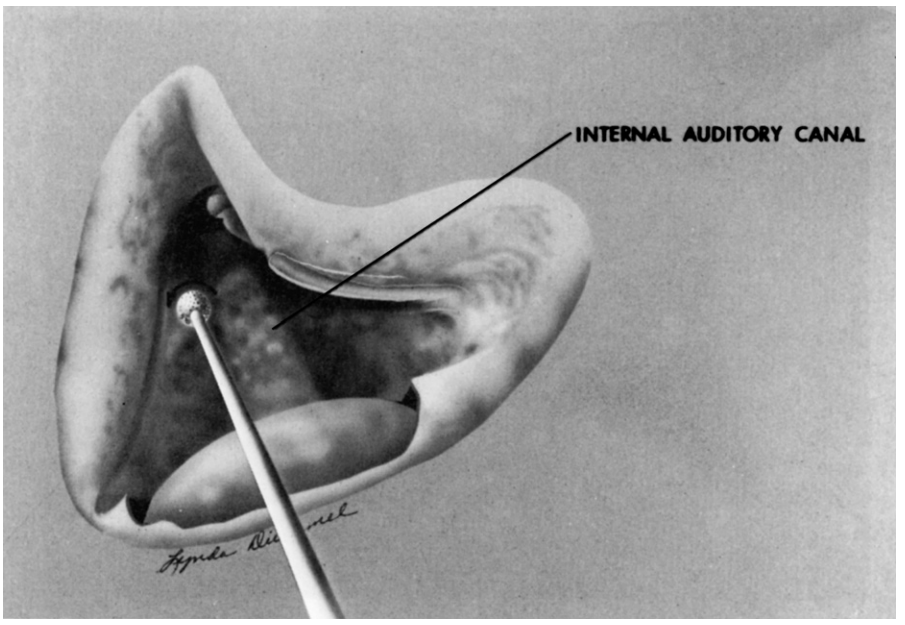

Fig. 6. The internal auditory canal is skeletonized. Note rotation of direction of bur.

confined to the area superior to the cochlear aqueduct, these nerves are not injured.

After the cochlear aqueduct has been identified, bone removal is continued around the internal auditory canal to the porus acusticus until the entire posterior lip of the internal auditory canal is removed. The diamond bur is used for these later parts of the dissection. The bone of the posterior fossa dura is then removed inferiorly until the sigmoid sinus is skeletonized. This completes the dissection inferiorly.

Dissection is then carried superiorly and anteriorly around the inter nal auditory canal. This bone removal is tedious because the facial nerve often underlies the dura along the anterior-superior aspect of the internal auditory canal. The surgeon must be very careful not to allow the bur to slip into the internal auditory canal. We prefer to remove the entire porus and the medial portion of the internal auditory canal first, leaving the dissection of the lateral end of the internal auditory canal until last. In this way the facial nerve is not exposed until most of the bone removal is completed.

Removal of the superior lip of the porus acusticus is tedious, but it is one of the most important parts of the dissection. If this is not entirely removed, the facial nerve underlies the ridge of bone at the porus and makes identification and removal of the tumor from the nerve in this area very difficult. Diamond burs are used to continue the dissection until two thirds of the porus acusticus is removed. Bone removal is then carried laterally, and the end of the internal auditory canal is exposed.

Dissection of the lateral end of the internal auditory canal begins inferiorly. The singular nerve is first identified, and bone removal done inferiorly then exposes the inferior vestibular nerve. As dissection proceeds superiorly, the transverse crest is identified. The superior aspect of the internal auditory canal is then dissected, and the facial nerve is identified as it exits the internal auditory canal and begins its labyrinthine segment. Finally the bar of bone (*Bill's bar*) separating the superior vestibular nerve from the facial nerve is identified. This completes the dissection around the internal auditory canal.

During the dissection of the internal auditory canal, an eggshell thickness of bone was left on the sigmoid sinus and the posterior and middle fossa dura. At this stage, this is removed completely, and the surgeon is ready to open the posterior fossa dura to expose the cerebellopontine angle. It is noteworthy that until this point all of the dissection has been extradural and the morbidity of the approach has been minimal.

Dural incision

The dura of the posterior fossa is then incised over the midportion of the internal auditory canal (Fig. 7). The incision then extends around the porus acusticus superiorly and inferiorly. Care is taken to avoid vessels on the surface of the tumor, and anteriorly-superiorly care is exercised to avoid injury to the facial nerve, which lies directly beneath the dura in this area. Posteriorly the petrosal vein lies just beneath the dura, and insertion of a Rosen elevator separates underlying blood vessels from the dura before incision. Keeping the deep blade of the scissors just beneath the dura prevents injury to underlying blood vessels as the incision progresses. The dural flaps are then retracted superiorly and inferiorly and Cottonoids are placed between the tumor and the cerebellum posteriorly.

An arachnoid cyst is often encountered around the posterior aspect of the tumor. The cyst is opened, and the plane of the tumor and cerebellum is further developed around the posterior aspects of the tumor. Cottonoids are advanced into this plane. It is extremely important to develop this plane accurately because doing so separates the major vessels of the cerebellopontine angle from the tumor. The operating microscope makes it possible to follow this proper plane and to a large extent has eliminated the major bleeding often associated with removal of cerebellopontine angle tumors.

As the dura is retracted posteriorly, it lies over the petrosal vein, which originates in the cerebellum and drains into the superior petrosal sinus near the level of the internal auditory canal. At times this vein is torn near its entry into the superior petrosal sinus as it is retracted posteriorly.

Bleeding from the proximal portion of the vein can be controlled by a clip. Bleeding from the superior petrosal sinus, however, is often much more difficult to manage. One means of controlling this bleeding is to fill the superior petrosal sinus with Surgicel. Another technique is to pack Surgicel extradurally over the petrous ridge at the anterior limit of the dissection. This produces extradural compression of the superior petrosal sinus and thus controls proximal bleeding. Distal back-bleeding from the sinus is controlled by

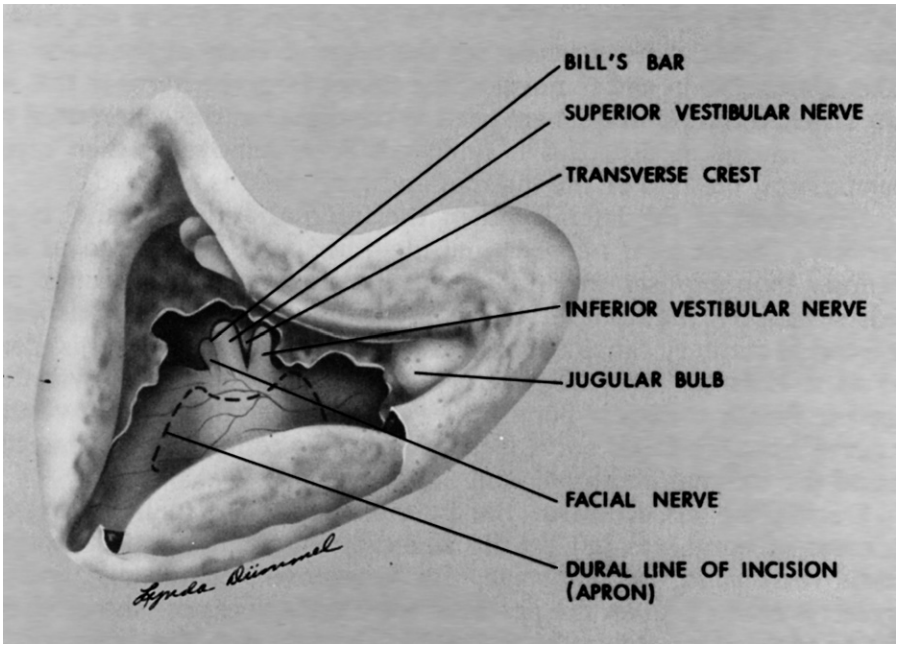

Fig. 7. Dural incision is outlined by the dashed line. An alternative method is to make a single incision over the mid portion of the internal auditory canal and then around the porus acusticus.

placing a clip on the sinus between the sinodural angle, where it enters the sigmoid sinus, and the petrosal vein.

Partial tumor temoval

In the case of a small tumor, the surgeon can begin development of the inferior and superior planes of the tumor. With a medium or large tumor, it is better to begin intracapsular removal of the tumor to reduce its size before developing the other planes.

The posterior surface of the tumor is first carefully inspected for nerve bundles. On rare occasions, the facial nerve may lie on the posterior surface of a tumor. After it has been determined that no nerve bundles are present on the posterior surface of the tumor, the capsule of the tumor is incised (Fig. 8), and intracapsular removal of the tumor is begun with the House-Urban dissector (Fig. 9). During intracapsular removal of the tumor, it is important to avoid excessive movement and pressure on the tumor because this may stretch and injure the facial nerve.

Isolating the tumor

Once the interior of the tumor has been extensively gutted, the development of the tumor plane is carried out further inferiorly and superiorly (Fig. 10). Small Cottonoids are used to develop the plane of the tumor and to separate surrounding structures. Avoidance of injury to surrounding structures is greatly facilitated by use of the fenestrated neurotologic suction tip [5]. Because the tumor has been extensively gutted, the capsule is displaced into the interior of the tumor. The surface of the capsule is then followed to the brain stem. We attempt to develop the posterior aspect of the tumor to the point where it can be seen at the brain stem, and Cottonoids are placed into this plane.

Inferiorly an attempt is made to localize the IXth cranial nerve, which can be best identified near its exit medial to the jugular bulb. In larger tumors, the IXth cranial nerve may be stretched over the surface of the tumor. This plane is carefully developed, and the IXth cranial nerve is isolated from the field with Cottonoids. During manipulation of the Vth, IXth, and Xth cranial nerves, changes in the pulse rate often occur. If these occur, we stop manipulation of the nerves and allow the vital signs to stabilize.

Often large vessels are located around the inferior aspect of the tumor, and these must be carefully separated from the tumor capsule and preserved. After the inferior aspect of the tumor

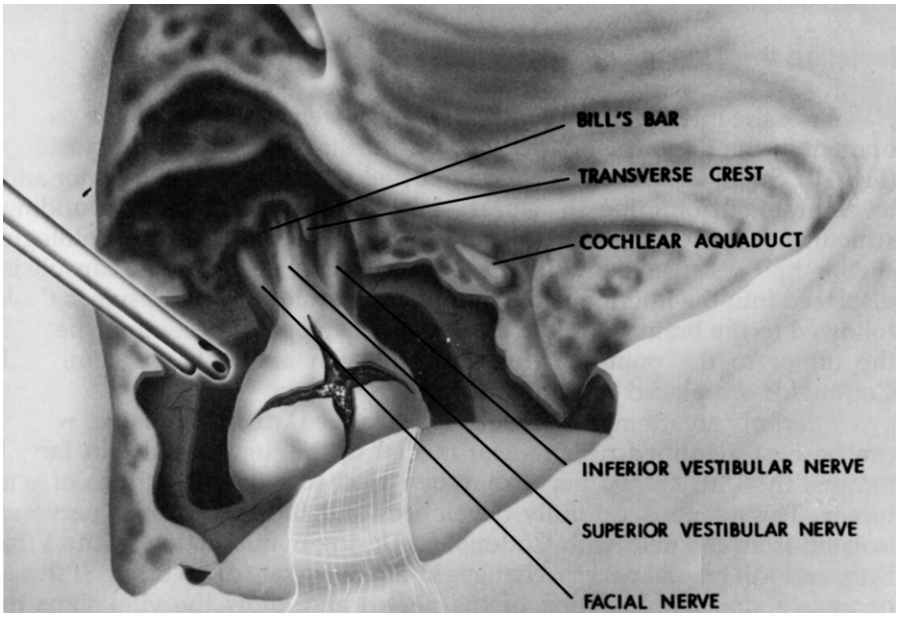

Fig. 8. The tumor capsule has been incised. Note the multiple openings in the fenestrated neurotologic suction irrigator.

has been developed down to the brain stem, additional debulking of the tumor and removal of a portion of the capsule can be completed.

The superior aspect of the tumor capsule is next developed. The petrosal vein is encountered in this location and must be carefully separated from the tumor. The facial nerve usually lies more anteriorly, but it is not unusual for it to come over the top of the tumor. The Vth cranial nerve is identified at the medial superior aspect of the

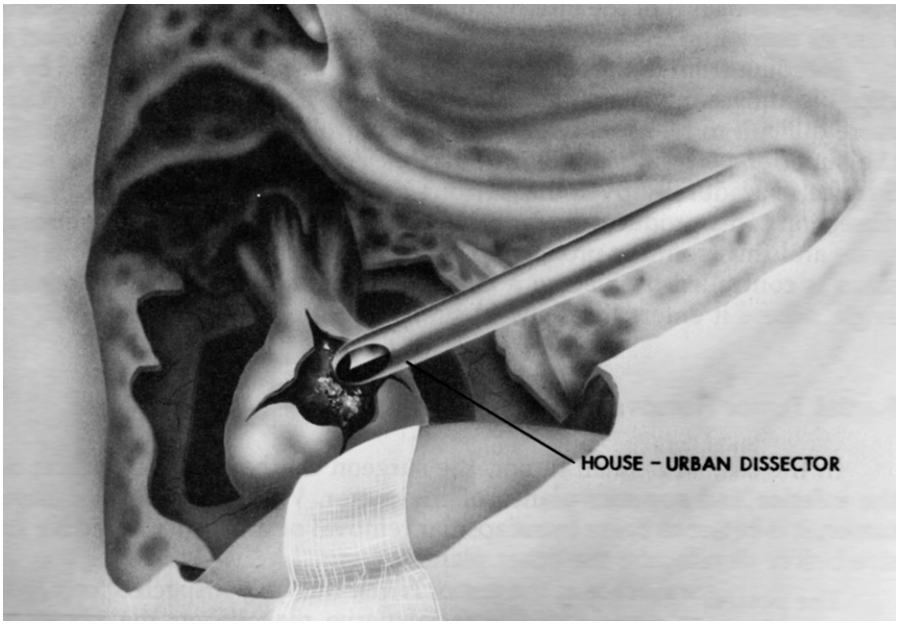

Fig. 9. The House-Urban rotating vacuum dissector begins gutting the tumor within its capsule.

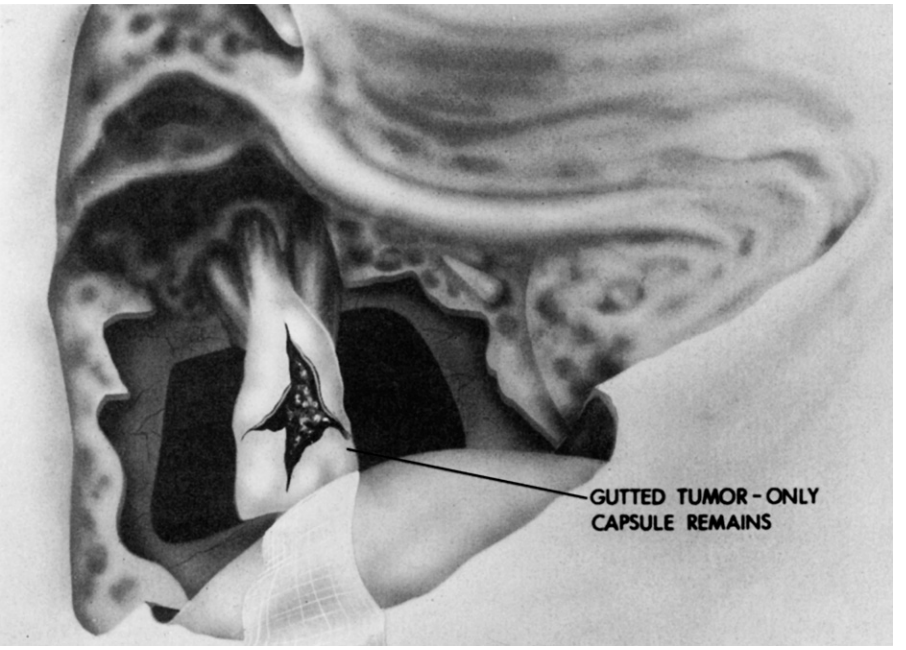

Fig. 10. The tumor is markedly reduced in size. Only the capsule remains.

tumor, and all these structures are carefully separated from the capsule and packed away from the field with Cottonoids.

Identification of the facial nerve

The lateral end of the internal auditory canal is dissected, and the plane of the facial nerve is established by uncovering the labyrinthine segment of the facial nerve. During bone removal, the vertical crest of bone (Bill's bar) separating the facial nerve from the superior vestibular nerve has been clearly identified. A long fine hook is then inserted lateral to Bill's bar to identify the superior vestibular nerve. The hook is gently passed medially and slightly anteriorly until it falls over Bill's bar into the facial nerve canal, which positively identifies the facial nerve. The hook is then withdrawn and placed beneath the superior vestibular nerve, turned inferiorly, and the superior vestibular nerve is pulled out from its canal (Fig. 11). At this point, the underlying facial nerve is seen, and the plane of the facial nerve from the tumor is definitely identified. Positive identification of the facial nerve at the lateral end of the internal auditory canal is one of the principal advantages of the translabyrinthine approach. Continuous intraoperative facial nerve monitoring is routinely used.

Next the hook is used to remove the inferior vestibular nerve, and the dura of the internal auditory canal is opened along the inferior aspect of the tumor. The dura is also opened superiorly, with great care taken to avoid the facial nerve. Incision of the dura of the internal auditory canal frees the tumor so that it can be gently retracted posteriorly away from the facial nerve. The Rosen separator and hooks are used to develop the plane carefully between the facial nerve and the tumor. The tumor is gently retracted posteriorly to bring this plane into relief. After the lateral end of the facial nerve has been definitely identified and separated from the tumor, all tumor remnants are removed from the lateral end of the internal auditory canal. The cochlear nerve is usually removed along with the tumor and the vestibular nerve (Fig. 12).

Facial nerve dissection

Usually it is relatively easy to develop the plane along the facial nerve within the internal auditory canal, but considerable difficulty often arises when the porus acusticus is reached. Dural adhesions to the surface of the tumor at the porus acusticus invariably make dissection of the facial nerve from the tumor very difficult in this area. The facial nerve usually can be followed past the area

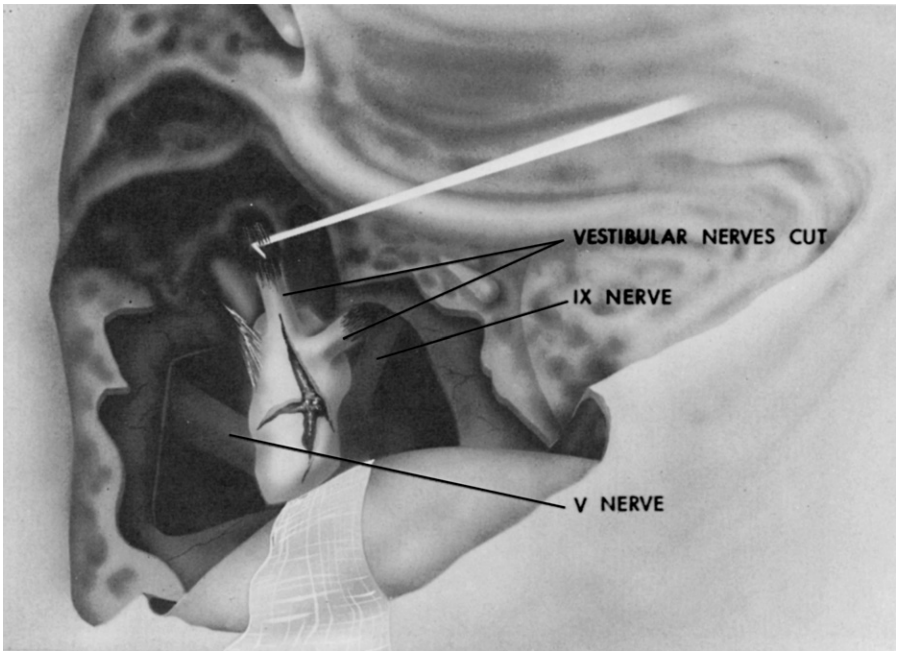

Fig. 11. A sharp hook pulls the superior vestibular nerve from the lateral end of the internal auditory canal. The inferior vestibular and cochlear nerves are also removed. Note the Vth nerve and IXth nerve in the posterior fossa.

of adhesions without undue difficulty. At times, however, this plane becomes very difficult, and we rotate the tumor posteriorly to identify the facial nerve on the tumor medially nearer the brain stem. The facial nerve is then followed medially to laterally until the plane becomes apparent, and tumor removal can be completed at the porus acusticus. During this entire dissection, the

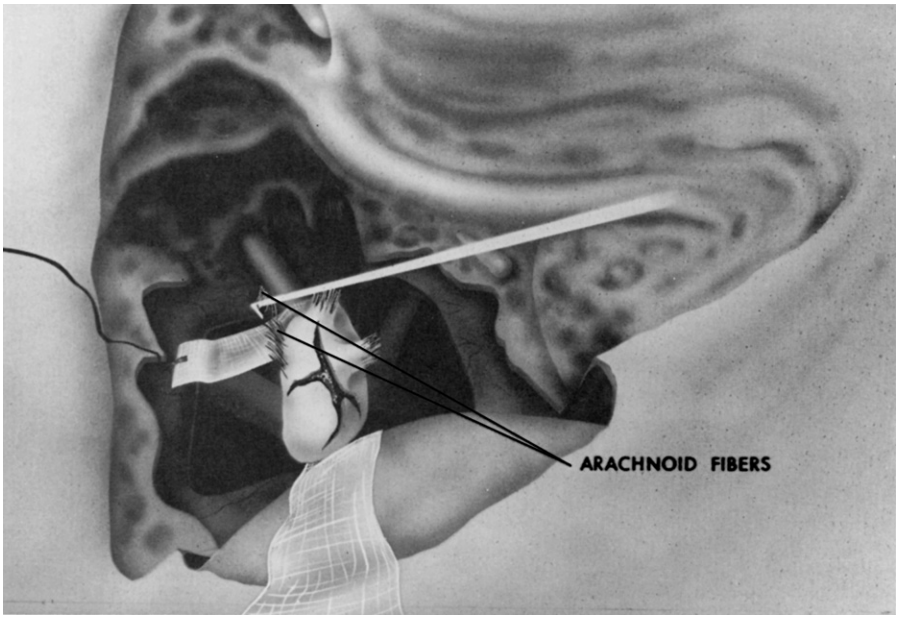

Fig. 12. The tumor is progressively freed from the facial nerve with sharp dissection.

surgeon must be careful not to push the tumor forward or medially, which would stretch the facial nerve. It is better to retract the tumor gently posteriorly and laterally, removing the stretch from the facial nerve.

Completion of tumor removal

Once the facial nerve has been separated from the tumor to the brain stem, the bulk of the tumor is removed with the House-Urban dissector, leaving only a small portion of tumor attached to the brain stem (Fig. 13). Removing the bulk of the tumor allows greater visibility of the tumor–brain stem plane. The last bit of tumor is then removed from the brain stem under direct vision. The adhesions between the tumor and the brain stem are usually not dense and can be easily separated. Bleeding in this area is controlled with bipolar cautery. Only those vessels that actually enter the tumor capsule are coagulated; the others are carefully freed from the tumor capsule. Often a small artery accompanies the VIIIth cranial nerve into the tumor. Bleeding from this artery is controlled by placing a clip on the VIIIth cranial nerve and contained artery or by bipolar coagulation.

Hemostasis and closure

After total tumor removal, the wound is profusely irrigated with Ringer's solution to remove any blood clots. The Cottonoids are then removed, and all bleeding points are controlled with either clips or bipolar cautery. Absolute hemostasis must be obtained, and this may require considerable time and effort. It is best to control bleeding with clipping or cautery rather than Surgicel packing. Using large amounts of Surgicel must be avoided because this substance expands considerably with fluid absorption and can consequently cause pressure on surrounding vessels or the brain stem.

After hemostasis is complete, the dura is sutured and the area of the mastoidectomy obliterated with strips of fat taken from the abdomen. A small piece of muscle is used to obliterate the aditus. The mastoid cavity is then filled to the surface of the cortex with strips of abdominal fat, and the postauricular incision is closed in layers. The skin is closed with interrupted subcuticular sutures. The patient is kept on the operating table until responding well and then is transferred directly to the intensive care unit.

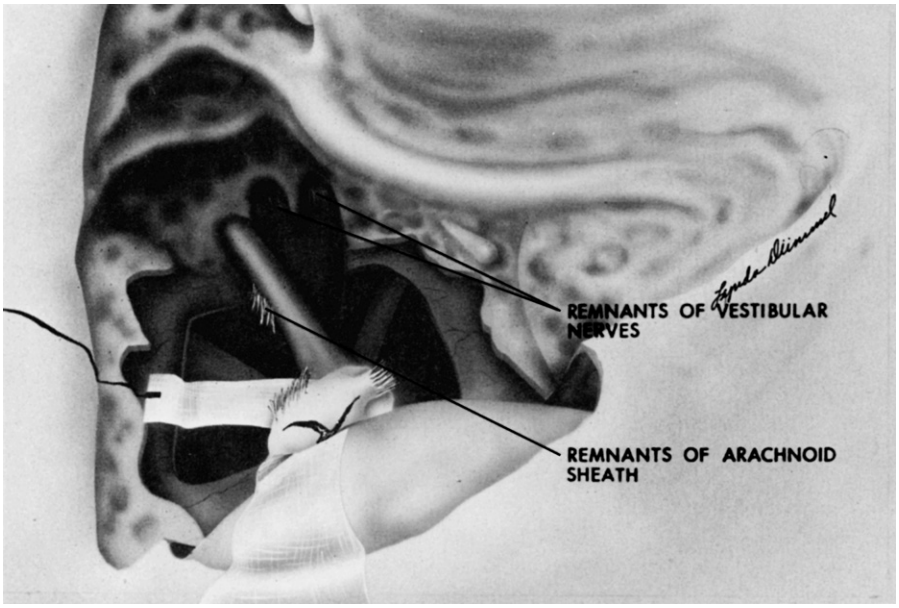

Fig. 13. The tumor has been freed from the facial nerve. It is now carefully dissected from the brain stem, completing tumor removal.

Perioperative management

The patient is observed in the intensive care unit for a period of 36 hours postoperatively. Steroids or antibiotics are not routinely used. In some patients with very large tumors that exhibit signs of cerebellar swelling, we do use steroids in the immediate postoperative period. Osmotic diuretics, fluid restriction, or lumbar drainage is not routinely employed. A standard mastoid dressing remains in place for 4 days, and the patient is instructed not to lift or strain during the early postoperative period.

We encourage early ambulation, and the patient is walking by the 2nd or 3rd postoperative day. It is rare for patients to require postoperative physical therapy, although this may be used for patients with unusual degrees of unsteadiness or ataxia.

Complications and management

Although rare, the most common early postoperative complication is a hematoma in the cerebellopontine angle. This is manifested by signs of cerebellopontine angle pressure and is managed by immediate opening of the wound and removal of the fat in the intensive care unit. This is a further advantage of the translabyrinthine approach in that the angle may be rapidly decompressed for this uncommon complication. The patient is then taken to surgery, where complete hemostasis is obtained and the wound again closed with abdominal fat.

Meningitis is an uncommon complication and is managed in the usual manner with appropriate antibiotics following culture and identification of the offending organism.

Postoperative cerebrospinal fluid leakage is an unusual complication of the translabyrinthine approach. Rarely these leaks occur through the postauricular incision. These may be controlled by resuturing the wound with an interlocking stitch and reapplication of the mastoid dressing. An occasional patient may have cerebrospinal fluid rhinorrhea after the mastoid dressing is removed. We first reapply a pressure dressing. If that is ineffective, a lumbar drain may be inserted for 3 or 4 days and the patient placed on bed rest. If these conservative measures fail, the wound is reopened and the fat is repositioned. The usual source of leakage is at the anterior margin of the labyrinthine bone removal, and additional fat is placed in this area.

If these measures fail to control the leak, another approach is used. The ear canal is transected and the skin of the meatus everted and closed. The mastoid periosteum is freed and sutured anteriorly to reinforce the meatal closure. The skin of the ear canal is removed completely along with the tympanic membrane, malleus, and incus. The eustachian tube is then firmly packed under direct vision with Surgicel and temporalis muscle. The middle ear is filled with muscle and the postauricular incision closed. A firm pressure dressing is applied and left in place for 3 to 4 days. In our experience, this technique has always been successful in closing cerebrospinal fluid leaks.

If facial weakness occurs, eye care is provided by a consulting ophthalmologist. Conservative measures are first used, including artificial tears, moisture chambers, and soft contact lens. If these measures fail to provide adequate corneal protection, a palpebral spring is inserted. The latter procedure is more commonly necessary when there is a concomitant Vth cranial nerve deficit with corneal anesthesia.

One of the advantages of the translabyrinthine approach, particularly compared with the retrosigmoid approach, is the reduced incidence of postoperative headache. It is unusual for headache to persist beyond the immediate postoperative period.

Because there is little cerebellar retraction, cerebellar dysfunction is unusual with the translabyrinthine approach.

Discussion of series

The House Ear Clinic series numbers over 2700 acoustic tumors as of October 1989. The results in the first 700 patients have been previously published [4]. With experience and refinements in technique, the results have improved throughout the years. Most recently we have studied the results in 216 patients who underwent surgery for acoustic tumors in 1980 and 1981 [2]. The average age of these patients was 47.3 years, and the group was equally divided into male and female.

Tumor size, measured on cranial computed tomography, is reported as extension into the cerebellopontine angle. Small tumors are those that extend less than 0.5 cm into the cerebellopontine angle; 20 of the 216 (9%) fit this category. Medium tumors, which are considered those that extend from 0.5 to 2 cm into the cerebellopontine angle, numbered 110 (51%). Large tumors are those extending from 2 to 4 cm into the

cerebellopontine angle; this group included 67 (31%) of the tumors. Tumors that extended more than 4 cm into the cerebellopontine angle are considered giant tumors; 19 patients (9%) had tumors of this size.

The average length of surgery was 3 hours and 12 minutes. There was one death in this series (0.4%). The patient had a postoperative hemorrhage and, despite early evacuation of the clot, sustained brain stem infarction and died.

Facial nerve function was studied 1 year after surgery. At that time, 180 patients (83%) had normal facial function. There was a partial paralysis in 34 patients (16%). In four of these patients, the facial nerve had been divided during tumor removal; they underwent immediate facial anastomosis in the cerebellopontine angle and had satisfactory recovery of facial function. Two patients had total facial paralysis 1 year after surgery; they then underwent hypoglossal facial anastomosis and had satisfactory recovery of facial function.

There was a direct correlation between preservation of normal facial nerve function and size of tumor. All 20 patients with small tumors had normal facial function at 1 year. Of the patients with medium-sized tumors, 85% had normal facial function. In the group with large tumors, 81% of the patients had normal facial function. Of those patients with giant tumors, 63% had normal facial function 1 year after surgery. Despite this correlation, it must be noted that some patients with relatively small tumors may have invasion of the facial nerve and thus incomplete recovery. Therefore the surgeon must be careful not to be overly optimistic in patient discussion even when the tumor is a small one.

Summary

The translabyrinthine approach is the preferred method for removal of all sizes of acoustic tumors when there is nonserviceable hearing.

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Transotic Approach to the Cerebellopontine Angle

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The development by House in the early 1960s of the translabyrinthine approach to lesions in the cerebellopontine angle revolutionized their treatment. Until the late 1970s, this was the primary approach used in the University of Zurich Department of Otolaryngology for the treatment of acoustic neuromas. At that time, an extension in the degree of temporal bone dissection was introduced to provide improved facial nerve identification and tumor visualization in larger lesions [1]. This new approach featured the complete exenteration of all pneumatic cell tracts of the temporal bone, total removal of the otic capsule with transposition of the facial nerve, and obliteratior of the middle ear cleft. The procedure was described as the *transotic approach* to distinguish it more clearly from the less extensive transcochlear approach introduced by House et al for the resection of skull base tumors [2].

The original transotic approach eliminated many exposure problems encountered with the translabyrinthine procedure, yet introduced a temporary postoperative facial paralysis from transposition of the facial nerve. To address this problem, a modification was developed that involved preservation of the facial nerve within the fallopian canal, thereby eliminating the temporary paralysis of permanent transposition while maintaining improved exposure [3,4]. As a result of the success of this procedure for the removal of larger tumors, the transotic approach has replaced the translabyrinthine approach in the treatment of virtually all acoustic neuromas treated in the Department of Otolaryngology. The purpose of this article is to present a detailed summary of the advantages, disadvantages, and technical aspects of this method as it relates to the surgical therapy of acoustic neuromas.

Surgical technique

The principal objective of the transotic approach is the direct lateral exposure of the cerebellopontine angle via the medial wall of the temporal bone. No cerebellar retraction is required, with the limits of dissection along the medial wall extending from the superior petrosal sinus to the jugular bulb and from the internal carotid artery to the sigmoid sinus. The facial nerve is left undisturbed within the tympanic and mastoid segments of the fallopian canal. The component steps in the transotic approach consist of (1) a subtotal petrosectomy with preservation of the tympanic and mastoid segments of the fallopian canal, (2) total removal of the otic capsule with wide exposure of posterior fossa dura along the medial temporal bone, (3) tumor removal with maximal facial nerve exposure, and (4) dural reconstruction with cavity obliteration.

Subtotal petrosectomy

The foundation of the transotic approach is the successful completion of a subtotal petrosectomy before advancing to otic capsule removal and tumor exposure (Figs. 1, 2, and 3). This involves the obliteration of the eustachian tube isthmus, closure of the external auditory canal, and complete exenteration of all air cell tracts to

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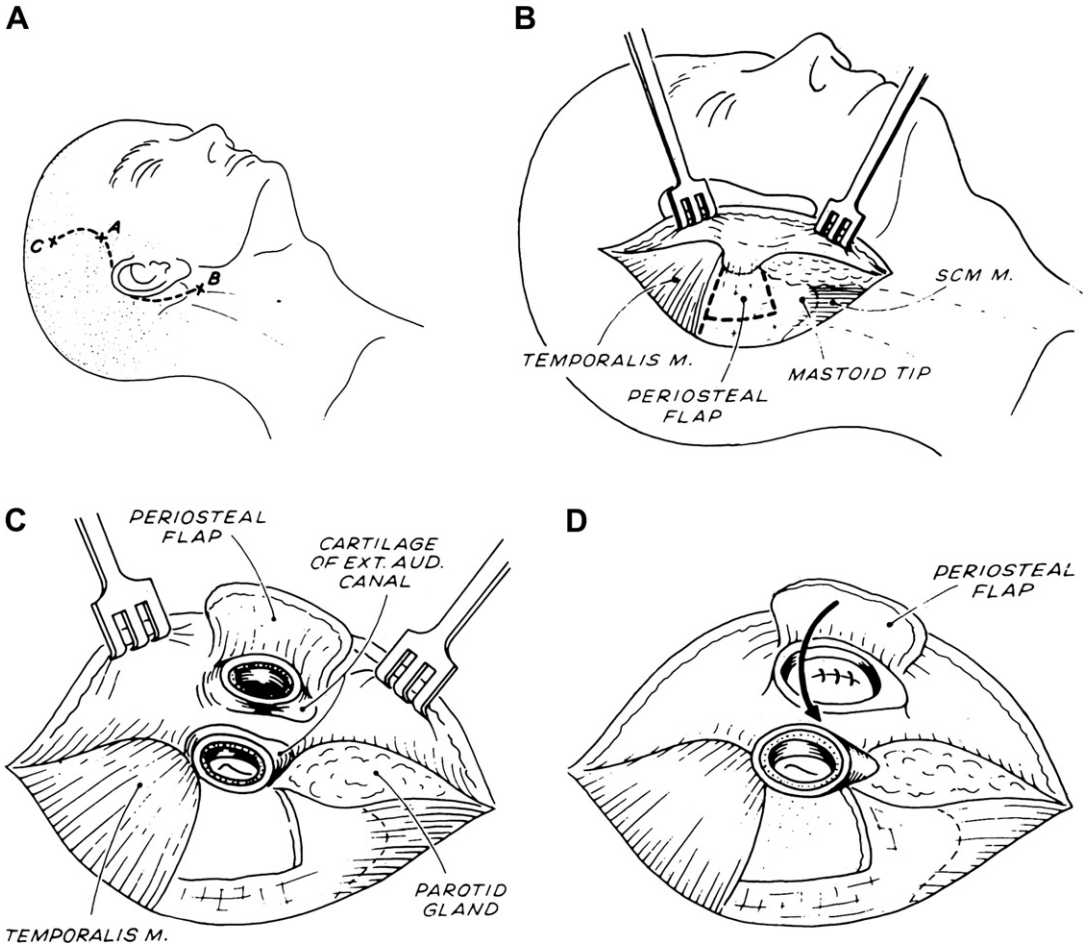
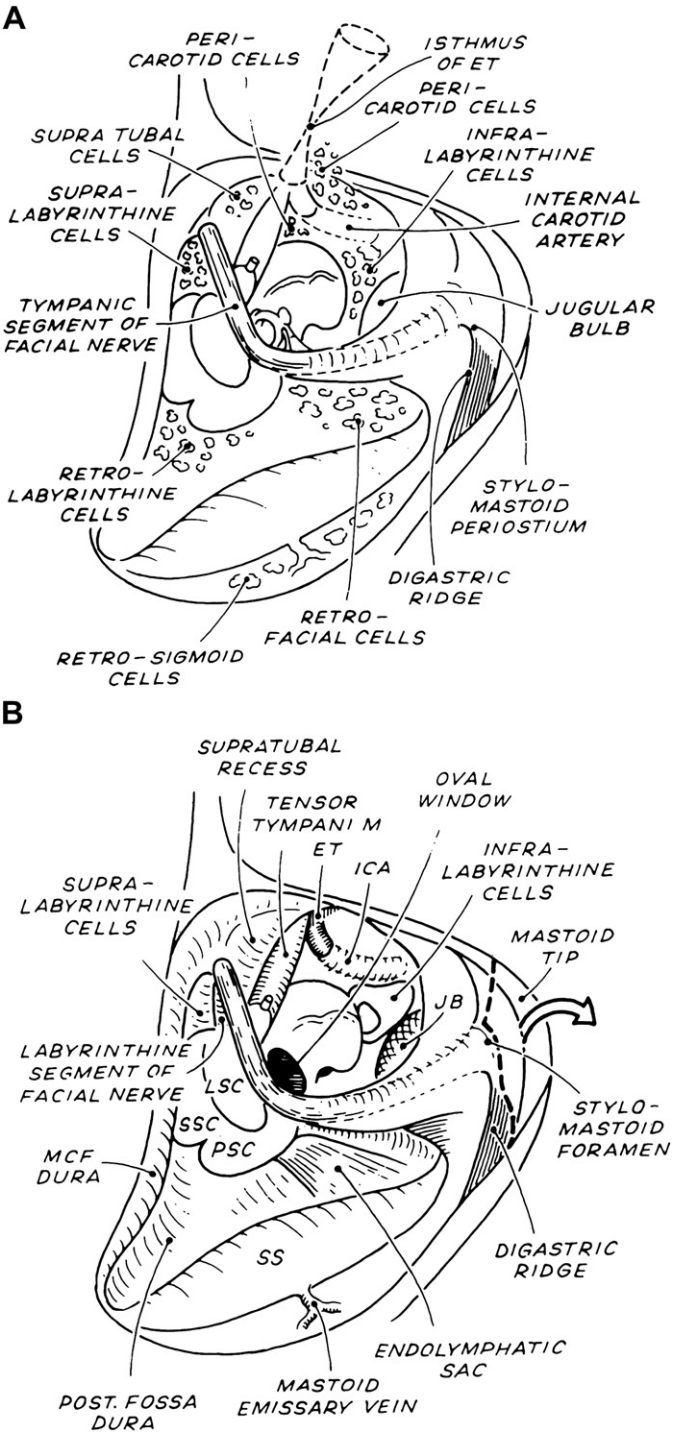


Fig. 1. (*A*) An S-shaped incision is made from the temporal region to 2 cm below the mastoid tip (A-B), with the superior portion (A-C) made at the conclusion of the procedure if needed for the temporalis muscle flap. (*B*) The postauricular skin is anteriorly reflected superficial to the temporalis muscle. An anteriorly based flap is created for mastoid periosteum and soft tissues. (*C*) The external auditory canal is transected at the level of the bony-cartilaginous junction. (*D*) The skin edges of the cartilaginous canal are separated slightly from underlying cartilage, everted laterally, and closed. The preserved periosteal flap is folded over the closed meatus and sutured to the cartilage for a two-layered closure. (*Adapted from* Fisch U, Mattox D. Microsurgery of the Skull Base. Stuttgart, Germany, Georg Thieme Verlag; 1988; with permission.)

include the retrosigmoid, retrofacial, antral, retrolabyrinthine, supralabyrinthine, infralabyrinthine, peritubal, and pericarotid cells. At the end of this initial dissection, the internal carotid artery, jugular bulb, and mastoid segment of the fallopian canal have been skeletonized.

Otic capsule removal

Exposure of the internal auditory canal contents requires a complete removal of the otic capsule with continued preservation of the fallopian canal as a bridge of bone from the geniculate ganglion to the stylomastoid foramen (Figs. 4 and 5). Posterior exposure is achieved through removal of the semicircular canals and vestibule. The inferior and anterior exposure inherent to this technique follows removal of the cochlea by drilling under and anterior to the fallopian canal bridge. Special care is taken to expose all dura between the jugular bulb, internal carotid artery, and semicanal of the tensor tympani muscle. Before completion of the superior exposure, the remaining bone over the posterior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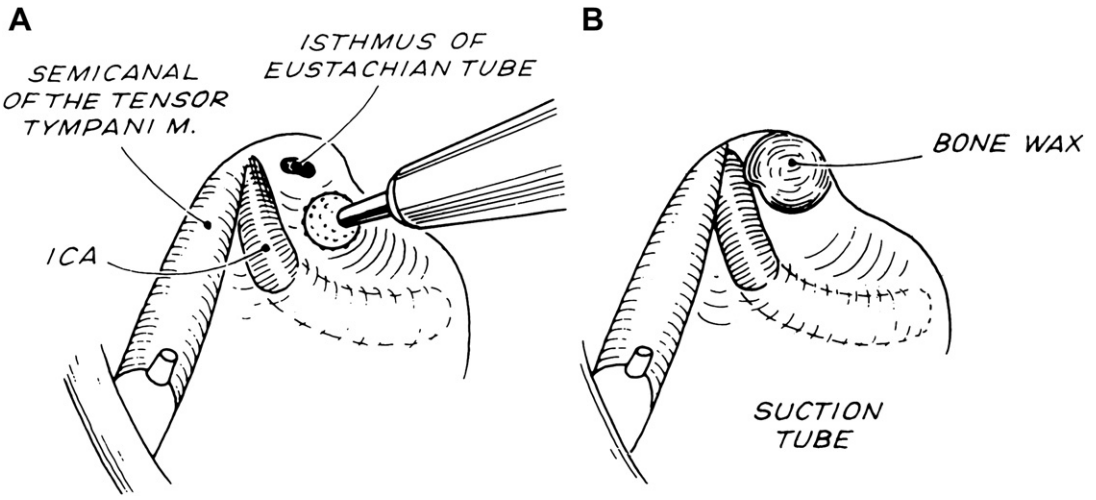

Fig. 3. (*A*) The bony eustachian tube is removed as far as the isthmus with diamond burs, taking care to avoid injury to the carotid artery. (*B*) After bipolar coagulation of the mucosa of the bony-cartilaginous junction, the eustachian tube is obliterated with bone wax. (*Adapted from* Fisch U, Mattox D. Microsurgery of the Skull Base. Stuttgart, Germany, Georg Thieme Verlag; 1988; with permission.)

fossa dura is removed between the superior petrosal sinus, sigmoid sinus, and posterior internal auditory canal.

Completion of the important superior exposure follows identification of the labyrinthine segment of the facial nerve at the meatal foramen by careful removal of the anterior-superior bone overlying the internal auditory canal. This identification is enhanced through the use of facial nerve monitoring. Subsequently exposure of posterior fossa dura is completed along the superior petrosal sinus and the posterior-superior acoustic porus. The completed exposure is diagrammatically illustrated in Fig. 5.

Tumor removal

Tumor removal begins with separation of the neuroma from the facial nerve at the meatal foramen (Figs. 6, 7, and 8). Working laterally to medially, the intracanalicular portion of the tumor is freed from the facial nerve. As it is separated from the nerve, the tumor is displaced into the space created by removal of the otic capsule. Once the porus is reached, the mobilized portion of tumor can be excised and the remaining intradural tumor debulked by intracapsular removal.

After complete hemostasis, removal of the intradural portion may then proceed with a posterior fossa incision, which begins between the sinodural angle and the posterior edge of the porus and is subsequently extended anteriorly below the porus. By retraction of the dural edges with 4–0 Vicryl sutures, the complete circumference of the tumor can be uncovered. Removal of the neuroma is accomplished by additional intracapsular removal, in conjunction with meticulous dissection of the tumor capsule from feeding vessels and the facial nerve. Identification and

Fig. 2. (*A*) The mastoid cortex is widely exposed. The remaining skin of the external auditory canal is removed, as well as the tympanic membrane, ossicles and posterior canal wall. All pneumatic cell tracts associated with the middle ear are thoroughly removed, the mastoid tip is removed to facilitate cavity obliteration. The tympanic and mastoid segments of the facial nerve are identified, leaving only a thin covering of bone over the nerve. The air cells are removed in the following order: retrosigmoid, retrofacial, retrolabyrinthine, supralabyrinthine, infralabyrinthine, pericarotid, and supratubal. (*B*) The surgical site following removal of all air cell tracts: ET, eustachian tube; ICA, internal carotid artery; JB, jugular bulb; SS, sigmoid sinus; LSC, lateral semicircular canal; PSC, posterior semicircular canal; SSC, superior semicircular canal; MCF Dura, middle cranial fossa dura. All middle ear mucosa has been removed. (*Adapted from* Fisch U, Mattox D. Microsurgery of the Skull Base. Stuttgart, Germany, Georg Thieme Verlag; 1988; with permission.)

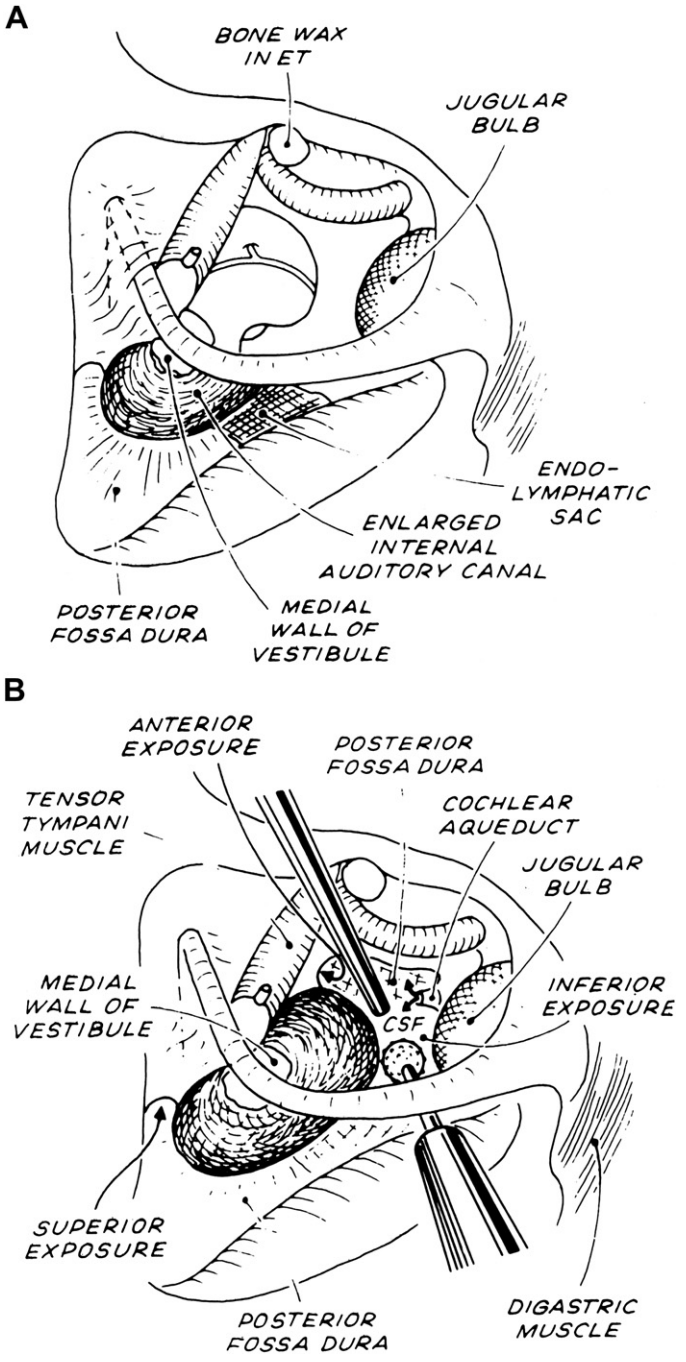

Fig. 4. (*A*) Posterior exposure. As in the translabyrinthine approach, the semicircular canals are removed and vestibule opened. The meatal dura is exposed from the posterior fundus of the canal to the porus. The bone removal is extended interiorly around the enlarged internal auditory canal and under the preserved fallopian canal. The endolymphatic sac is exposed. (*B*) Inferior and anterior exposure. The cochlea is drilled away and the posterior fossa dura is gradually exposed by working under and anterior to the fallopian canal, preserved as a bridge of bone. The dura is exposed between the jugular bulb, internal carotid artery, and the semicanal of the tensor tympani muscle. With this exposure, the cochlear aqueduct is encountered. Arachnoid within the aqueduct may be opened to allow decompression of the posterior fossa before the dura is incised. (*From* Fisch U, Mattox D. Microsurgery of the Skull Base. Stuttgart, Germany, Georg Thieme Verlag; 1988; with permission.)

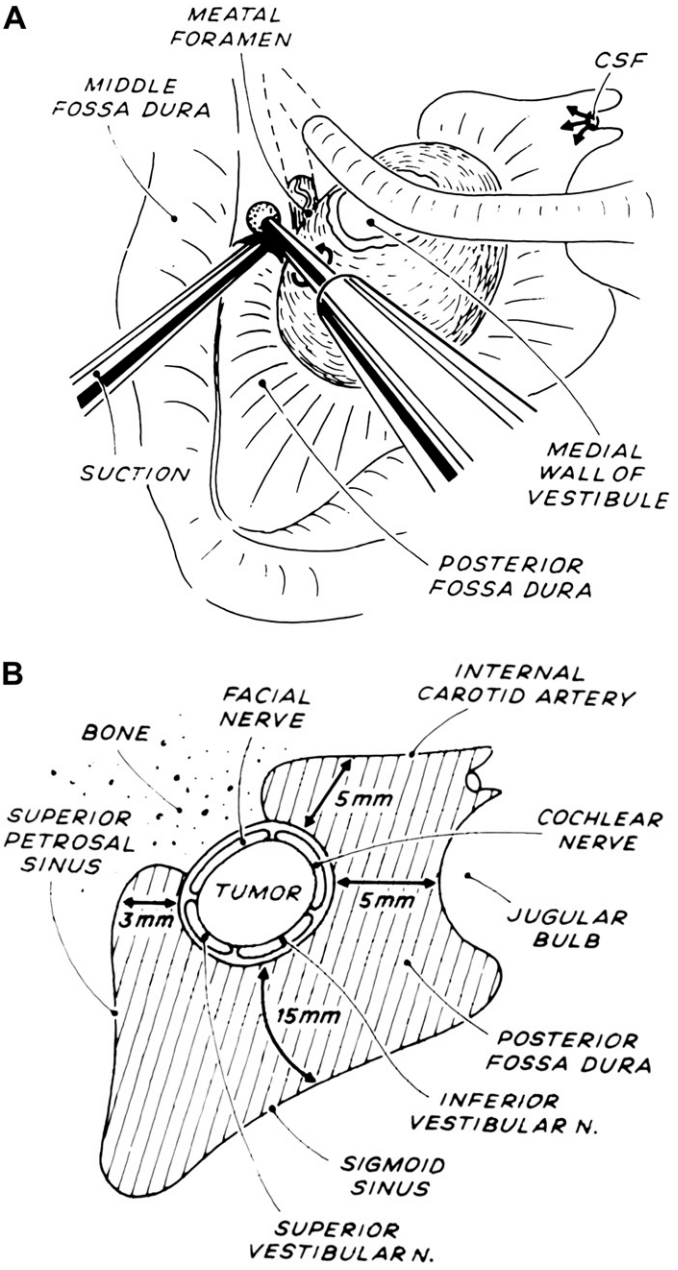

Fig. 5. (*A*) Superior exposure. Identification of the labyrinthine segment of the facial nerve must precede completion of the dural exposure superior to the porus. The nerve is usually 2 mm anterosuperior to the medial wall of the vestibule. Localization of its position is enhanced by the use of the nerve integrity monitor. Following this identification, bone is removed anteriorly along the superior petrosal sinus as far as the meatal foramen. (*B*) Extent of bone removal around the internal auditory canal. For protection of the facial nerve, the anterior-superior wall is left intact. The measurements shown of this maximal exposure represent averages from surgical cases. (*Adapted from* Fisch U, Mattox D. Microsurgery of the Skull Base. Stuttgart, Germany, Georg Thieme Verlag; 1988; with permission.)

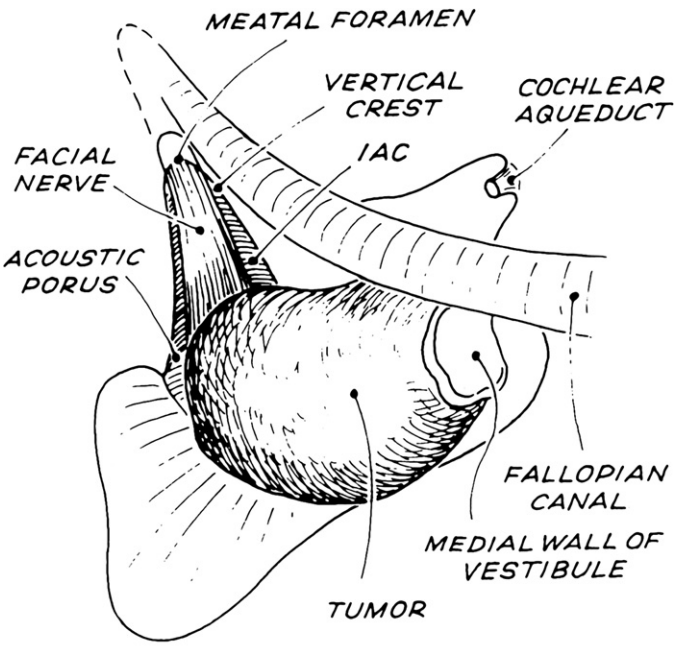

Fig. 6. The facial nerve is identified in the internal auditory canal proximal to the meatal foramen. As the tumor is gently separated from the nerve, it is pushed into the space created by complete otic capsule removal. Once the tumor is mobilized to the porus, the extradural tumor can be amputated and intradural tumor decompressed by intracapsular removal. (*From* Fisch U, Mattox D. Microsurgery of the Skull Base. Stuttgart, Germany, Georg Thieme Verlag; 1988; with permission.)

dissection of a displaced and thinned facial nerve at the porus is facilitated by the additional anterior exposure of its entire intracranial portion used in the transotic approach.

Defect reconstruction and closure

The defect in the posterior fossa dura is reconstructed with a fresh musculofascial graft harvested from the temporalis muscle fixed to the dura using the traction sutures previously placed (Fig. 9). A smaller musculofascial graft and fibrin glue are used to supplement the eustachian tube obliteration. Large strips of abdominal wall fat are then placed over the dural patch, secured under the fallopian canal bridge, fibrin glue applied, and the temporalis muscle transposed. Additional abdominal wall fat is placed under the transposed muscle to increase the pressure on the dural closure slightly. Following a layered skin closure, a pressure dressing is applied and maintained for 5 days.

Discussion

The transotic approach has advantages that are shared with the more posteriorly oriented translabyrinthine procedure. Cerebellar retraction is not necessary, and the surgical orientation is otologically oriented for secure facial nerve identification and reconstruction. Additionally, tumor growth in the lateral internal auditory canal can be dealt with easily because the dissection is initiated laterally.

In the transotic approach, however, the extension of bone removal to include the anterior medial wall of the temporal bone affords the surgeon a superb view of the cerebellopontine angle *anterior* to the tumor (Fig. 10). With this added exposure, the operative field has superior illumination for dissection of tumor from surrounding vessels and facial nerve without increased morbidity or mortality (Table 1). In contrast to the transcochlear approach and its modifications, [2,5] facial nerve transposition is unnecessary because the fallopian canal bridge does not significantly impair exposure. With the entire intracranial segment of the facial nerve visualized before tumor removal, the expeditious separation of nerve from tumor may occur with increased safety to facial function (see Table 1; Tables 2 and 3). If tumor invasion of the facial nerve is significant enough to require segmental

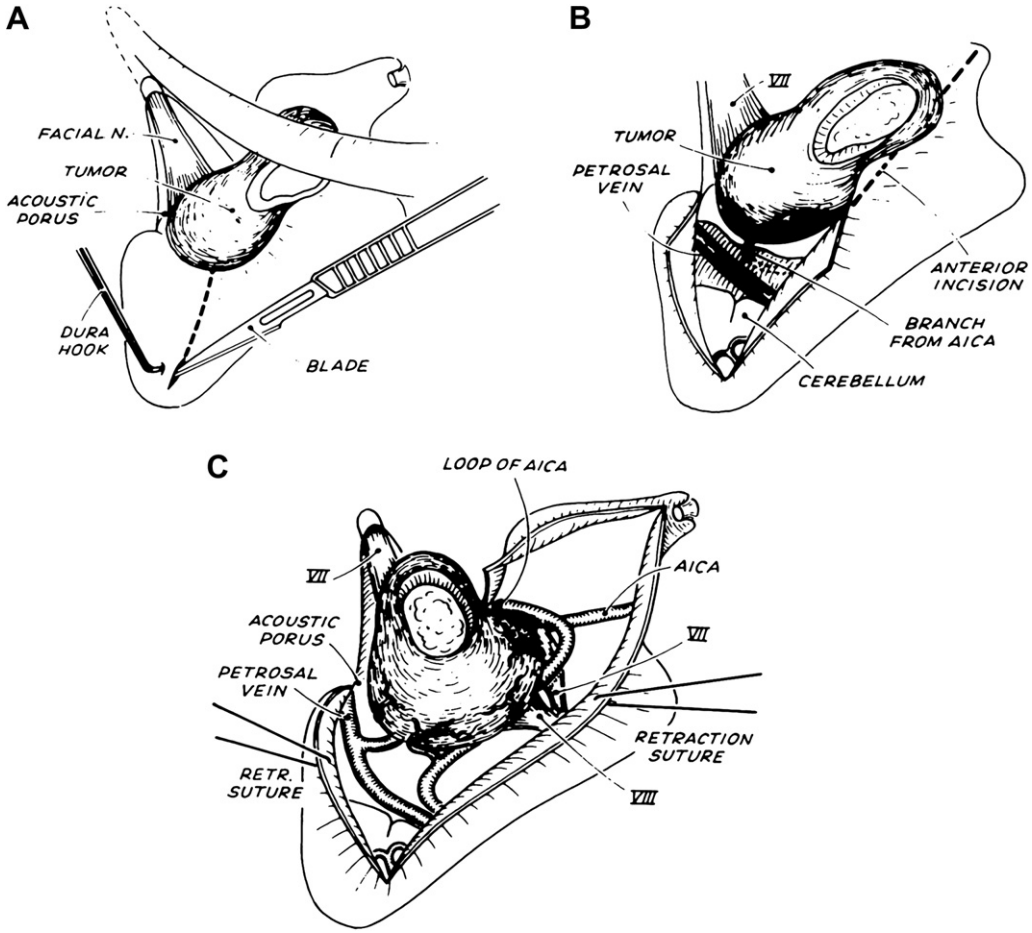

Fig. 7. (*A*) Once all bone removal is completed and hemostasis achieved, the posterior fossa dura is incised between the sinodural angle and posterior edge of the porus. (*B*) The incision is extended below and anterior to the porus, taking care to look for AICA or other vessels beneath the dura before cutting. (The bony fallopian canal bridge has been omitted for ease of illustration.) (*C*) The superior and inferior dural flaps are retracted with 4-0 Vicryl sutures. The posterior and inferior tumor margins are well seen, as well as the anteriorly displaced facial nerve. (Bony fallopian canal bridge omitted.) (*Adapted from* Fisch U, Mattox D. Microsurgery of the Skull Base. Stuttgart, Germany, Georg Thieme Verlag; 1988; with permission.)

resection, exposure is optimal for graft placement (Fig. 11). The cervical portion of the skin incision may be easily extended to facilitate a hypoglossal-facial anastomosis at the conclusion of tumor resection if there is no useful proximal facial nerve stump.

Postoperative cerebrospinal fluid leaks continue to plague other approaches to the cerebellopontine angle (Table 4), with various techniques introduced for the prevention of this complication [6–9]. In contrast, the use of the transotic approach at the University of Zurich has avoided this problem with a high degree of consistency (see Table 1). Additionally an important improvement has been the elimination of *delayed* cerebrospinal fluid leaks in our series that occasionally presented with meningitis several years after the original translabyrinthine surgery.

The technical basis for this improved rate of dural closure begins with the initial subtotal petrosectomy. Inherent in this portion of the procedure is the meticulous obliteration of the eustachian tube orifice, preceded by the removal of all middle ear mucosa and pneumatic cells connecting with the middle ear space. Removal of all middle ear mucosa with fat obliteration of the middle ear

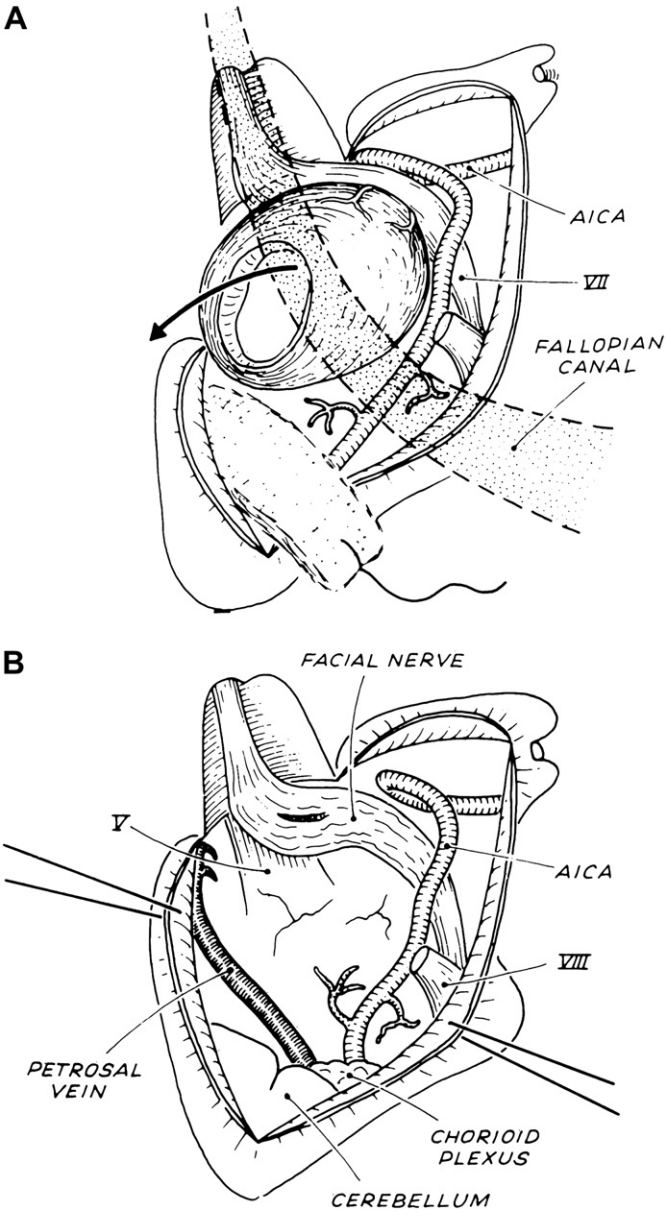

Fig. 8. (*A*) The separation of tumor from intracranial facial nerve is expeditious because of the visualization of its complete intracranial portion. Direct vision anterior to the tumor is advantageous because the anteriorly displaced facial nerve is frequently spread paper thin and nearly transparent. (*B*) Following complete tumor removal, the intracranial facial nerve is well visualized and accessible to grafting if necessary. (Bony fallopian canal omitted.) (*From* Fisch U, Mattox D. Microsurgery of the Skull Base. Stuttgart, Germany, Georg Thieme Verlag; 1988; with permission.)

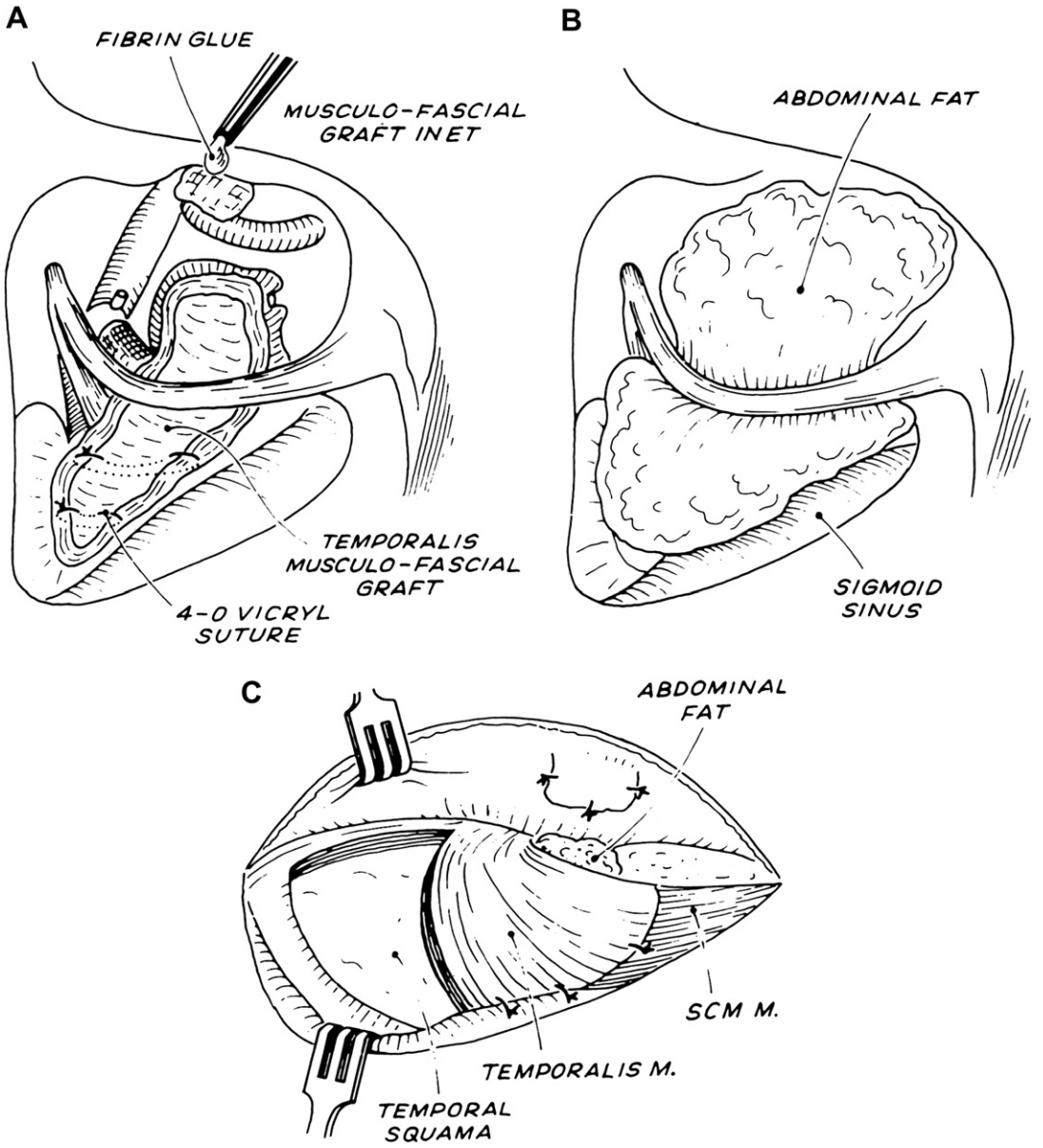

Fig. 9. (*A*) Posterior fossa dura is reconstructed using a musculofascial graft taken from the temporalis muscle, the graft is placed under the dura and fixed by using the previously placed 4-0 Vicryl traction sutures. A small musculofascial graft is also used to supplement the eustachian tube orifice. Fibrin glue is used to stabilize both grafts. (*B*) Large strips of abdominal fat pulled under the bony fallopian canal bridge are used to provide added pressure to keep the musculofascial graft against the dura. (*C*) The skin incision is extended superiorly for exposure of the temporalis muscle (A-C in Fig. 1 *A*). The posterior two thirds of the muscle are mobilized, rotated inferiorly, and sutured to the sternocleidomastoid (SCM) and tissues of the posterior occiput. Additional adipose tissue is then placed under the muscle and fibrin glue injected around the grafts. The subcutaneous tissue and skin are closed in iayers. The wound is drained with a suction drain that is removed as soon as a pressure dressing is placed. (*From* Fisch U, Mattox D. Microsurgery of the Skull Base. Stuttgart, Germany, Georg Thieme Verlag; 1988; with permission.)

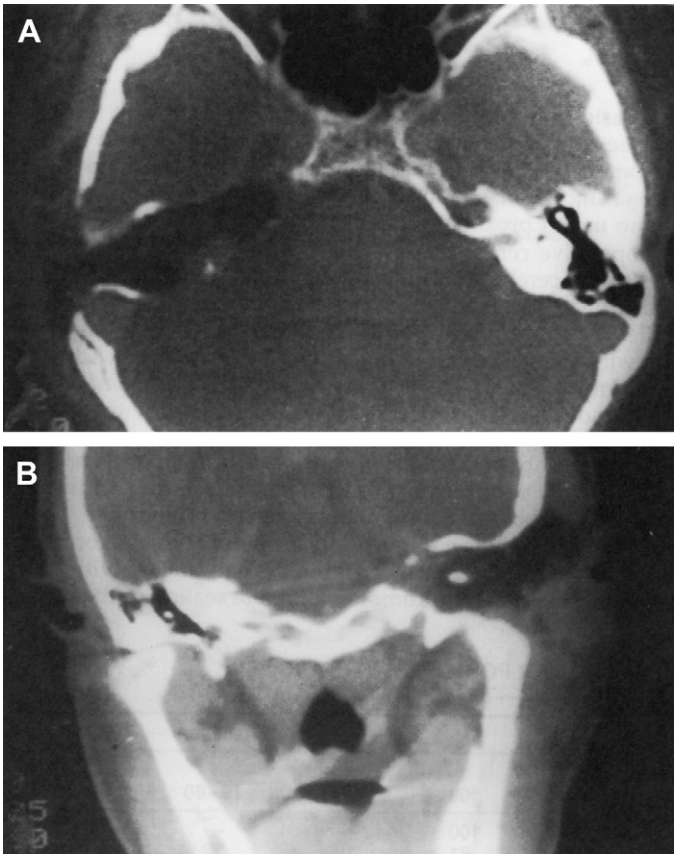

Fig. 10. Postoperative computed tomography scan following a transotic approach to the cerebellopontine angle for tumor removal. Note the wide exposure and bony defect created in the medial temporal bone. (*A*) Axial view. (*B*) Coronal view.

Table 1
Postoperative complications of surgical approaches*

Complication	Transotic (n = 147)(%)	Translabyrinthine (n = 114)(%)
Mortality	1	1
CSF leak		
Immediate	3	15
Requiring wound revision	1	7
Delayed (4–8 yr postoperatively)	0	5
Requiring wound revision†	0	5†
Meningitis	1	5
Facial function (2 yr postoperatively)		
Grades I,II	85	65
Paresis	13	31
Paralysis	2	4

* 1–2.5 cm diameter acoustic neuromas.
† Three patients presented with meningitis over 4 years following surgery.

Table 2
Two-year postoperative facial function—transotic approach

		Percent recovery				
Tumor Size (cm)	n	100	99–80	79–60	59–40	39–0
1–1.4	14	100	—	—	—	—
1.5–2.5*	52	61	19	12	4	4
Total	66	70	15	9	3	3

* No differences noted in facial function following removal of tumors 1.5–1.9 cm versus 2–2.5 cm.

cleft promotes complete sealing of the dural closure without the potential development of mucosal cysts [10]. The closure of the eustachian tube eliminates an important conduit for cerebrospinal fluid contamination; exenteration of all air cells removes occult tracts that may lead to postoperative cerebrospinal fluid leakage. Additionally, the bony fallopian canal bridge and temporalis muscle flap provide a stable means to apply constant pressure on the musculofascial dural patch.

In the Department of Otolaryngology at the University of Zurich, safety of tumor removal with preservation of facial function is paramount in the rationale of the surgical approach. A middle fossa approach is used for the treatment of intracanalicular tumors up to 8 mm in size in patients with usable hearing in the involved ear. The suboccipital approach has not been used for hearing conservation procedures owing to their generally unpredictable, low success rates at the possible expense of incomplete tumor removal [11,12]. Therefore larger tumors up to 2.5 cm are excised using the transotic approach in all patients with usable contralateral auditory function. Tumors that are believed to be adherent to the brain stem or larger than 2.5 cm are generally removed through the suboccipital approach. Additionally, the presence of unresolved otitis media or mastoiditis is a contraindication to the use of the transotic technique.

Table 3
Results of intracranial grafting

			Facial function	
Surgical approach	n	Mean follow-up (yr)	Percentage	Grade
Transotic	8	4	66	III
Translabyrinthine	2	11	63	III

The transotic approach to the cerebellopontine angle is a procedure that rewards the extra work required for full otic capsule removal with an excellent view of the anterior cerebellopontine angle. The additional 11/2 to 2 hours required beyond a translabyrinthine technique for this bone work is easily compensated by less time necessary for tumor removal (40 to 60 minutes). With this exposure, straightforward separation of tumor from intracranial contents can proceed expeditiously with emphasis on facial nerve preservation. The benefits of this technique are seen in the preservation of normal facial function (grade I) in all patients with tumors less than 1.5 cm in diameter and in 61% of those with larger lesions (see Table 2). Although the major utilization of a transotic procedure has been in removal of acoustic neuromas, other useful applications have been in the surgical treatment of meningiomas, hemangiomas, arachnoid cysts, and mucosal cysts invading the internal auditory canal.

Summary

The transotic approach to the cerebellopontine angle for resection of tumors invading the internal auditory canal provides superior illumination and exposure for optimal preservation of facial nerve function. Separation of facial nerve from tumor is enhanced with an anterior exposure that allows visualization of the intracranial segment of the nerve before tumor removal without significantly increasing total operative time. Facial nerve grafting or hypoglossal-facial anastomosis may be incorporated into the procedure at the time of tumor resection using the transotic approach. When combined with a musculofascial patch secured to the dural defect, the initial subtotal petrosectomy with eustachian tube and middle ear cleft obliteration generally avoids the complication of an immediate or delayed postoperative

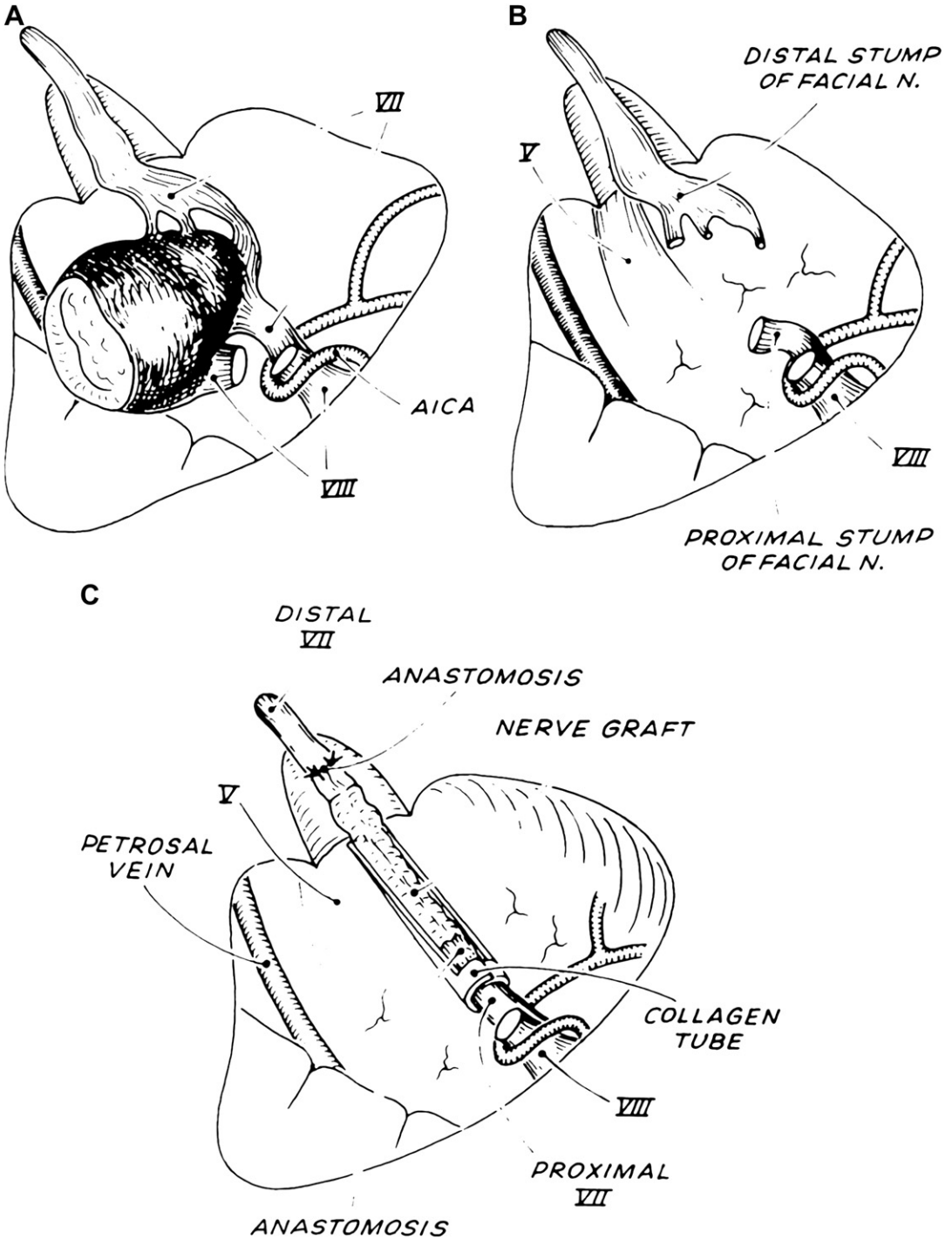

Fig. 11. (*A–B*) In some cases, a plane of dissection between facial nerve and tumor can not be easily developed. In these instances, a portion of nerve is resected if there is persistent tumor or loss of function in residual nerve. (*C*) The transotic approach provides excellent exposure for freshening nerve stumps and graft anastomosis. (*Adapted from* Fisch U, Mattox D. Microsurgery of the Skull Base. Stuttgart, Germany, Georg Thieme Verlag; 1988; with permission.)

Table 4
Postoperative cerebrospinal fluid leaks in literature

Series (yr)	Total no of series patients	Patients with postoperative cerebrospinal fluid leak (%)		
		Translabyrinthine	Combined	Suboccipital
Bryce, et al [6] (1991)	319	11	29	10
Robson, et al [13] (1989)	98	—	—	17
Hardy, et al [14] (1989)	100	13	—	—
Glasscock, et al [15] (1986)	616	11	25	27
Harner, et al [16] (1985)	162	—	—	12
Tos and Tomsen [17] (1985)	200	10	—	—
Gardner, et al [18] (1983)	105	9	22	—

cerebrospinal fluid leak. The transotic approach is indicated for tumors up to 2.5 cm in size that are not adherent to the brain stem.

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Neurosurg Clin N Am 19 (2008) 279–288

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Middle Fossa Approach for Acoustic Tumor Removal

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The middle fossa approach for vestibular nerve section was reported as early as 1904; however, hammer and chisel were used for surgery at that time, which put the facial nerve at risk [1]. The middle fossa approach did not have widespread application until refined by the senior author (WFH) in 1961, when it was used for decompression of the internal auditory canal in cases of extensive otosclerosis [2]. That therapy was later abandoned, but it became evident that this approach was suitable for removal of acoustic tumors. Initially the middle fossa approach was used for tumors of all sizes. Further experience demonstrated, however, that it was most suitable for small tumors [3–5] and that preservation of hearing and facial nerve function was possible in a significant proportion of operated patients [6]. The middle fossa approach was used infrequently until the development of gadolinium-enhanced magnetic resonance imaging (MRI). With this development, a larger number of acoustic tumors are diagnosed when they are small and before hearing has been significantly affected, making an attempt at hearing preservation desirable.

The primary indications for the middle fossa approach are a small acoustic tumor, with less than 5 mm extension into the cerebellopontine angle, and good preoperative hearing. For hearing conservation surgery, we use the arbitrary audiometric criteria of speech reception threshold better than 30 dB and speech discrimination score better than 70%, although these indications must be individualized to the needs of the patient [7]. Some advocate attempted hearing preservation in the removal of small acoustic tumors if any measurable preoperative hearing exists [8].

The middle fossa approach provides complete exposure of the contents of the internal auditory canal, allowing removal of laterally placed tumors without the need for blind dissection [9]. This exposure ensures total removal and is well suited for the removal of very small acoustic tumors [10]. The facial nerve can be located in its bony canal, allowing positive identification in a location not involved by tumor.

The middle fossa approach is technically difficult because of the lack of robust landmarks and the limited exposure. Bleeding in the posterior fossa can be difficult to control because of the limited access. Because of its location in the superior aspect of the internal auditory canal, the facial nerve is subjected to more manipulation than in the other approaches [11,12]. In the past, facial nerve results in middle fossa cases have not been as good as those using the translabyrinthine approach for similar-sized tumors [13]. The routine use of the facial nerve monitor, however, has helped to improve these results. Patients older than 60 years do not tolerate the middle fossa approach as well as younger patients because of the fragility of the dura and retraction of the temporal lobe.

Several authors use an extended middle fossa approach for large tumors [14–16]. The tentorium is divided to give wider access to the posterior fossa. Some also perform a labyrinthectomy to enlarge the exposure when hearing preservation is not attempted [17–19].

Perioperative Management

We do not usually use preoperative or postoperative antibiotics, but despite this, the

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incidence of postoperative meningitis is very low. Because of the long-distance referral nature of our practices, we prefer that if a patient is to develop postoperative meningitis, it happen while still in southern California, instead of a partially treated meningitis occurring at home.

Intraoperative furosemide and mannitol are given to allow easier temporal lobe retraction. The junior author (CS) also routinely uses a single dose of dexamethasone (Decadron) intravenously at the beginning of surgery. It is his clinical impression that the incidence of delayed facial paralysis is reduced by this measure. This single dose of steroid does not seem to affect wound healing adversely. Long-acting muscle relaxants are avoided during surgery so as not to interfere with facial nerve monitoring.

Surgical anatomy

The surgical anatomy of the temporal bone from the middle fossa approach is compact but complex (Fig. 1). Landmarks are not as apparent as with other approaches through the temporal bone, so laboratory dissection is useful for the surgeon to become familiar with the anatomy from above.

Anteriorly the limit of the dissection is the middle meningeal artery, which is lateral to the greater superficial petrosal nerve. The arcuate eminence marks the position of the superior semicircular canal and may be readily apparent in some patients but obscure in others. Kartush and colleagues [20] have cautioned that the relationship between the arcuate eminence and the superior semicircular canal may be variable in some patients, but the superior canal tends to be perpendicular to the petrous ridge. Medially the superior petrosal sinus runs along the petrous ridge.

Surgical tolerances are tight in the area of the lateral internal auditory canal. The labyrinthine portion of the facial nerve lies immediately posterior to the basal turn of the cochlea. Bill's bar separates the facial and superior vestibular nerves. Slightly posterior and lateral to this area are the vestibule and ampullated end of the superior semicircular canal.

Identification of the geniculate ganglion can be accomplished by tracing the greater superficial petrosal nerve posteriorly to it. If the tegmen is unroofed, the geniculate is found to be slightly anterior to the head of the malleus.

The internal auditory canal lies approximately on the same axis as the external auditory canal; this relationship is useful in orienting the surgical field [14]. The more medial one progresses along the internal auditory canal, the more space exists

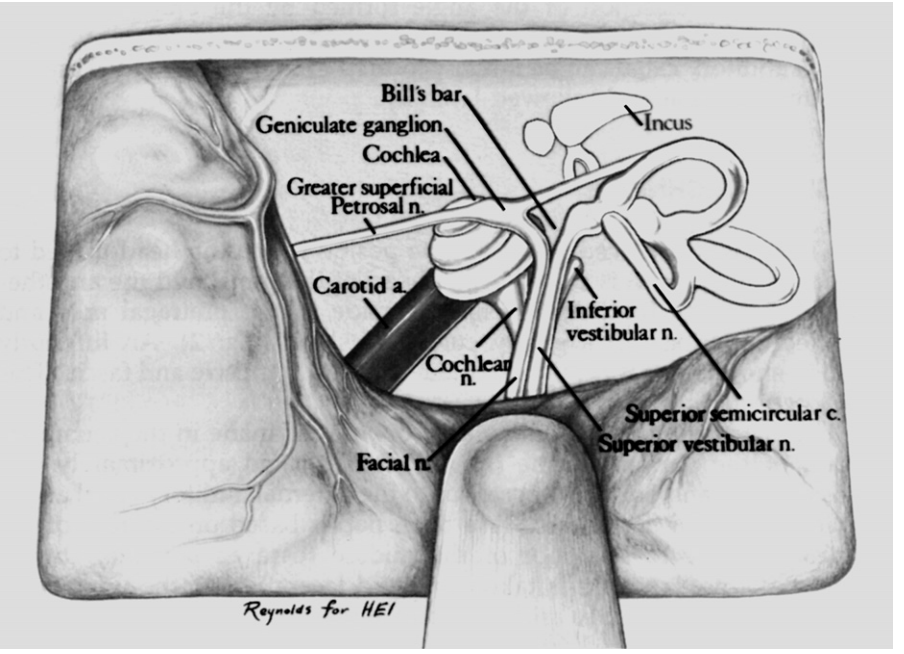

Fig. 1. Surgical anatomy of the temporal bone as viewed from the middle fossa approach. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

around it, [21] allowing for safe dissection in this area.

Several methods can be used to locate the internal auditory canal, which are reviewed in detail elsewhere [14,20]. We prefer to follow the facial nerve in a retrograde fashion to the internal auditory canal (see later). In some cases, after the geniculate ganglion has been identified, the junior author (CS) employs the technique of Garcia-Ibanez, [22] which involves drilling on the bisection of the angle formed by the blue line of the superior semicircular canal and greater superficial petrosal nerve. The internal auditory canal can be initially located in the "safe" medial area of the temporal bone and followed laterally.

Surgical technique

The patient is placed in the supine position with the head turned to the side. The surgeon is seated at the head of the table, and the anesthesiologist is at the foot. An incision is made in the pretragal area and extended superiorly in a gently curving fashion (Fig. 2). An inferiorly based U-shaped flap is fashioned of the temporalis muscle and fascia. This flap is reflected inferiorly.

Using a cutting bur, a craniotomy opening is made in the squamous portion of the temporal bone (Fig. 3). It is located approximately two thirds anterior and one third posterior to the external auditory canal and is approximately 2.5 cm square. This bone flap is based on the root of the zygoma as close to the floor of the middle fossa as possible. During creation of this flap, care is taken to avoid laceration of the underlying dura. The bone flap is set aside for later replacement.

The dura is elevated from the floor of the middle fossa. The initial landmark is the middle meningeal artery, which marks the anterior extent of the dissection. Frequently venous bleeding is encountered from this area and can be controlled with oxidized cellulose (Surgicel). Dissection of the dura proceeds in a posterior to anterior fashion. In approximately 5% of cases, the geniculate ganglion of the facial nerve is dehiscent, but injury can be avoided with dural elevation in this manner. The petrous ridge is identified with care not to injure the superior petrosal sinus. The arcuate eminence and greater superficial petrosal nerve are identified. These are the major landmarks to the subsequent intratemporal dissection. Once the dura has been elevated, typically with a suction irrigator and a blunt dural elevator, the House-Urban retractor is placed to support the temporal lobe. To maintain a secure position, the teeth of the retaining retractor should be locked against the

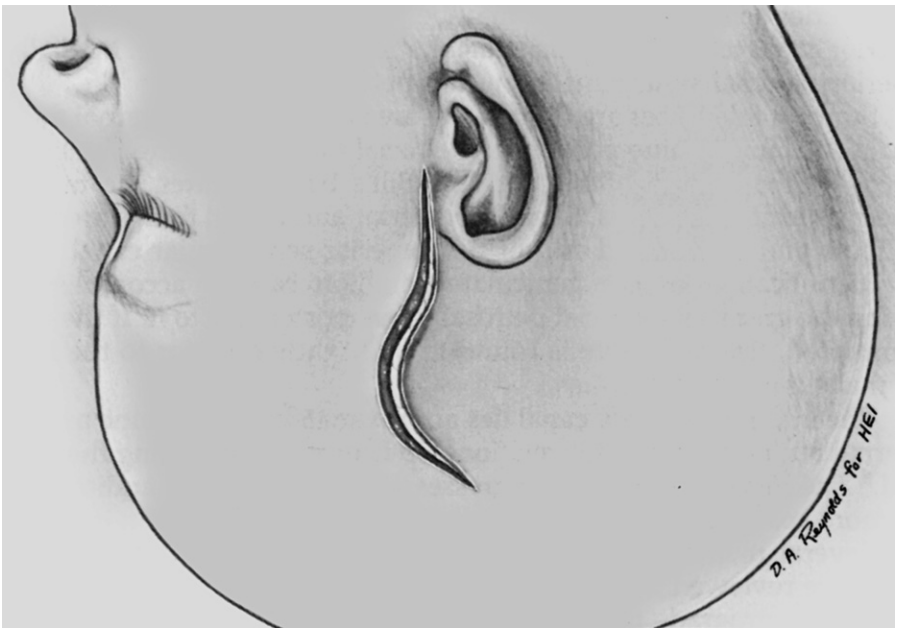

Fig. 2. Incision begins in the pretragal area and extends 7 to 8 cm superiorly in a gently curving fashion. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

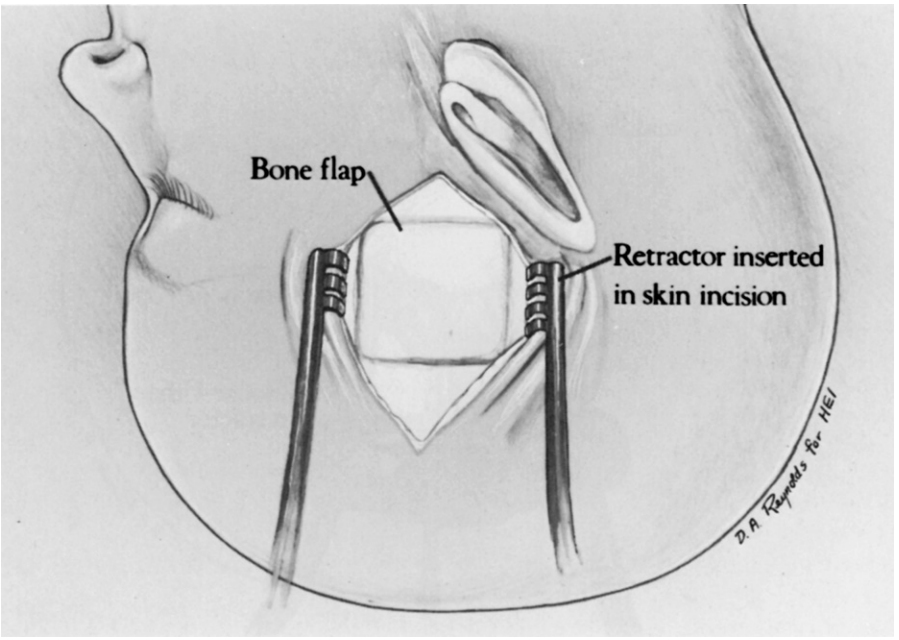

Fig. 3. Two thirds of the craniotomy window is located anterior to the external auditory canal. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

bony margins of the craniotomy window (Fig. 4). Using a large diamond bur and continuous suction irrigation, the blue line of the semicircular canal is identified at the arcuate eminence. This structure makes an approximately 45 to 60 degree angle with the internal auditory canal.

The greater superficial petrosal nerve is located medial to the middle meningeal artery (Fig. 5) then followed posteriorly to the geniculate ganglion (Fig. 6). The labyrinthine portion of the facial nerve is identified medial to the ganglion. Care must be taken to avoid the cochlea, which lies only a few millimeters anterior to the labyrinthine portion of the facial nerve.

Bone is removed from the superior surface of the internal auditory canal down to the porus acusticus. The lateral end of the internal auditory canal is dissected with identification of Bill's bar and the superior vestibular nerve (Fig. 7). Medially 180 degrees of bone can be removed from its circumference (Fig. 8). This exposure must narrow laterally because of the location of the inner ear.

The dura of the internal auditory canal is divided along the posterior aspect (Fig. 9). The facial nerve is clearly identified in the anterior portion of the internal auditory canal.

The superior vestibular nerve is cut at its lateral end, and the vestibulofacial anastomotic fibers are divided. The tumor is separated from the facial and cochlear nerves (Fig. 10). Using a right angle hook, the inferior vestibular nerve is divided, and the tumor is gently freed from the lateral end of the internal auditory canal. The tumor is separated from the cochlear and facial nerves and removed (Fig. 11). To preserve hearing, it is essential to preserve the internal auditory artery. The vessel typically runs between the facial and cochlear nerves but may not be visible during the dissection.

After irrigation of the tumor bed and establishment of hemostasis, abdominal fat is used to close the defect in the internal auditory canal. The House-Urban retractor is removed and the temporal lobe is allowed to reexpand.

The wound is closed with absorbable subcutaneous sutures over a Penrose drain if indicated. This drain is typically removed on the 1st postoperative day. A mastoid-type pressure dressing is maintained for 4 days postoperatively.

Postoperative care

The patient is observed in the intensive care unit for the initial 2 postoperative days and typically has a hospitalization of 6 to 7 days. Once leaving the intensive care unit, ambulation is

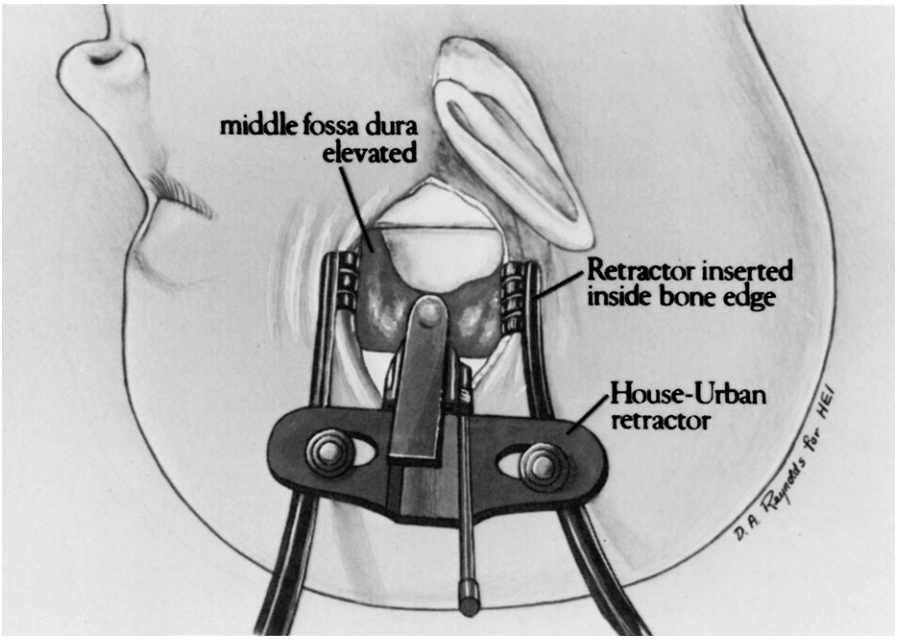

Fig. 4. Temporal lobe is supported by House-Urban retractor. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

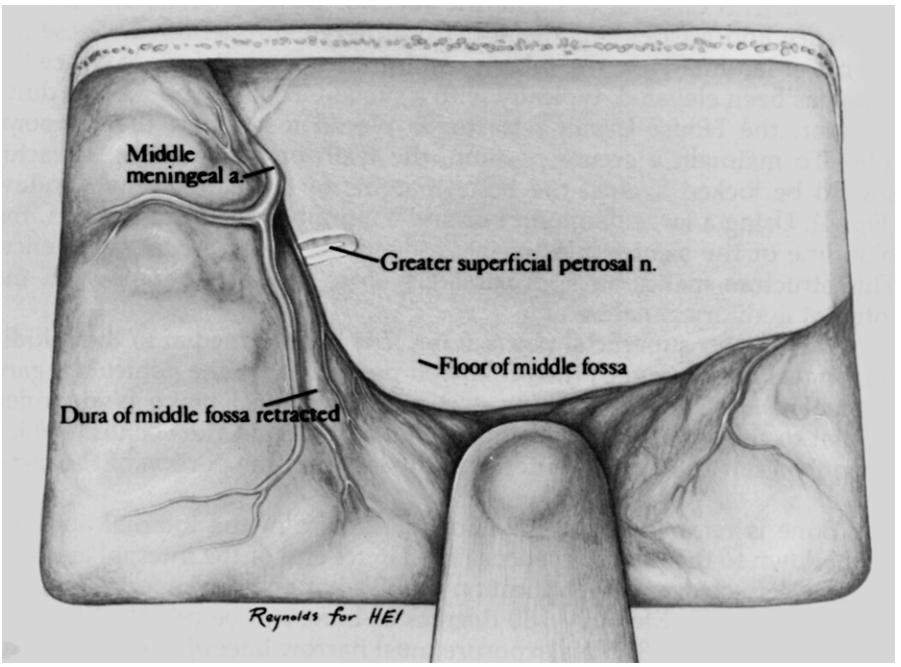

Fig. 5. The greater superficial petrosal nerve is identified medial to the middle meningeal artery. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

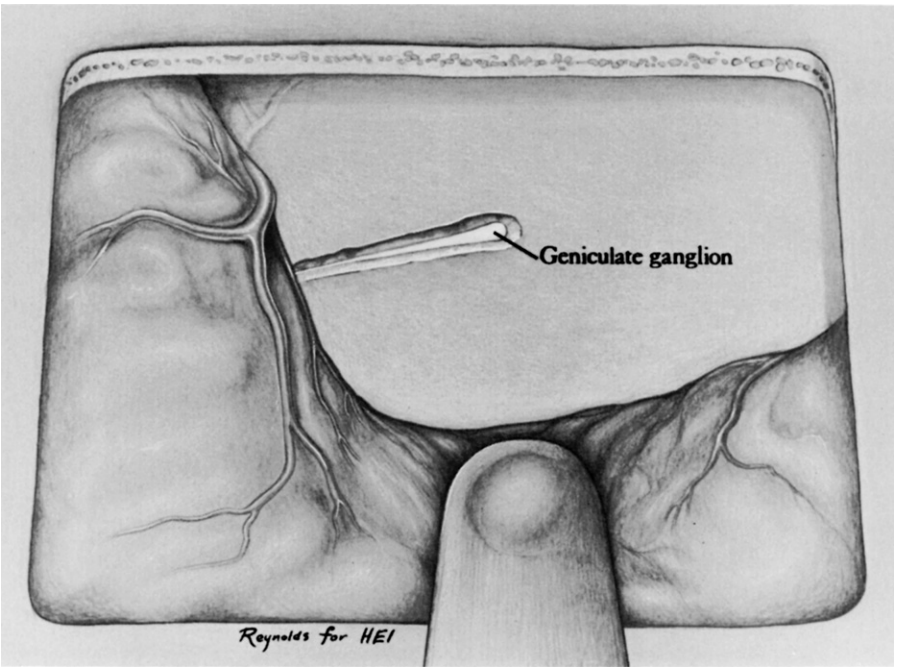

Fig. 6. The geniculate ganglion is found by following the superficial petrosal nerve posteriorly. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

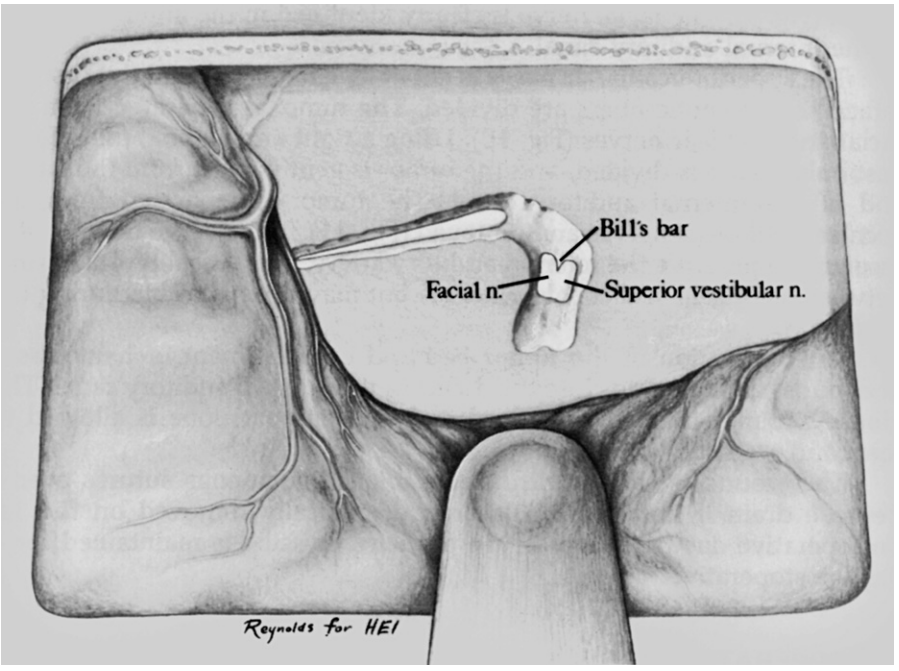

Fig. 7. Bill's bar separates the facial from the superior vestibular nerve at the lateral end of the internal auditory canal. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

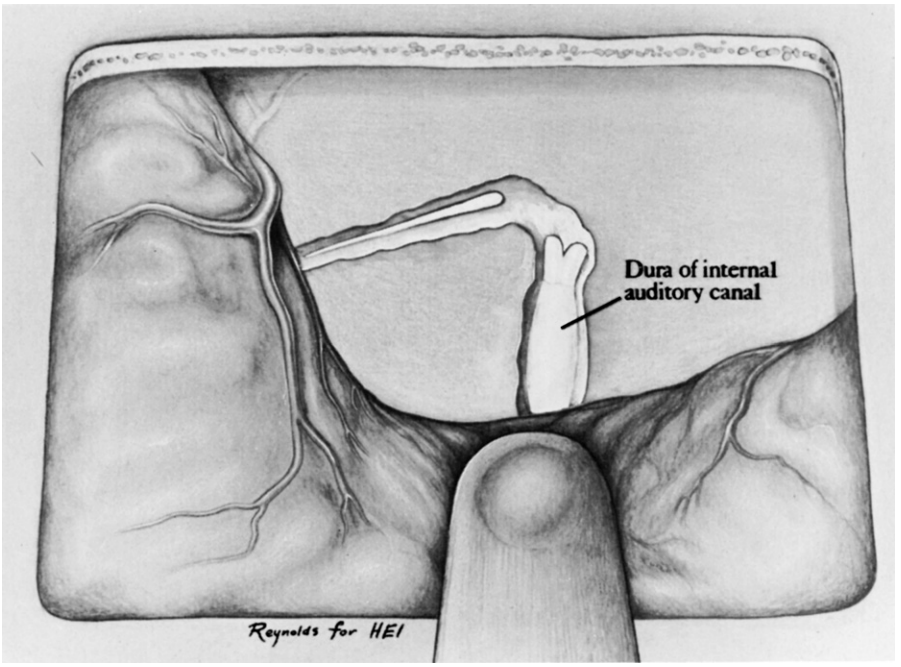

Fig. 8. The internal auditory canal is skeletonized through the entire length. Bone is removed around the porus acusticus, uncovering dura of the posterior fossa. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

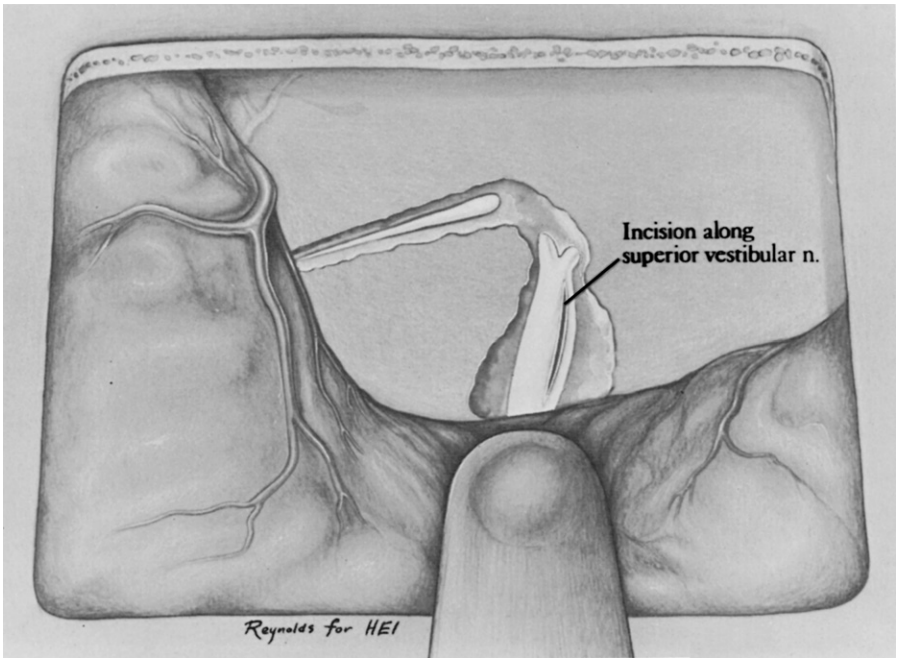

Fig. 9. The dura of the internal auditory canal is incised along the posterior aspect. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

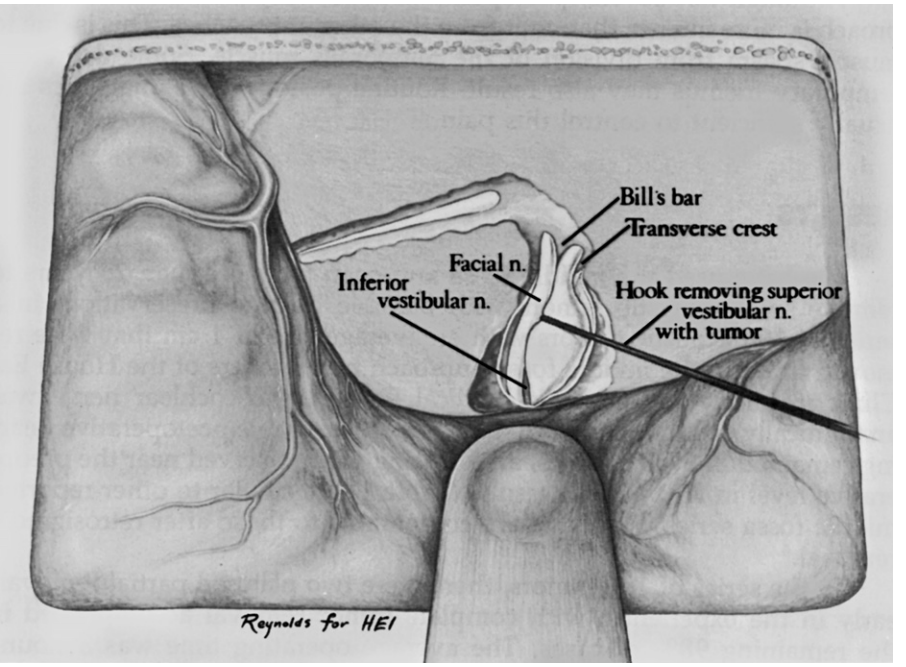

Fig. 10. The intracanalicular acoustic tumor is dissected from the facial and cochlear nerves. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

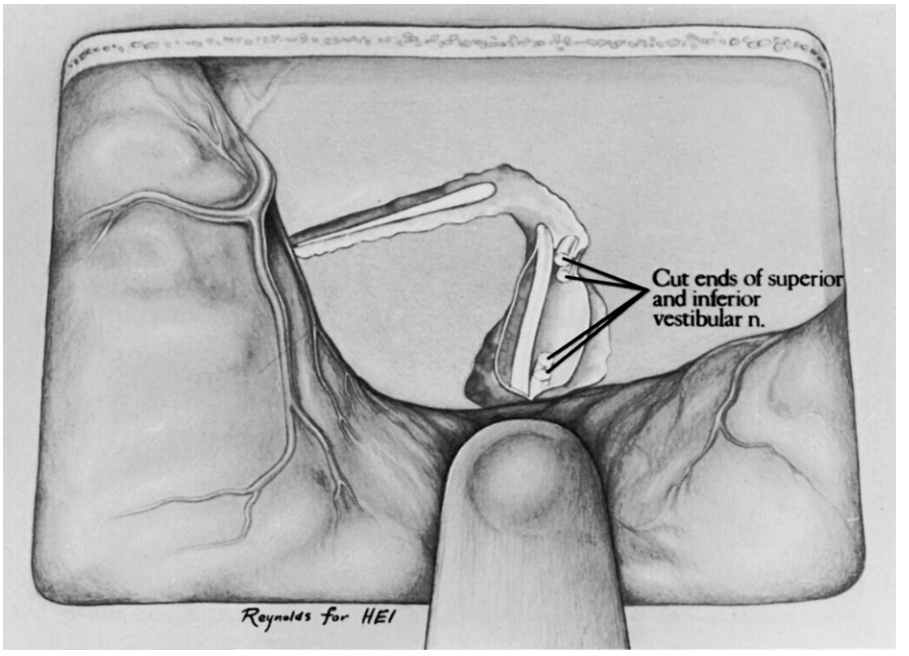

Fig. 11. Divided vestibular nerves are visible after tumor removal. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

Table 1
Postoperative facial nerve and hearing results

Series	n	Measurable postoperative hearing (%)	Anatomic facial nerve preservation (%)	Postoperative facial nerve function (house grade I or II) (%)
Shelton, et al [13]	106	59	100	89
Gantz, et al [8]	42	50	98	86
Glasscock, et al [23]	32	31	100	Na
Sanna, et al [24]	20	50	95	Na

Abbreviation: Na, not available.

encouraged. We believe early ambulation is important for rapid vestibular compensation.

Although not severe, postoperative pain after the middle fossa approach is more intense than that from the other approaches. This is due to muscle spasm from division of the temporalis muscle. Some degree of temporary trismus may also result. Routine postoperative analgesics are usually sufficient to control this pain.

Results

We have used the middle fossa approach for more than 25 years to remove small acoustic tumors with possible hearing preservation. In a series of 106 acoustic tumors with an average size of 1 cm that were removed through the middle fossa approach by members of the House Ear Clinic (formerly the Otologic Medical Group), the cochlear nerve was anatomically preserved in 89% of cases [13]. Measurable postoperative hearing remained in 59% of cases, and hearing was preserved near the preoperative level in 35%. These results (Table 1) are similar to other reported middle fossa series [8,23–26] and are comparable to those after retrosigmoid removal [27].

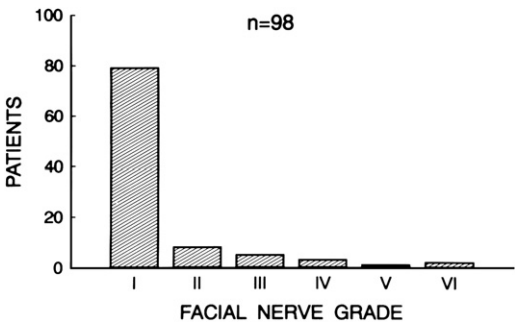

Fig. 12. Distribution of postoperative facial nerve function at 1 year or greater. Normal or near normal function occurred in 89% of patients. (*Reprinted by* permission of the House Ear Institute, Los Angeles.)

In the series of 106 tumors, there were two planned partial removals early in the experience, with complete tumor removal accomplished in the remaining 98% of cases. The average operating time was 3 hours, 12 minutes, with a range of $1\frac{1}{2}$ to 6 hours.

The facial nerve was anatomically preserved in all cases. Of the 98 patients with 1-year follow-up, 89% had House grade I or II function (Fig. 12).

Complications occurred in 18% of the patients. Two patients with cerebrospinal fluid leaks required surgical closure. No patients had postoperative seizures or hydrocephalus. Six patients had meningitis that responded to antibiotic therapy.

Serious complications were rare. There were two postoperative deaths, both in the 1960s, early in our experience. The middle fossa approach was used then for tumors that were larger than we would now consider appropriate. One patient died after a posterior fossa bleed, the other from anterior-inferior cerebellar artery thrombosis.

Postoperative seizures have been reported only in two patients from one series [23]. Electroencephalogram studies in both of these patients were consistent with an ipsilateral temporal lobe source for the epileptic activity, which was believed to be due to temporal lobe retraction.

Summary

The middle fossa approach is well suited for the removal of small acoustic tumors with possible hearing preservation. The most appropriate candidates have tumors with less than 5 mm extension into the cerebellopontine angle and good preoperative hearing (speech reception threshold $\leq$30 dB, speech discrimination score $\geq$70%). Measurable postoperative hearing can be preserved in 31% to 59% of patients, and normal or near normal facial function occurs in 86% to 89%. Serious postoperative complications are rare with this approach.

With the advent of gadolinium-enhanced MRI, it is now possible to diagnose acoustic tumors reliably when small and before hearing has been significantly affected. The middle fossa approach provides excellent access for the removal of these small tumors.

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Intraoperative Monitoring of Facial and Cochlear Nerves During Acoustic Neuroma Surgery

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COMMENTARY

While most of the information in this article is still useful and relevant, there have been several developments in cranial nerve monitoring during the 16 years since its initial publication. This brief addendum is not meant to be a comprehensive update, but rather a concise summary of some of the most relevant developments with references to more recent literature. For a more comprehensive treatment of many of these topics, see Yingling and Ashram [1]. More information specific to cochlear nerve monitoring may be found in Martin and Stecker [2].

In 1992, many of the systems used in intraoperative monitoring were cobbled together from laboratory equipment. Today, commercial systems specifically designed for intraoperative monitoring are available from several manufacturers, including Cadwell Laboratories, Axon Systems, and Nihon-Kohden. These systems typically include specialized low voltage and/or current stimulators suitable for direct cranial nerve stimulation, the ability to perform EMG and evoked potential recordings simultaneously, and can be configured to monitor many other types of surgery with appropriate software protocols.

A major limitation of EMG-based methods for facial nerve monitoring has been their inability to be used during electrocautery, which creates large electrical artifacts that obliterate EMG signals at times when cranial nerves may be at significant risk from thermal injury if cautery is applied in the vicinity of the nerve. Thus methods not based on electrical recordings are a useful adjunct to EMG, which remains the most sensitive indicator of facial nerve irritation. While methods based on direct detection of facial motion by attached sensors have been attempted [3], the best alternative to EMG may be a video-based system [4].

Another recent development is the identification of a specific EMG response to stimulation of the nervus intermedius [5]. This response has a characteristic low amplitude, prolonged latency, and restricted distribution compared to stimulation of the facial nerve itself. If this distinction is not recognized, the n. intermedius may be mistaken for the facial nerve; since these nerves are sometimes widely separated by the growth of the tumor this may lead to inadvertent section of the facial nerve.

Since the NIH Consensus Statement on Acoustic Neuroma [6] unequivocally recommended routine intraoperative monitoring of the facial nerve, there have been no formal clinical trials to assess the efficacy of facial nerve monitoring in improving outcome. However, numerous studies have shown than parameters derived from responses to intraoperative stimulation (ie, as threshold, amplitude, pre-post surgery, or proximal/distal ratios) are strong predictors of postoperative facial function [7–20]. While the consistency of these reports is encouraging, the optimum predictive variables have yet to be determined; this will require a larger population studied with a consistent set of parameters. In a different context (middle ear and mastoid surgery), Wilson and colleagues [21] demonstrated the cost-effectiveness of facial nerve monitoring.

Finally, one of the remaining issues in facial nerve monitoring is the necessity for surgeon-applied stimulation of the nerve itself, which is often difficult in larger tumors until substantial resection has taken place, sometimes without knowledge of the location of the nerve until it is too late. A method for continuous assessment of facial nerve function without the necessity for direct intracranial stimulation would help mitigate this problem. An obvious candidate

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is the blink reflex, which is elicited by stimulation of the supraorbital branch of the trigeminal nerve and, via a polysynaptic reflex arc, elicits responses in muscles innervated by the facial nerve. Unfortunately, although the blink reflex has shown promise as a prognostic indicator in the clinical setting [5], it has proven difficult to reliably elicit under general anesthesia [22].

The most promising solution to this problem may be elicitation of facial nerve motor evoked potentials by transcranial electrical stimulation of the contralateral face area of motor cortex [23]. Questions remain as to the specificity of this response to facial nerve activation (for example, trigeminal nerve activation could produce similar responses, and latency-based techniques for differentiation of V vs. VII as described in the main article may not be applicable to transcranial stimulation). Nevertheless, the method of transcranial stimulation has come into widespread use for monitoring corticospinal tracts during spinal surgery, and its application to facial nerve monitoring may prove to be the most significant advance in this field since the advent of EMG monitoring in the late 1970's.

The first use of cranial nerve monitoring during posterior fossa surgery was almost a century ago. On July 14, 1898, Dr. Fedor Krause performed a cochlear nerve section for tinnitus and noted that "… unipolar faradic irritation of the (facial) nerve-trunk with the weakest possible current of the induction apparatus resulted in contractions of the right facial region, especially of the orbicularis oculi, as well as of the branches supplying the nose and mouth…" [24]. The patient awoke with a slight facial paresis, which mostly resolved within a day. Krause also noted contractions of the shoulder, which he attributed to stimulation of the spinal accessory nerve, which "… had undoubtedly been reached by the current, because it was, together with the acusticus, bathed in liquor that had trickled down… ." He thus anticipated not only the use of electrical stimulation to locate cranial nerves but also the enduring problem of artifactual responses from current spread.

Frazier [25] described a similar technique used in 1912 during an operation for relief of vertigo, pointing out the importance of facial nerve preservation and the fact that it could be identified by "galvanic current." Similar methods were later described by Olivecrona [26,27], Hullay and Tomits [28], Rand and Kurze [29], Pool [30], and Albin and colleagues [31]. Givre and Olivecrona [26] and Hullay and Tomits [28] even recommended removal of acoustic neuromas under local anesthesia to facilitate assessment of facial function. This early technique, in which the face was observed for visible contractions after electrical stimulation, remained the state of the art for facial nerve identification until the late 1970s, when the use of facial electromyography (EMG) was introduced by Delgado et al in 1979 [32].

There are, to our knowledge, no reports of VIIIth cranial nerve monitoring until the late 20th century, undoubtedly because the development of techniques for signal averaging and the discovery of the human auditory brain stem response (ABR) by Jewett and Williston in 1971 [33] were necessary preconditions for attempting to monitor cochlear nerve function. Also, during the early days of acoustic neuroma surgery, the generally large size of the tumors when diagnosed and the relatively crude state of surgical techniques made mortality the main issue, rather than cranial nerve preservation. As advances in diagnosis and microsurgical techniques have made such surgery safer, increasing emphasis has been placed on preservation of cranial nerve function, with a resultant growth in development of techniques for monitoring these nerves during surgery.

Now VIIth cranial nerve monitoring during acoustic neuroma surgery has become routine, and anatomic preservation of the facial nerve is regularly achieved in all but a few of the largest tumors. Facial motility is still often compromised in the immediate postoperative period, but the prognosis for eventual recovery of function is good if the nerve is intact and can be electrically stimulated after tumor removal. Preservation of hearing has been more difficult to achieve, owing to the more intimate relationship of the tumors with the cochleovestibular nerve, but can now often be achieved in smaller tumors with the aid of VIIIth cranial nerve monitoring techniques. This article, based on our experience in over 500 posterior fossa procedures as well as a review of the literature, describes the methods currently available for cranial nerve monitoring, emphasizing facial and cochlear nerve monitoring during acoustic neuroma surgery.

Technical issues

Personnel

Successful performance of intraoperative monitoring is not simply a matter of bringing another piece of equipment into the operating room. Applying neurophysiologic techniques in the time-pressured and electrically hostile environment of

the operating room requires specialized skills that may make the difference between successful monitoring and no monitoring or, even worse, inadequate monitoring that provides inaccurate feedback to the surgeon. As a result, a new specialty field of intraoperative neurophysiologic monitoring is evolving, and a professional organization, the American Society of Neurophysiological Monitoring (ASNM), has been founded. Specialists in intraoperative monitoring have come from diverse backgrounds, including neurology, neurophysiology, audiology, and anesthesiology; regardless of background or professional degree, however, such personnel share a common fund of knowledge including the relevant neuroanatomy and neurophysiology, principles of biomedical instrumentation, knowledge of the variety of intraoperative monitoring techniques and their uses and limitations, and practical experience in performing these techniques and interpreting their results. Given the potentially catastrophic consequences of inappropriate application of monitoring techniques, we believe that the participation of professional monitoring personnel is highly desirable, despite the additional costs incurred. Third-party reimbursement should be facilitated by the recent addition of a CPT code (95920) specific to intraoperative neurophysiologic monitoring.

Anesthetic considerations

Unlike cortical evoked potentials, which are notoriously sensitive to many anesthetic agents, the ABR and EMG responses that are monitored during acoustic neuroma surgery are essentially unaffected by any commonly used anesthetic regimens. The one exception to this is a contraindication to the use of any muscle relaxants because blockade of the neuromuscular junction is incompatible with meaningful monitoring of EMG activity. A recent report [34] has suggested that partial blockade can be used to prevent patient movement while still retaining the ability to elicit EMG responses with facial nerve stimulation. Our experience has verified this observation but indicates that although electrically evoked EMG is relatively preserved, both spontaneous EMG and *mechanically elicited activity* appear to be obliterated by these agents. This compromises two of the more important indicators of facial nerve injury.

We therefore recommend that *no paralytic agents* be used during acoustic neuroma surgery. This, of course, creates its own problems for anesthetic management because patient movement could have disastrous consequences and must be prevented by maintaining an adequate level of anesthesia. Fortunately, the ABR and EMG are not affected by routine concentrations of common anesthetics, such as nitrous oxide, opiates, or halogenated agents, so no other constraints on anesthetic technique are necessary. Short-acting agents such as succinylcholine may be given to facilitate intubation, but it must be verified that such agents have cleared before any manipulations that might affect the facial nerve are undertaken. For a suboccipital approach, this would be the time of opening the dura and retraction of the cerebellum; in a translabyrinthine approach, the facial nerve is first at risk during skeletonization of the horizontal portion in the temporal bone. Fortunately these events typically occur far enough into the procedure that any relaxants given at intubation will have cleared in time.

Instrumentation

Electromyography instrumentation

The essential requirements for facial EMG monitoring are a stimulator that can be precisely controlled at low levels, one or more low noise amplifiers capable of amplifying microvolt level signals, an oscilloscope, and an audio monitor with a squelch circuit to mute the output during electrocautery. The NIM-2 (Nerve Integrity Monitor), manufactured by Xomed-Treace (Jacksonville, Florida), is a commercial device offering two channels (only one of which is displayed at a time) and appropriate stimulation and squelch circuits. It is relatively easy, however, to put together a system from off-the-shelf components that can provide more channels at a substantially lower cost. At the University of California, San Francisco (UCSF), we use a four-channel system with Grass amplifiers and stimulator (Quincy, Massachusetts) and a Tektronix oscilloscope (Beaverton, Oregon), with a custom audio monitor. Another possibility, although generally more expensive, is to use a commercial multichannel EMG machine, provided that low enough levels of stimulation are available. Although several multichannel machines are available, most are designed for percutaneous stimulation at higher levels (ie, 1 to 300 V or 1 to 50 mA), whereas the levels needed for safe intracranial stimulation are less than 1 V or 1 mA. A qualified biomedical engineer can usually modify such systems to lower the stimulation range, although care must be

taken not to compromise patient safety features. The availability of more channels allows simultaneous monitoring of multiple divisions of the facial nerve independently as well as other cranial motor nerves such as V and XI, which are often involved in acoustic tumor surgery (see later).

Auditory brain stem response

The primary requirements for ABR monitoring are an averaging computer with appropriate high gain, low noise electroencephalogram (EEG) amplifiers, and an acoustic stimulus generator capable of delivering clicks of calibrated intensity, with control of polarity (condensation, rarefaction, or alternating) and repetition rate. Most commercial evoked potential systems meet these essential specifications and can be adapted to use in the operating room. Typical clinical systems include modules that accomplish two- to four-channel, high-gain (100 to 500 K) differential amplification with multipole, band-pass filtering capabilities; acoustic stimulus generation with a stimulus intensity range from threshold to at least 70 to 80 dB normal hearing level (NHL); response averaging with real time display of the evolving averages as well as the raw trace; and permanent record keeping on a disk medium with hard copy printout. Several additional features, however, are desirable for optimum monitoring performance. Key design features of the ideal monitoring system are versatility, portability (and size), and degree of automation.

General technical considerations

Ideally, systems for use during acoustic neuroma surgery would be capable of simultaneous EMG and ABR monitoring. This would require independent control of the time base, stimulation, and averaging parameters for the EMG and ABR channels, features that are not generally available in clinical EMG/evoked potential (EP) machines, which are designed to perform a single test at a time. The only exception that we are aware of at this writing is the Nicolet Viking II (Nicolet Instrument Corporation, Madison, Wisconsin), which has a recently released software package for intraoperative monitoring that allows for such simultaneous protocols. Simultaneous collection of ABRs from left and right ears is also desirable to control for nonspecific effects, such as anesthesia, acoustic artifact, and patient temperature. Again, this feature is not typically available in commercial systems; see under "Monitoring the VIIIth Cranial Nerve" later for details on how such a protocol has been implemented on a custom system.

Degree of automation and size are also important design issues. In general, the more compact and portable the system, the more likely it will be accommodated without major complaints from surgical personnel, especially if it is transported between various operating rooms. Automated data collection protocols, with simultaneous display of baseline traces and recent trends as well as the current trace, facilitate continuous monitoring and assessment of intraoperative changes, although the capability for manual override of automated protocols is desirable.

Surgical monitoring is done in an electrically hostile environment. Every effort must be marshalled to eliminate or reduce 50 or 60 Hz power line interference as well as the frequently broadband noise originating in other operating room equipment (eg, electrocautery, lasers, ultrasonic aspirators, microscopes, anesthesia machines, electrified beds, light dimmers, patient warmers, compression stockings). The 60 Hz notch filters found on most equipment are of limited utility because they remove only 60 Hz sinusoidal activity; more common is noise that recurs at the line frequency but consists of complex spikes with a high fundamental frequency that is not affected by notch filters. Therefore every effort should be made to identify such sources and eliminate their interference if possible. Frequently this can be done by grounding these items, plugging them into a different AC outlet, rerouting cables away from monitoring equipment, or even disconnecting them during crucial periods for monitoring. Unfortunately it is not always possible to eliminate or even identify some sources of interference (at UCSF, one particularly noisy operating room turned out to be upstairs over a magnetic resonance imaging scanner, which generated large pulsatile magnetic fields that were of sufficient strength to cause problems a floor away). Techniques for distinguishing residual artifact from physiologic activity are discussed later under "Monitoring the VIIth and Other Cranial Motor Nerves."

Another important technique is to ensure that the patient is adequately grounded to the recording apparatus and that no alternate ground paths exist. The patient ground should be placed close to the recording electrodes and care taken to obtain a low impedance ground by removing surface oils with alcohol, then rubbing conductive paste into the skin before applying a ground pad. All equipment should be grounded to the same

spot with heavy-duty cables to avoid ground loops. A detailed analysis of these issues is beyond the scope of this article, but an excellent tutorial is provided by Møller [35].

Type and placement of recording electrodes

Either surface or needle electrodes can be used. Surface electrodes are less specific, more prone to artifact, and more timeconsuming to apply, so their use has largely been supplanted by needle electrodes, which can be quickly inserted and taped into place. The most commonly used are platinum needle electrodes designed for EEG recording (Grass E2), which have a larger uninsulated surface than electrodes designed for single-fiber EMG recording and thus are more likely to detect EMG activity arising anywhere in the desired muscle. Prass and Liiders [36] recommend the use of intramuscular hook wire electrodes, which are inserted with the aid of a hypodermic needle; in our experience, these are more traumatic and offer no major practical advantage, so we routinely employ the simpler needle electrodes.

The first uses of facial EMG primarily employed a single recording channel, typically with a bipolar configuration with one electrode in orbicularis oculi and another in orbicularis oris [37]. This montage provides coverage of muscles innervated from both superior and inferior branches of the facial nerve. It has several disadvantages, however, which have led to increasing use of multiple channels. First, the wider the spacing between two electrodes, the greater is the sensitivity to artifact pickup, which in the electrically hostile environment of the operating room can lead to difficult or erroneous interpretations. Second, mechanical trauma to the VIIth cranial nerve frequently causes sustained EMG activity that can make the identification of responses to electrical stimulation difficult. With two or more independent channels, there is a greater likelihood that at least one will be quiet enough to allow stimulation to be used even during high ongoing EMG activity.

For these reasons, we advocate the use of at least two channels of facial EMG as well as recordings from muscles innervated by other cranial nerves. For ABR recording in hearing conservation, one electrode is placed in the ear canal and another on the forehead or vertex; the placement of this electrode is not critical as long as it is near the midline. (See under "Monitoring the VIIIth Cranial Nerve" for further details on ABR recording procedures.) The positioning of the recording electrodes for a suboccipital approach with an effort to preserve hearing are shown in Fig. 1. For translabyrinthine approaches, the same configuration is used, with the exception of the earphone and electrodes for ABR recording.

Monitoring VIIth and other cranial motor nerves

Three main techniques for monitoring cranial motor nerve activity can be distinguished: (1) monitoring ongoing EMG activity for increased activity or changes in activity patterns related to irritation of the nerves by intraoperative events, such as retraction, tumor dissection, use of electrocautery, lasers, and ultrasonic aspiration; (2) identifying and mapping the course of the nerves with activity evoked by intracranial electrical stimulation; and (3) determining nerve functional integrity using evoked EMG methods.

Activity evoked by electrical stimulation

Until the late 1970s, the typical method for facial nerve identification involved someone (usually the anesthesiologist) observing the patient's face for evidence of movement related to intraoperative events or electrical stimulation. Unfortunately in many cases a complete facial palsy resulted even though the face was observed to move with stimulation. It is likely that the high level of stimulation necessary to produce gross movement from a nerve both chronically stretched by the tumor and acutely traumatized during surgery was itself damaging to the nerve and thus contributed to this apparently contradictory outcome. As a result, considerable effort has gone into developing more sensitive measures of facial activity that can be elicited with lower and safer levels of stimulation.

Modalities for monitoring

Several early efforts focused on the use of more sensitive detectors of facial motion, using photoelectric devices, strain gauges, or accelerometers mounted on the face [38,39]. A commercial device is available that uses this technique [40,41]. A low-tech version of this method has been described in a whimsically titled paper "Bells against palsy," [42] which uses small "jingle bells" sutured at the points of maximum excursion of the facial musculature. A technique has also been described

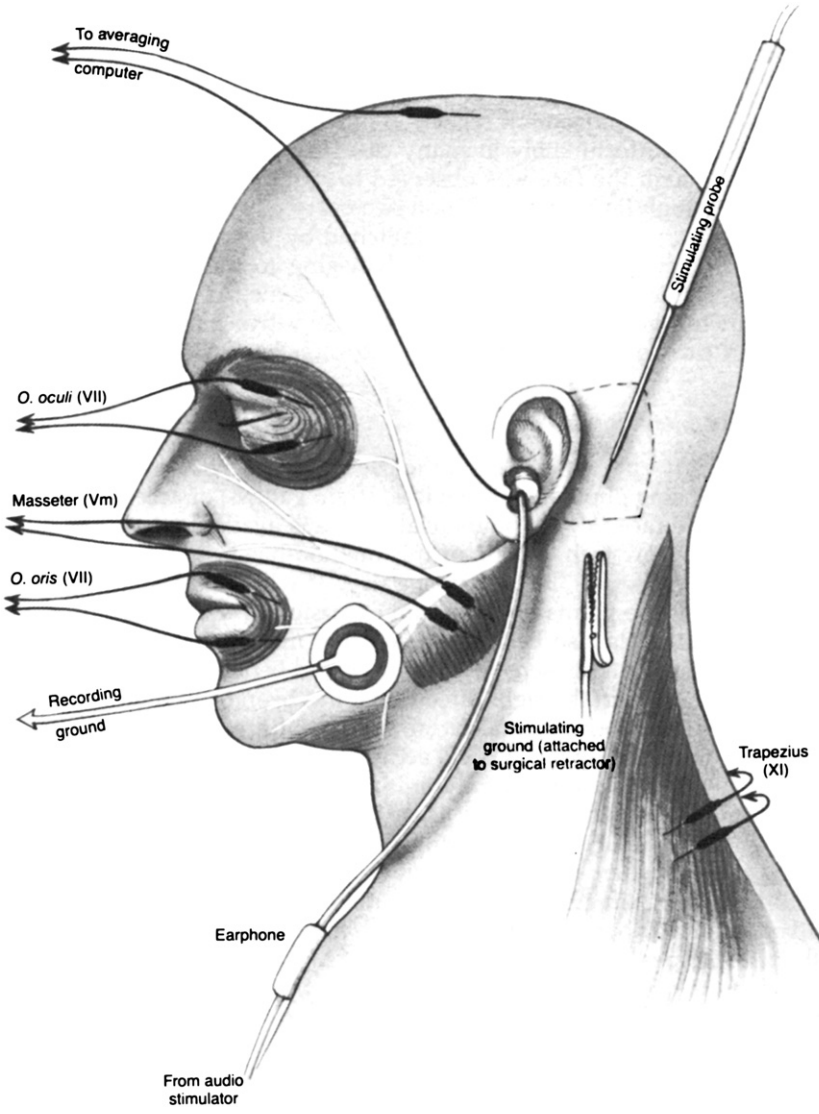

Fig. 1. Diagrammatic representation of electrode placement for monitoring acoustic neuroma surgery with attempted hearing conservation. Pairs of needle electrodes are placed in the following muscles: masseter (Vm); orbicularis oculi and o. oris (VII); and trapezius (XI). Click stimuli from a small transducer are fed into the ipsilateral ear through a foil-covered sponge insert that also serves as a recording electrode, referred to a needle electrode on the vertex. A ground electrode is placed on the cheek. A flexible-tip probe is used to stimulate cranial motor nerves. (*From* Jackler RK, Pitts LH. Acoustic neuroma. Neurosurg Clin North Am 1990;1:199–223; with permission.)

that measures pressure variations in air-inflated rubber sensors that are placed beneath the upper lip [43].

Although a step in the right direction, it has been our experience that techniques for monitoring actual facial movement are less sensitive than those based on recording facial EMG activity, which has become the most widely used technique following the initial report in 1979 by Delgado and colleagues [32]. It should be noted, however, that a major limitation of the EMG method is the difficulty in monitoring during the use of electrocautery, which is a time when the facial nerve is potentially at high risk. The amplitude of the artifact from bipolar cautery can be reduced by using a solid-state unit that operates at a single high

frequency (ie, Davol System 5000, Bard Biomedical Division, Billerica, Massachusetts), rather than a spark-gap unit such as the Codman-Malis (Codman and Shurtleff, Inc., Randolph, Massachusetts), which generates a broad-band noise that is difficult to filter out. The use of techniques based on detection of motion, which are not subject to electrical interference, may provide an important adjunct to EMG monitoring despite their relatively lower sensitivity.

A novel method was described by Prichep and colleagues [44,45] that was based on the crossed auricular reflex, elicited by stimulation of the contralateral ear and recorded from the ipsilateral mastoid-forehead (Fpz). This reflex, which mediates movement of the pinnae in lower animals in response to sounds, is present in vestigial form in humans. It is mediated through a crossed (and uncrossed) pathway with the motor outflow through the facial nerve and appears as a positive-negative-positive complex at a latency of 12 to 16 msec, following the contralateral ABR. This brain stem facial evoked response (BFER) is so small in amplitude that it can be detected only with the use of digital filtering before signal averaging (see "Extension of Techniques to Other Cranial Motor Nerves, Other Posterior Fossa Procedures," later). Prichep and colleagues [44,45] give several examples of changes in the BFER that were associated with surgical manipulation of the VIIth cranial nerve, with recovery of the response when the surgeon reversed the manipulation. Although this novel technique deserves more study, the lack of ready availability of systems incorporating online digital filtering has limited its application, in contrast to EMG-based methods, which can be accomplished with much simpler instrumentation.

Finally, a method that uses recording of compound nerve action potentials (CNAP) from the facial nerve at the stylomastoid foramen after intracranial stimulation has been described by Schmid and colleagues [46]. Similarly, Richmond and Mahla [47] used antidromic recording, stimulating the facial nerve distal to the stylomastoid foramen and recording within the surgical field. These methods have the advantage that they can be used even when the patient is paralyzed, which prevents coughing and allows the use of lower levels of narcotics or other anesthetic agents. Another potential advantage is that the entire nerve can be monitored with a single electrode placed proximal to the divergence of the various branches in the face. On the other hand, the CNAP cannot be easily made audible for direct feedback to the surgeon, and it is not clear whether it is sensitive to facial nerve activity because of injury or manipulation of the nerve. Further investigation of these techniques is warranted.

For the remainder of this section, we focus on techniques using EMG recordings, which are the most commonly used method and the one that we have primarily employed in our own experience.

Types of stimulating electrodes

Both monopolar and bipolar stimulating electrodes have been employed. Theoretically a bipolar electrode should show more specificity and precision of localization because there would be less likelihood of spread of current to adjacent structures than with a distant reference monopolar configuration. In practice, however, this appears not to be the case. The effectiveness of bipolar stimulation is highly dependent on the orientation of the two tips of the probe with respect to the axis of the nerve [48]. The increased bulk of a bipolar electrode makes maintenance of the desired orientation difficult in the close confines of the posterior fossa. A mono-polar electrode does not have this disadvantage and if the stimulus intensity is kept at the appropriate level (see later) can provide spatial resolution of less than 1 mm.

Several types of monopolar electrode have been described. Møller and Jannetta [37] used a simple malleable wire on a probe handle with the distal tip bared of insulation. Prass and Lüders [49] described a similar electrode except that the insulation was continuous to the flush-tip, which could be bent so that only the central portion of the tip contacted the desired tissue, minimizing spread of current to adjacent structures. Yingling and colleagues [50] developed a probe with a flexible Pt-Ir tip, insulated except for a 0.5-mm ball on the end, which can be used to probe within dissection planes or behind the tumor out of direct visualization without the danger of inadvertent damage to delicate neural or vascular structures (Fig. 2). The flexible tip thus frequently allows the facial nerve to be located electrically before its course is apparent visually, and dissection can then proceed in the most advantageous manner to avoid neural damage (Fig. 3).

These probes are all designed for the single purpose of stimulation, and thus dissection must be temporarily halted each time stimulation is performed. Kartush [51] has developed a set of

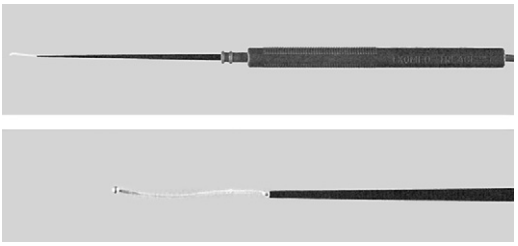

Fig. 2. Flexible-tip probe used for intracranial stimulation. The entire probe and the flexible wire are insulated except for the 0.5-mm ball on the end in order to achieve localized stimulation.

dissecting instruments that are insulated to just above the cutting surface and can be interchangeably connected to the electrical simulator, allowing simultaneous dissection with constant stimulation. According to Kartush [51], sharp dissection, as opposed to traction or prolonged dissection, may evoke little or no EMG response even with complete transection of the nerve. These "stimulus dissectors" are of particular value in removing the last portions of the tumor capsule, which are closely adherent to the nerve. They can also be used for intermittent stimulation during dissection in other regions because they can easily be electrified on desire.

Constant voltage versus constant current

The question of whether to use constant current or constant voltage stimulators has been a source of continuing controversy. Because transmembrane current is ultimately the effective stimulus for a nerve axon, constant current stimulators have generally been preferred for transcutaneous nerve stimulation, since the current delivered to the nerve is maintained at a constant level despite changes in electrode impedance. The same considerations may not apply, however, for intracranial stimulation, in which the degree of shunting of the nerve by blood, cerebrospinal fluid, or irrigant may vary

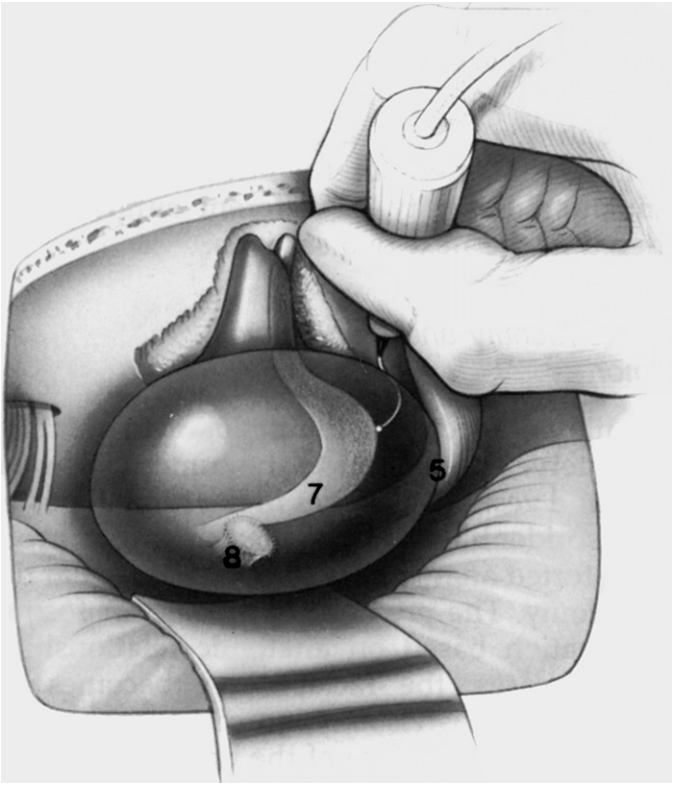

Fig. 3. Surgical view of large acoustic neuroma (suboccipital approach) showing use of flexible-tip probe to locate the facial nerve on the medial surface of tumor, out of direct view. Tumor is drawn as if transparent to show details of anatomy on the hidden surface.

widely from one second to the next. Møller and Jannetta [37] have articulated the case for constant voltage stimulation. Consider a nerve bathed in a conducting fluid. Much of the current delivered through the stimulating electrode flows through the relatively lower impedance fluid, rather than through the nerve. A constant current stimulator may thus have to be turned up to a relatively high level to depolarize the nerve effectively. If the fluid is then suddenly removed (ie, by suction) or a drier portion of the nerve is contacted, the same total current now flows through the nerve, with potentially damaging consequences. The current delivered to a nerve from a constant voltage stimulator depends on the impedance of the nerve itself, according to Ohm's law, regardless of the amount of shunting. A varying total current is delivered as the overall nerve/fluid environment changes, but the current delivered to the nerve itself, paradoxically, is more constant with a constant voltage stimulator.

On the other hand, Prass and Lüders [49] argued in favor of constant current stimulation, claiming that their flush-tip probe design eliminates the problem of current shunting by fluids. Kartush and colleagues [48] offered some data to confirm this view; they compared bare-tip with flush-tip probe designs and showed that significantly greater response amplitudes were obtained with flush-tip stimulators. Note, however, that these results were obtained with constant current stimulators; it is not clear that the same results would have been obtained with constant voltage devices.

Further research, preferably in animal models, is necessary to resolve this debate finally. In the meantime, most groups will probably continue to use the method with which they have the most experience and feel most comfortable. Whether constant voltage or constant current is used, the question still remains as to what actual level of stimulation is most appropriate. Although some have argued for a "set it and forget it" approach, we believe that more useful information can be gained by varying the stimulation intensity in different surgical contexts. These issues are considered in the next two sections.

Use of stimulation to identify and map nerves in relation to tumor

The primary utility of electrical stimulation is to identify the facial or other cranial motor nerves in relation to the tumor. The relations among the facial, cochlear, and vestibular nerves in the normal posterior fossa are relatively constant, so identification produces less of a problem in cases with relatively undistorted anatomy such as microvascular decompression or vestibular neurectomy. The presence of a posterior fossa tumor, however, makes identification based on anatomic relationships difficult or impossible. In many cases, the facial nerve becomes stretched and widened to the extent that it is visually indistinguishable from arachnoid tissue, and vasculature on the surface of the brain stem may even be seen through a gossamer-thin, yet functionally intact nerve. In such situations, often the only way to identify and trace the facial nerve is with electrical stimulation.

The procedures we use at UCSF are as follows. First, the integrity of the stimulating and recording system must be confirmed at the earliest opportunity to avoid potentially catastrophic false-negative results. The presence of a stimulus artifact is *not* an infallible test; it is sometimes possible to see a stimulus artifact with only one lead connected, either the anodal return or the cathodal stimulator. Conversely, the *absence* of any artifact is usually indicative of an incomplete connection somewhere in the system. To avoid this ambiguity, we try to confirm the functional integrity of the entire system before commencing tumor dissection from the VIIth cranial nerve. In a suboccipital approach, this can usually be done by stimulating the XIth cranial nerve at the jugular foramen as soon as the dura has been opened and the cerebellum retracted, confirming the presence of a response in the trapezius muscle. Fortunately this is usually possible before tumor resection begins except in very large acoustic tumors. With monopolar constant voltage stimulation, using cathodal pulses of 0.2 msec duration at a rate of 5 to 10/sec, the threshold for obtaining an evoked EMG response from normal bare nerves is usually between 0.05 and 0.2 V, averaging about 0.1 V. (Thresholds reported for constant-current stimulation have ranged from $<$0.1 to 0.5 mA.) If the Xlth cranial nerve cannot be visualized at the outset, the stimulating electrode can be placed directly on any visible muscle and a direct muscular response obtained, although this requires a higher level of stimulation than is necessary to obtain an EMG response from nerve stimulation. In translabyrinthine procedures, the facial nerve can be stimulated within the mastoid bone before the tumor is exposed, although the threshold is higher, depending on the thickness of the overlying bone.

Once functional integrity has been verified, we then attempt to locate and stimulate the facial nerve. In smaller tumors (cerebellopontine angle component of 1 cm or less), the nerve can usually be visually identified and confirmed with stimulation before dissection begins. Once the threshold has been established, the voltage can be increased to 3 × threshold and the stimulator used to sweep across the exposed surface of the tumor to confirm that there are no facial nerve fibers in the area to be dissected. In larger tumors, the location of the facial nerve may not be immediately apparent. In this case, we start with 0.3 V and map the accessible region and, if no response is obtained, try again at 0.5 and 1 V. We do not exceed a stimulation level of 1 V; if no response is obtained at this level, it can be safely assumed that the facial nerve is not in the immediate vicinity, and dissection can proceed.

The dissection is begun at the brain stem end of the tumor, attempting to identify the facial nerve at the brain stem root entry zone before dissecting the lateral aspect of the tumor in or near the internal auditory canal. This is because the most common site of injury to the facial nerve is just outside of the porus acusticus, where it frequently is compressed against the temporal bone by the tumor. If this region is dissected first, the nerve may be compromised to the extent that it is not possible to identify it at the brain stem with electrical stimulation because of a conduction block in the more distal segment. Once the facial nerve is identified at the brain stem and traced as far laterally as possible, with the tumor-nerve interface under direct vision, the dissection can move to the lateral end, working back toward the mid–cerebellopontine angle until the nerve is freed from both ends.

As dissection proceeds, the stimulator is used repeatedly to scan the tumor capsule for the presence of facial nerve fibers as the tumor is mobilized, using stimulus intensities as already described. The flexible tip probe already described is particularly useful in this regard because it can be used to probe within dissection planes and often identify the general location of the nerve before it is visually apparent. The great advantage of the flexible tip is that it can be used to probe portions of the capsule that are out of view on the far side of the tumor, since the VIIth cranial nerve usually courses on the anterior surface of the tumor and the common surgical approaches are from posterior. Once a response is obtained, stimulus intensity is reduced to 0.1 or 0.15 V, and the region where responses are obtained is narrowed. Once the nerve is in sight, the electrode is placed directly on the nerve and a threshold is obtained. Further stimulation for mapping the location of the nerve is carried out at approximately 3× this threshold, which should be periodically rechecked as dissection proceeds.

With monopolar stimulation, spatial resolution of electrical mapping is partly determined by stimulus intensity; thus, for the most accurate localization, the stimulus is kept at a relatively low level as just described. This allows a spatial resolution of less than 1 mm, so that the facial nerve can be easily distinguished from the adjacent vestibulocochlear complex. On the other hand, if the immediate aim is to confirm that the nerve is *not* in an area about to be cut or cauterized, higher levels of stimulation up to 1 V can be used to reduce the likelihood of false-negative results. As more and more tumor is removed, the course of the facial nerve can be mapped from brain stem to internal auditory canal. It is important to note that while the nerve may be relatively cylindrical at each end, it is frequently compressed by the tumor in the cerebellopontine angle to such an extent that it may be a broad, flat expanse of fibers splayed across the surface of the tumor, which can be identified and distinguished from arachnoid tissue only with electrical stimulation.

Another important point is that other cranial motor nerves may often be encountered in unexpected locations, particularly in larger tumors. By noting the distribution and latency of response in the various channels, it is usually possible to distinguish among several nerves and thus gain more insight into the anatomic relationships. The latency of the facial response to stimulation of the VIIth cranial nerve in the cerebellopontine angle (measured to the onset of the first inflection) is 6 to 8 msec in an intact nerve. The exact latency varies depending on the site of stimulation from the brain stem root entry zone to the internal auditory canal. Stimulation of the motor fibers of the trigeminal nerve (Vm) produces EMG activity in the masseter and temporalis muscles; because of the proximity of these muscles to the facial muscles, there is typically considerable crosstalk between channels, so that activity elicited by stimulation of the VIIth cranial nerve may be volume conducted to the masseter channel, and that from stimulation of Vm may be seen in facial channels. Responses to Vm versus VIIth cranial nerve

stimulation, however, can be distinguished from one another by their different onset latencies. Stimulation of Vm produces EMG responses that are of a considerably shorter latency (3 to 4 msec to onset) than those produced by VIIth cranial nerve stimulation (6 to 8 msec), allowing these nerves to be distinguished despite overlap in the responding channels. (Mnemonic: CNVII about 7, CNV less than 5). As already mentioned, stimulation of the XIth cranial nerve produces responses restricted to the trapezius channel; because of the greater distance, there is generally no crosstalk between channels with XIth cranial nerve stimulation. Finally the VIth cranial nerve may occasionally be encountered. Stimulation of the VIth cranial nerve produces activity that can be seen as a short latency response (~2 msec) restricted to the orbicularis oculi channel, where it is seen by volume conduction from the lateral rectus. (One of the bipolar electrodes in this pair should be positioned near the lateral canthus to optimize pickup of this response, which is of smaller amplitude than those recorded directly from the lateral rectus muscle.) These patterns are indicated schematically in Fig. 4.

Use of stimulation to assess functional status of nerves following tumor removal

In addition to localizing and mapping the course of cranial nerves in relation to cerebellopontine angle tumors, electrical stimulation may also be used to determine changes in the functional status of these nerves and thus may help to predict postoperative function. We have found that the ability to elicit facial EMG responses by low-threshold stimulation of the VIIth cranial nerve at the brain stem after total tumor resection is usually predictive of good postoperative function, although transient facial palsies may still be seen. Conversely, a substantially elevated threshold or the inability to elicit a response with stimulation up to 1 V is generally associated with significant facial dysfunction, although if the nerve is anatomically preserved there is still the possibility of return of function as the nerve fibers regenerate.

Other methods have been proposed to quantify further VIIth cranial nerve status after acoustic tumor removal. Harner and colleagues [52] stated that a decrease in the amplitude of the compound muscle action potential (CMAP) with

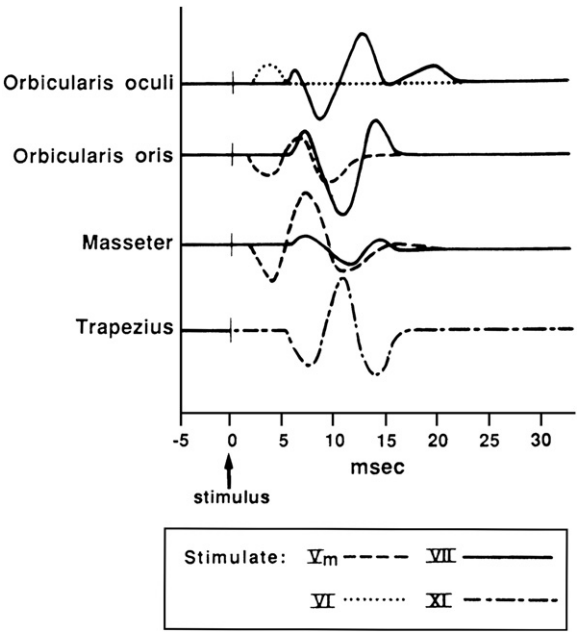

Fig. 4. Schematic representation of responses obtained in four-channel montage (see Fig. 1) with intracranial stimulation of cranial nerves (CN)Vm, VI, VII, and XI. Despite crosstalk in CNV and VII channels, these nerves can be clearly distinguished by the shorter latency of responses to Vm stimulation. Stimulation of CNVI produces a short latency response localized to the orbicularis oculi channel, due to volume conduction from the nearby lateral rectus; responses to CNXI stimulation are restricted to the trapezius channel (see text for details). (*From* Jackler RK, Pitts LH. Acoustic neuroma. Neurosurg Clin North Am 1990;1:199–223; with permission.)

supramaximal stimulation was associated with an increase in the degree of facial weakness. This is presumably due to a decrease in the proportion of facial nerve fibers remaining functional after tumor removal. Schmid and colleagues [46] have suggested calculating intracisternal latency intervals by noting the difference in latency between EMG responses elicited by stimulation at the brain stem versus internal auditory canal. They obtained a mean intracisternal latency interval of 0.24 msec; three patients with transient postoperative facial palsies had values of 0.5 to 0.54 msec. A combination of methods based on preoperative and postoperative comparisons of threshold, latency, and CMAP amplitude may provide a better predictive index of postoperative facial nerve function.

Spontaneous and mechanically evoked activity

In addition to EMG responses elicited by electrical stimulation, spontaneous EMG activity and EMG responses related to intraoperative events are also frequently encountered. Patients with significant preoperative facial deficits may exhibit tonic EMG activity even before the craniotomy is performed; this may decrease as the nerve is decompressed with opening of the dura and draining of cerebrospinal fluid. Virtually all patients exhibit at least some mechanically evoked facial EMG activity during tumor dissection, retraction, irrigation, or other intraoperative events. Such activity is frequently the earliest indicator of the location of the facial nerve, which can then be more precisely localized with electrical stimulation as described earlier. It is important to note that a simultaneous increase in spontaneous EMG activity on all channels is unlikely to result from localized dissection. When such a generalized increase occurs, the anesthesiologist should be notified immediately because this is frequently an early indication that the depth of anesthesia is too light, and overt patient movement often occurs within a few seconds after the increased EMG activity.

Distinguishing artifacts from electromyographic activity

A number of causes other than muscle activity can produce activity on the oscilloscope screen or loudspeaker, and it is important to distinguish them from true EMG activity. Some of these are obvious artifacts associated with electrocautery equipment, ultrasonic aspirators, and lasers and can be readily identified by their association with use of these devices and generally large amplitude. Such artifacts can be rejected from the audio monitor by use of interlock devices or squelch circuitry, which mutes the audio during their use. More troublesome are smaller artifacts produced by bimetallic potentials as a result of contact between surgical instruments made of different metals; because these may be associated with similar intraoperative events as those producing true EMG responses, they can be difficult to distinguish. Some useful criteria include the fact that artifacts are typically higher in frequency content than EMG activity and thus sound more "crackly" than true EMG activity, which has more of a "popping" sound, and the tendency for artifacts to appear simultaneously on several channels, which is unlikely for an EMG response. Experienced monitoring personnel are in a better position to make such decisions than surgeons who are focused on the operative field.

Phasic versus tonic electromyography

Prass and Lüders [36] distinguished two types of EMG activity associated with intraoperative events. The phasic "burst" pattern, characterized by short, relatively synchronous bursts of motor unit potentials, was thought to correspond with a single discharge of multiple facial nerve axons. Such activity was associated with direct mechanical nerve trauma, free irrigation, application of Ringer's-soaked pledgets over the facial nerve, and electrocautery and could be easily associated with such events. In contrast, tonic or "train" activity, episodes of prolonged asynchronous grouped motor unit discharges that could last up to several minutes, were most commonly associated with facial nerve traction, usually in the lateral to medial direction. Such train activity was further divided into higher frequency trains (50 to 100 Hz), which were dubbed "bomber potentials" because of their sonic characteristics, and lower frequency discharges (1 to 50 Hz), which were more irregular and had a sound resembling popping popcorn. The onset and decline of "popcorn" activity was more gradual than the more abrupt onset and decline of "bomber" activity.

It should be noted that, particularly in larger tumors in which there is significant compression of the facial nerve, tonic EMG activity may be observed even in baseline recordings. This can complicate the detection of changes in EMG activity associated with intraoperative events as well as the use of stimulus-evoked EMG for nerve

identification and mapping. As discussed earlier (under "Technical Issues"), the use of multiple channels can help in identification of changed patterns of tonic activity or of stimulus-evoked activity.

Does tonic electromyographic activity imply nerve injury?

Prass and Lüders [36] suggested that episodes of "burst" activity were probably due to the mechanoreceptor properties of nerve axons, as they tended to be directly associated with intraoperative compression of the facial nerve. Such mechanically evoked activity was distinguished from injury discharges and thought to have no necessary relationship to nerve injury. In fact, they point out that the ability to elicit burst activity with mechanical stimuli indicates functional integrity of the nerve distal to the site of stimulation and that a trend of decreasing burst activity despite continued mechanical stimulation may indicate nerve injury has already occurred.

In contrast, they argued that frequent and prolonged "train" responses, especially of the "bomber" type, were more likely to be associated with either nerve ischemia or prolonged mechanical deformation and thus potentially correspond to injury potentials and poor postoperative function. Daube and Harper [53] have described cases in which prolonged train activity was associated with inability to stimulate the nerve electrically after tumor removal and lack of postoperative facial motility. As with the various methods for determining the functional integrity of the facial nerve with electrical stimulation, described earlier, more work is necessary to associate such intraoperative events firmly with ultimate clinical outcome.

Extension of techniques to other cranial motor nerves, other posterior fossa procedures

The methods described for facial nerve monitoring are easily adaptable to virtually any cranial motor nerve by placing recording electrodes in the appropriate muscles. We have, for example, monitored the IIIth, IVth, and VIth cranial nerves during removal of cavernous sinus tumors with electrodes in the extraocular muscles and IXth, Xth, XIth, and XIIth cranial nerves during a variety of skull base procedures with electrodes in the soft palate, false vocal cords, trapezius, and tongue, respectively. Both mechanically and electrically elicited activity may be observed in such cases, just as described for the facial nerve, with the exception that the characteristic latencies of EMG responses differ depending on the particular nerve studies. For further details on such procedures, see Møller [35], Desmedt [54], and Lanser and colleagues [55].

Effects of neural monitoring on clinical outcome

Several studies have appeared comparing postoperative preservation of facial nerve function in series of cases with and without facial nerve monitoring. Leonetti and colleagues [56] compared 23 unmonitored cases with 15 monitored cases of infratemporal approaches to the skull base, all involving rerouting of the facial nerve in the temporal bone. In the unmonitored group, 11 of 23 (48%) showed a House grade V or VI facial palsy [57] at discharge, whereas none of the monitored group fell into this category, and 12 of 15 (80%) were in grade I or II. Niparko and colleagues [58] reported the outcome for 29 monitored patients with translabyrinthine acoustic neuroma removals versus 75 unmonitored cases using the same approach. A nonsignificant trend for better facial function in the monitored group was seen at the end of the 1st postoperative week. One-year follow-up revealed that satisfactory facial function was significantly associated with monitoring ($p < 0.05$); analysis of subgroups showed this effect to be significant only for tumors larger than 2 cm, although there was a nonsignificant trend ($p = 0.08$) in the same direction for smaller tumors.

The best-controlled study to date is that of Harner and colleagues [52] who reported outcome data from 91 consecutive acoustic neuroma removals. The unmonitored control group consisted of 91 patients selected from a larger pool of 173 cases to match the monitored group on the basis of (in order): (1) tumor size, (2) most recent year of operation, and (3) age of the monitored patient. The resulting groups were closely matched for tumor size (median 3 cm) and age (median 54 yr). The facial nerve was anatomically preserved in 92% of the monitored group and 84% of the unmonitored group, a nonsignificant difference. The most meaningful comparisons were at 3 months and 1 year postoperatively. At 3 months, 46% of the monitored and 20% of the unmonitored group had House grade I function; 15% of the monitored and 35% of the unmonitored group had a House grade VI palsy. At 1 year, 45% of the monitored versus 27% of the unmonitored group had no deficit (House grade 1), whereas only 2% of the monitored and

6% of the unmonitored group had no facial function whatsoever (House grade VI).

A potential confounding factor in all these studies is the fact that the unmonitored cases were always operated on earlier than the monitored ones, raising the possibility that the improvements in outcome could be due simply to greater experience on the part of the surgeons. Harner and colleagues [52] point out, however, that part of the surgeon's technical improvement is directly attributable to the use of monitoring. As surgeons become more aware of the types of maneuvers that produce EMG discharges, they naturally adapt their operative technique to avoid such maneuvers whenever possible. Intraoperative monitoring may thus contribute to improved facial nerve preservation in more than one way. A quote from Harner probably typifies the attitude of most surgeons who have used intraoperative facial nerve monitoring: "I don't think I could convince anybody at our institution (the Mayo Clinic) with experience to give up monitoring under any circumstances." Similarly our surgical team at UCSF refuses to proceed with an acoustic neuroma operation unless cranial nerve monitoring capability is available.

Monitoring the VIIIth cranial nerve modalities for monitoring

Although the VIIIth cranial nerve is the cranial nerve at greatest risk during the majority of cerebellopontine angle surgeries, it is also the most likely to have significant preoperative deficits and is the least important to preserve since vestibular and auditory function remain relatively intact with only one surviving ear. Monitoring of VIIIth cranial nerve function during posterior fossa surgeries is most appropriate for (1) smaller acoustic neuromas (especially those that are confined to the medial portions of the internal auditory canal) with well-preserved hearing (slight pure tone loss and good to excellent speech discrimination scores), (2) nonschwannoma posterior fossa tumors (eg, meningiomas), or (3) microvascular decompression of posterior fossa cranial nerves [35]. In larger acoustic neuromas in which hearing conservation is not a realistic goal but in which the tumor is large enough to displace the brain stem significantly with possible collapse of the 4th ventricle or for other surgical procedures such as vascular aneurysms of the brain stem or resection of arteriovenous malformations, monitoring of auditory function of the opposite (contralateral) ear may be useful in detecting brain stem compromise [50,59,60].

Only the auditory portion of the VIIIth cranial nerve is actually monitored, employing either *far-field* or *near-field* techniques. The two monitoring methods are distinguished by the proximity of recording electrodes to the VIIIth cranial nerve. In far-field methods, electrodes are positioned at a distance from the VIIIth cranial nerve, usually on the scalp surface. The most common method of far-field recording is the scalp-recorded ABR [33]. widely used in clinical diagnosis of auditory system dysfunction. In contrast, near-field methods employ the placement of one or both active electrodes near or actually on the VIIIth cranial nerve. The most commonly used near-field recording in surgical monitoring is the auditory whole CNAP, but transtympanic recording of the cochlear microphonic potential in conjunction with the auditory CNAP has also been used. There are distinct advantages and disadvantages to the use of both near-field and far-field techniques; state of the art monitoring of auditory function may include the use of both techniques during different stages of posterior fossa surgeries.

Intraoperative auditory nerve monitoring techniques used in posterior fossa surgery were first described in 1978 by Levine and colleagues [61]. Since the initial report, other authors have extended the intraoperative use of ABR and CNAP measures and concluded that they were a highly reliable and efficacious test of VIIIth cranial nerve function during posterior fossa craniotomies [62–69]. Further work over the last decade has detailed the application of these monitoring techniques to include a wide range of surgical procedures of the posterior fossa, including acoustic neuromas; other cranial motor neuromas; cerebellopontine, petrous apex, and transtentorial meningiomas; microvascular decompression of the Vth, VIIth, VIIIth, IXth, and Xth cranial nerves, and restricted neurectomies of the vestibular portion of the VIIIth cranial nerve and 2nd and 3rd divisions of the Vth cranial nerve [35,60,70–78].

The ABR is typically elicited by repetitive click stimuli, with 1000 to 2000 trials at repetition rates of 8 to 33/sec averaged to produce a replicable response. The relatively large number of trials is necessary because of the small size of the far-field potentials (200 to 500 nV) in relation to ongoing EEG and EMG activity. Averaged ABRs consist of a sequence of 5 or more reproducible waves occurring within the 1st 10 msec after the

stimulus. Of these, waves I, III, and V are the most commonly used. Wave I reflects the compound action potential generated in the distal segment of the cochlear nerve, wave III is probably generated by 2nd order neurons exiting the cochlear nucleus complex, and wave V originates higher in the brain stem, probably at the level of the upper pons bilaterally [79].

In normal subjects, the latency intervals between these peaks are very consistent, and thus increases in latency of the interpeak intervals are evidence of compromised transmission between the sites of generation of each wave. Normal interpeak latencies are approximately 2.1 msec (I to III); 1.9 msec (III to V); and 4.0 msec (I to V). The absolute latency of wave I may be affected by peripheral factors such as conductive or cochlear hearing loss, but the interpeak latencies are usually not affected. In contrast, acoustic neuromas typically produce an increase in the I to III interpeak latency (and consequently I to V as well) owing to compromise of the cochlear nerve in the cerebellopontine angle. The III to V interpeak latency is generally normal in smaller tumors but may also become increased if the tumor is large enough to cause significant compression of the brain stem. In many cases, the waveforms are so degraded by the presence of the tumor that only wave V, the most prominent component, may be recordable. In such cases, the interaural time difference in the absolute latency of wave V (IT5) may be used to help establish a diagnosis of unilateral retrocochlear dysfunction. For a more thorough discussion of ABR basic techniques and their diagnostic use, see Moore [80], and Jacobson [81].

Employing ABR and related techniques in surgery presents some novel problems not encountered in normal clinical application. The goal for the balance of this section is to detail important technical considerations for the successful use of these procedures in a surgical setting as well as discuss how these protocols have been employed by various surgical teams to improve the chances for hearing preservation in acoustic neuroma surgery.

Methodologic considerations

In addition to the general methods for ABR recording, there are special considerations for equipment used in the operating room, the most important of which are as follows:

1. Standard audiometric earphones are not useful in the operating room because they are too large and would interfere with surgical access. Instead, it is necessary to use miniature earphones that fit within the ear and do not compromise the surgical field. Møller and colleagues have successfully used small in-the-ear transducers designed for use with portable cassette players (Radio Shack). Inexpensive earphones, however, may vary considerably in the acoustic waveform delivered for a given electrical input [82], with consequent changes in ABR latencies. Stimulus artifact also poses a problem since such earphones are not shielded. A better solution is to use higher-quality transducers such as Etymotic Tubephones (Etymotic Research, Elk Grove Village, Illinois), which duplicate the frequency response characteristics of standard audiometric ear phones. These can be placed at the level of the neck with acoustic output passed along a short tube that terminates in an ear insert foam plug. Use of such earphones greatly reduces stimulus artifact production owing to the distance between differential leads and the earphone's speaker. If the foam plug is covered with a conductive gold foil (TipTrode, Nicolet, Madison, Wisconsin), it can also serve as a recording electrode. In addition to providing acoustic isolation from operating room background noise, this electrode provides definition of wave I as good as that obtained with a needle electrode in the ear canal, owing to closer proximity to the distal VIIIth cranial nerve than the earlobe or mastoid electrodes routinely used in clinical ABR testing.
2. It is desirable to use a protocol in which stimuli can be delivered to either ear in an alternating fashion, with the recording montage automatically switched to record from the electrode at the currently stimulated ear, with the vertex or forehead electrode always connected to the other input of the differential amplifier. This allows immediate comparison of the ABR from the operated side with that obtained from the contralateral ear, a useful control for nonspecific effects on the ABR from factors such as anesthesia and temperature. Most commercial systems do not have the capacity for such interleaved recording. One of us (JNG), however, has developed such

a system based on a portable IBM-compatible computer, which presents stimuli and records responses in the following manner. The ears are stimulated alternately with 100 μsec, alternating polarity square waves. EEG activity for 15 msec following each stimulus presentation is recorded from electrodes at the vertex and the stimulated ear, automatically selected by the computer. Alternate trials from each ear are accumulated in separate memory buffers, providing an automatic replication for assessment of reliability. The cycle repeats at 33.3 stimuli/sec/ear, so that duplicate 1500-trial averages for each tested ear are obtained roughly every 2 minutes, assuming minimum artifact. With the simple addition of software macro capabilities, this monitoring scheme can be repeated endlessly, with automatic data storage to disk for permanent documentation and to a printer for hardcopy trend analysis.

3. Finally it is imperative to take whatever steps are necessary to minimize or control both electrical and acoustic artifacts. Electrical artifacts, which are of concern for both EMG and ABR monitoring, have already been discussed under "Technical Issues." In addition, acoustic interference becomes a significant issue when either ABR or direct VIIIth cranial nerve action potentials are recorded. Drilling of the skull, especially with high-speed drills used in opening the internal auditory canal, can pose serious obstacles to appropriate interpretation of auditory nerve monitoring results as a result of acoustic masking, which can degrade or even obliterate the ABR or CNAP.

Unfortunately the VIIIth cranial nerve and especially the inner ear itself are at great risk during this period in typical suboccipital approaches. Interpretations of these results can be complicated by erroneous conclusions based on acoustic masking from drilling. For surgical teams who wish to conserve hearing in selected patients, it is important to control for masking effects in two simple ways. First, simultaneous monitoring of the opposite, unoperated ear can serve as a control. Although it is unlikely that ABR wave I through V latency differences and overall peak amplitudes will be equivalent in the two ears, interpretation of relative differences can be a great help. It is also likely that the operated ear may be masked to a greater degree than the opposite ear as a result of the closer proximity of the drilling.

Second, deliberate halting of drilling can be done when it is necessary to obtain unmasked results from the test ear. Unfortunately time becomes a major concern in this regard, and techniques have to be employed to reduce greatly data collection times. This is a primary motivation for the emphasis on near-field recording techniques when attempts are made to conserve hearing. Comparing the amplitude and latency of the Nl response collected over the course of temporal bone drilling can be done in seconds because averaged CNAP recordings can be obtained within 5 to 10 seconds compared with much greater times (1 to 2 minutes) for ABR averages. Therefore the capability to perform both far-field and near-field recordings during attempts at hearing conservation in acoustic neuroma surgeries is desirable.

Routine uses of auditory brain stem responses and VIIIth cranial nerve compound action potentials during acoustic neuroma surgery

Auditory brain stem response

Stimuli for eliciting auditory brain stem response. Although ABRs can be elicited by many auditory stimuli, including clicks and tone pips of various frequencies, broad-band clicks produced by square wave stimulation are the most often employed. Although the electrical waveform is a simple pulse, the mechanical characteristics of most earphones produce an acoustic output with energy distributed over a wide portion of the high-frequency auditory spectrum. Such a stimulus is desirable for clinical use because it activates a wider range of the cochlea than pure tone pips, increasing the signal amplitude and minimizing the potential problem of stimulating at a frequency corresponding to an individual's major hearing deficit.

Typically stimuli consist of 100-*μsec* duration pulses delivered at a rate of from 8 to 33 stimuli/sec. More rapid rates produce a faster updating of the average and thus are desirable for use during surgery, when timely feedback is important. Slower repetition rates have the advantage of increasing response amplitude, however, and may be necessary in cases with poor signal to noise ratio Rarefaction clicks or alternating polarity stimuli are most often used. Alternating stimulus polarity has the advantage of eliminating residual stimulus artifacts when insert

Tubephones are used. Stimulus intensity is always maintained at a high level, usually at 105-dB peak sound pressure level (SPL) or higher, to obtain the best possible signal to noise ratio (this is at least 70 dB above subjective click threshold levels). Although this would be an unacceptably high level for truly continuous stimulation, the short duty cycle with 100-μsec clicks (roughly 0.3% at 30/s) does not appear to pose a problem; we are not aware of any reports of compromised hearing traceable to ABR recording over extended periods. For ABRs, each ear is ideally tested separately in an alternating fashion with averages superimposed to eliminate spurious interpretations caused by noise. For VIIIth cranial nerve CNAPs, only delivery of stimuli to the ear ipsilateral to the surgical site is necessary.

Auditory brain stem response recording. For ABRs, electrodes are placed at the vertex (Cz) or at any point along the midsagittal plane between midforehead (Fpz) and vertex (noninverting lead) as well as in the ipsilateral ear canal or earlobe (inverting lead). The ground may be placed at any convenient location, usually on the forehead contralateral to the surgical field. Electrodes themselves can be scalp electrodes, paste electrodes, or subdermal needle electrodes. Needle electrodes have the advantage of more discrete placement, more stable impedance over the long course of surgery, and equal impedance across differential leads.

For auditory CNAPs, in which the electrode is intentionally placed directly on the nerve, electrodes are one of three basic types. Two are monopolar electrodes with the active electrode either a cotton wick sutured on the end of a malleable wire or a flexible ball-tipped wire (usually platinum-iridium); in either case, the electrode is held in place by a separate adjustable clamp, the cerebellar retractor, or brain cotton or bone wax restraint [35,83–85]. The reference electrode is usually connected to the wound musculature. To take maximum advantage of the near-field electrode to increase signal to noise ratios, accurate placement in close proximity to the auditory portion of the VIIIth cranial nerve at the root entry zone is essential for quick turnaround of averaged CNAPs, especially if the patient has a significant preoperative hearing deficit (Fig. 5).

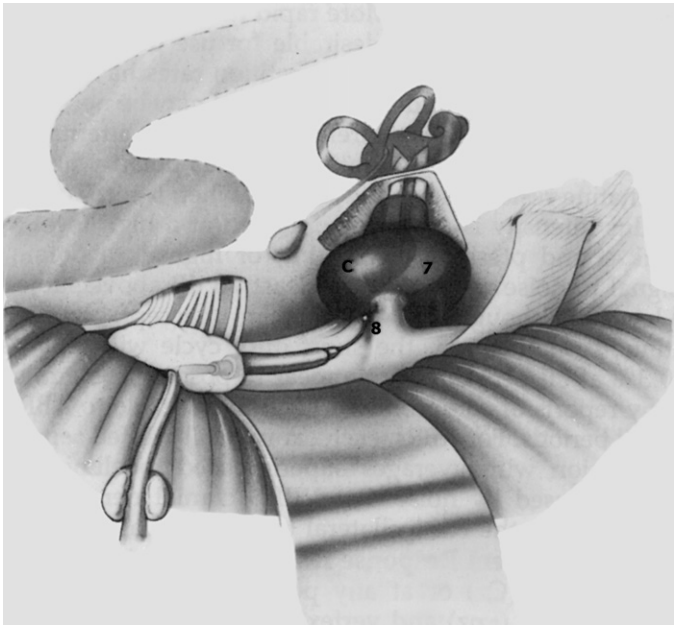

Fig. 5. Surgical view of suboccipital approach to small acoustic neuroma showing flexible-tip electrode in place on cochlear nerve at brain stem for recording of VIIIth cranial nerve compound action potentials. A malleable solid-core wire, attached rigidly outside the craniotomy, is attached to the flexibletip and used to hold the tip in place, slightly indenting the surface of the nerve.

The second type of near-field electrode is a true bipolar electrode with two closely spaced contacts, both of which are positioned on or near the auditory portion of the VIIIth cranial nerve near the root entry zone. In principle, such a bipolar arrangement should provide greater spatial selectivity than a monopolar electrode. In practice, however, this is not necessarily the case; the amplitude and waveform of the CNAP may change unless the orientation of the electrode in relation to the nerve is held absolutely constant. Also, bipolar electrodes are inherently bulkier and thus more difficult to position correctly within the tight confines of the posterior fossa. In our experience, the spatial selectivity of a monopolar electrode is more than adequate to distinguish cochlear from vestibular components of the VIIIth cranial nerve since only the cochlear division produces a CNAP in response to acoustic stimulation.

Some of these techniques have been combined in useful ways. For example, the use of a transtympanic electrode on the promontory of the cochlea has been coupled with a scalp electrode on the midline of the forehead or vertex to increase signal to noise ratios and avoid placement of a near-field electrode on the VIIIth cranial nerve root entry zone [74,86]. Another variation of this scheme is placement of one electrode of a bipolar pair at the root entry zone and the other on the cochlear promontory, thus allowing simultaneous CNAP and cochlear microphonic or summating potential recording [83]. In this case, rarefaction pulses rather than alternating polarity pulses are used as acoustic signals. Electrodes are flexible, ball-tipped bipolar, or concentric bipolar. The ground remains the same as that used for ABRs. Possible complications with either of these techniques include infection or cerebrospinal fluid leaks owing to the violation of the eardrum. The ability to assess independently cochlear versus neural function, however, may add new and potentially useful information. For example, if the cochlear microphonic or summating potential is preserved but the CNAP lost, the likely site of damage is the nerve itself, with little that can be done to reverse the deficit. On the other hand, if the cochlear microphonic and summating potential are also lost, transient cochlear ischemia is another possible mechanism, which is potentially reversible by raising systemic blood pressure or administering vasodilators.

Finally we have occasionally encountered patients with good to excellent hearing, using bipolar electrodes with both contacts placed in the near-field directly on or adjacent to the VIIIth cranial nerve, in whom the CNAP signal is so large that the need for signal averaging is eliminated. (We are not aware of any such cases in the literature.) In these fortunate cases, changes in CNAP amplitude can be played over a loudspeaker for feedback to the surgeon in real time, analogous to how spontaneous EMG signals are monitored for cranial motor nerves.

Cautions in interpreting auditory brain stem response changes. ABRs and CNAPs are relatively unaffected by the level of anesthesia or the type of anesthetic agents used, provided that normal core temperature or, more importantly, brain temperature is maintained. Core temperature rarely drops below 31 to 32°C over the course of surgery. Within this range, ABR absolute and interpeak latencies increase as a function of decreasing temperature at a rate of about 0.17 to 0.2 msec/°C [87,88], so that below 32.5°C, the values become abnormal relative to normothermic normal subjects [89]. Below 27°C, waveforms become difficult to identify [89] or disappear [82], although response amplitudes may also increase before being lost at about 18°C [90]. Even though core temperature is well controlled and maintained near normal values, brain stem temperature may decrease, especially within tissue bordering the exposed cerebellopontine angle if it is repeatedly irrigated with saline that is insufficiently warmed to body temperature. To the extent that core temperature is *not* maintained, recording ABRs from the contralateral ear can be of some value in determining whether any changes are systemic or localized.

Another factor that can affect the ABR is the change in pathways for current flow accompanying surgical exposure of the tumor. The craniotomy itself, changes in the local environment of the VIIIth cranial nerve with removal of cerebrospinal fluid and exposure of the nerve to air, and insertion of metallic retractors into the opening all create differences in the geometry of the relationship between the sites of ABR generation in the VIIIth cranial nerve and brain stem and the recording electrodes. These changes, which are of no clinical significance, can nevertheless lead to differences in amplitude, latency, and waveform configuration, which may be as large as those associated with intraoperative events significantly affecting the auditory pathway. Fortunately most of these changes occur relatively early in the

procedure before the VIIIth cranial nerve is in serious jeopardy; they may, however, necessitate obtaining a new intraoperative baseline because the ABR may be changed enough that the preincision baseline is no longer appropriate.

Typical auditory brain stem response findings in acoustic neuroma surgery

Changes in auditory brain stem response latency and amplitude. Fig. 6A shows typical ABR results encountered in a posterior fossa exposure with cerebellar retraction for removal of a small (<1.0 cm) intracanalicular acoustic neuroma. Postanesthesia, pre–initial incision results show well-defined ABRs with reproducible wave I, III, and V peaks to both right and left stimulation. There is almost always a significant difference in interpeak I to III and I to V latency values in the test ear (see left in Fig. 6A) compared with those from the opposite ear (see right in Fig. 6A). Initial ABRs are often much worse than the example shown here, depending on the degree of preoperative hearing loss and the extent to which the tumor compresses the nerve within the internal auditory canal or stretches it in the cerebellopontine angle.

Fig. 6B shows initial ABR results from another case in which wave V is desynchronized and greatly reduced in amplitude, wave III is absent (presumably as a result of lack of sufficient neural synchrony), and the I to V interpeak latency value is considerably extended (>5 msec). In the majority of acoustic neuroma surgeries, test ear ABR findings progressively worsen over the course of surgery as a result of one or more variables, mimicking the results shown in Fig. 6B. Retraction of the cerebellum, dissection of arachnoid support tissue, acoustic trauma, decreasing localized brain temperature, and disruption of cochlear blood supply may all affect ABR peak I, III, and V amplitude and latency values [62,75]. Retraction of the cerebellum is thought to be one of the

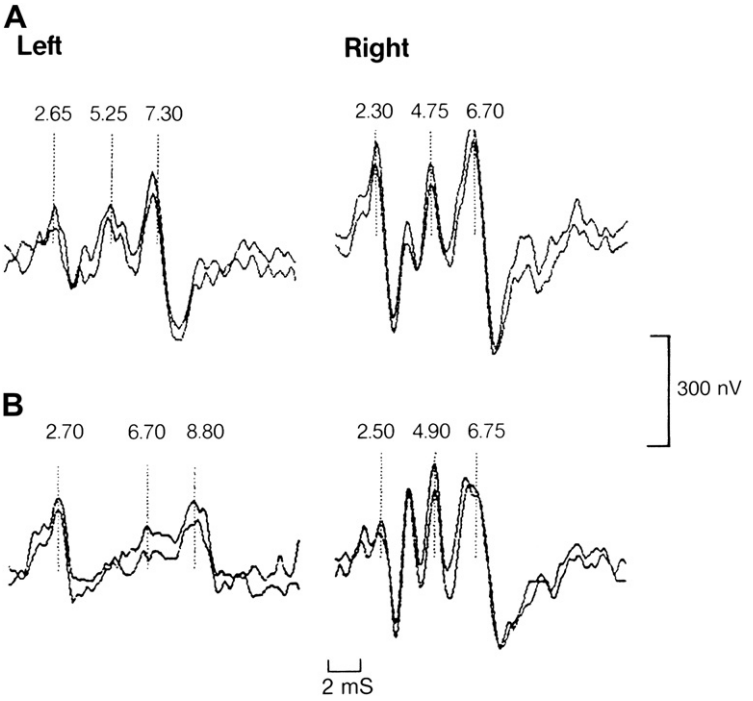

Fig. 6. Representative examples of intraoperatively recorded auditory brain stem responses (ABRs) from two patients. Recordings were obtained post induction, but before first incision, (*A*) 38-year-old woman with an 0.8-cm L acoustic neuroma, mild high frequency hearing loss, and speech discrimination scores of 92% (L.E.) and 100% (R.E.). (*B*) 52-year-old woman with a 1.8-cm L acoustic neuroma, moderate to moderate-severe sloping hearing loss, with speech discrimination scores of 56% (L.E.) and 90% (R.E.). Stimuli were alternating polarity, 100-μsec square wave pulses, presented at 80 dB nHL, 33.3/sec; 0.9-msec acoustic delay; averaged responses (n = 4000) were recorded vertex to ipsilateral ear canal. Duplicate averages are overlaid.

principal maneuvers responsible for significant ABR degradation [62,63]. It is often possible to reverse some of these effects by adjusting the cerebellar retractor, by temporarily halting dissection of tumor capsule, or by attempting dissection from a different angle or direction [66]. Occasionally wave I amplitude is enhanced, presumably because of mechanical trauma to inhibitory auditory efferent fibers, which travel with the vestibular nerve. This is usually followed by a rapid decrease in wave I amplitude values, suggesting disruption of the blood supply to the cochlea.

Correlation with postsurgical auditory evaluation. In the majority of cases in which ABR wave V is preserved after the tumor has been completely removed, subjective reports of preserved hearing are obtained. Even with such a favorable intraoperative outcome, however, hearing may still be lost; in some cases, hearing may be present immediately after surgery only to disappear within the next 2 or 3 days. The mechanism of this delayed loss is unclear but may involve vasospasm of the cochlear artery. If only ABR wave I is preserved, preservation of subjective hearing is much less likely. In more than one case, we have recorded an intact and unchanged wave I for more than an hour after complete transection of the cochlear nerve at the brain stem, a condition that is unlikely to be compatible with hearing preservation! Complete loss of waves I and V almost always results in loss of hearing; however, even this indicator is not infallible, and a surgeon should not decide to cut an otherwise healthy-looking cochlear nerve solely on the basis of ABR findings.

It is relevant to note that following surgical recovery, it is possible that postsurgical hearing is adversely affected even in patients who report subjectively unchanged hearing and in whom a reproducible ABR wave V peak is maintained (although of longer latency than presurgical controls). Administration of psychoacoustic tests of central auditory function, which rely on preservation of neural synchrony, is likely to pinpoint these deficits, especially dichotic listening tasks. Little effort has been directed at addressing such questions because most surgical and monitoring teams are pleased if there is little change in presurgical and postsurgical pure tone thresholds and speech discrimination scores.

Real-time analysis of VIIIth cranial nerve activity. In an attempt to make auditory nerve monitoring more reflective of real-time changes, most monitoring teams attempting to preserve hearing have turned to near-field recording paradigms in which VIIIth cranial nerve CNAPs are recorded in real time or with little averaging, allowing for updates on nerve status to be communicated within 5 or 10 seconds [70,74,83]. If manipulation of tumor and nerve or dissection of support tissue begins to affect the amplitude of the CNAP, it may be possible to switch surgical tactics to lessen these effects. An example of such a successful maneuver is shown in Fig. 7. The surgeon's manipulation of the tumor produced a decrease in CNAP amplitude, which returned when the tumor was released and approached from a different angle.

Other authors have decreased averaging time by employing strategies of optimal digital filtering [35,91,92]. In studies such as these, traditionally recorded (vertex to mastoid) ABR baseline waveforms are established postinduction and spectrally decomposed to determine optimum filtering for waveform reconstruction. It is important that the filter characteristics be individually determined for each patient, since the baseline ABR is typically abnormal and filters based on normative data might not be optimal in a given case. Subsequently ABRs are acquired by applying this unique filter to each single trial before averaging, allowing for reduction in the number of sweeps necessary for identifying critical waveform features such as changes in amplitude or latency for given peaks. Digital filtering may allow ABRs to be collected with as few as 128 sweeps, allowing updating of the averages within 10 seconds or less. Although these techniques are computationally intensive and generally not available in commercial devices, they hold the promise of extending successful hearing preservation outcomes to patients with larger tumors that cannot be easily monitored using near-field VIIIth cranial nerve CNAPs. They also have the advantage over near-field recording techniques because placement of an electrode within the surgical field is unnecessary.

Used in tandem, ABR and CNAP recordings may help increase the likelihood of hearing preservation in small acoustic neuromas [77]. Certain limitations of the successful use of these techniques must be kept in mind, however. Placement of a near-field electrode near or on the VIIIth cranial nerve at the root entry zone is critical for real-time recordings of the CNAP. This limitation greatly reduces the number of tumors in which this technique can be used.

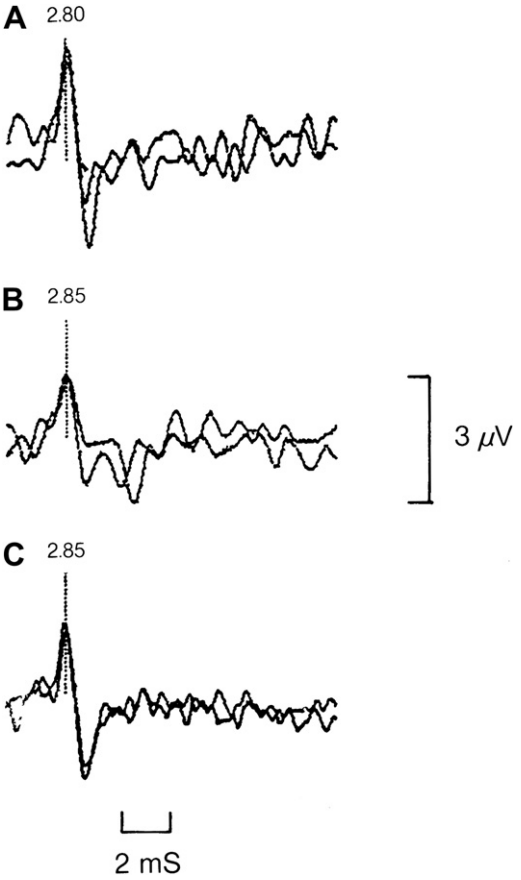

Fig. 7. Representative examples of changes in near-field VIIIth cranial nerve compound action potentials (CNAP) over the course of 30 seconds from a patient with an 0.6-cm intracanalicular acoustic neuroma, (*A*) Just prior to mobilization of tumor tissue adherent to the VIIIth CN, (*B*), mobilization of tumor caused a sharp reduction (>50%) in N_1 amplitude, (*C*), followed by partial recovery. Stimuli same as described in Fig. 5. CNAPs (100 trials/ average) were recorded from a flexible, ball-tipped electrode placed on the auditory portion of the VIIIth CN adjacent to the brain stem root entry zone. The indifferent electrode was placed in wound musculature. Duplicate averages are overlaid.

The use of ABR and VIIIth cranial nerve CNAP techniques is also appropriate in other posterior fossa surgeries and may be particularly helpful in such cases when the cochlear nerve is not as adherent to the tumor as in the typical acoustic neuroma. Microvascular decompression procedures, especially of the Vth, VIIth, and VIIIth cranial nerves and cerebellopontine angle meningiomas in which the VIIIth cranial nerve root entry zone is free, are the other most common surgical procedures that benefit from auditory nerve monitoring.

Impact of VIIIth cranial nerve monitoring on success in preservation of hearing

Five major confounding issues in published reports make it difficult to assess clearly the usefulness of VIIIth cranial nerve intraoperative monitoring techniques in regard to hearing preservation.

1. There is little agreement concerning the definition of success in preservation of hearing in the literature. Some authors consider any useful speech discrimination or pure tone thresholds less than 70 dB between 0.5 and 2.0 kHz as preservation [93], whereas most others define preservation as pure tone thresholds less than 50 dB and speech discrimination scores greater than 50% [94]. Others suggest a classification scheme in which hearing preservation is expressed as a percentage of patients whose postoperative hearing remained in one of three categories compared with preoperative assessment: (1) good = speech reception threshold (SRT) <30 and speech discrimination score (SDS) >70%, (2) serviceable = SRT <50 and SDS >50%, (3) measurable = any measurable hearing [95]. An excellent qualitative scheme for classification of hearing following acoustic neuroma surgeries can befound in Silverstein and colleagues [96].
2. Many reports on attempts at hearing preservation in posterior fossa surgeries do not correlate electrophysiologic data obtained intraoperatively with behavioral data obtained postsurgically [60,97].
3. Well-designed studies employing presurgical and postsurgical behavioral data may lack intraoperative correlates [98].
4. Many electrophysiologic reports group hearing preservation results from acoustic neuromas with other surgeries of the posterior fossa [60,78].
5. Preservation of hearing is directly related to presurgical hearing status and tumor size, making comparisons of results difficult across studies in which there are no controls for these independent variables.

Therefore, certain cautions have to be exercised in drawing conclusions from published

reports. The most useful reports are those that contain both intraoperative electrophysiologic data and postsurgical behavioral follow-up data. Furthermore tumor size and preoperative hearing status need to be well documented.

In general, if tumor size is controlled for and restricted to less than 2 cm, useful to adequate hearing preservation is maintained in roughly 30% to 45% of patients as determined by preoperative and postoperative pure tone averages and speech discrimination scores [78,93,94,96,98,99]. ABR and CNAP results obtained intraoperatively suggest that if waves V and I are preserved, there is an excellent chance that hearing will be preserved [78,93,100], although exceptions have been reported [78]. If waves I and V are lost intraoperatively, there is little or no chance of hearing preservation [78,93], although rare exceptions have been reported. When wave V is lost with preservation of wave I, hearing conservation is not always maintained and cannot be accurately predicted. Furthermore, even if the response recovers over the course of surgery, transient changes in wave I and the cochlear microphonic potential may reflect pathogenic changes to the nerve with serious long-term consequences for hearing preservation [74,83]. Recovery of wave V without loss of wave I over the course of surgery does not always appear to lead to hearing loss [93].

The predictability of hearing conservation based on identifiable waves V and I at the end of surgery has been best addressed in a study by Watanabe and colleagues [78]. Their patient group included acoustic neuromas as well as other, nonacoustic surgeries, but it points up the utility of electrophysiologic assessment in a large group of patients. Out of a group of 80 patients in whom ABR wave V could be recorded at the onset of surgery, 68 patients had identifiable wave Vs at the end of surgery and 12 did not. Of these 68, 66 had hearing preservation at the conclusion of surgery and 2 did not. Of the 12 patients without wave V at the conclusion of surgery, 2 patients demonstrated postoperative hearing preservation despite the loss of wave V. Likewise, out of a group of 75 patients in whom wave I could be recorded at the onset of surgery, 62 patients had identifiable wave Is at the end of surgery and 13 did not. Of these 62, 57 had hearing preservation and 5 did not. Of the 13 patients without wave I at the conclusion of surgery, 3 patients demonstrated postoperative hearing preservation despite the loss of wave I.

These results taken together suggest that only four predictions (5%) were inaccurate based on wave V methods and only 8 predictions (10.6%) were inaccurate based on wave I methods. It is likely that some of these errors are due to changes in auditory nerve status subsequent to surgical recovery. For example, a certain small percentage of pathologic changes in the auditory nerve may only manifest themselves over a long time course (ie, >14 days), whereas other changes, including spontaneous recovery [101,102], may explain discrepancies in which hearing was preserved despite loss of wave I or V responses.

There are several possible outcomes of ABR monitoring, each with distinct consequences for hearing prognosis and pathogenic mechanisms. Preservation of both waves I and V after total tumor removal, even with increased I to V interpeak latency, indicates that the cochlea, VIIIth cranial nerve, and lower auditory brain stem pathways are intact and should be predictive of at least some preserved hearing postoperatively; however, even this most favorable outcome may still be associated with delayed hearing loss. Preservation of wave I with loss of wave V is more problematic. Wave V may be lost simply because the cochlear fibers are disrupted enough that the volley entering the brain stem is no longer synchronous enough to produce a recordable wave; in this case, good postoperative hearing may still be obtained. On the other hand, wave I can be preserved even with transection of the cochlear nerve at the brain stem; thus interpretations of such a pattern should be made cautiously and in the context of the degree of anatomic preservation of the VIIIth cranial nerve. Finally loss of all waves including wave I generally indicates an outcome that is incompatible with preservation of hearing; however, even this seemingly straightforward prediction is not foolproof. If wave I is gradually lost during a long and difficult dissection, the loss may still represent only desynchronization of the afferent volley and be compatible with intact transmission into the brain stem and some recovery of hearing postoperatively. Sudden and precipitous loss of wave I, however, is likely to be associated with compromise of the cochlear blood supply and represents an outcome unlikely to result in any useful hearing.

Electrophysiologic monitoring of the VIIIth cranial nerve is technically challenging, but when performed by skilled professionals who work as a team with their surgical colleagues, VIIIth

cranial nerve morbidity can be significantly reduced in selected acoustic neuroma cases.

Conclusions and future directions

The introduction of techniques for cranial nerve monitoring in acoustic neuroma surgery has led to significant improvements in surgeons' ability to identify and preserve the function of the VIIth and possibly VIIIth cranial nerves while achieving total tumor removal. It is equally obvious that the state of the art is still developing and that further improvements are likely as techniques become more refined and integrated.

Part of the improvement will come as integrated hardware-software systems, optimized for intraoperative monitoring, become available in the marketplace. Computer-based systems with simultaneous capacity for EMG and ABR recording, rapid data collection with online digital filtering, better artifact rejection, automated control of stimulation and recording parameters, and user-friendly interfaces and displays of current data as well as trends during the operation will bring the full range of currently available techniques into more widespread use.

In addition, there is still much to be learned about the relationship between intraoperative recordings and ultimate clinical outcome. More well-controlled studies, with carefully characterized patient populations and standardized monitoring techniques, need to be carried out to address many of the ambiguities discussed here. Various techniques need to be compared in terms of their predictive efficacy. For example, is the best predictor of postoperative facial nerve function likely to be (1) the threshold for obtaining any evoked EMG response with stimulation of the VIIth cranial nerve after tumor resection, (2) the amplitude of the surface EMG response to supramaximal VIIth cranial nerve stimulation, (3) the total amount of mechanically elicited EMG activity during the case, (4) the intracisternal conduction velocity obtained by comparing the latency of facial responses to stimulation of the VIIth cranial nerve at the brain stem root entry zone versus the porus acusticus, (5) the shape of an input/output curve relating EMG amplitude to stimulation amplitude, (6) the recovery function of the response to paired stimuli at short intervals, (7) some combination of the above-mentioned, or (8) some measure not yet envisioned? Similarly what aspect of the intraoperatively recorded ABR or VIIIth cranial nerve CNAP will prove the best indicator of postoperative hearing preservation? The answers to such questions will be obtained only by more systematic studies, using the full range of techniques now available. The only certain outcome is that improvements in monitoring techniques and routine integration of professional monitoring personnel into the surgical team, coupled with advances in early diagnosis and microsurgical techniques, will continue to improve the prognosis for preservation of cranial nerve function in patients undergoing acoustic neuroma surgery.

Summary

The likelihood of successful preservation of facial and cochlear nerve function during acoustic neuroma surgery has been improved by the advent of intraoperative monitoring techniques. The facial nerve is monitored by recording EMG from facial muscles, with no muscle relaxants used; mechanical irritation of the nerve during surgery causes increased EMG activity, which can be detected in real time using a loudspeaker. Brief episodes of activity associated with specific surgical maneuvers aid the surgeon in avoiding damage to the nerve, whereas prolonged tonic EMG activity may reflect significant neural injury. Electrical stimulation with a hand-held probe elicits evoked EMG responses, which can be used to locate and map the nerve in relation to the tumor. The threshold for eliciting evoked EMG responses provides a rough indicator of the functional status of the nerve. Different nerves in the posterior fossa (trigeminal, facial, spinal accessory) can be identified in multichannel recordings by the spatial distribution and latency of responses to electrical stimulation. The ability to elicit EMG responses from low amplitude stimulation of the facial nerve at the brain stem after tumor removal is a reasonable predictor of postoperative facial function. Cochlear nerve function is assessed by recording the ABR from ear canal and scalp electrodes or the CNAP with an electrode placed directly on the nerve at the brain stem root entry zone. The ABR is a well-known, noninvasive technique that can be adapted to intraoperative use relatively easily but is of limited utility owing to the delay inherent in signal averaging. Direct CNAP recordings require placement of an intracranial electrode in such a way as to contact the cochlear nerve without interfering with surgical access but have the distinct

advantage of rapid feedback on changes in cochlear nerve status.

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Cochlear and Brainstem Implantation

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Cochlear and auditory brainstem implants offer safe and effective hearing habilitation and rehabilitation for profoundly deafened adults and children. Brainstem implant technology is currently approved for use in patients with neurofibromatosis type 2, who have lost integrity of auditory nerves following vestibular schwannoma removal. An update on implant devices, speech processing strategies, candidacy criteria, and perceptual performance are provided in this article.

Cochlear implants

Cochlear implantation is an established treatment for selected individuals with bilateral severe to profound sensorineural hearing loss (SNHL) who derive limited benefit from conventional hearing aids. The first cochlear implants, developed in the early 1960s, comprised single electrodes that were surgically placed within the scala tympani, in an effort to electrically stimulate the auditory nerve in patients with absent or dysfunctional cochlear hair cells. These early devices restored some degree of sound awareness to recipients and, in many cases, facilitated lip-reading far better than their hearing aids had. The introduction of multichannel devices in the early 1980s, development of advanced speech coding strategies, and refinement of candidacy criteria have led to substantial improvements in postimplant performance, evidenced by improved open-set speech understanding in both children and adults.

This article provides an update on issues related to cochlear implantation, including device design, speech processing strategies, candidate selection, surgical technique, and perceptual performance.

Implant device

Basic components

All cochlear implant systems possess an externally worn device and an implanted internal component (Fig. 1). The external hardware consists of an ear-level microphone, an ear-level or body-worn speech processor, and a transmitter placed behind the ear. The internal component consists of a receiver–stimulator, linked to an intracochlear electrode array via a lead wire. Some implant devices have a second electrode which serves to ground the stimulating electrode. Sound received by the microphone is transduced into electrical signals, which are filtered, analyzed, and digitized by the speech processor and forwarded to the transmitting coil. The encoded signals are then delivered to the implanted receiver–stimulator by radio-frequency electromagnetic induction. This signal is reconverted to an electrical signal, which is then delivered to the implanted electrode within the scala tympani. Current applied to the electrodes radiates into the fluid of the scala tympani, spreads through the habenula perforata of the osseous cochlear modiolus, and stimulates the auditory nerve.

Electrode design

The design of the electrode array differs in the presently available commercial implants. Four implant systems have been FDA approved for

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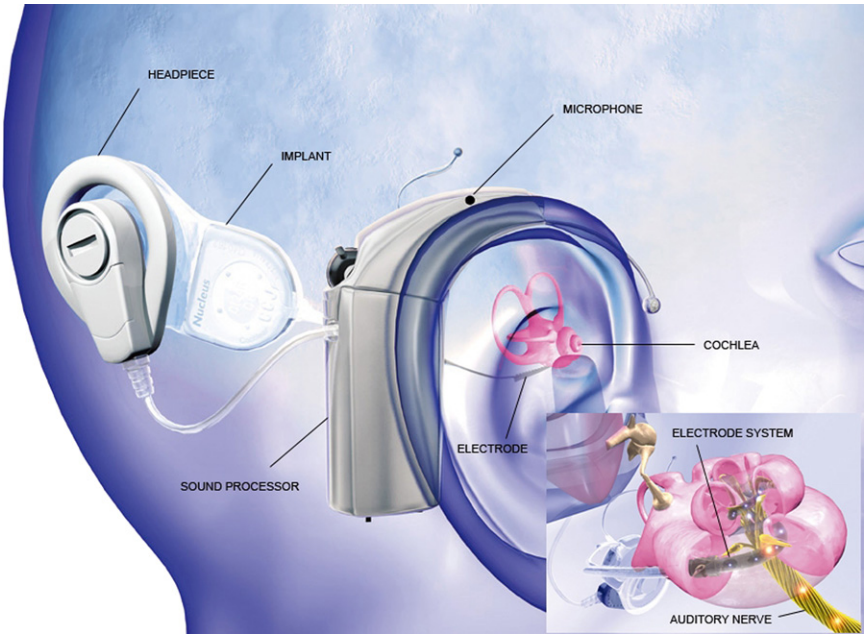

Fig. 1. Essential components of cochlear implant system (Cochlear Corp., Englewood, CO).

use in adults and children in the United States (the Nucleus Freedom Implant [Cochlear Corp., Englewood, Colorado], the HiRes 90K Implant [Advanced Bionics Corp., Sylmar, California], and the PULSAR CI100 and SONATA TI100 Implants [MED-EL Corp., Innsbruck, Austria]). There has been a trend in the past decade toward implantation of modiolar-hugging electrode arrays. The closer proximity of these electrode arrays to spiral ganglion cells offers theoretic advantages of improved sound quality, speech recognition, and power efficiency [1].

The Nucleus Freedom Implant system currently uses the Contour Advance electrode with Softip (Fig. 2). This consists of a 25mm long precurved modiolar-hugging electrode with 22 platinum electrode contact plates held in a straight position with a soft platinum wire stylet. The electrode tip comprises a conical tapered silicone elastomer designed to improve the insertion characteristics of the original Contour electrode and minimize tip fold-over during the insertion process (Fig. 3). The Contour Advance with Softip electrode inserted using the Advance Off Stylet (AOS) technique has been shown to significantly reduce trauma to the intracochlear structures during he insertion process [2]. Using the AOS technique, the electrode is inserted until its tip reaches near the back of the basal turn of the cochlea, then is advanced off the stylet (Fig. 4). A marker on the outer surface of the electrode (11 mm from the tip) delineates the insertion point at which the AOS technique should begin. As with earlier generation Nucleus implant systems, the Freedom Implant includes a ground or reference electrode, allowing monopolar stimulation of all 22 electrodes in the array, thus reducing power consumption.

Similar to the Nucleus Freedom implant, the HiRes 90K implant is housed in a titanium case with a removable magnet and telemetry coil attached and encased in silastic (Fig. 5). The device comes with 2 electrode options. The HiFocus Helix electrode is 24.5 mm long and consists of 16

Fig. 2. Nucleus Freedom implant (Coclear Corp., Englewood, CO).

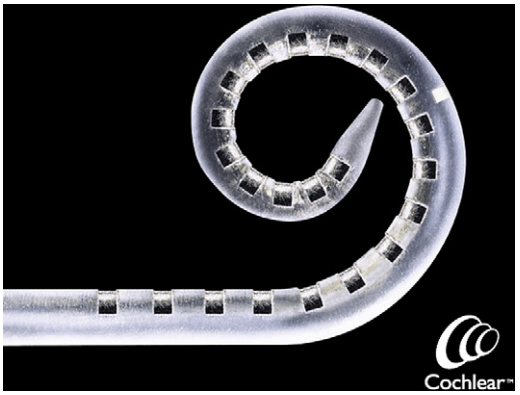

Fig. 3. Advance Contour electrode with Softip (Cochlear Corp., Englewood, CO).

planar platinum-iridium contacts arranged along the medial surface of the silicone electrode array. Dielectric partitions between the electrode contacts prevent channel interaction resulting from longitudinal spread of electrical current toward neighboring groups of nerve fibers. This precurved electrode comes preloaded on a stylet assembly which advances the electrode array off the stylet to an insertion depth of 21.5 mm. This configuration theoretically allows for the electrodes to lie close to the cochlear modiolus, thus providing improved sound fidelity and hearing performance. The HiFocus 1j electrode has the same number of electrode contacts on a slightly longer, narrower, and less curved silicone electrode array, and is designed to be inserted to a depth of 25 mm.

The PULSAR CI100 and SONATA TI100 devices developed by MED-EL Corporation house the same I100 electronics in a ceramic and titanium casing respectively (Fig. 6). The standard electrode array for these 2 devices features 12 pairs of electrode contacts on a soft and flexible straight electrode. The design of this array allows for the deepest insertion depth (approximately 31 mm) which then enables stimulation of a larger number of nerve fibers within the cochlea.

In addition to the standard electrode inventory provided by all the manufacturers, modified electrode configurations including straight, compressed and split electrodes (Fig. 7) are generally available with each device to implant congenitally malformed and ossified cochleae.

In addition to the standard electrode inventory offered with each of these cochlear implant systems, modified electrode designs such as the straight electrode, short electrode and split electrodes, are available for implanting malformed or ossified cochleae.

Speech-coding strategies and speech processors

Speech-coding strategies are software programs stored within the speech processor, which convert pitch, loudness and timing of sound into useful electrical signals [3]. Strategies are typically either non-simultaneous or simultaneous.

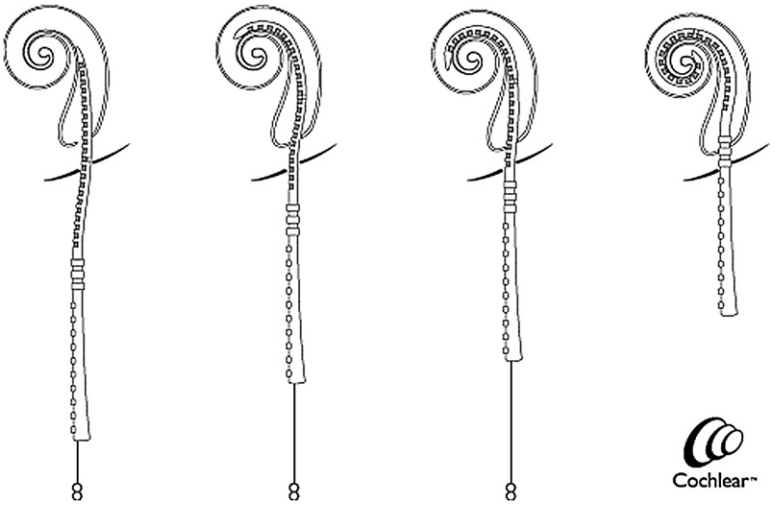

Fig. 4. Advance off stylet technique for insertion of Advance Contour electrode with Softip (Cochlear Corp., Englewood, CO).

Fig. 5. HiRes 90K implant (Advanced Bionics Corp., Sylmar, CA).

The most widely used speech coding strategy, SPEAK, or spectral peak, samples sound approximately every 4 ms and processes this information into 20 different frequency bands [4]. The processor selects an average of six filter bands that have the highest energy in each 4 ms interval and presents pulses sequentially to the six corresponding electrodes. Up to 10 maxima can be sampled in this fashion. This results in stimulation of up to 10 electrodes every 4 ms, representing the spectral energy levels of the sound input during that interval. The ACE, or advanced combined encoder, strategy is similar to the SPEAK strategy but uses a much higher rate of stimulation.

Another popular strategy is the continuous interleaved sampling (CIS) strategy [5,6]. With this strategy, each electrode receives pulses at a rate of 600 to 1000 pulses per second. Speech is divided into several frequency bands, and the amplitude envelope is extracted from each band. This information is then translated into an electrical impulse that drives the electrode representing that frequency band. In the SPEAK and CIS strategies, the electrode pulses are generated sequentially so that no two electrodes are active at the same time. This strategy avoids the problems of electrode interactions.

The Nucleus Freedom processors (Fig. 8) use a combination of the SPEAK, ACE and CIS strategies. The original ACE strategy allows stimulation rates up to 14,400 pps (pulses per second), whereas the more recent ACE (RE) strategy allows for higher stimulation rates of up to 34,000 pps.

HiResolution (HiRes) Sound is available in the HiRes Implant System from Advanced Bionics. HiRes is designed to offer a wide, programmable dynamic range, preserve spectral and temporal details of sound and stimulate at rates of up to 83,000 pps. In the HiRes 90K implants, the number of sites of stimulation can be increased beyond the number of electrode contacts. Through simultaneous delivery of current to pairs of adjacent electrodes, stimulation can be ''steered'' to sites between the contacts by varying

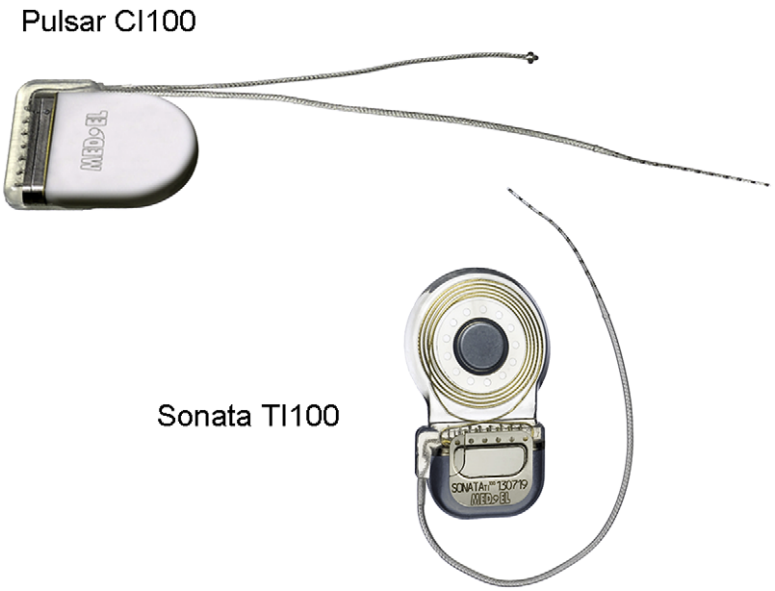

Fig. 6. MED-EL Pulsar CI100 and Sonata TI100 implants (MED-EL Corp., Innsbruck, Austria).

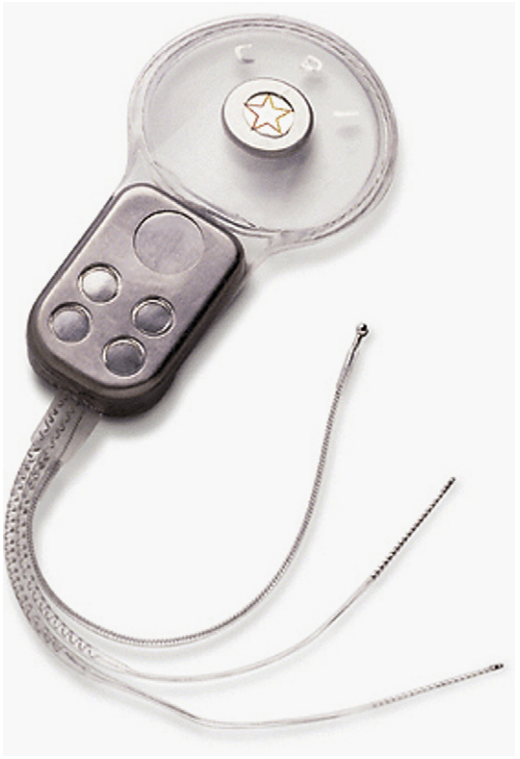

Fig. 7. Nucleus Freedom implant with split electrode (Cochlear Corp., Englewood, CO).

the proportion of current delivered to each electrode of the pairs. The Advanced Bionics strategies currently in use are the Hi-Res-P (Pulsatile), HiRes-S (Simultaneous) and HiRes Fidelity 120.

MED-EL coding strategies provide high stimulation rates up to 50,700 pps and individual current sources for each channel. With the introduction of the I100 electronics platform and the OPUS speech processors, MED-EL developed the FSP (Fixed Place strategy). The timing of stimulation is used to code the temporal structure of the sound signal in the low and mid frequency range by using channel-specific sampling sequences [7].

Telemetry

Neural response telemetry is a method that enables direct measurement of auditory nerve action potentials from cochlear implant patients. This technology is currently available for all four implant devices [7]. Initial recordings are obtained intraoperatively once the implant electrode has been inserted into the scala tympani and the receiver-stimulator has been secured in place.

The information obtained is useful for troubleshooting device failures and optimizing parameters for speech-processing strategies. This is particularly useful in mapping cochlear implants for younger pediatric patients who lack auditory experience.

Patient selection

Candidate selection for cochlear implantation has evolved as the devices and patient performance evolved. In general, adults and children with bilateral severe to profound SNHL, who receive little or no benefit from conventional hearing aids, are in good physical and mental

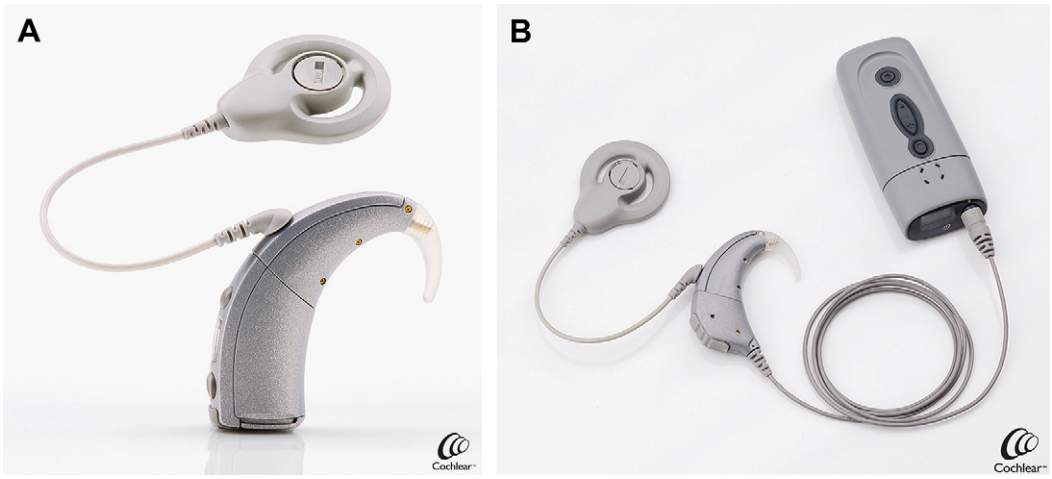

Fig. 8. Nucleus Freedom (*A*) BTE sound processor, (*B*) Bodyworn sound processor (Cochlear Corp., Englewood, CO).

health, and possess the aptitude and motivation to participate meaningfully in the auditory rehabilitation program are potential candidates for cochlear implantation.

Medical evaluation

A complete history and physical examination are necessary to detect problems that may contraindicate surgery or interfere with the patient's ability to complete postimplantation rehabilitation.

The cause of hearing loss is rarely a contraindication to implantation. Profound hearing loss associated with cochlear nerve aplasia is a rare congenital anomaly, in which the lack of auditory innervation obviates the option of cochlear implantation [8]. Prior meningitis with cochlear ossification or fibrosis does not exclude a patient from implantation but may necessitate modification of the surgical technique.

Two crucial factors influencing auditory performance following cochlear implantation include age of onset of deafness and duration of profound hearing loss. The ideal adult candidate has profound acquired SNHL. A period of auditory experience adequate for development of normal speech, speech perception, and language offers a significant advantage in learning to use the implant [9]. These postlingually deafened patients represent the majority of adults undergoing implantation. In these patients, there is a significant correlation between duration of profound hearing loss and performance. Those with prolonged auditory deprivation receive similar auditory information as do other implant patients but are not able to use the information as effectively in the recognition of running speech [10], which is thought to be due to the loss of central auditory processing.

A small number of adult implant recipients are congenitally or prelingually deafened, with prolonged auditory deprivation and little to no experience with sound. These patients typically have greater difficulty assimilating the new auditory information and, in general, have performed less well than those with some degree of auditory memory [11].

For children, the later the onset of deafness, the greater the auditory memory and, generally, the better the speech language skills. Both of these factors contribute to a higher likelihood of successful implant use. For congenitally or prelingually deafened children, increasing data support early implantation for maximal auditory benefit and speech language development [12]. Current FDA guidelines require a child to be at least 1 year old, have profound SNHL, demonstrate no benefit from conventional hearing aids, and be free of medical contraindications. Under exceptional circumstances, infants may be implanted earlier if hearing loss is the result of meningitis and there is concern for cochlear ossification. In addition to these criteria, the commitment of the family and the child's educational setting to postimplantation rehabilitation is a crucial determinant of successful implant use. Bilateral cochlear implantation is currently being recommended in both adults and children where applicable. The additional input from bilateral cochlear implantation has been shown to improve hearing in noisy situations and improve sound localization with the use of intensity cues [13]. The second device may be implanted simultaneously or sequentially.

Audiologic assessment

The basic testing battery includes unaided and aided warble tones and speech-detection thresholds, environmental sound recognition, and speech perception tests, with and without visual cues [14]. In general, a patient is considered a candidate for cochlear implantation when the following criteria are met: bilateral profound SNHL, with a three-frequency pure-tone average (500, 1000, and 2000 Hz), unaided threshold in the better ear of 70 dB hearing loss or poorer, or a speech discrimination score of 50% or less in best-aided conditions in the ear to be implanted and 60% or less in the non-implanted ear or bilaterally.

Audiologic screening in children requires auditory brainstem evoked response testing and otoacoustic emissions testing, in addition to conventional behavioral audiometry. An aggressive 6-month trial period using appropriate amplification and intensive auditory and speech training is an integral component of candidacy assessment in children. The global evaluation of cochlear implant candidacy in children is considerably more challenging than in adults and is best approached by a dedicated team comprising speech and hearing professionals, social workers, psychologists, and educators. The ultimate candidacy of a child is determined by not only a demonstrated physiological need but also the strength of the child's social and educational background.

Imaging

Preoperative imaging serves to complete the candidacy evaluation process and assist in surgical planning. A high-resolution CT scan of the temporal bones using a bone algorithm is the study of choice in most centers. MR imaging is the study of choice in a few centers. These images allow the surgeon to identify cochlear malformations and assess cochlear patency, mastoid pneumatization, and the course of the facial nerve. Observation of a narrow internal auditory canal on CT scans of the temporal bone should alert the surgeon to the possibility of cochlear nerve agenesis, which contraindicates implantation. This may be confirmed on sagittally reconstructed T2-weighted MR images through the internal auditory canal. MR imaging is also superior to CT imaging in assessing soft tissue obliteration of the cochlear lumen.

Electrical testing

Electrical stimulation of the promontory or round window has been used by some implant teams as part of the preoperative candidacy assessment [15]. Patients detecting the stimulus may possess better residual auditory neuronal stimulability. Such testing has not been uniformly adopted by all implant teams because patients with a negative response, particularly at the promontory, may respond to intracochlear stimulation with an implant.

Surgical technique

Selection of the side for implantation is governed by several factors. The most patent cochlea is typically chosen for implantation. It is generally believed that the ear with the shortest duration of deafness may serve as the best ear for implantation; however, if the patient uses a hearing aid in only one ear (the side that is perceived as the better-hearing ear), implanting the contralateral ''worse'' ear does not negatively impact performance [16]. When no specific factors lead to the choice of one ear over the other, the ear on the side of the dominant hand is chosen to facilitate device manipulation [17].

The implant is inserted via a transmastoid facial recess approach to the round window–scala tympani. Placement of a cochlear implants in children is essentially the same as in adults. By age 1 year, the mastoid antrum and facial recess are adequately developed. In patients with mastoid cavities or absent posterior ear canal, obliteration of the mastoid cavity with blind sac closure of the external auditory canal is preferably done at the time of disease removal. Cochlear implant placement is then performed at a second stage approximately 4 to 6 months later.

Surgery is performed with the patient under general anesthesia with the use of continuous intraoperative facial nerve monitoring. Many different incisions have been designed to allow placement of the receiver–stimulator. In general, the skin flap developed must be large enough to cover the receiver–stimulator completely. In adults and older children, temporalis muscle is removed around the receiver–stimulator area to minimize the thickness of the scalp over the internal device. This technique enhances magnetic coupling of the internal receiver and the external transmitter and reduces power consumption. In younger children with thin scalps, the temporalis muscle is elevated with the skin in a single layer. A depressed seat is created in the skull posterosuperior to the pinna, with adequate allowance for placement of a behind-the-ear microphone piece. More recently, minimal access incisions have been developed for placement of cochlear implant devices. In conjunction with minimal access surgery, receiver-stimulators may be tucked under the temporalis muscle without drilling a bony pedestal in the skull to secure the device in.

A complete mastoidectomy is performed, preserving a bony overhang around the margins of the mastoid cavity, to aid stabilization of the carrier coil within the cavity. The short process of the incus and its buttress are then used as bony landmarks to guide development of the facial recess. The chorda tympani is generally left intact unless a narrow recess limits visualization and access to the round window. The lip of bone overhanging the round window niche is then gently removed to allow for visualization of the round-window membrane. The cochleostomy is made anterior and inferior to the round-window membrane, in the basal turn of the cochlea. The electrode array is carefully advanced into the scala tympani. The cochleostomy is then sealed with a small plug of temporalis muscle or fascia.

The postauricular flap is closed in layers without drainage. For purposes of hemostasis, only bipolar electrocautery should be used after insertion of the cochlear implant device.

Surgical complications

The risks of implant surgery are similar to those of routine mastoid surgery and include infection, facial paralysis, dizziness, cerebrospinal fluid (CSF) leak, and meningitis. Skin flap breakdown and wound infection are the most common complications of cochlear implant surgery [18]. Placement of the device too close to the incision, or an excessively thin flap, can lead to device extrusion. It is imperative to seat the device approximately 1 or 2 cm from the wound edge and maintain a flap thickness of at least 6 to 7 mm. Flaps thicker than this can lead to inadequate magnet contact between the outer and inner hardware, with subsequent diminished performance.

During opening of the facial recess, the facial nerve is at particular risk for injury. Heat injury can be avoided by careful dissection and copious irrigation. The use of facial nerve monitoring may reduce the risk for injury, although it is no substitute for knowledge of temporal bone anatomy and good surgical technique.

Cerebrospinal fluid leaks, although unusual, can occur in two locations. The recessed site for the internal receiver often extends down to the dura. Inadvertent injury to the dura can result in CSF leak and should be repaired at the time of surgery with temporalis fascia. In congenitally malformed cochleae, the modiolar base may be deficient, resulting in a gush of CSF subsequent to the cochleostomy. When this situation is anticipated preoperatively based on imaging findings, the eustachian tube should be temporarily obliterated with Surgicel before the cochleostomy. Rarely do patients require further management of this complication, such as placement of a lumbar drain.

In the initial phase of cochlear implant development and insertion, there was a concern about the possibility of bacterial seeding of the CSF through the cochleostomy during bouts of acute otitis media. In 2003, several cases of fatal meningitis were reported in implanted children. Subsequent analysis of these cases demonstrated a significant correlation between the use of silastic intracochlear positioners to enhance perimodiolar electrode placement and post-operative meningitis. The use of these modiolar positioners has since been abandoned. The presence of cochlear malformations was also found to be a significant risk factor for meningitis. Streptococcus pneumoniae was found to be the predominant microbe in these cases. The United States Food and Drug Administration currently recommends preoperative vaccination with Prevnar or Pneumovax, along with the use of perioperative antibiotics for meningitis prophylaxis in all implant patients [19].

In the elderly population, the possible effects of cochlear implantation on the vestibular system should be considered. Transient dizziness and imbalance have been reported in up to 30% of implant recipients [20].

Rehabilitation

The external device is fitted 4 weeks after surgery, allowing for resolution of edema in the postauricular flap. Electrode mapping is also performed at this time. The initial goals of this fitting are to establish thresholds and comfortable loudness levels for each electrode. Most cochlear implant processors assign successive bands of frequencies to each electrode in tonotopic order. In some patients, processor programming may involve adjusting this electrode-to-frequency assignment if the pitch of the electrodes is not tonotopically ordered. All of these parameters are then stored in the speech processor and are collectively referred to as the "MAP."

In very young children with little or no auditory experience, determining these settings can be challenging. Electrically evoked auditory potentials and acoustic reflex thresholds can be useful adjuncts to the mapping process in children [21].

Rehabilitation continues at regular intervals for several years following implantation. Global rehabilitation often includes simple auditory training, speech reading practice, and counseling of the patient and family.

Results

Despite differences in device design and function, postimplant performance has not differed significantly among the devices. Virtually all patients receiving multichannel cochlear implant devices experience substantial benefit. Up to two thirds of adults undergoing implantation obtain open-set speech recognition and comprehend speech to some degree while using the telephone [22–24]. Even poor performers benefit from the awareness of environmental sounds and enhancement of lip-reading abilities.

One of the crucial determinants of performance is memory of previous auditory experience. Postlingually deafened adults with some auditory

experience and a short duration of deafness generally learn to use the sound information provided by the implant more quickly and effectively than do those who were born with profound hearing loss or those who lost their hearing early in life [25].

In children, a supportive family and educational environment, combined with intensive auditory and speech rehabilitation, are crucial for successful device use. Postlingually deafened children often demonstrate rapid improvement in speech perception, usually achieving 100% correct responses on closed-set testing, reaching a maximal benefit within 12 to 18 months [26]. Prelingually deafened children achieve average word recognition scores of 30% to 44% using newer speech-processing strategies [27,28]. Onset of deafness after 2 years of age and duration of deafness of less than 2 years convey the best prognosis for performance [29]. Congenitally deaf children undergoing implantation at a young age demonstrate the most improvement after implantation [12,30,31]. Congenitally deaf adolescents undergoing implantation do less well and have a high nonuser rate [32].

Acquisition of speech and language in children undergoing implantation often parallels speech-perception abilities. Furthermore, speech intelligibility and language skills continue to improve over time and, on average, exceed those of age- and hearing-matched peers using hearing aids [33,34]. Patients in oral-communication settings appear to perform at a higher level than do those in total-communication settings [35–37]. Research efforts are currently focused on developing atraumatic electrodes and insertion techniques to preserve residual hearing in the implanted ear. Investigational devices with shorter electrodes are being implanted in individuals with significant residual low frequency hearing who currently receive limited benefit from conventional hearing aids. The successful implantation of these devices allow for bimodal hearing in the implanted ear with auditory stimulation using a conventional aid to amplify low frequency tones and electrical stimulation using the cochlear implant to restore hearing in the high frequency spectrum [REFERENCE].

Auditory brainstem implants

The auditory brainstem implant (ABI) was designed to restore some hearing to patients lacking functionally intact cochlear nerves and who were, therefore, not candidates for cochlear implantation. These are principally patients with neurofibromatosis type 2 (NF-2), in whom both cochlear nerves are nonfunctional as the result of the vestibular schwannoma or its surgical removal. In such individuals, the cochlear nucleus complex within the brainstem is stimulated directly by placing the ABI electrode into the lateral recess of the fourth ventricle.

The first ball electrode ABI was placed by House and Hitselberger in 1979 in a patient with NF-2 during the removal of her second vestibular schwannoma. Early prototype single-channel devices were replaced by multichannel devices in 1992. The Nucleus multichannel ABI device received FDA approval for implantation in October 2000.

Device design

The current Nucleus multichannel ABI24 device was developed collaboratively by the House Ear Institute and Cochlear Corporation [38]. The device consists of an implanted component and an externally worn component, similar to the cochlear implant device. The implanted receiver–stimulator, modeled after the Nucleus cochlear implant device, is attached to an electrode array composed of 21 platinum disks mounted on a silicone and Dacron mesh carrier (Fig. 9). The external component consists of a microphone, speech processor and a transcutaneous transmitter coil. Unlike the cochlear implant device, however, the receiver magnet is removed at the time of surgery, allowing patients with NF-2 to undergo MRI

Fig. 9. Nucleus multichannel ABI24 device (Cochlear Corp., Englewood, CO).

surveillance of tumors. A small retainer disk is used instead to secure the external transmitter coil in place.

Functionally, the ABI device differs little from the cochlear implant device. A microphone converts sound to an analog electrical signal, which is then modified by a speech processor and delivered to the electrode array via either a percutaneous pedestal or a transcutaneous coil.

Patient selection

The criteria for placement of ABIs include a diagnosis of NF-2, age of at least 12 years, requiring either first or second side surgery, proficiency in the English language, and reasonable expectations.

Initially, the ABI was placed at the second acoustic neuroma surgery. With increased experience, several patients have undergone device placement during surgery for the first tumor, even though they had serviceable hearing in the opposite ear. Earlier implantation allows the patient time to learn to use the device before complete loss of hearing occurs in both ears. Also, waiting to implant the second side often results in placing the device after removal of large tumors, which may have distorted normal landmarks, thus significantly complicating placement of the device. Finally, implanting the first side gives the patient a chance to have the second side implanted should the initial device fail to provide useful auditory sensation [39].

Surgical technique

The cochlear nucleus complex, composed of the dorsal and ventral cochlear nuclei, is the target for placement of the ABI electrode array. The ventral nucleus is the main relay nucleus for nerve VIII input, and its axons form most of the ascending pathway. Both nuclei are not visible from a surgical approach and must be located using surface landmarks. The lateral termination of the fourth ventricle, the foramen of Luschka, is found between the roots of the facial and glossopharyngeal nerves. Usually only a stump of nerve VIII remains, and this may be a useful landmark for the lateral recess as well. Experience has demonstrated that the ideal position for the electrode placement is completely within the lateral recess, adjacent to the dorsal nucleus and the inferior aspect of the ventral nucleus. This position results in optimal auditory stimulation and the least stimulation of adjacent structures, including cranial nerves V, VII, and IX, or the overlying flocculus of the cerebellum. Also, placement completely within the lateral recess aids in the stabilization of the electrode [40].

The approach used for tumor removal in ABI cases is the translabyrinthine craniotomy. This approach offers the most direct access to the lateral recess after tumor removal. Intraoperative monitoring of the trigeminal, facial, and glossopharyngeal nerves is helpful. The translabyrinthine craniotomy skin incision is modified to allow for placement of the internal receiver–stimulator. Once the tumor is removed, a seat is created in the lateral surface of the skull for placement of the receiver, and a groove is created as a path for the electrode lead. Meticulous hemostasis is obtained in the cerebellopontine angle, and then the landmarks leading to the surface of the cochlear nuclei are identified. Normally, the opening of foramen of Luschka is marked by intact choroid plexus and the taenia traverses the roof of the lateral recess. After removal of larger tumors, however, these landmarks may be obscured. In such cases, the stump of nerve VIII can usually be followed to the opening of the lateral recess, and nerve IX reliably leads to the floor of the recess. Once identified, the location of the lateral recess can usually be confirmed by observing the egress of spinal fluid during a Valsalva maneuver.

The electrode array is then placed completely within the lateral recess as noted earlier. This placement situates the array immediately adjacent to the dorsal cochlear nucleus and the posterior tip of the ventral cochlear nucleus. Orienting the electrode array such that the electrodes face medially and superiorly maximizes stimulation of the cochlear nuclei.

After placement of the electrode, tests for electrical auditory responses are performed, as are stimulation of adjacent cranial nerve nuclei and vital sign changes. The position of the electrode array is adjusted to maximize auditory stimulation while nonauditory stimulation is minimized. The electrical ABR is very useful in confirming placement of the array, especially in cases in which the tumor has distorted normal anatomy. The electrode array is secured by placing a small piece of fat or polytetrafluroethylene (Teflon) felt within the opening of the lateral recess. The electrode lead is then positioned in the mastoid cavity, while the ground electrode is inserted under the temporalis muscle. The magnet in the receiver–stimulator is removed

and replaced with a silicone plug to allow for postoperative MR imaging. The mastoid defect is packed with fat and the wound closed in layers.

Surgical complications

The most common complications related to ABI placement include CSF leak, electrode migration, and nonauditory side effects. CSF leak in these cases may be related to tracking of CSF along the electrode lead, from the subarachnoid space to the subcutaneous plane. These leaks may be managed in a similar fashion to leaks occurring after conventional neuro-otologic surgery. Rarely is re-exploration necessary for control of the leak [39].

Migration of the electrode may occur as a result of unstable positioning or changes in shape and position of the brainstem after tumor removal. Electrode position may be confirmed on high-resolution CT scans in which soft tissue windows of the posterior fossa are overlaid with images enhanced for high-contrast objects [41].

Nonauditory side effects have occurred in 42% of multichannel implant users and seem to be related to electrode position [38]. In patients with a low electrode placement, as demonstrated on CT scans, symptoms related to glossopharyngeal nerve stimulation have occurred and are typically a sense of tingling or constriction in the throat. One patient with an exceptionally low placement had nausea and shoulder contraction related to vagal and accessory nerve stimulation respectively [40]. High electrode placement has caused facial twitching due to stimulation of the intact facial nerve. A mild sense of jittering of the visual field also has been reported, possibly related to activation of the flocculus of the cerebellum. Nonauditory side effects in the multichannel device generally occur with stimulation of the more medial or lateral electrodes. They can usually be reduced by switching reference electrodes, increasing the duration of the stimulus pulse, or turning off the electrode. The severity of the nonauditory sensations often decreases over time, sometimes allowing for reactivation of electrodes previously turned off.

Rehabilitation

Initial device stimulation is usually carried out 4 to 8 weeks after implantation and consists of measuring threshold and comfort levels and detecting nonauditory side effects, which may be controlled with modification of stimulus parameters.

Results

With some notable exceptions, the overall performance of patients with ABIs have paralleled that of patients with earlier single-channel cochlear implants. Eighty-five percent of patients undergoing implantation receive auditory sensations [42]. Combined with lip-reading cues, 93% of patients demonstrate improved sentence understanding at 3 to 6 months. Most patients have environmental-sound awareness and understanding of closed-set words, consonants, and vowels. Open-set speech recognition in patients receiving ABIs is the exception rather than the norm. Sound recognition and speech perception generally continue to improve for up to 8 years following implantation [43], although overall progress tends to be slower and of a lesser magnitude than that seen with multichannel cochlear implants. The ABI device is currently being implanted in non-NF2 patients in Europe who are not candidates for cochlear implantation. These include patients with extensive cochlear ossification, cochlear nerve aplasia, or cochlear nerve injuries. Early results indicate better hearing outcomes in these patients compared to NF2 patients. A significant number of these non-tumor patients are able to understand speech at a level comparable to that of the most successful cochlear implant users, including the ability to converse on telephones [44]. Clinical trials are currently underway in the United States for this clinical indication.

Summary

Cochlear implantation is an established habilitative and rehabilitative option for profoundly deafened individuals over 1 year of age who derive limited benefit from conventional hearing aids. Auditory performance varies among individuals and is determined primarily by age at implantation, preexistence of speech and language skills, and the time interval between onset of deafness and implantation. Successful implant users generally demonstrate improved auditory abilities and speech production skills beyond those achieved with hearing aids.

Multichannel ABIs can provide useful auditory information to patients with NF-2 who have lost integrity of auditory nerves following removal of vestibular schwannomas. The implant allows for awareness of environmental sounds and, potentially, speech recognition. Most patients undergoing implantation demonstrate improved

lip-reading skills, and exceptional performers achieve understanding of open-set speech.

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Surgical Approaches and Complications in the Removal of Vestibular Schwannomas

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Vestibular schwannomas (VS) are benign tumors of Schwann cells that originate on the vestibular portion of the eighth cranial nerve. Found in the internal auditory canal (IAC) and cerebellopontine angle, VS account for nearly 6% of intracranial tumors. Although surgical excision previously had high mortality rates, the advent of the operating microscope and neurotologic and neurologic monitoring has allowed surgical techniques to evolve and morbidity and mortality to be reduced significantly. Currently, complete surgical excision with preservation of facial nerve function is often an achievable goal. The increasing sensitivity of MRI has allowed for diagnosis of smaller tumors and has increased the ability to preserve hearing. Three basic surgical approaches are used for removal of VS: the translabyrinthine, retrosigmoid, and middle cranial fossa (MCF) approaches, which are described in this article. Although many factors influence the choice of surgical approach, personal experience of the surgeon often determines the surgical approach. This article presents the different approaches, their advantages and disadvantages, common complications, and mechanisms to prevent complications.

Surgical history

The first documented case of VS was an autopsy report during the late eighteenth century by Eduard Sandifort [1]. It was nearly 100 years later when Thomas Annandale performed the first complete surgical excision with patient survival. Surgical excision had high mortality rates—in excess of 50%—until the early 1920s, when Harvey Cushing refined techniques and lowered mortality rates to 21% [2]. Cushing's protégé, Walter Dandy, further lowered mortality rates to 10% by the early 1940s [3].

The modern era of tumor dissection began in the early 1960s with the introduction of the operating microscope and the introduction of the translabyrinthine and MCF approaches by William House. Sterile technique, microscopic magnification, and precise otologic drills have continued to lower mortality rates to approximately 1%. Continued earlier diagnosis with the advent of MRI with gadolinium has changed the goals of surgery from complete excision and survival to maintenance of facial nerve function and preservation of hearing when possible [4].

Treatment options

After proper diagnosis, four treatment options are available for VS: observation, stereotactic radiation therapy, complete surgical excision, and subtotal resection with planned radiation therapy.

Observation

The indolent nature of VS has led many physicians to observe tumors that may remain dormant or grow slowly enough to never require treatment. Treatment decisions may be based on patient characteristics, such as age, general health, status of hearing in the contralateral ear, and patient preference, or tumor characteristics, such

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as size, location, and growth rate [5]. Observation is performed via a set schedule of MRI. We scan our patients initially at 6 months and then every year until a general growth rate can be established. If minimal growth has been observed, the MRI every 2 years is possible.

The natural history of VS is enigmatic. The percentage of tumors that grow is generally unknown and has varied widely in studies from 40% to 80% [5–7]. A long-term follow-up of 14 years in more than 1800 patients with observed acoustic neuroma was published recently in which 83% of intrameatal tumors remained intrameatal and 70% of extrameatal tumors did not grow more than 2 mm [6]. All tumors that exhibited growth did so in the first 4 years and maintained consistent growth rates throughout observation [6]. Tumor growth rates can be stratified into slow growing (<2 mm/y) and fast growing (>2 mm/y). Tumors with slower growth rates often do not require treatment [5].

There are difficulties with observation. First, MRI is expensive and patients may be lost to follow-up. Rarely, tumors may undergo accelerated rates of growth after multiple years of stable growth. Patients who require surgery after observation also are older and may suffer from more comorbidities, which makes surgery potentially more risky. Finally, tumors with continued growth may no longer meet criteria for hearing preservation approaches or radiation therapy and may make facial nerve preservation more difficult.

Advantages to observation are the obvious delay and potential elimination of the need for surgical or radiosurgical intervention. Older patients may develop or have exacerbation of existing disease that may take precedence over treatment of the tumor (ie, cancer, stroke, cardiac disease). Younger patients may choose to observe the tumor to determine growth and choose intervention only when growth has been established. Economic reasons, such as retirement, job changes, or insurance issues, may compel a patient to choose observation as an initial option. Family issues, such as a wife/husband or child with an illness or other family issues (child starting school/college), also may compel a patient to choose observation.

Radiation therapy

Although surgical resection has the goal of complete tumor removal, radiation therapy has the primary goal of tumor control. Leksell performed the first stereotactic radiosurgery for VS in 1969. Techniques have evolved and outcomes with fewer side effects are currently possible. Although most tumors are controlled successfully, nearly 9% of tumors exhibit growth after radiation therapy. Surgery after failed radiation therapy is more difficult to perform because intense scarring and fibrosis obscure surgical planes. As a result, facial nerve outcomes are poorer than in patients with nonirradiated tumors [8–10]. Because VS are known to be slow growing, it is difficult to study techniques (radiation therapy) that slow or eliminate tumor growth. Long-term studies are needed to determine the effectiveness of radiation therapy over observation and surgery to treat VS.

Surgery

The goal of surgery is complete tumor removal while preserving neurologic function and hearing, if possible. Each of the three main approaches for surgical removal of VS has advantages and disadvantages (Table 1). The retrosigmoid and MFC are the two surgical techniques most commonly used in VS surgery when attempting to preserve hearing. Hearing preservation may be attempted when the pure-tone average is 50 dB or less and the speech discrimination is more than 70%. Tumor size also determines whether hearing preservation is attempted, because a tumor ≥ 2 cm is rarely amenable to hearing preservation despite initial hearing status. Location also may play a role in hearing preservation because tumors far lateral in the IAC with extension into the cochlea or vestibule may not be amenable to hearing preservation.

All procedures are performed under general anesthesia with neurophysiologic monitoring. Facial EMG electrodes are inserted into the obicularis oris and occuli muscles for continuous facial nerve monitoring. Auditory brainstem response is used in hearing preservation cases with an acoustic ear insert in the external auditory canal, a recording electrode placed at C0 on the vertex, a reference in the ipsilateral ear lobule, and a ground in the shoulder. Perioperative antibiotics and steroids are used. Mannitol (1 g/kg) is given just before opening the dura in all cases.

The translabyrinthine approach provides wide access to the posterior fossa with little or no need for brain retraction. The anatomy is familiar for the neurotologist but often is unfamiliar to the neurosurgeon. The facial nerve is identified early in the case laterally at the fundus of the internal IAC and medially at the brainstem. This lateral surgical approach allows for the facial nerve to be

Table 1
Advantages and disadvantages of surgical approaches for removal of vestibular schwannoma

	Translabyrinthine	Middle fossa	Retrosigmoid
Advantages	Consistent facial nerve identification No tumor size limitation No intradural drilling Wide exposure posterior Fossa Low recurrence rates ABI placement possible Low rate of headaches	Best hearing preservation No intradural drilling Low rate of headaches	No tumor size limitation Hearing preservation possible Wide exposure brainstem ABI placement possible Consistent facial nerve identification Neurosurgeon familiarity
Disadvantages	Complete hearing loss Neurosurgeon unfamiliarity Abdominal fat graft	Limited tumor size Temporal lobe retraction Limited exposure posterior Fossa Increased risk of recurrence of tumor in an unfavorable position related to the facial nerve	Limited exposure of lateral IAC Intradural drilling Postoperative headaches Cerebellar retraction Facial nerve identification is relatively late in the dissection

Abbreviation: ABI, auditory brainstem implant.

in a favorable place deep in the IAC and places the vestibular nerves laterally, where the surgeon encounters the vestibular nerves initially on IAC dissection. The main disadvantage of this approach is the lack of ability to preserve hearing.

The MFC approach was seldom used for tumors until recently because most tumors remained undiagnosed until they were too large to be removed by MCF. Its popularity has increased recently as imaging technology has allowed for detection of small tumors and tumors with up to 1 cm in the cerebellopontine angle are amenable to this technique. Although this approach has the most favorable hearing preservation rates, the technique is challenging because the facial nerve lies between the surgeon and the tumor and the principal anatomic landmarks for identification of the IAC are less reliable and may be obscured. The superior approach to the IAC makes the facial nerve and the superior nerve the first encountered by the surgeon on dissection through the temporal bone. Elevation and retraction of the temporal lobe also carry the risk of brain injury, including aphasia, seizures, and stroke.

The retrosigmoid approach traditionally has been the most widely used approach because of its popularity among neurosurgeons and its wide versatility. It affords wide exposure of the posterior fossa, can be used for excision of small and large tumors, and offers an opportunity for hearing preservation. Intradural drilling of the IAC is required. Like the translabyrinthine approach, lateral surgical approach allows for the facial nerve to be in a favorable position deep in the IAC and places the vestibular nerves laterally, where the surgeon encounters the vestibular nerves initially on IAC dissection.

Surgical approaches

Retrosigmoid approach

The patient is positioned in a modified park bench position, lying supine with the ipsilateral shoulder and hip bumped with rolls and padding. The patient's head is flexed and rotated toward the opposite shoulder and secured with Mayfield pinions. This simple and straightforward position enables excellent visualization of the contents of the posterior fossa. Extreme rotation and flexion may cause venous occlusion and should be avoided. Somatosensory evoked potentials are monitored throughout the case. Potential changes in the waveforms may indicate compromise of the vascular system or spinal compression. Hair is prepped with betadine solution and shaved for four finger breadths behind the ear.

As shown in Fig. 1, a curvilinear "C" incision is made into the posterior scalp down onto the neck. The incision is approximately 4 cm posterior to the ear canal. The incision is carried through

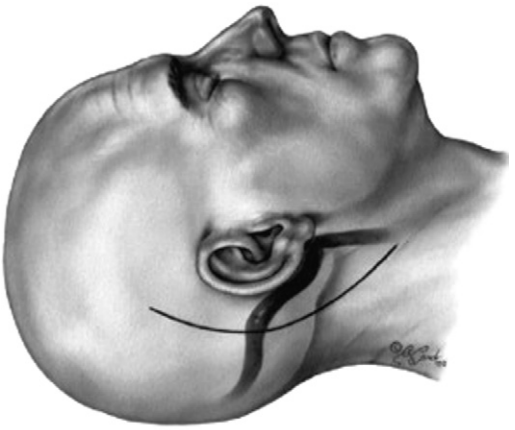

Fig. 1. Skin incision for retrosigmoid approach.

the skin and subcutaneous tissues and an anterior flap is dissected for nearly 1 cm. Electrocautery is used to incise the musculoperiosteum down to the bone. Offset incisions allow for overlapped closure and a reduced incidence of incisional cerebrospinal fluid (CSF) leaks.

As shown in Fig. 2, a 4 × 4 cm craniectomy is performed. The anterior and superior limits of the craniotomy are the sigmoid and transverse sinuses, respectively. As a general rule, the external ear canal approximates the level of the transverse sinus. Laterally exposed mastoid air cells near the sigmoid sinus are occluded with bone wax to prevent transgression of CSF at initial opening

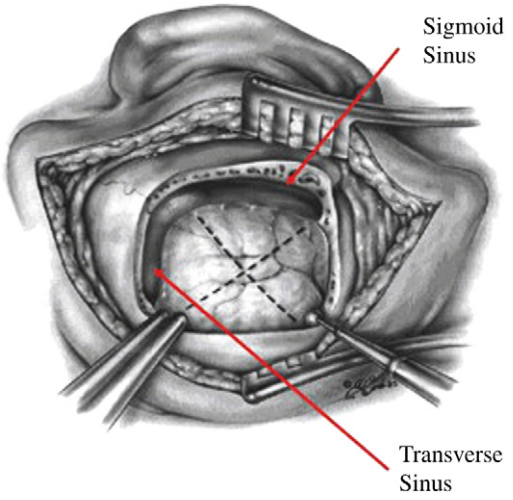

Fig. 2. Craniotomy for retrosigmoid approach.

and again at the end of the case. The dura is then opened, as shown along the dashed lines in Fig. 2, and the dural flaps are reflected laterally and secured with sutures. The dural flaps are routinely irrigated throughout the case to avoid desiccation. Moist cottonoid pledgets are placed over the cerebellum to avoid intraoperative injury and prevent desiccation throughout the case. The cisterna magna is opened anteroinferiorly to the cerebellum with a right angle pick to allow CSF egression, which maximizes posterior fossa relaxation and minimizes the need to retract the cerebellum during the case.

The tumor and the seventh to eighth nerve complexes are identified at the brainstem and dissection is performed in a cooperative fashion between the neurosurgeon and neuro-otologist. The lateral surface of the tumor is examined for a rare lateral displacement of the facial nerve or major vascular structure. Visual inspection of the IAC is often difficult until large tumors are internally debulked. If still difficult to visualize, the IAC can be palpated with blunt instrumentation. The dura above the IAC is coagulated with bipolar cautery in an arc several centimeters above the posterior lip of the IAC before making a similar incision. The dura is then elevated down to the porus acousticus of the IAC and reflected over the tumor. Several cotton balls are placed medially to the IAC to prevent bone dust from accumulating in the cerebellopontine angle.

Dissection begins with an otologic drill and medium-sized cutting burs until the IAC is approached. Diamond burs are then used to create superior and inferior troughs closer to the IAC until nearly 180° to 270° of bone is removed around the posterior IAC (Fig. 3). Exposure of the IAC continues laterally until the IAC appears normal and the end of the tumor is encountered. For hearing preservation to be accomplished, the common crus of the bony labyrinth and the vestibule must not be injured during dissection. Drilling continues until a thin layer of bone remains over the IAC dura. This bone is removed with careful dissection and the IAC dura is then opened. It is preferred to gain exposure of the IAC lateral to the tumor before opening the dura. Keeping the dura intact provides a protective layer to the contents of the IAC during drilling. Opening the dura too early requires further drilling laterally to gain tumor control with an open IAC. The superior vestibular nerve, inferior vestibular nerve, and tumor are identified in the posterior aspect of the canal. Gentle retraction

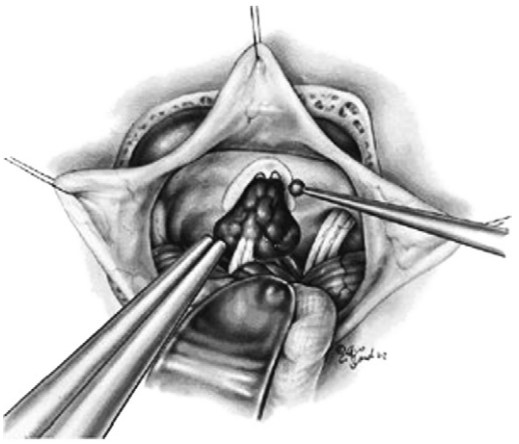

Fig. 3. IAC dissection in retrosigmoid approach.

of the superior vestibular nerve reveals the facial nerve, which is identified physiologically with a stimulator at minimal settings (0.05 mA). The vestibular nerves are sectioned laterally, and tumor dissection is performed in the plane between the tumor and the facial and cochlear nerves in a lateral to medial fashion. Endoscopic assistance with a 30° endoscope also may be valuable to inspect the lateral IAC and reduce the risk of residual disease.

Once the tumor dissection is complete, the operative field is copiously irrigated and hemostasis is achieved with bipolar cautery. The entire bone surrounding the IAC is sealed with bone wax applied with a cotton pledget. Gelfoam is then placed over the remaining seventh and eighth nerve complex and fibrin glue is used to reinforce the seal of the IAC. The dura is then closed with running 3-0 nylon closure. Fibrin glue is placed over the closure of dura. The subcutaneous tissues and skin are closed in several layers with absorbable 2-0 and 3-0 vicryl sutures and staples are placed in the skin. A compressive dressing is placed over the wound to apply pressure in the postoperative period. The patient is kept in the neurointensive care unit for 24 to 48 hours and kept on steroids and antibiotics.

Translabyrinthine approach

The patient is placed in the supine position and the head is turned away from the operative side. The initial positioning is much simpler with this approach than with the retrosigmoid approach—no pins, head holders, or park bench positioning is required. The hair is prepped with betadine solution and four fingerbreadths of hair are shaved above and behind the auricle. As shown in Fig. 4, a C-shaped incision is made extending from one finger breadth above the ear and two finger breadths behind the postauricular crease down to one finger breadth posteroinferiorly to the mastoid tip. The incision is made through the skin and a skin flap is elevated for 1 cm toward the ear through the entire incision. The stepped incision allows for easier closure and prevention of CSF leak. A large superficial temporal fascial graft is harvested and placed on a block. As shown in Fig. 4, a C-shaped musculoperiosteal incision is made with electrocautery from just above the temporal line to the mastoid tip. This incision is offset from the cutaneous incision by 1 cm throughout the incision. The periosteum is elevated to the external auditory canal and retracted with dura hooks. Several large pieces of digastric or sternocleidomastoid muscle are harvested for later use in packing the middle ear.

The bony exposure is performed in three stages: complete mastoidectomy, labyrinthectomy, and IAC dissection. Most of the drilling is performed with cutting burs; however, diamond burs are used when dissection is around critical areas. A complete mastoidectomy is performed, skeletonizing the bone overlying the sigmoid sinus and the tegmen. As shown in Fig. 5, the facial nerve is identified throughout its mastoid course with a large diamond bur. In previous reports the facial recess was opened, the incudostapedial

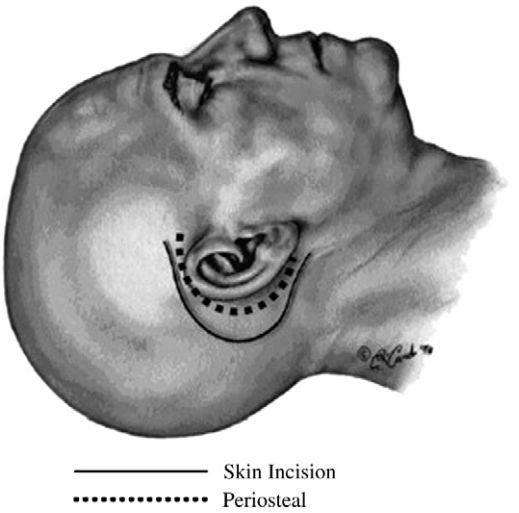

Fig. 4. Skin and periosteal incisions for translabyrinthine approach.

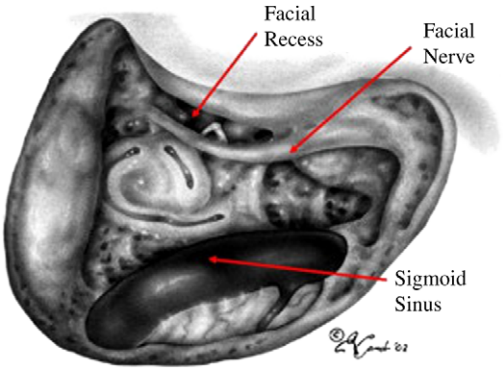

Fig. 5. Mastoidectomy and start of labyrinthectomy.

joint was separated, and the incus was removed. Currently, the senior author does not open the recess or remove the incus unless needed for exposure, leaving less exposed area to seal from CSF leak. All bone is removed from the middle fossa dura, sinodural angle, sigmoid sinus, and posterior fossa down to the level of the labyrinth. Nearly 1 cm of bone is also removed from the posterior fossa dura behind the sigmoid to allow for sinus retraction if necessary.

A complete labyrinthectomy is performed using a diamond bur after the labyrinth is skeletonized and the jugular bulb is identified. The three semicircular canals are removed starting with the horizontal canal. The posterior canal is then opened from its midportion to the common crus. The superior canal is removed and the inferior portion of the posterior canal is removed. The vestibule is opened widely.

Bone from the inferior, posterior, and superior aspects of the IAC is removed with diamond burs. The orientation of the IAC is roughly parallel to the external auditory canal. All bone surrounding the IAC is skeletonized to an eggshell thickness from the middle and posterior fossa dura onto the IAC before removing any bone or opening the dura. The inferior border of the IAC is skeletonized before the superior as the facial nerve is in the superior IAC. The fundus of the canal is exposed just medial to the vestibule, and the transverse crest that separates the vestibular nerves is identified.

The dura of the IAC is then sharply opened. The transverse and vertical crests in the lateral aspects of the IAC are identified with a right angle pick. The superior vestibular nerve is gently displaced while palpating the vertical crest or ''Bill's bar'' to allow visualization of the facial nerve, which is confirmed electrophysiologically with stimulation at minimal settings (0.05 mA) (Fig. 6). A small right angle hook is used to avulse the superior vestibular nerve laterally and expose the facial nerve. The inferior vestibular nerve and cochlear nerves are then separated laterally. The contents of the IAC are dissected from the facial nerve in a lateral to medial fashion. The posterior and middle fossa dura surrounding the IAC is opened for visualization of the cerebellum, brainstem, and cerebellopontine angle component of the tumor. Internal debulking of the tumor is performed with ultrasonic aspiration or a laser to allow for infolding of the tumor and dissection away from the facial nerve and brainstem. The remainder of the tumor is dissected from the facial nerve and brainstem until completely removed.

The surgical field is then copiously irrigated. Meticulous attention to closure is important for preventing CSF leak. The packing of the eustachian tube orifice and the middle ear is important in that regard. The bone of the promontory is abraded with a small right angle pick and the middle ear, vestibule, and eustachian tube are occluded with previously harvested muscle. The fascia graft is then used to cover the antrum. No attempt at primary reconstruction of the opened posterior fossa dura is performed at our institution because it is removed at initial opening. Abdominal fat is cut into strips and used to pack the mastoid defect down to the level of the IAC. The mastoid periosteum is then closed with interrupted 2-0 vicryl sutures. The skin is closed watertight with 3-0 vicryl sutures and staples externally. A compressive dressing is placed on the incision.

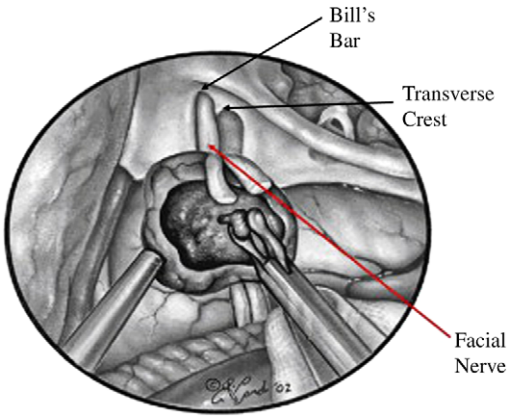

Fig. 6. IAC dissection in translabyrinthine approach.

Middle fossa approach

The patient is placed in a supine position with the head turned to expose the ear of interest. The hair is prepped with betadine solution and four fingerbreadths of hair are shaved above the auricle. The patient is then prepped and draped in the usual fashion. Unlike other surgeries, the surgeon sits at the head of the bed. Although some surgeons prefer a vertical incision through the skin, a broad, posteriorly based, U-shaped skin flap is designed. The anterior aspect of the incision should be left posterior to the natural hairline to camouflage the scar. As shown in Fig. 7, an anteriorly based U-shaped incision is made through the musculoperiosteal layer to skull after the entire skin flap is elevated posteriorly. A Lempert elevator is used to elevate the periosteum to the anterior limit of the incision.

Careful palpation with blunt instrumentation allows identification of the external auditory canal. A 4 × 4 cm craniotomy is performed with the opening roughly two-thirds anterior to the external auditory canal and one-third posterior. A 4-mm cutting bur is used for most bone removal, but a diamond bur is used to remove the last layers of bone over the dura. The bone flap is dissected free from the underlying dura and preserved for reconstruction at the end of the case. The bone inferior to the craniotomy is removed down to the level of the external auditory canal.

Using the microscope and a freer elevator, the middle fossa dura is then elevated off the temporal bone medially to the petrous ridge. Dissection proceeds in a posterior to anterior direction to avoid trauma to the geniculate ganglion and greater superficial petrosal nerve, which may be dehiscent in up to 15% of patients. Anteriorly, dissection stops short of the foramen spinosum and middle meningeal artery.

As shown in Fig. 8, a House-Urban retractor facilitates retraction after dural elevation. Pressure from CSF is relieved with hyperventilation to lower pCO_2 and mannitol. The temporal floor is inspected for clues to identifying the IAC, including the arcuate eminence overlying the superior semicircular canal, the greater superficial petrosal nerve, and the external auditory canal. The semicircular canal is blue-lined in an anterolateral direction to expose the lateral extent of the canal near the IAC.

Although there are several methods available to identify the IAC, we prefer to identify the IAC medially away from the labyrinth and cochlea at the porus acousticus. The plane of the IAC can be approximated by bisecting the angle between the greater superficial petrosal nerve and superior semicircular canal. Drilling along the petrous ridge helps localize the IAC safely. As drilling is extended laterally toward the fundus, smaller diamond burs are used and dissection is performed directly over the canal to avoid injury to the underlying cochlea anteriorly and labyrinth posteriorly. The facial nerve is traced laterally to expose the geniculate ganglion and the labyrinthine segment of the facial nerve.

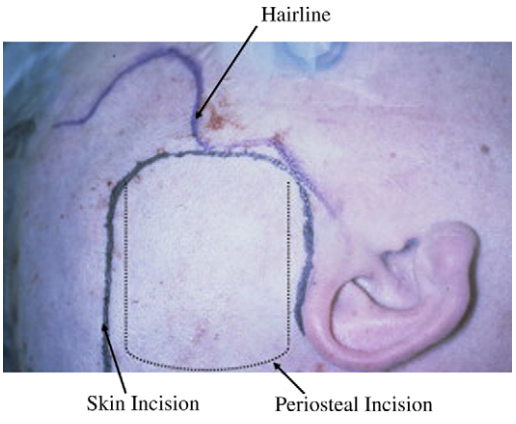

Fig. 7. Skin and periosteal incisions in middle fossa approach.

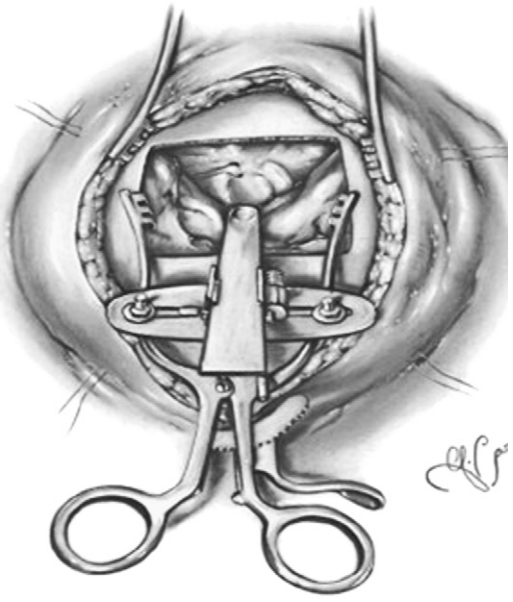

Fig. 8. Middle fossa exposure after dura retraction.

The dura of the IAC is then opened parallel to the long axis of the IAC. The facial nerve is identified in the anterior aspect of the IAC and physiologically with a facial nerve stimulator on minimal settings. The tumor is dissected from the facial nerve throughout the IAC. Once the facial nerve is separated, a small right angle hook is used to avulse the superior and inferior vestibular nerves laterally. As shown in Fig. 9, the tumor and vestibular nerves are then dissected free from the facial and cochlear nerves in a lateral to medial fashion. The dissection may be extended medially along the petrous ridge for larger tumors. Once the tumor is dissected free from the IAC, the vestibular nerve is sharply sectioned medially and the tumor is removed.

The surgical field is then copiously irrigated. The peri-IAC air cells are sealed with bone wax to avoid postoperative CSF leak. Muscle or fat is placed in the lateral IAC. The temporal lobe is then released. The craniotomy is replaced and secured with small titanium reconstruction plates. The musculoperiosteal flap is then secured with interrupted 2-0 vicryl sutures and the skin with 3-0 vicryl sutures. The skin is reinforced with staples, and a compressive dressing is placed.

Complications

Although complications rates are reduced with increasing experience, they still occur at a predictable rate [11]. The key to managing complications is twofold. First, prevent complications from occurring with meticulous technique and attention to detail. Second, detect problems early and initiate appropriate therapy in a timely fashion.

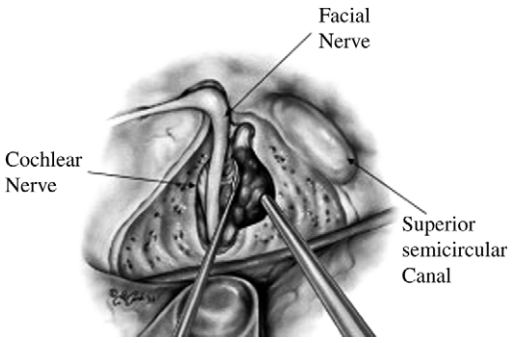

Fig. 9. IAC dissection in middle fossa approach.

Recurrence

Recurrent tumor is usually the result of regrowth of residual tumor fragments. Despite total removal of all microscopically visible tumor, remnants of nerves may contain undetectable tumor cells. Because recurrent tumors do not cause symptoms until they become extremely large, imaging with gadolinium-enhanced MRI is the primary way to detect a recurrence [12]. Because imaging protocols can be used to suppress the signal from fat (fat suppression protocols) and other reconstruction materials, including muscle and fascia, may become indistinguishable from tumor, fat should is best used to reconstruct surgical defects [12]. Linear enhancement of the dura is consistent with inflammation or scar, whereas nodular enhancement is suspect for recurrent or residual tumor [13]. Postoperative MRI within the first year often shows contrast enhancement of dura in involved surgical resection secondary to inflammation and scar. If the image shows an abnormality such as dural enhancement, we proceed with imaging 2 years later. If the first image is normal, a single follow-up scan is obtained at 5 years, with the exception of patients with NF2 who require continuing surveillance.

Subtotal removal of tumor is sometimes necessary to preserve the integrity of the facial nerve or brainstem. Rigorous hemostasis during surgery reduces the vascularization of the remaining tumor and may stop regrowth of tumor [12].

Shelton reported 0.3% recurrence rate on more than 1500 translabyrinthine approaches. No predictive factor of recurrence has been identified for recurrence, including tumor size or patient age [14]. Recurrences are rare after 10 years, regardless of the approach. One disadvantage of the hearing preservation approaches is that the dissection in the lateral IAC dissection is often performed blindly and tumor may be missed in the fundus of the IAC. Blevins and Jackler [15] demonstrated that the lateral one third of the IAC must be left unexposed to avoid injury to the underlying labyrinth in the retrosigmoid approach. In the middle fossa approach, Driscoll and colleagues [16] found that the lateral 14% to 34% of the inferior IAC is obscured by the transverse crest. This would only present a problem in lateral tumors that originate on the inferior vestibular nerve. The translabyrinthine approach allows visualization of the entire IAC and allows dissection of tumor from facial nerve from the fundus of the IAC to the brainstem. The cochlear nerve

is usually removed, which reduces the risk of residual tumor cells being left behind.

Recently, there has been growing interest in the use of endoscopes during surgery to see beyond the limits of the microscope and "to look around the corner." Endoscopes offer outstanding illumination and allow for dissection of neoplasm in the most lateral aspect of the IAC without disruption of the labyrinth [17]. Difficulties passing angled scopes without damaging vital structures and concerns over increased local temperature caused by the light have limited its use [18].

Medical complications

The mortality for VS surgery is approximately 1% in most large series and usually is the result of neurovascular insult [19]. A preoperative medical evaluation by an internist who follows patients postoperatively helps minimize nontumor complications, including myocardial infarction, pneumonia, pulmonary embolus, and deep venous thrombosis, that occur in frequencies similar to other major surgical procedures [20].

Tinnitus

Although more than half of patients without tinnitus preoperatively experience it postoperatively, nearly 50% of patients with preoperative tinnitus experience a significant reduction in their tinnitus postoperatively [21]. Most patients compensate well to the tinnitus and rarely are distressed by symptoms.

Equilibrium

Most patients experience acute vertigo after surgery for VS. The severity of postoperative vertigo is directly proportional to the preoperative vestibular function seen on preoperative electronystagmography. Preoperative electronystagmography also identifies patients with bilateral vestibular hypofunction who may have more difficulty compensating after surgery. Patients begin vestibular rehabilitation early in the postoperative period and are encouraged to participate in activities that challenge their vestibular system after discharge. Recovery of balance is nearly universal by 6 to 9 months after surgery.

Cerebrospinal fluid leak

CSF leak is the most common postoperative complication after VS resection, and it occurs in approximately 10% of patients [22]. The MCF approach has a slightly lower rate than the other two approaches [22]. CSF leaks complicate the postoperative course because they can lead to meningitis, increased hospital stay, and reoperation. CSF leaks increase the risk of meningitis. Although the overall risk of meningitis is between 1% and 3%, a CSF leak raises the risk to roughly 14% [22]. Leaks may be evident immediately after surgery or after discharge from the hospital.

CSF may escape through the skin or the nose via the eustachian tube. Although most leaks after the translabyrinthine and MCF approaches present as rhinorrhea, rhinorrhea and incisional leaks are nearly equal after the retrosigmoid approach. CSF leaks through the incision are secondary to failure to obtain a watertight closure. They generally respond to conservative therapy, such as bedside placement of a suture, pressure dressings, and bed rest, in 60% to 70% patients [23]. Rarely is a lumbar drain or operative intervention required. CSF rhinorrhea is the result of spinal fluid gaining access to the middle ear cleft or eustachian tube, and it usually requires more invasive measures because spontaneous resolution occurs in only approximately 10% to 20% of cases [19]. An additional 30% to 70% of cases may resolve with the placement of a lumbar drain [23]. Typically, lumbar drains are used for 2 to 5 days. The drain is then clamped and, if the leak does not recur, it is removed. Patients must be closely monitored, which often necessitates a prolonged intensive care unit course. Although lumbar drains are successful in most patients, operative intervention has the highest success rate (80%–90%) [23].

Closure of the middle ear and eustachian tube with temporalis muscle creates a significant conductive hearing loss, which is insignificant after the translabyrinthine approach or other nonhearing conservation procedures. Although opening a facial recess affords better visualization of the eustachian tube, it also opens a larger route to the middle ear cleft. The vestibule is also packed with muscle and sealed with fibrin glue to prevent CSF from leaking through the vestibule and stapes footplate into the middle ear. If a CSF leak occurs in the immediate postoperative period, conservative therapy with head-of-bed elevation and a pressure dressing may be successful. Persistent drainage requires a lumbar drain or re-exploration of the surgical field. If the CSF leak persists, eustachian tube obliteration may be necessary.

The lateral dura contracts and hardens during the case, which often makes a water-tight closure difficult after the retrosigmoid approach. Reinforcement with synthetic dura lateral to the dural closure helps reduce incisional leaks. CSF also may present as rhinorrhea after the retrosigmoid approach. CSF may enter the middle ear cleft through nonoccluded peri-IAC air cells directly or mastoid air cells near the craniotomy site. The lateral cells should be occluded with bone wax initially after the craniotomy and again at closure. The peri-IAC cells are more difficult to obliterate and require diligent application of bone wax after tumor dissection in the IAC. Sealing this area with fibrin glue also helps to reduce the incidence of CSF leak. Postoperative CSF rhinorrhea is usually controlled with conservative measures, including bed rest and lumbar drain placement. Recalcitrant cases may require reoperation with resealing of the mastoid and peri-IAC air cell system. If the CSF leak persists, a mastoidectomy, packing of any retrosigmoid-mastoid fistula, or obliteration of the middle ear and eustachian tube may be indicated.

CSF leaks after MCF almost exclusively present as rhinorrhea. CSF may leak through peri-IAC air cells or defects between the IAC and middle ear. The defects and air cells around the IAC are occluded with bone wax and fat is used to plug the lateral IAC defect at the end of the case. If a leak develops postoperatively, observation, bed rest, and lumbar drainage are often successful. Recalcitrant cases may require exploration through a transmastoid approach to identify the problem. If no useful hearing is present, obliteration of the middle ear and eustachian tube often stops the CSF rhinorrhea.

Meningitis

Increased survival after VS surgery is in no small part secondary to the dramatic reduction in bacterial meningitis since the advent of antibiotics. Meningitis occurs in 1% to 8% of cases and may be either bacterial or aseptic [24]. Aseptic meningitis occurs more commonly than bacterial and is usually secondary to inflammation from bone dust or blood. Meningitis rates are several folds higher in the presence of a postoperative CSF leak [22]. The diagnosis is difficult because most patients already have symptoms of headache, neck pain, and low-grade fevers in the immediate postoperative period. Any suspicion necessitates a lumbar puncture and aggressive therapy with broad-spectrum antimicrobial therapy and steroid prophylaxis while awaiting cultures. Although spinal fluid analysis is often difficult because of contamination with normal postoperative inflammatory mediators and blood, protein counts >200 mg/dL, glucose <40 mg/dL, and white blood cell counts $>1000/mm^3$ are important clues in making the diagnosis of bacterial meningitis [25]. Aseptic meningitis responds quickly to steroids, which are slowly tapered over 3 to 4 weeks to prevent recurrent symptoms.

Facial nerve

Standard universal reporting of outcomes with the House-Brackmann facial nerve scale allows for easy comparison between studies. Generally, grade I or II is defined as good postoperative facial nerve function, grade III or IV as intermediate, and grade V or VI as poor. The importance of neurophysiologic monitoring toward facial nerve outcomes cannot be stressed enough in skull base cases.

The facial nerve is thinned with tumor growth, which reduces the nerve's resilience to manipulation. In studies with large series, facial nerve outcomes are reduced with increasing tumor size or preoperative nerve dysfunction [19]. Tumors may be defined by the size of the tumor in the cerebellopontine angle as intracanalicular (none), small (<15 mm), medium (15–30 mm), and large (>30 mm). The facial nerve is anatomically preserved in 95% to 100% of cases and nerves that stimulate with minimal settings have a 90% chance of grade I-II outcomes at 1 year [26]. In the event of facial nerve transaction, immediate repair should be accomplished; postoperative care with artificial tears, lubricant, and gold weight insertion are encouraged while the facial nerve regains function. Good facial nerve outcomes should be seen in at least 95% of intracanalicular tumors, 85% of small tumors, 75% of medium tumors, and 40% of large tumors [19]. Although a surgeon's experience correlates directly with facial nerve outcomes, there does not seem to be a difference among the surgical approaches [7,27,28]. Tumors resected by a retrosigmoid or MFC approach are more likely to have immediate facial nerve weakness because of neuropraxic injury from intraoperative manipulation. At 1 year there was no difference between approaches, however [29–40].

Delayed facial nerve dysfunction may occur in up to 40% of cases [41]. Similar to Bell's palsy, the delayed weakness has been attributed to edema of the facial nerve or reactivation of virus molecules in the geniculate ganglion. Fortunately, steroids reduce this edema; most patients (90%) recover completely and the remaining patients regain most of their facial nerve function.

Hearing preservation

The three most important factors in determining hearing preservation are preoperative hearing thresholds, tumor size, and the distance between the lateral extension of tumor and the vestibule [42–44]. Two surgical approaches allow for hearing preservation by leaving the labyrinth and cochlear nerves intact: the MCF and the retrosigmoid. Intraoperative monitoring allows for detection of hearing function and preservation. Alterations in the intraoperative auditory brainstem responses should prompt cessation of surgical manipulation until tracings return to baseline. Although there are various classification systems, the American Academy of Otolaryngology–Head and Neck Surgery system is the most universally adopted system. The system is useful to help gauge overall hearing, but useful hearing depends on preoperative hearing and more importantly the status of the contralateral ear. This classification scheme is particularly misleading in patients who fall near division lines because clinically insignificant changes may result in class changes (Table 2).

The overall rate of serviceable hearing preservation, class A or B, for tumors <2 cm is 40% to 50% [24]. The MFC yields better hearing preservation than the retrosigmoid approach. The MFC yields hearing preservation rates between 50% and 70%, whereas the retrosigmoid approach yields hearing preservation between 30% and 40% [24,45]. The better results of the MFC route may be related to better preservation of the vascular supply to the cochlea and cochlear nerve or smaller tumors generally removed through this approach. The internal auditory artery runs in the anterior IAC between the facial and cochlear nerves and is protected by the facial nerve from manipulation in the MCF approach. One advantage of the retrosigmoid approach is the unique possibility of real-time intraoperative monitoring via direct recordings from the eighth nerve at the brainstem.

Table 2
American Academy of Otolaryngology—Head and Neck Surgery hearing classification scheme

Class	Pure-tone threshold (dB)	Speech discrimination (%)
A	≤ 30	≥ 70
B	>30 but ≤ 50	≥ 50
C	>50	≥ 50
D	any	<50

The likelihood of hearing preservation also depends on the distance between the tumor and the IAC fundus. Generally, if the distance from the IAC fundus is <3 mm, the likelihood of hearing preservation is lowered [46]. Hearing is vulnerable when the tumor extends laterally for two reasons: (1) The cochlear nerve is vulnerable to avulsion from the modiolus when dissecting tumor laterally, which is the precise reason for sharp and not blunt dissection. (2) The labyrinth or cochlea also may be injured in extending exposure laterally.

Headaches

The incidence of headache that lasts beyond the initial postoperative period ranges from 0% to 65% [47–50]. It does not seem to be correlated with age, gender, and operative time. Patients with smaller tumors tended to have more headaches. Persistent headaches are not common in patients undergoing resection of VS via either MFC or translabyrinthine approaches [47,49]. Initial discomfort after surgery is expected and related to the incision, reduced CSF pressure, dural irritation, and muscle spasm [50]. Fortunately, most headaches resolve over subsequent months to years, and only 10% of patients continue to experience headaches.

Several mechanisms have been proposed for headache after retrosigmoid excision of acoustic neuroma. Tight dural closure may result in excess tension and irritation of the dura. Dural adhesions to nuchal muscles also may result in intermittent stretching and traction of the dura with head movements or straining. Finally, intradural drilling results in bone dust, which may be the source of aseptic meningitis and associated headache. The surgeon must attempt to prevent the incidence of headaches when possible. First, careful placement of Gelfoam in the posterior fossa may collect bone dust during drilling and reduce postoperative inflammation. Second, periodic moistening of the dural flaps helps prevent

dessication and allows easier closure. Finally, replacement of bone flap at the end of surgery significantly reduces postoperative headaches [50–53]. Most headaches can be controlled with anti-inflammatory medicines, including aspirin, acetaminophen, and ibuprofen. Pain that requires narcotic analgesia necessitates a neurologist–headache specialist referral.

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NEUROSURGERY
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LINAC Radiosurgery and Radiotherapy Treatment of Acoustic Neuromas

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Acoustic neuromas (ANs) are benign, slow-growing tumors that arise from the Schwann cells of the vestibulocochlear nerve. Although these tumors are benign, their expansion in the internal auditory canal and cerebellopontine angle compresses the cranial nerves and the brainstem [1]. Once diagnosed, the tumors are commonly resected microsurgically, managed conservatively with radiologic surveillance, or treated with radiation therapy [2]. This article focuses on the effectiveness of linear accelerator (LINAC) stereotactic radiosurgery and radiotherapy in AN treatment.

Radiation biology

Radiation-induced damage to the cell machinery has been used to control the proliferation of tumors. Interruption of cell division is a desired effect in cells that have lost the ability to respond to appropriate internal and external surveillance because of mutation. The therapeutic role of ionizing X-rays and gamma rays lies in their ability to compromise the integrity of DNA and other important biologic molecules of the target cell. Specifically, excitation of electrons in molecules exposed to radiation produces reactive, free radicals, which in turn degrade structures that are important for cell survival. Double strand breaks in the DNA are most detrimental to the cell. Effective mechanisms for fixing the double strand breaks, which include nonhomologous end joining and a more precise homologous recombination, are often inadequate to deal with the overwhelming damage to the genetic material, especially in highly mutated tumor cells. These lesions prevent the cell from completing the replication cycle and arrest the growth of the tumor. Excessive radiation damage to DNA and the arrest of mitosis lead to induction of the apoptotic—or programmed—cell death pathway. Radiation damage to phospholipids in the membrane of the cell also acts to trigger cascades that lead to cell death [3,4].

There are two types of ionizing radiation: electromagnetic, which consists of photons or packets of energy, and particulate radiation. The common feature of these forms of radiation is ionization, a process that ejects an excited electron from the target atom. This process occurs when a photon or particle transfers its energy to the target tissue. X-rays and gamma rays are

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indirectly ionizing, whereas charged particles are directly ionizing. X-rays and gamma rays are types of electromagnetic radiation and differ in that an X-ray is a result of a collision between an electron and a target, whereas gamma rays are produced when the contents of the nucleus of an atom return to their initial energy state from an excited level, a process known as gamma decay. As these electromagnetic waves pass through tissue, they are absorbed and produce fast recoil electrons. As this occurs, they lose intensity.

Protons are heavy charged particles that cause damage directly. They have an opposite charge from electrons and have considerably more mass. When a proton beam strikes a target, it deposits almost all of its energy at the end of its range. This characteristic, known as the Bragg peak, can be exploited to deliver high doses to tumors with almost no fall-off of radiation. Helium ions act similarly. Neutrons have no charge and are equivalent to a proton in mass. They are not affected by an electric field. They are also indirectly ionizing, and after interacting with matter they produce recoil protons and alpha particles. Although all other radiation modalities work best on parts of tumors in which oxygen is available, neutron radiation provides an opportunity to target the center of large tumors that are oxygen deficient and types of tumors known to be poorly aerated [5].

Radiosurgery (RS) is a term used for the delivery of ionizing radiation to an intracranial target with the use of stereotactic technique. The exposure of normal, healthy tissues to radiation should be limited, yet significant doses must be delivered to the tumor to have a therapeutic effect. Stereotactic technique facilitates the accomplishment of these goals through accurate localization and targeting of the lesion. The target volume is represented in a three-dimensional space with the use of markers on a fixed frame, the location of which is electronically linked with either CT or MRI scans. Fig. 1 shows the planning of a treatment based on the MRI of a patient with AN. Special care must be taken to isolate and protect the brainstem, which is in close proximity to the target volume. The three-dimensional target created is irradiated from several angles; beams all meet and deliver a high cumulative dose of radiation to a single isocenter. For irregularly shaped targets, more than one isocenter can be used. Three modalities are used to deliver radiation in stereotactic RS [6].

As of 2003, approximately 200,000 patients were treated with gamma knife technology. Gamma knife contains 201 cobalt 60 sources

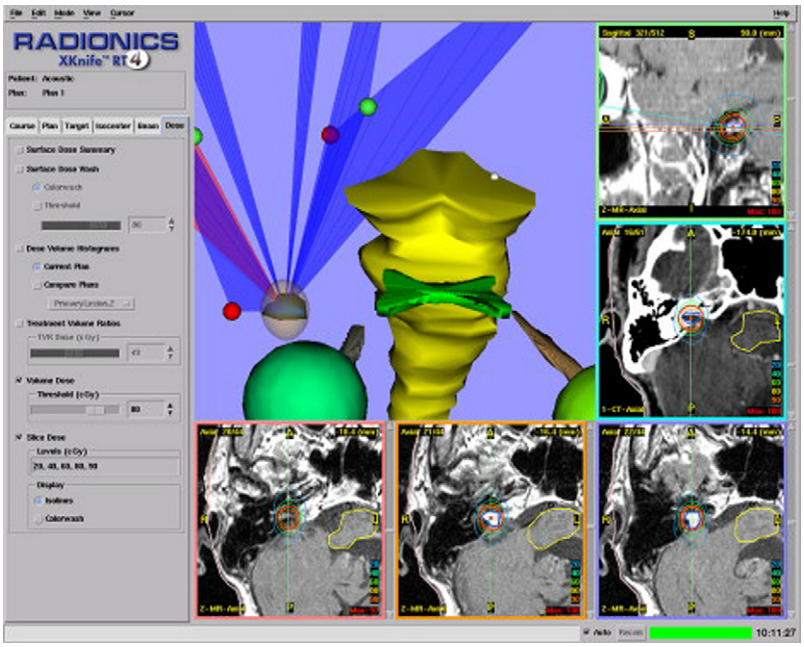

Fig. 1. This computerized treatment plan reveals MRI defining the target (encircled in red) and the area of concern to be isolated, the brainstem encircled by an oblong shape. The mock treatment plan shows the eyes and the brainstem, which are isolated from the target. The radiation beams are shown converging on the target, in this case an intracanalicular AN.

that are collimated using a helmet fitted for patients. Once the treatment is planned using the three-dimensional representation of the target, a patient's head is fixed and the beams from the sources align on multiple isocenters [6].

LINAC is a tool that has long been used to generate X-rays. LINAC uses electromagnetic waves of microwave frequency to accelerate electrons. This first became possible with technology borrowed from high-energy microwave generators used in military radars during World War II. LINAC was first used for radiotherapy in 1953 [7]. More recently, in the early 1980s, it was adapted for use in stereotactic RS. Many LINAC units have been modified for RS, whereas others are designed specifically for this use. It is estimated that more than 30,000 people have undergone LINAC-based RS. LINAC delivers a photon beam of high-energy X-rays through a series of arcs or fixed static fields. Conformality is maximized with the use of micro-multileaf collimators. Conformality can be enhanced further with the use of intensity modulated RS, which varies the intensity of dose within a field to treat tumor and spare normal tissue.

Another modality that has been used in RS over the past 40 years is particle beam irradiation. Two to six beams of charged particles are focused on the target. One example is the use of protons in radiation of intracranial tumors. As in the other two modalities used for RS, the radiation dose that reaches structures outside the target volume is minimized by the effect of the Bragg peak [6].

Stereotactic radiotherapy, or fractionated stereotactic radiotherapy (FSRT), is an alternative to RS. It differs in that the full radiation dose is not administered at one time but is instead divided into several doses. The accuracy of irradiation is reproduced with the use of a head frame, such as the GTC (Gill Thomas Cosman) (Fig. 2), which ensures that the treatment is consistently targeted each time. A bite plate may be used to individualize the frame for each patient and achieve reproducibility with multiple treatments (see Fig. 2; Fig. 3). Using smaller doses over many fractions reduces late side effects, such as hearing loss, when compared with single fraction RS [8].

Natural history of acoustic neuromas

When evaluating the effect of radiation therapy on the growth of ANs, the natural history of these tumors must be considered. One of the options used in the management of patients who have

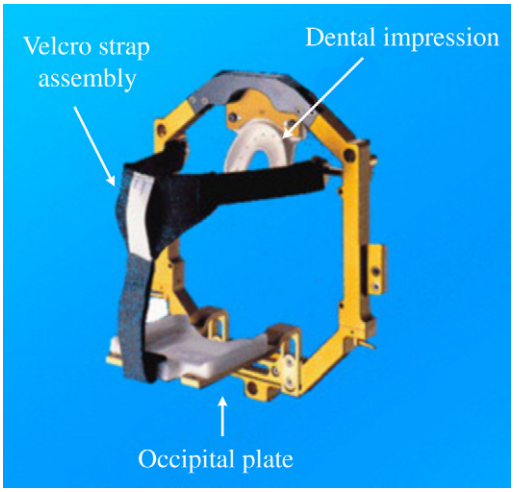

Fig. 2. A removable and reusable stereotactic device using an oral bite plate to ensure placement reproducibility.

ANs is radiologic surveillance of the tumors with repeat MRI scans. Since the introduction of gadolinium-enhanced MRI, tumors of smaller size can be detected and followed over time. A significant amount of data on the natural history of AN tumors has been collected from patients who choose this conservative option.

A review of studies on the surveillance of ANs was published by Selesnick and Johnson [9] in 1998. The authors presented a meta-analysis of 13 studies on the topic published between 1985 and 1997. In all, 571 patients across the studies

Fig. 3. This patient has been placed in the removable reusable stereotactic device using an oral bite plate to ensure placement reproducibility. The patient is positioned for treatment.

evaluated were followed for an average of 36 months. The mean diameter of tumors at the beginning of the surveillance was 11.8 mm, and 54% of the tumors (range 14%–74%) grew at an average rate of 1.8 mm per year. Slightly less than half of the patients included in the meta-analysis had no AN growth at a mean follow-up of 3 years. Among the patients whose tumor size increased ($n = 178$), the growth rate was 4 mm per year. Initial size of the tumors and the age of the patients were not found to be predictive of growth of AN.

A more recent meta-analysis published by Yoshimoto [10] in 2005 reviewed 26 studies examining the natural course of AN growth published between 1991 and 2002. In the 1340 patients included, the mean initial tumor diameter was 11 mm, and 46% (range 15%–85%) had an increase in tumor size during the mean follow-up period of 38 months (range 6–64 months). A mean growth rate of 1.2 mm per year was calculated from the 16 studies ($n = 964$) that contained this information. Eight percent of patients had a spontaneous decrease in tumor size. The author also analyzed a subgroup of 12 studies that used only MRI and not CT for tumor surveillance. In this group, 39% of tumors increased in size in the mean follow-up time of 33 months.

Since the Yoshimoto's review, Stangerup and colleagues [11] reported on 552 patients from a single institution who had ANs not associated with neurofibromatosis type 2 (NF2) and who underwent radiologic surveillance of tumors with repeated MRI scans. The data were collected for the patient population managed from 1975 to June 2005, and the mean follow-up was 43 months (range 12–180 months). Tumors were characterized as either intrameatal or extrameatal and were analyzed separately. Of the 230 intrameatal tumors, 17% grew to extend extrameatally. Growth occurred 64% of the time during the first year (growth rate, 10.32 mm/y), 23% during the second year (growth rate, 3.83 mm/y), 5% during the third year (growth rate, 2.17 mm/y), and 8% during the fourth year of observation (growth rate, 0.92 mm/y). Extrameatal tumors observed in 322 patients were defined as growing when the largest diameter increased by 2 mm and were defined as shrinking when the diameter decreased by 2 mm. In total, 70.2% of tumors remained stable, 28.9% grew, and 0.9% shrank. Sixty-two percent of growth occurred during the first year (growth rate, 4.90 mm/y), 26% occurred during the second year (growth rate, 2.79 mm/y), 10% occurred during the third year (growth rate, 1.15 mm/y), and 2% occurred during fourth year of follow-up (growth rate, 0.75 mm/y). The difference between growth of intrameatal and extrameatal ANs of 17% and 28.9%, respectively, was reported to be statistically significant. Intrameatal or extrameatal tumor growth was not observed after the fourth year of follow-up.

It is difficult to compare AN growth rates with and without radiosurgical intervention because of differences in follow-up time and methods used to report tumor size and control. Battaglia and colleagues [12] addressed the issue by comparing 111 patients followed radiologically for an average of 38 months at a single institution with data reported in the literature on radiosurgical treatment of AN. Only patients treated with the maximum marginal dose of 12 to 13 Gy administered in a single treatment with any radiosurgical modality were included in the analysis. Mainly, data from studies in which gamma knife surgery was used were represented. Only studies that reported a mean or median follow-up of 24 months were used for comparison. The natural tumor growth rate reported by the authors was 0.7 mm/y, with 18% of tumors growing faster than 1 mm/y and 13% growing faster than 2 mm/y. On average, change in size was noted at 2.2 years after surveillance was initiated. No growth during the follow-up period was noted in 50.5% of the patients. Tumor control in RS studies selected by the authors was defined as no growth more than 2 mm/y or more than 1 mm in two dimensions. Using this same definition, 87% of ANs were controlled by conservative management, a result not significantly different from when the tumors were treated with radiation.

There is some indication that AN tumors do not grow in a linear fashion. In 2000, Tschudi and colleagues [13] reported growth followed by stabilization in some of the patients whose tumors were observed over time. Charabi and colleagues [1] described a group of cases in which the tumor size was stable for an average of 19 months and then experienced a period of growth. This irregularity in the natural progression of the tumors suggests that a period of stability does not necessarily predict long-term control, whereas a period of growth does not predict continuous enlargement.

The fact that the natural history of AN growth undergoes periods of quiescence is important when trying to judge the impact of radiation therapy on these tumors.

Tumor size measurement

The current, preferred method for measuring the size of AN tumors involves the use of MRI. Accuracy and precision are important in determining the initial size of the tumor at presentation and evaluating tumor changes. The consistency of these measurements and the precision with which tumor progression can be followed have limitations.

In 2003, Slattery and colleagues [14] addressed the issue of reliability of MRI-based measurement. Factors that contribute to the quality of the scan include the MRI machine used, how the image is acquired, the training of the technician and the radiologist, whether contrast is administered, what dimension is used to measure the size of the tumor, and how much the patient moves during the scan.

A large source of variability could originate in the method of measurement. The radiologist can measure a distance by eye or use computer software. (The latter approach is considered more precise.) Studies evaluated in our review varied with respect to what constitutes the size of the tumor. Greatest diameter of the AN was commonly used. The American Academy of Otolaryngology–Head and Neck Surgery recommends that two measurements be taken: one parallel and one perpendicular to the petrous ridge. Computer algorithms allow tumor volume to be calculated to evaluate size. Each of these methods varies in its accuracy and precision.

To answer a question of how much consistency there is among data obtained from MRI scans and what is the minimum amount of change that can be detected in tumor size, Slattery and colleagues [14] performed six scans on three different MRI machines (two consecutive scans on each) for seven patients with NF2. T1-weighted post-gadolium-enhanced gradient three-dimensional sequences were used in this study. In total, 20 meningiomas and ANs were used to evaluate inter- and intrascanner reliability. The volume and the greatest diameter were measured using computer software. The authors reported that calculated tumor size was consistent across the two studies conducted on one machine and across the three machines used. They concluded that the minimal difference in tumor diameter that can be detected on an MRI is 1.1 mm, whereas the minimum reliable volume difference is 0.15 mL. The authors suggested that a more consistent protocol be used to measure tumor size in patients with AN.

This finding is important when judging the effect of radiation on acoustic tumors, because small changes in size may not be appreciated or may be overread.

Methods

For the purpose of this article, MEDLINE was searched for studies using LINAC for AN treatment. The terms "vestibular schwannoma," "acoustic neurilemmoma," "acoustic neurinoma," "acoustic neurilemoma," "acoustic schwannoma," and "acoustic neuroma" were crossed with "radiosurger*," "stereotactic radiosurger*," "linear accelerator," "LINAC," "linear accelerator-based surger*," and "X knife." (The use of [*] after a word indicates that all possible endings of the word were incorporated in the search.) Only articles published in English since 1996 were considered for analysis. The abstracts were screened to exclude articles pertaining to the use of gamma knife and other stereotactic radiation modalities. The references of selected articles were reviewed for additional relevant sources. Articles from a singles institution reporting on redundant patient populations were identified, and the more recent and complete studies were chosen for analysis.

Radiosurgery

In one of the earliest publications on LINAC RS for ANs, Chakrabarti [15] presented 11 patients in whom surgery failed to control the tumors and who were treated with stereotactic radiation. The diameter of the tumors ranged from 15 to 35 mm (mean 26 mm), and the lesions were targeted with 12.5 to 20 Gy administered to the 90% isodose contour. Ten patients were followed radiologically with repeat CT or MRI scans or both for 3 to 36 months (mean 15 months). Seven of the patients had observable necrosis in the center of the tumor 6 months after RS. During the period of 7 to 20 months after treatment, tumors decreased in size in 4 patients. No change was observed in four cases, and 1 mm increase was seen in 2 patients. Hearing was diminished in 7 patients before treatment; it was stabilized or improved in 5 of these patients, whereas 2 patients became completely deaf. Although no new facial palsy was found after RS, 4 patients experienced it before treatment. In 2 patients, their preexisting facial palsy was unchanged; it improved in 1 patient at 14 months after RS; it deteriorated

to complete facial palsy in 1 patient. In 1 patient, radiation damage to the brainstem caused imbalance [15].

The experience with LINAC stereotactic RS at Cleveland Clinic Foundation was described by Suh and colleagues [16]. Twenty-nine patients were treated in this study, with 4 suffering from NF2 and 12 having been operated on previously for ANs. The tumor volume ranged from 0.18 to 28.7 mL (median 2.1 mL). Tumors were targeted using a single isocenter in 24 cases and two isocenters in 5 cases. The treatment protocol called for a dose to the periphery that ranged from 8 to 24 Gy (median 16 Gy). Out of concern for possible damage to the brainstem, lower doses were used at first, ranging from 8 to 12 Gy. A 50% to 80% isodose line (median 80%) was used.

Patients were followed for a period of 4 to 110 months (median 49 months), during which they were seen 6 to 8 weeks after the procedure and then at 6-month intervals. The result of the treatment was local control in 28 of 29 patients defined as no increase in tumor size. Decrease in tumor size was achieved in 11 patients, and no size change was seen in 17 patients. This finding translated into an actuarial 5-year control rate of 94%. One tumor grew 41 months after the treatment.

The incidence of hearing complications was 74%, with 7 patients having a reduced level of hearing and another 7 becoming deaf. Because no audiograms were performed, the hearing data are subjective. Acute complications were noted in 2 patients and involved nausea and vomiting soon after the procedure. More serious complications included new or worsening facial numbness in 5 patients, 4 of whom experienced persistent facial numbness. Transient ataxia was noted in 3 patients (10%). New or worsening facial nerve deficit was observed in a total of 8 patients (28%) and was permanent in 7. In 1 patient, surgery to correct difficulty in closing the eye was performed. The 5-year actuarial incidence rates of trigeminal and facial neuropathy were calculated at 15% and 32%, respectively. Hydrocephalus was a problem in 2 patients, both of whom had shunts inserted. Brain stem edema on T2 images was noted on 1 patient's MRI scan and was associated with balance problems.

Statistical analysis of the results of this study did not show any association between history of previous surgery, age, sex, history of neuropathy, NF2, maximal tumor diameter, or tumor volume and the risks for long-term complications [16].

Foote and colleagues [17] described 149 patients treated with RS and analyzed the risk factors associated with this procedure. In this large study population of patients with AN, 8 patients (5.4%) had bilateral tumors associated with NF2 and 42 patients (28%) had undergone previous resection of their tumors. The mean tumor volume was 4.8 mL (range 0.3–22 mL). The number of isocenters used ranged from 1 to 11, increasing over the duration of the study, with 2 being the average. The doses of radiation administered ranged from 10 to 22.5 Gy, including only doses at increments of 2.5. A mean dose of 14 Gy was usually given to the 70% or 80% isodose line.

The results reported were based on the radiologic follow-up of 6 to 94 months (median 34 months). Control of tumor growth occurred in 93%, with 78 tumors decreasing in size and 45 tumors remaining stable. Ten lesions enlarged, giving the overall control rate at 5 years of 87% (95% CI, 76–98). Six of the patients participating in the study had to undergo surgery to excise the tumor.

In terms of complications, the risks of facial and trigeminal neuropathies 2 years after treatment were 11.8% (95% CI, 6.2–17.1) and 9.5% (95% CI, 4.5–14.3), respectively. Most of the patients who suffered these complications had transient symptoms.

The incidence of facial and trigeminal neuropathies decreased as the study continued. Of the 108 patients who were treated after January 1994, 5 patients suffered facial and 2 suffered trigeminal neuropathies. The actuarial 2-year incidence of facial neuropathy for the first 41 patients was 29%, whereas for the last 108 patients the incidence was reduced to 5%. Likewise, the risk of trigeminal neuropathy decreased from 29% to 2%. The authors suggested that this rate was representative of imaging and radiation dose planning and dose selection improvement.

Certain risk factors were shown to increase the incidence of complications. Radiation doses to the brainstem of >17.5 Gy increased the risk of facial neuropathy by 45-fold when compared with the doses <17.5 Gy. When comparing the 2-year incidence of any cranial neuropathy at doses of ≤ 12.5 Gy to doses above this value, the difference was marked at 2% and 24%, respectively. The authors also noted that having surgery before receiving radiation increased a patient's risk of developing delayed cranial neuropathy by fivefold [17].

In another study on LINAC single fraction RS for AN, Spiegelmann and colleagues [18] described 48 patients, 44 of whom were followed for 12 to 60 months (mean 32 months). Seven patients underwent prior surgery for ANs. The patients presented with maximum tumor diameters ranging from 10 to 31 mm (mean diameter 20 mm). In 40 cases the tumors were irradiated at one isocenter, 3 cases used two isocenters, and 1 case used three isocenters. Tumor margins were irradiated with an average of 14.55 Gy (range 11–20 Gy). Radiation doses ranging from 15 to 20 Gy were used for the first 2 years of the study. Later, tumors considered small and measuring <16 mm in diameter received a maximum dose of 14 Gy, whereas tumors that exceeded this size were treated with a minimum of 11 Gy at 68% to 90% isodose. This treatment was given over 20 to 45 minutes.

Based on the MRI scans obtained every 6 months earlier in the study and yearly during the last 2 years of the study, 98% of tumors were controlled. During the first year of follow-up, 11 tumors increased in size, and enlargement was combined with facial neuropathy in 8 patients. All tumors returned to smaller size at a later time. In 33 patients, tumor reduction was noted. The magnitude of shrinkage ranged from 15% to 90% and occurred at least 12 months after RS was administered. Most shrinkage occurred between 24 and 36 months. Ten tumors (23%) remained unchanged in terms of size, and 1 tumor enlarged at 48 months.

Serviceable hearing, defined as speech discrimination score of 70% or higher, was preserved in 71%. One improvement of speech discrimination score from 50% to 80% 24 months after therapy was noted.

Complications of hydrocephalus did not occur in this study. Eighteen percent of patients developed a new trigeminal neuropathy that was concomitant with facial neuropathy in all cases. In total, nine cases (24%) of facial neuropathy were reported, all of which occurred within 1 year of follow-up and experienced improvement over time. Facial weakness persisted in 3 patients. The rate of facial neuropathy, defined with the House-Brackmann scale, was 8%. The authors also noted that this rate depended on the dose of radiation administered: 5.5% of patients who received the dose of 14 Gy developed facial neuropathy, whereas patients treated with 15 to 20 Gy had a 42% incidence of neuropathy. Tumor size also had an effect. The risk of facial neuropathy was 9.5% and 54% in patients with small (0.8–3.7 mL) and large (4–11 mL) tumors, respectively. Conversely, 2 patients had improved facial nerve function after RS [18].

In the 2003 publication by Meijer and colleagues [19], single fraction RS and fractionated radiotherapy outcomes in treatment of ANs were compared. The two groups were not different with respect to tumor size (mean 2.5 cm); however, patients who received single treatment tended to be older (mean age 63 years) compared with a mean age of 43 years for the fractionated treatment group. This section describes the single fraction RS experience. Forty-nine patients were selected for the treatment with the single fraction of radiation based on their lack of teeth. The first 7 patients received a regimen of 10 Gy at 80% isodose line; the other 42 patients were treated with 12.5 Gy at 80%, with all treatments using a monoisocenter. The difference in outcomes between the patients receiving two different doses of radiation was not statistically significant. The mean follow-up for these patients was 30 months of yearly MRI scans, with tumor control defined as no increase of the diameter by 2 mm or more. The actuarial 5-year tumor control rate for RS patients was 100%.

Hearing was preserved in 75% of patients, with the data based on subjective patient reporting of perceived hearing level. The 5-year rate of facial nerve preservation was 93%, with 45 patients retaining normal function, whereas the preservation probability for the trigeminal nerve was 92%.

One patient who had undergone RS treatment developed hydrocephalus and was required to undergo an operation [19].

Two groups of patients, one treated with FSRT and the other with RS, were reported by Chung and colleagues [20] in an article entitled "Audiologic and Treatment Outcomes After LINAC-based Stereotactic Irradiation for AN." The two groups were analyzed independently, and outcomes of RS are described herein. RS was administered to 45 patients. In that group, 10 patients had prior surgery and 1 had ventriculoperitoneal (VP) shunt placed for a pre-existing hydrocephalus. The tumor diameter ranged from 4 to 34 mm (median 20 mm). All tumors were treated with a monoisocenter, with the exception of three tumors, which were treated with two isocenters. Forty-four of 45 patients received a dose of 12 Gy at 50% (3 patients) and 80% (41 patients) isodose line. One patient was treated with 15 Gy to the 80% isodose line, which

produced a tumor control rate of 100% during the follow-up period of 8 to 61 months (median 27 months).

Among the complications reported, 2 patients (4.4%) developed hydrocephalus for which VP shunts were placed. One 92-year-old patient died from this complication. Permanent mild facial numbness developed in 3 patients (7.5%), and 2 patients (4.4%) had permanent House-Brackmann grade 2 facial paresis. One patient developed an infection at the pin site of the stereotactic headring. The pons (1 patient) and the cerebellum (1 patient) became edematous because of a vasogenic process, which was mild and responded to the administration of steroids. Tumor swelling, with nonenhancing necrotic core and concurrent swelling of the tumor, caused the compression of the brainstem in 1 patient and resulted in ataxia, ipsilateral numbness at the maxillary distribution, and diplopia [20].

A 2005 study by Okunaga and colleagues [21] reported on 46 patients with unilateral ANs who were treated with LINAC RS. Of these patients, 12 (26.1%) had previously undergone resection. The tumor volume in this patient population ranged from 0.4 to 7.01 mL (median 2.29 mL) and they were targeted with one to four isocenters (median of two). Tumor margins were targeted with the median radiation dose of 14 Gy (range 10–16 Gy), and median maximal dose was 23.2 Gy (range 17–36.1 Gy). Radiation to the brainstem was limited to 10 Gy. During the 12 to 120 months of follow-up (median 56.5) with MRI studies performed every 3 to 4 months, eight tumors (19%) showed enlargement. This result was persistent and continuous in 3 patients (2 of whom had to repeat RS at 29 and 36 months), whereas in 5 patients, initial enlargement reached a plateau. No change was noted in two lesions (4.8%), whereas transient enlargement followed by shrinkage occurred in 19 patients (45.2%). Another 13 lesions (31%) shrunk directly with the minimum reduction ratio of 0.05-fold. Two tumors regrew approximately 5 years after RS.

Tumor control was achieved in 31 (73.8%) of 42 patients followed for more than 1 year, 31 (81.6%) of 38 patients followed for more than 2 years, and 18 (100%) of 18 patients followed for more than 5 years. In 11 patients, tumor size decreased more than 7 years after RS; in 5 patients tumor size decreased more than 9 years after RS. Central enhancement was lost in 37 (88.1%) of 42 tumors monitored longer than 1 year.

With respect to hearing preservation, useful hearing was maintained at Gardner-Robertson classes I or II in 66.7% of patients.

Among the procedural complications reported, 4 (7.5%) of 53 patients had hydrocephalus noted on initial visit and were treated with VP shunts and RS. Nine (21.4%) of the 42 patients monitored for longer than 1 year had ventricular enlargement. Of these patients, 3 (7.1%) required placement of a VP shunts and 4 (9.5%) improved spontaneously within 1 year of the finding. The study noted that 4.8% of patients developed new facial palsy, and new trigeminal neuropathy developed in 1 patient (2.4%) [21].

The most recently published data on the use of LINAC for treatment of ANs was found in the 2006 update of the RS experience at the Royal Adelaide Hospital. Roos and colleagues [22] reported on 65 patients with AN who underwent RS, 61 of whom had sporadic and unilateral tumors and 4 of whom had tumors associated with NF2. Six patients had prior surgeries for the tumors, which have since recurred. The median tumor diameter was 22 mm (range 11–40 mm). Radiation was given in a single dose of 12 to 14 Gy to one or two isocenters. The patients were followed with annual MRI scans and audiometry for the first 2 to 3 years and once every 2 years afterwards. The median follow-up for this study group was 48 months (range 12–134 months).

A transient enlargement and central necrosis was noted in tumors of 9 patients (16%). The median increase in size was 4 mm (range 2–5 mm) and occurred from 4 to 25 months after the treatment (median 12 months), typically persisting for 1 to 2 years. Tumor control, which was defined as a reduction or stabilization of tumor size, was achieved in 59 of the 62 patients (95%). Seventeen tumors were stable over 12 to 85 months, and 42 shrank by 2 to 12 mm (median 4 mm) over 13 to 134 months. Of the patients treated with primary RS, 98.5% did not require surgical resection of the disease, and only 1 patient required an operation.

Loss of objectively useful hearing was noted in 18 of the 34 patients with assessable, useful pretreatment levels. The loss occurred by 8 to 77 months (median 24 months). Sixteen patients (47%) maintained a useful level of hearing over 20 to 108 months (median 60 months) of follow-up. Other complications of the procedure included partial trigeminal neuropathies in 7 patients, 2 of whom also developed facial neuropathies. Of these complications, 4 were new findings, whereas

the other 3 patients had pre-existing numbness. Three cases of hydrocephalus necessitating a placement of VP shunts were reported [22].

Fractionated stereotactic radiotherapy

In a publication by Kalapurakal and colleagues [23], 19 patients with ANs were treated with FSRT. This patient group was selected for having large tumors with a mean pons-petrous diameter of 28 mm (range 15–35 mm) and a mean mid-porous transverse diameter of 35 mm (range 23–49 mm). These tumors were treated with either 36 Gy (first 6 patients) or 30 Gy (the remaining 13 patients) given in six fractions. The total dose was reduced because of ataxia experienced during the high-dose treatment by 2 patients.

The patients were followed with CT or MRI scans or both for a median of 54 months (range 34–65 months). The treatment resulted in tumor regression in 10 patients and stabilization of tumor size in the other 9. Of the 9 patients who had hearing before the procedure, 1 was noted to have an improvement and hearing was preserved at a pretreatment level in the other 8. None of the patients in this series experienced any facial or trigeminal nerve injury [23].

In 1999, Poen and colleagues [24] reported on 33 patients in whom a total of 34 AN tumors were treated with FSRT. Of these patients, 10 had NF2. Seven patients had previous operations after which the disease recurred. Tumor diameter ranged from 7 to 42 mm (median 20 mm), with 4 tumors being considered small (diameter $\leq$15 mm), 27 were moderate (16–30 mm), and 3 were classified as large ($>$30 mm). In this study, the tumors were most commonly targeted at two isocenters; however, the number of isocenters used ranged from one to four. The total radiation dose administered was 21 Gy, which was split into three fractions of 7 Gy each. Two of the first 3 patients treated received doses of 25.5 Gy and 19.5 Gy, whereas 1 patient in the study received a standard 21 Gy dose in two fractions rather than three because of a calculation error.

The patients were followed at 6-month intervals for the first 2 years and annually thereafter for a median clinical follow-up of 24 months (range 6–48 months). Tumor control was defined as no change in tumor size of 3 mm or more. Shrinkage of tumors was observed in 11 patients (34%), whereas stabilization of tumor size occurred in 20 patients (63%). One patient had an enlargement of an AN after FSRT. This patient's disease was associated with NF2, and although growth was noted at 1.8 years, regression occurred at a later time. At 2 years follow-up, 93% of patients in the study had tumor control.

Hearing was reported according to the Gardner and Robertson system and was considered useful at classes I and II. At 2 years of follow-up the rate of useful hearing preservation was 77% when NF2 patients were included in the analysis. Serviceable hearing (classes I–III) was preserved in 92% of patients with sporadic ANs and 67% of patients diagnosed with NF2 at 2 years.

The trigeminal nerve was affected in 5 patients (16%) after the treatment. In 3 of these patients the dysfunction was new, in 1 patient worsening of a pre-existing trigeminal dysesthesia was noted, and in another patient, severe ipsilateral herpes zoster in the previously affected branch was reported. In 97% of patients, facial nerve function was preserved, with a single case of injury to the nerve (House-Brackmann Grade III) 7 months after FSRT. No other treatment complications were reported [24].

Williams described the FSRT experience in 125 patients with AN, 4 of whom had previous surgical intervention for the disease and 1 of whom had a diagnosis of NF2. The tumor diameter was $<$30 mm in 111 patients and $\geq$30 mm in 14 cases. Radiation dosing was prescribed according to tumor size, with tumors $<$30 mm in diameter receiving 25 Gy in five fractions and larger tumors ($\geq$30 mm) receiving 30 Gy administered in ten fractions. Both treatments were given to the 80% isodose.

Patients were followed with MRI scans every 3 months for the first year, every 6 month after the first year, and yearly thereafter. The median follow-up time was 21 months (range 12–68 months), during which no new growth of the tumors was observed. Although the data for stabilization and shrinkage of the disease were not provided, the author reported that the tumors that did decrease in size did so by 12% and 13% in 25- and 30-Gy treatment groups, respectively.

Of the 56 patients who had audiometric follow-up for a median of a year, Gardner-Robertson classification was maintained in 26 patients, hearing worsening was noted in 20 cases, and improvement occurred in 10 patients. No difference in hearing preservation was noted across the two different radiation doses.

Two patients experienced temporary trigeminal nerve dysfunction, and none had facial nerve complications [25].

In a study by Meijer and colleagues [19], 80 dentate patients—out of a total of 129 patients involved in the study—were given FSRT for ANs. The mean tumor diameter for this group was 2.5 cm (range 0.8–3.3 cm) and was targeted with one isocenter and five doses of either 4 Gy (12 patients) or 5 Gy (68 patients) at 80% isodose line. The total radiation dose was either 20 Gy or 25 Gy. The patients were followed with yearly MRI scans for a period of 12 to 107 months (mean 35 months) and had a 94% 5-year probability of tumor control, defined as no diameter increase of 2 mm or more. In all cases tumor enlargement took place within 3 years of treatment. The actuarial 5-year facial and trigeminal nerve preservation probabilities in the fractionated treatment group ($n = 73$) were 97% and 98%, respectively. The 5-year hearing preservation probability, measured subjectively, was 61%. Radiation damage to the cerebellopontine angle was seen on MRI of 1 patient who developed gait problems 6 months after the treatment [19].

In the next study, Sawamura and colleagues [26] described 106 patients with ANs treated by FSRT. In 5 of these patients the ANs were associated with NF2. The remaining 101 patients had sporadic, unilateral solitary tumors. Twelve patients underwent surgery for their disease. Tumor diameter ranged from 3 to 40 mm (median 15.5 mm). The treatment protocol involved the administration of 40 to 50 Gy (median 48 Gy) in 20 to 25 fractions (median of 23 fractions).

The follow-up in this study was 6 to 128 months (median 45 months). MRI, neurologic, and otologic examinations were performed every 5 months for 5 years and every 12 months thereafter. The result of treatment was a 91.4% (95% CI, 85.2–97.6) 5-year actuarial rate of tumor control. Continuous tumor growth after the FSRT was seen in 3 patients (3%) who had to undergo surgery to remove the ANs.

The 5-year actuarial rate of hearing preservation (Gardner-Robertson classes I and II) was 71.7% (95% CI, 54.5–88.9). The 5-year rate of class preservation of classes I to V was 64.6% (95% CI, 53.3–75.9). These data were obtained via audiologic examinations.

Twelve patients (12%) developed hydrocephalus and required placement of a VP shunt. Among the 12 patients who had tumor resection before FSRT, 1 developed hydrocephalus as a complication of radiation treatment. When risk for hydrocephalus was analyzed with regard to tumor size, the mean size (25.5 mm) of the 11 tumors in patients who developed this complication was significantly larger than in the 86 tumors unrelated to hydrocephalus (18.2 mm). Other complications included transient facial nerve palsy (4%), trigeminal neuropathy (13.9%), and disequilibrium (16.8%). Prior existing tinnitus improved in 11 of 37 patients. Dizziness or vertigo improved in 11 of 20 [26].

In a study by Chung and colleagues [20], the group treated with FSRT contained 27 patients with ANs. Of these patients, 4 had NF2 and 1 had undergone a previous surgical resection of the tumor. They were assessed every 6 months for the first 2 years and annually thereafter with MRI and audiogram studies. The tumor diameter ranged from 7 to 37 mm (median 16 mm), and the lesions were targeted with one isocenter in 26 of 27 cases (1 patient was treated with two isocenters). Twenty-five patients were treated with 45 Gy radiation dose, which was administered in 25 fractions over 5 weeks. In 1 patient the same dose was given in 28 fractions. A 90% isodose line was used in both treatment protocols. One patient received 25 fractions for a total dose of 47.5 Gy to the 50% isodose line.

The patients in the FSRT treatment group were followed for 13 to 59 months (median 26 months), with a reported tumor control rate of 100%. Tumor progression was defined as enlargement on two consecutive MRI scans and did not occur in the study population. Hearing rate preservation was 85% at 1 year after the FSRT and decreased to 57% at 2 years follow-up. Two patients (7%) complained of transient facial numbness, and 1 patient (4%) developed transient facial paresis. One patient (4%) developed hydrocephalus that required placement of a VP shunt [20].

In a study entitled ''Stereotactic Radiotherapy for the Treatment of AN,'' Selch and colleagues [27] described 48 patients who underwent FSRT with LINAC. There was a history of partial resection of the ANs in 6 patients. The patients were followed with MRI and physical examination every 6 months for a median of 36 months (range 6–74 months). Radiologic data for at least 4 years was available for 13 patients, and for 6 patients it was available for at least 5 years. The diameter of the tumors in this study ranged from 0.6 to 4 cm (median 2.2 cm), and a single isocenter was used to target all the lesions. A total radiation dose of 54 Gy was split into in 30 fractions of 1.8 GY and was prescribed to the 90% isodose line. This dose produced tumor shrinkage of 1 to 14 mm (median 2 mm) in 12 patients (27%) at a median

of 6 months (range 6–24 months). MRI scan indicated a loss of central tumor contrast enhancement in 32 patients (67%) at a median of 6 months (range 3–12 months) after the FSRT. In 8 patients contrast enhancement returned. Increase in tumor diameter by 1 to 2 mm was observed in 12 patients (25%) at a median of 6 months (range 3–18 months). Four of these tumors decreased to original size and 2 shrank to a size smaller than the original.

Useful hearing, defined subjectively as inability to use the telephone, was preserved in 39 patients (93%), with a loss occurring in 3 patients. The 5-year actuarial probability of preserving useful level of hearing was reported at 91.4%. Facial nerve dysfunction occurred in 1 patient (2.1%) with no prior history of problems with the nerve. The actuarial 5-year probability of preserving the facial nerve function was 97.2%. One patient reported improvement of pretreatment facial nerve dysfunction. The actuarial 5-year rate of preserving trigeminal nerve function was 96.2%, with new dysfunction being noted in 1 patient (2.2%) in whom the tumor enlarged 1 mm. None of the patients treated with FSRT in this study developed hydrocephalus.

Other sequelae of FSRT treatment included worsening of tinnitus symptoms in 6 patients, 4 of whom had improvement to levels below the pretreatment levels. Pretreatment ataxia improved in 1 patient. Statistical analysis performed by the authors suggested that patient age, tumor size, and tumor volume did not relate to the outcomes after FSRT [27].

Combs and colleagues [28] reported on the use of FSRT in a population of 106 patients with ANs. Prior surgical interventions for the tumors were performed in 14 patients. Tumor diameter of $\leq$1 cm was reported in 13 patients, 1- to 2-cm tumors were found in 48 patients, 2- to 3-cm tumors were found in 30 patients, and 3- to 4-cm tumors were found in 13 patients. Two tumors had a diameter $>$4 cm. The range of target volumes as measured on MRI was 2.7 to 30.7 mL (me 3.9 mL). The total median radiation dose prescribed was 57.6 Gy and was administered in a median of 32 fractions, with 1.8 Gy per fraction.

Follow-up MRI scan was performed 6 weeks after the FSRT, then at 3- to 6-month intervals for the first 2 to 3 years, and annually afterwards. Median follow-up time was 48.5 months (range 3–172 months), and 95.3% of tumors were locally controlled on the last MRI scan. The actuarial control rate was 94.3% at 3 years and 93% at 5 years.

Hearing preservation in patients who presented with useful hearing (Gardner-Robertson classes I and II) before the FSRT ($n = 65$) was 94% at 5 years. New trigeminal neuralgia occurred in 5 patients and proved to be transient in 2 patients. Irreversible damage to the trigeminal nerve occurred in 3 of 87 patients at risk (3.4%). Pretreatment trigeminal dysesthesia resolved in 8 patients after the FSRT treatment. Two (2.3%) of the 88 patients with a normal facial nerve sustained irreversible damage [28].

In another recent study, Chan and colleagues [29] reported on 70 patients with AN treated with FSRT. Eleven of these patients were diagnosed with NF2, and 21% had previous surgeries for AN tumors. The median size of the tumors in this patient population was 2.4 mL (range 0.05–21.1 mL), and the lesions were targeted at one isocenter. A total dose of 54 Gy was administered in 30 fractions of 1.8 Gy each to the 95% isodose line.

The patients were followed for a median of 45.3 months after the treatment. The range of follow-up periods was not provided by the authors. Of the 70 patients, radiologic data were available for 68; data showed decrease in central enhancement at median of 7 months in 46% of all tumors treated with FSRT. Tumor shrinkage was observed in 53 cases, with actuarial 3- and 5-year size decrease in 36% and 62%, respectively. Tumor enlargement was noted in 4 patients occurring 2 years after treatment in all cases. At 3 and 5 years, the actuarial tumor control rates were 100% and 98%, respectively.

Because hearing preservation was assessed using a subjective scale, the data from this study are not used in the overall analysis.Among the complications reported, one patient had a change in facial weakness from House-Brackmann grade 2 to grade 5. The 5-year actuarial probability of facial nerve function preservation was 99%. Eight cases of facial twitching and spasming (defined as facial hyperfunction) were reported. Two cases of transient facial numbness were recorded, with the actuarial 5-year trigeminal nerve preservation rate of 96%. Statistical analysis performed by the authors indicated that previous tumor resections increased the risk of trigeminal neuropathy [29].

Data summary

Tumor control

When comparing microsurgical treatment of AN tumors to radiation therapy, it is important to

Table 1
Tumor control after stereotactic radiosurgery with the linear accelerator

City	Year	N	No. of sessions	Total dose (Gy)	Follow-up (mos)	Tumor size	Result	% Grew	% Stable	% Shrank
Florida	2001	133	1	10–22.5	6–94 (median 34)	0.3–22 mL (mean 4.8 mL)	87% 5-year actuarial control	7	34	59
Adelaide	2006	62	1	12–14	12–134 (median 48)	11 mm–40 mm	95% control = no increase of $\geq$2 mm	4.8	27.4	67.7
Nagasaki	2005	53	1	Margins: 10–16 Max: 17–36.1	12–120 (median 56.5)	0.4–7.01 mL (median 2.29 mL)		19	4.8	76.2 (after transient enlargement in 45.2)
		42	—		12		73.8% control in 42 patients	26.2	—	
		38	—		24		81.6% control in 38 patients	18.4	—	
		18	—		60		100% control in 18 patients	0	—	
Amsterdam	2003	49	1	10 & 12.5	12–107 (mean 30)	8–38 mm (mean 25)	100% 5-year actuarial control	0	100%	
Vancouver	2004	45	1	12	8–61 (median 27)	4–34 mm (median 20 mm)	100% control rate	0	100%	
Tel Hashomer	2001	44	1	11–20	12–60 (mean 32)	10–31 mm (mean 20 mm)	98% control rate	2	23	75 (by 15%–90%)
Cleveland	2000	29	1	8–24	4–110 (median 49)	0.18–28.7 mL (median 2.1 mL)	94% 5-year actuarial control	3	59	38
London	1996	10	1	2.5–20	3–36 (mean 15)	15–35 mm (mean 26 mm)	80% control rate	20 (1 mm)	40	40

Table 2
Tumor control after linear accelerator fractionated stereotactic radiotherapy

City	Year	N	No. of sessions	Total dose (Gy)	Follow-up (mo)	Tumor size	Result	% Grew	% Stable	% Shrank
Baltimore	2002	125	5 10	25 30	12–68 (median 21)	<30 mm: 111 ≥30 mm: 14	100% control (no new growth)	0	100	
Heidelberg	2005	106	32	57.6	3–172 (median 48.5)	0–10 mm: 13 11–20 mm: 48 21–30 mm: 30 31–40 mm: 13 >40 mm: 2	94.3% 3-year actuarial control; 93% 5-year actuarial control	4.7	95.3	
Sapporo	2003	101	20–25	40–50	6–128 (median 45)	3–40 mm (median 15.5 mm)	91.4% 5-year actuarial control	3	97	
Amsterdam	2003	80	5	20 or 25	12–107 (mean 35)	8–33 mm (mean 25 mm)	94% 5-year actuarial control = no increase in the largest diameter >2 mm	N/A	N/A	N/A
Boston	2005	70	30	54	median 45.3	0.05–21.1 mL (median 2.4 mL)	100% 3-year actuarial, 98% 5-year actuarial control rate	6	18	76
Los Angeles	2004	48	30	54	6–74 (median 36)	6–40 mm (median 22 mm)	100% 5-year actuarial control in 6 patients	12.5 (1–2 mm)	60.5	27 (1–14 mm, median 2 mm)
Stanford	1999	33	3	21	24	7–42 mm (median 20 mm)	93% control at 2 years = no increase of >3 mm	3	63	34
Vancouver	2004	27	25	45	13–59 (median 26)	7–37 mm (median 16 mm)	100% control = no enlargement on two consecutive MRIs	0	100	
Philadelphia	1999	19	6	30 or 36	24–65 (median 54)	23–49 mm (mean 35 mm)	100% control	0	47	53

Table 3
Hearing preservation after stereotactic radiosurgery

City	Year	N	No. of sessions	Total dose (Gy)	Follow-up (mo)	Tumor size	Result
Adelaide	2006	34	1	12–14	12–134	11–40	47% useful hearing preservation
Tel Hashomer	2001	13	1	11–20	12–60	10–31 mm (mean 20 mm)	71% hearing preservation rate; serviceable hearing, defined as speech-discrimination score of $\geq$70%
Nagasaki	2005	9	1	17–36.1	12–120	0.4–7.01 mL (median 2.29 mL)	66.7% patients maintained useful hearing (GR I and II)
London	1996	7	1	12.5–20	3–36	15–35 mm (mean 26 mm)	Stabilized or improved in 5, and deteriorated to complete deafness in 2

Table 4
Hearing preservation after fractionated stereotactic radiotherapy

City	Year	N	No. of sessions	Total dose (Gy)	Follow-up (mo)	Tumor size	Result
Sapporo	2003	106	20–25	40–50	6–128 (median 45)	3–40 mm (median 15.5 mm)	71.7% GR I and II preservation
Heidelberg	2005	65	32	57.6	3–172 (median 48.5)	0–10 mm: 13 11–20 mm: 48 21–30 mm: 30 31–40 mm: 13 >40 mm: 2	94% 5-year actuarial rate of hearing preservation (GR I and II)
Baltimore	2002	56	5 10	25 30	12–68 (median 21)	<30 mm: 111 $\geq$30 mm: 14	46.4% maintained GR class, hearing worsened in 35.7%, and improved in 17.8%
Stanford	1999	33	3	21	24	7–42 mm (median 20 mm)	92% serviceable hearing (GR I-III) preservation rate at 2 years for patients with sporadic AN;67% for patients who have NF2
Vancouver	2004	22	25	45	12 24	7–37 mm (median 16 mm)	85% (GR I and II) 57%
Philadelphia	1999	9	6	30 or 36	24–65 (median 54)	23–49 mm (mean 35)	100% preservation; one case of improvement

Table 5
Facial nerve (VII) preservation after stereotactic radiosurgery

City	Year	N	No. of sessions	Total dose (Gy)	Follow-up (mo)	Tumor size	Result
Florida	2001	41 108	1	10–22.5	6–94	0.3–22 mL (mean 4.8 mL)	29% 2-year actuarial incidence of facial neuropathy 5% 2-year actuarial incidence of facial neuropathy; 11.8% overall incidence at 2 years (mostly transient)
Adelaide	2006	57	1	12–14	12–134	11–40	3.5% had mild neuropathy
Amsterdam	2003	49	1	10 and 12.5	12–107 (mean 30)	8–38 mm (mean 25)	93% 5-year rate of facial nerve preservation
Vancouver	2004	45	1	12	8–61	4–34 mm (median 20 mm)	4.4% developed permanent House-Brackmann Grade 2 facial paresis
Nagasaki	2005	42	1	17–36.1	12–120	0.4–7.01 mL (median 2.29 mL)	4.8% had new facial palsy
Tel Hashomer	2001	37	1	11–20 14 15–20	12–60	10–31 mm (mean 20 mm) 0.8–3.7 4–11	24% of transient new facial neuropathy; 8% had persistent facial weakness; 2 patients had facial nerve improvement; 5.5% developed facial neuropathy 42% developed facial neuropathy 9.5% facial neuropathy 54% facial neuropathy
Cleveland	2000	29	1	8–24	4–110	0.18–28.7 mL (median 2.1 mL)	28% had deficit, permanent in 24%; 32% 5-year actuarial rates of facial neuropathy
London	1996	11	1	12.5–20	3–36	15–35 mm (mean 26 mm)	0% new facial nerve palsy; 2 of 4 cases of old facial palsy were unchanged; 1 of 4 improved (at 14 months); 1 of 4 deteriorated to complete facial palsy

Table 6
Facial nerve (VII) preservation after fractionated stereotactic radiotherapy

City	Year	N	No. of sessions	Total dose (Gy)	Follow-up (mo)	Tumor size	Result
Baltimore	2002	125	5 10	25 30	12–68 (median 21)	$<$30 mm: 111 $\geq$30 mm: 14	0% damage
Sapporo	2003	106	20–25	40–50	6–128 (median 45)	3–40 mm (median 15.5 mm)	4% developed transient facial palsy
Heidelberg	2005	88	32	57.6	3–172 (median 48.5)	0–10 mm: 13 11–20 mm: 48 21–30 mm: 30 31–40 mm: 13 $>$40 mm: 2	2.3% sustained irreversible damage
Amsterdam	2003	73	5	20 or 25	12–107 (mean 35)	8–33 mm (mean 25 mm)	97% 5-year actuarial nerve preservation
Boston	2005	70	30	54	median 45.3	0.05–21.1 mL (median 2.4 mL)	1.5% facial weakness (House-Brackman II progressing to V); 99% 5-year actuarial facial nerve preservation
Los Angeles	2004	48	30	54	6–74	6–40 mm (median 22 mm)	2.1% had new facial palsy (House-Brackmann V); improvement from V to IV in one patient
Vancouver	2004	45	1	12	8–61	4–34 mm (median 20 mm)	4% transient facial paresis
Stanford	1999	33	3	21	6–48	7–42 mm (median 20 mm)	3% facial nerve injury (House-Brackmann Grade III)
Philadelphia	1999	16	6	30 or 36	24–65 (median 54)	23–49 mm (mean 35 mm)	0% damage

Table 7
Trigeminal nerve (V) preservation after stereotactic radiosurgery

City	Year	N	No. of sessions	Total dose (Gy)	Follow-up (mo)	Tumor size	Result
Florida	2001	41 108	1	10–22.5	6–94	0.3–22 mL (mean 4.8 mL)	29% 2-year actuarial risk of trigeminal neuropathy 2% 2-year actuarial risk of trigeminal neuropathy; 9.5% overall incidence at 2 years (mostly transient)
Adelaide	2006	62	1	12–14	12–134	11–40 mm	11.3% mild developed neuropathy
Nagasaki	2005	53	1	Margins: 10–16 Max: 17–36.1	12–120 (med 56.5)	0.4–7.01 mL (median 2.29 mL)	2.4% developed trigeminal neuropathy
Amsterdam	2003	49	1	10 and 12.5	12–107 (mean 30)	8–38 mm (mean 25)	92% 5-year nerve preservation
Vancouver	2004	45	1	12	8–61	4–34 mm (median 20 mm)	7.5% developed permanent mild facial numbness
Tel Hashomer	2001	44	1	11–20	12–60	10–31 mm (mean 20 mm)	18% developed neuropathy
Cleveland	2000	29	1	8–24	4–110	0.18–28.7 mL (median 2.1 mL)	17.2% new facial numbness, permanent in 13.8%; 15% 5-year actuarial rate of trigeminal neuropathy

keep in mind the different goals of these therapeutic modalities. The goal of microsurgery is to resect the tumor and leave little or no disease behind. Radiation therapy, however, aims to arrest the growth of the tumor or cause it to become smaller. In all the studies reviewed, successful control was defined as no enlargement of the tumor on serial MRI scans. Success of surgical treatment cannot necessarily be equated with the success of RS or FSRT.

As described in the section on natural history of AN in this article, AN tumors are slow-growing tumors that may be stable for periods of time. This nonlinear pattern of growth necessitates that patients be followed for long periods of time after radiation therapy to assess the true efficacy of this procedure. The minimum follow-up for patients treated with RS reported in the analyzed series was 3 months, with several other studies reporting minimum follow-up time less than 1 year. Patients treated with FSRT had a minimum follow-up time of 6 months (in two studies). These follow-up periods are unacceptable, because a lack of tumor growth during this period is not equivalent to the ultimate success of therapy. Although most studies for RS and FSRT had a median follow-up of more than 2 years and a maximum follow-up of 5 years and more, only one study [21] separated patients based on the length of follow-up, therefore strengthening the data. It would be more valuable to know the results of treatment at longer follow-up periods rather than the control rate across patients followed for various lengths of time. Lack of such data weakens any conclusions that can be made on the subject.

Reported tumor control after stereotactic RS ranged from 73.8% to 100%, whereas control in patients treated with FSRT was 91.4% to 100%. Stabilization of tumor growth in patients treated with RS ranged from 4.8% to 59%. Shrinkage was observed in 38% to 76.2% of tumors irradiated with a single dose, whereas 0% to 26.2% increased in size (Table 1). FSRT patients experienced stabilization in 18% to 63% of cases, shrinkage in 34% to 76% of cases, and tumor growth in 0% to 12.5% of cases (Table 2). Given the limitations stated previously, this finding suggests that fractionation of radiation treatment leads to better control of AN. Tumors targeted with a single dose of radiation decreased in size more often than tumors that underwent FSRT; however, they also grew more often.

Tremendous variability in the total amount of radiation (total Gy) administered is seen across

Table 8
Trigeminal nerve (V) preservation after fractionated stereotactic radiotherapy

City	Year	N	No. of sessions	Total dose (Gy)	Follow-up (mo)	Tumor size	Result
Baltimore	2002	125	5 10	25 30	12–68 (median 21)	<30 mm: 111 ≥30 mm: 14	1.6% temporary dysfunction
Sapporo	2003	106	20–25	40–50	6–128 (median 45)	3–40 mm (median 15.5 mm)	13.9% neuropathy
Heidelberg	2005	87	32	57.6	3–172 (median 48.5)	0–10 mm: 13 11–20 mm: 48 21–30 mm: 30 31–40 mm: 13 >40 mm: 2	5.7% total, 3.4% irreversible
Amsterdam	2003	73	5	20 or 25	12–107 (mean 35)	8–33 mm (mean 25 mm)	98% 5-year actuarial preservation
Boston	2005	70	30	54	median 45.3	0.05–21.1 mL (median 2.4 mL)	3% developed transient facial numbness; 96% 5-year actuarial trigeminal nerve preservation
Los Angeles	2004	48	30	54	6–74	6–40 mm (median 22 mm)	2.2% experienced new dysfunction
Stanford	1999	33	3	21	6–48	7–42 mm (median 20 mm)	16% nerve injury; new dysfunction in 9.7%; worsening of old symptoms in 6.3%
Vancouver	2004	27	25	45	13–59 (median 26)	7–37 mm (median 16 mm)	7% transient facial numbness
Philadelphia	1999	15	6	30 or 36	24–65 (median 54)	23–49 mm (mean 35)	0% damage

Table 9
Incidence of hydrocephalus after stereotactic radiosurgery

City	Year	N	No. of sessions	Total dose (Gy)	Follow-up (mo)	Tumor size	Result
Adelaide	2006	65	1	12–14	12–134	11–40	4.6%
Nagasaki	2005	53	1	17–36.1	12–120	0.4–7.01 mL (median 2.29 mL)	7.5%
Amsterdam	2003	49	1	10 and 12.5	12–107 (mean 30)	8–38 mm (mean 25)	2%
Tel Hashomer	2001	48	1	11–20	12–60	10–31 mm (mean 20 mm)	0%
Vancouver	2004	45	1	12	8–61	4–34 mm (median 20 mm)	4.4%
Cleveland	2000	29	1	8–24	4–110	0.18–28.7 mL (median 2.1 mL)	6.9%

the analyzed studies. In general, an increase in the total radiation dose provides better tumor control while elevating the probability of complications associated with treatment.

Hearing preservation

The location of AN tumors jeopardizes hearing. Many patients present with varying degrees of hearing loss, which unfortunately is not restored. In general, the best outcome after resection or irradiation of an AN is to maintain the pretreatment hearing level. In analyzing the data reported in the literature, we looked for the rates of hearing preservation measured on an objective scale. This rate involved a pre- and postradiation auditory examination. Any subjectively defined data were not included in our analysis. The probability rate of useful hearing preservation in patients treated with RS ranged from 47% to 71%, with useful hearing defined as Gardner-Robertson classes I and II (Table 3). This rate is based on a limited population size, with 56 cases reported over three studies [18,21,22]. Hearing preservation rate after FSRT was much more favorable, ranging from 57% to 100% across six studies (Table 4). One study reported an improvement of hearing in 17.8% (ten cases) [25].

Other complications

The complications of radiation therapy for AN that were cited most often in the studies evaluated in this article included facial and trigeminal nerve neuropathy and hydrocephalus. Facial nerve dysfunction was reported in 0% to 54% of patients whose tumors were treated with RS (Table 5). This large range is partly caused by the heterogeneity of treatment protocols and tumor sizes. When facial nerve complications were stratified according to radiation dose and tumor size, large tumors exposed to the most radiation were most likely to be associated with neuropathy [18]. Fractionated radiation treatment was associated with a much smaller incidence of facial nerve injury, ranging from 0% to 4% (Table 6).

A similar trend was found with regard to complications involving the trigeminal nerve. Trigeminal nerve dysfunction was seen in 2.4% to

Table 10
Incidence of hydrocephalus after fractionated stereotactic radiotherapy

City	Year	N	No. of sessions	Total dose (Gy)	Follow-up (mo)	Tumor size	Result
Sapporo	2003	101	20–25	40–50	6–128	3–40 mm (median 15.5 mm)	12%
Amsterdam	2003	80	5	20 or 25	12–107 (mean 35)	8–33 mm (mean 25 mm)	0%
Los Angeles	2004	48	30	54	6–74	6–40 mm (median 22 mm)	0%
Stanford	1999	33	3	21	6–48	7–42 mm (median 20 mm)	0%
Vancouver	2004	27	25	45	13–59	7–37 mm (median 16 mm)	4%

29% of patients treated with RS (Table 7) and in 0% to 16% of FSRT patients (Table 8). Complication of hydrocephalus was seen in 0% to 7.5% of RS patients (Table 9) and 0% to 12% of FSRT patients (Table 10). In most cases, an operation for VP shunt placement was required.

Summary

The review of literature on the use of LINAC stereotactic RS and FSRT in the treatment of AN leads us to conclude that these modalities are essentially safe. Although the incidence of therapies resulting in the most common complications—including facial and trigeminal nerve neuropathy and hydrocephalus—is low, hearing preservation after irradiation of the tumor is poor. The efficacy of these modalities to control tumor growth is still in question. Larger study populations with longer follow-up time are needed. In general, FSRT is better than RS in terms of tumor control and reduced complication rates.

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Stereotactic Radiation Techniques in the Treatment of Acoustic Schwannomas

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Medical decision-making is based on benefit-to-cost analysis. Optimally, treatment obtains a high degree of benefit while minimizing the physical, social, and financial costs. The goals of the treatment of acoustic schwannomas are prohibiting tumor growth and alleviation of symptoms caused by damage to local structures. These symptoms—tinnitus, ataxia, and hearing loss—secondary to eighth nerve dysfunction, as well as symptoms arising from damage to adjacent structures such as the facial nerve, trigeminal nerve, or pons, can be caused by tumor growth or treatment. Determination of optimal therapy must also take into account an understanding of the natural history of the disease, because acoustic schwannomas are slow-growing benign tumors that when left untreated, usually enlarge over time and cause problems.

Historical perspective

Archeological findings from 2500 BC provide evidence that acoustic nerve tumors have been present since antiquity. These tumors, called acoustic neuromas or, more properly, schwannomas, were diagnosed based on a recognized progression from deafness to death as early as 1810. The first documented successful removal of an acoustic schwannoma was performed by Thomas Annandale in 1896 in Edinburgh, Scotland. The patient, who was pregnant at the time of the operation, was able to go home and give birth to a healthy child [1].

During the twentieth century, different surgical approaches (suboccipital, translabrynthine, and middle fos) were developed for the resection of eighth-nerve tumors. Each sought to diminish the inherent anatomic issues associated with total removal. In addition to the obvious hearing loss associated with eighth nerve resection, there was also the risk of seventh nerve damage, cerebrospinal fluid leak, and hemorrhage. Because of difficulties with early diagnosis before modern imaging tests, the pattern of slow intermittent growth, and the morbidity of resection, observation instead of immediate intervention became a frequent consideration.

In 1951, Dr. Lars Leksell, a neurosurgeon, and Borje Larsonn, a physicist, using the Uppsala University cyclotron, ion Uppsala, Sweden, developed an approach to treating small brain lesions with multiple proton beams, using a fixed rigid Cartesian coordinate system to locate the target. Their idea was to develop a noninvasive therapy system to deliver ablative doses of radiation to a geometrically defined discrete volume of tissue, using multiple small beams of radiation. This concept evolved into the Gamma Knife (Elekta, Stockholm, Sweden) stereotactic radiosurgery. The Gamma Knife unit consists of 201 cobalt radiation sources placed in a helmet, within which lie shuttered channels directed toward the center of the helmet. The target lesion is placed at that center position of the helmet by using a stereotactic frame affixed to the patient's skull. Different dose patterns with sharp dose gradients can be obtained using multiple isocenters designed to match the shape of the tumor (Appendix).

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Dr. Leksell and his team working at the Karolinska Institute in Stockholm, Sweden were the first to treat acoustic neuromas with stereotactic radiosurgery (SRS). Their initial series (1969–1974) reported initial tumor control in eight of the nine cases; however, hearing loss was reported in the majority of patients treated [2]. In these early studies, high doses (25–35 Gray) were employed; targeting was crude compared with imaging studies available today. The first Gamma Knife unit in the United States was installed at the University of Pittsburgh in Pittsburgh, Pennsylvania under the direction of Dr. Dade Lunsford. Many of the early results involving radiosurgery for acoustic neuromas in North America were published by the University of Pittsburgh group.

An alternate stereotactic delivery platform was developed using linear accelerators (linacs). Linacs were already available in most modern radiation centers. Instead of multiple sources aimed at a central designated target, the linac systems rotated the treatment beam of the unit around the target in a varying number of rotational arcs. Betti and Colombo initially developed this technique in South America. Because the linac systems involve moving sources of radiation, special devices are needed to limit positional variation in beam delivery.

Both of these techniques require some method to limit patient and target movement. Fixation of target position in a geometric coordinate system can be achieved with metal frames screwed into the skull, which can obtain submillimetric accuracy, or with thermoplastic molded mask systems, which allow for more fractionated schedules, whereby treatment can be administered over several days without placing fixation screws into a patient's skull.

Treatment goals

The goal of treatment is to eradicate the effects of a tumor with the fewest side effects. In surgical series, the means of achieving this goal is the removal of the offending tumor. In most reports of radiation efficacy, the objective is to achieve tumor reduction; however, in slow-growing tumors such as acoustic schwannomas, the achievable goal in studies with shorter duration of follow-up is the prevention of both tumor growth and symptom progression. Tumor shrinkage assessed by radiographic size may take years before a complete response can be evaluated. Additionally, relapses might be late occurrences.

Increased tumor volumes and irregularly shaped tumor volumes are associated with increased integral dose (energy deposition) within the tumor for Gamma Knife and cone-based linac SRS systems. Gamma Knife doses are prescribed to the tumor periphery. These peripheral doses are frequently 50% of the peak dose delivered within the central volume treated. This dose profile with areas of very high dose within the target volume (hot spots) is less a problem with single isocenter treatment plans using a multileaf collimator, where dose is more uniform. Shaped-beam peripheral doses are usually prescribed to the 90% to 95% isodose line. Central hot spots are 10% to 20% higher than the dose prescribed to the tumor periphery; however, the multileaf collimated treatment plans do not produce dose patterns that are as conformal or tightly fitting as the multiple isocenter systems or Gamma Knife systems. Single isocenter treatment techniques typically treat a small rim of normal tissue surrounding an irregularly shaped tumor.

Stereotactic radiosurgery

The initial studies from Pittsburgh demonstrated the ability of stereotactic radiosurgery to achieve control rates of 95% or more with doses of 16 to 20 Gray; however, these doses were associated with an increased risk of trigeminal nerve injury, seventh nerve injury, and decreased hearing. Before 1992, when using these higher doses, reports of treatment results in patients who were in the Pittsburgh patient cohort receiving stereotactic radiation demonstrate that 34% developed facial nerve weakness, although one half of these were transient. Thirty-two percent developed new trigeminal neuropathy. Useful hearing could be preserved in 38% at 1 year [3]. Foote and colleagues [4] reported similar side-effect profiles from the University of Florida in Gainesville, Florida when higher doses were used, with fewer side effects when doses lower than 13 Gray were prescribed to the tumor margin.

Reduced toxicity to the trigeminal and facial nerves was accomplished by decreasing the marginal SRS dose given to the tumor. Rates of morbidity decreased from 29% to 5% for facial neuropathy, and similarly, to 2% or less for trigeminal neuropathy. Tumor control rates did not appear to be compromised until the marginal dose was decreased to 10 Gray or less [4]. Doses to the tumor margin of 12 to 13 Gray were

associated with 5-year tumor control rates of 92% to 98% [4–6]. Maximum doses were 20 to 26 Gray. Useful hearing was noted in 56% to 78% after treatment.

Optimal dose prescription balances tumor kill and normal tissue survival. Radiation oncologists have long recognized the limited tolerance of cranial nerves to radiation. The best-delineated toxicity data for stereotactic radiation relate to the optic nerves and chiasm. Limits of 8 Gray in one fraction and 50 Gray using standard fractionation (1.8 to 2 Gray per day), serve as dose restraints for this nerve. Radiation toxicity must also take into consideration the length of nerve radiated as well as the dose absorbed. The idea of volume at risk may be a function of vascular damage or of repopulation limits. The dose given to the tumor margin and more plausibly, the maximal dose within the volume treated reflect the degree of tissue damage.

A group from Seoul, Korea related hearing loss in relationship to the cochlear dose received during radiosurgery [7]. The mean dose to the cochlea in those maintaining useful hearing was 6.9 Gray. When the mean dose was greater than 11 Gray, hearing declined. Massager and colleagues [8] also found cochlear dose to be lower in patients retaining useful hearing. They found a significant relationship regarding intracanalicular tumor volume (<100 mm^3 versus 100 mm^3) as well as intracanalicular integrated dose as determinants of hearing loss. Their paper postulates that ''hearing worsening after the gamma knife radiosurgery (GKR) procedure can be attributed to cochlear injury inside the internal acoustic canal caused by the enlargement of the intracanalicular part of the vestibular schwannomas during the inflammatory edema phase after radiosurgery through an increase of the intracanalicular pressure.''

Fractionated stereotactic radiation therapy

Whereas some centers were investigating lowering the marginal peripheral dose with single dose treatment regimens, others investigated using fractionated radiation therapy. The theory behind fractionated or multiple treatment radiation therapy is that multiple smaller doses of radiation can achieve a similar tumor effect (cell death) while allowing normal tissue time to repair between each dose, and thereby limit toxicity. This approach involves a greater total dose of radiation than a single-dose treatment to overcome whatever repairs the tumor has been able to achieve. Standard fractionation involves doses of 1.8 to 2.0 Gray per day given daily for 25 to 30 treatments. A total dose to tumors as measured at their periphery is 45 to 60 Gray. Central portions of the tumor volume receive 5% to 10% more than the periphery. This regimen has been used for decades in the treatment of malignant tumors to maximize soft tissue repair from radiation damage.

Several institutions have reported their results for fractionated radiotherapy using radiosurgical techniques in the treatment of acoustic schwannomas, with excellent tumor control rates and with minimal toxicity. Relocatable, molded face masks have been used for skull immobilization. These treatments have been delivered using linac-based therapy, with tumor doses prescribed to a peripheral dose encompassing the tumor, plus a small margin (2 mm) to account for the small amount of movement that occurs between daily fractions and any movement within the face mask during treatment.

Chan and colleagues [9], from Massachusetts General Hospital-Harvard in Boston, Massachusetts, report a 5-year tumor control rate of 98% using a regimen of 54 Gray given in 1.8 Gray fractions as prescribed to the 95% isodose line. They note a distinct relationship between tumor sizes and tumor control. Surgical resection was required for three patients with larger tumors and increasing symptoms at a median of 37 months. At 5-year follow-up, freedom from any surgical intervention was 97% for tumors smaller than 8 cm^3, and 47% for tumors greater than 8 mm^3.

Selch and colleagues [10] from the University of California, Los Angeles (UCLA), using a similar radiation regimen—54 Gray in 30 treatments as prescribed to the 90% isodose line—reported a local control rate of 100% at 36-month median follow-up in 50 patients. Useful hearing was preserved in 93%, with a median follow-up of 36 months. Facial numbness occurred in 1 patient (2.2%) and 1 patient experienced the new onset of facial palsy. Twelve of their patients experienced tumor growth. In 6 of the 12, the growth was transient, and was felt to represent a treatment effect. The transient type of enlargement shows subsequent shrinkage within 2 years, and is frequently associated with loss of central enhancement on MRI. The phenomenon of transient enlargement has also been a common finding in other institutions for both SRS and fractionated stereotactic radiation therapy (FSRT) [11,12].

A Heidelberg, Germany group treated 106 patients who had acoustic neuromas using standard fractionation, given to a total dose of

57.6 Gray. Local control at 5 years was 93%. Trigeminal and facial toxicity were 3.4% and 2.3%, respectively. Useful hearing was preserved in 94% [13]. In a more recent publication by that same group, Combs and colleagues [14] report that hearing preservation in patients who had useful or serviceable hearing before radiation therapy was 55% at 9 years after SRS, compared with 94% showing serviceable hearing 5 years after FSRT.

Attempts to decrease toxicity by decreasing total tumor dose for ractionated stereotactic radiotherapy have also been described. Thomas Jefferson University in Philadelphia, Pennsylvania presented a retrospective analysis showing no tumor control difference in two cohorts of patients treated with either 50.4 Gray or 46.8 Gray. Although tumor control rates were equivalent (98% versus 100%) with a median follow-up of 3 years, hearing preservation was better in the lower dose group. Hearing preservation was measured by pure tone averages and speech discrimination. Corrected for follow-up and initial hearing, the rate of preservation was 93% for the low-dose group versus 67% for the higher-dose cohort. The median follow-up time for the low-dose group was 29 months [15]. A group at Hokkaido, Japan used 40 to 50 Gray in 20 to 25 fractions. Their actuarial tumor control rate at 5 years was 91%, with no new permanent facial weakness. The rate of useful hearing preservation (Gardner-Robertson Class I or II) was 71%. Complications were mild—transient facial nerve palsy was 4%, trigeminal neuropathy was 14%, and balance disturbance occurred in 17% of patients [16].

In another low-dose FSRT study, Shirato and colleagues [17] matched a group of patients who had vestibular schwannoma who underwent observation only against a cohort of patients treated with fractionated radiotherapy delivering 36 to 44 Gray in 20 to 22 treatments. The conclusion of the study was that there were no differences in the actuarial Gardner/Robertson hearing class preservation curves after the initial presentation. The rate of hearing deterioration in the treated arm was comparable to that of untreated patients. The mean growth of the tumor in the observation arm was 3.87 mm per year, whereas there was tumor reduction in the radiated cohort.

Hypofractionation

In an attempt to maximize hearing preservation rates without the need for several weeks of daily radiation treatments, a third alternative—hypofractionation—has also been studied. Using biological modeling to provide theoretically equivalent results as standard fractionation, hypofractionation gives higher doses per treatment for fewer treatments than standard fractionation schemes, but less dosage per day than single-dose prescriptions. Hypofractionation regimens use doses in the range of 3 to 7 Gray per day for 3 to 10 days, for total doses in the range of 21 to 30 Gray.

Meijer and colleagues [18] from Vrije Universiteit University Medical Center in Amsterdam used a hypofractionation schedule of 4 to 5 Gray for 5 days as measured at the 80% isodose line. The 20 to 25 Gray was delivered in 1 week. Five-year local control in 80 patients was 94%. Facial nerve function was preserved in 97%. The study authors compared these patients to a group of 49 patients treated at the same institution with a single fraction of 10 to 12.5 Gray, and found no significant differences in outcome in regard to tumor control or facial nerve damage. Five-year hearing preservation favored the fractionated group (75% versus 61%). At Johns Hopkins in Baltimore, Maryland, a similar rate (70%) of hearing preservation was also achieved using 5 Gray for 5 days for smaller tumors or 3 Gray for 10 treatments for larger tumors [19].

Large tumors

Tumor size can affect control. Foster and colleagues [20] showed that tumors larger than 3 cm had a control rate of 33%, whereas tumors of 2 to 3 cm had a control rate of 86%, and tumors of 2 cm or less could be controlled in 89% of their SRS series. Chan [9] also showed a relationship between increasing tumor volume and the need for surgical intervention (shunt or resection) following FSRT.

Park and colleagues [21] reviewed 50 cases of acoustic neuromas measuring over 3 cm on MRI. Microsurgery was performed on all patients. Among eight patients who underwent subtotal resection followed by radiosurgery, all had tumor control with a median follow-up of 113 months (9.4 years). Gross total resection alone resulted in failure in one patient, and subtotal resection without radiation resulted in a 32% recurrence rate. The facial nerve preservation rate was inversely proportional to the extent of tumor removal.

Neurofibromatosis-2

Approximately 5% of patients who have acoustic neuromas have neurofibromatosis type 2 (NF-2). These patients present a special management problem, because their tumors are often bilateral, placing them at risk for total hearing loss. Both microsurgical techniques and stereotactic radiosurgery have been associated with poorer rates of hearing preservation in NF-2 patients. Tumor control rates following single-dose stereotactic radiosurgery are reported as 50% to 98%, with preservation of functional hearing being achieved in 40% to 50% [22–25]. This decrease in functional hearing following therapy is also noted in some fractionated stereotactic radiotherapy series. Combs and colleagues [14] saw hearing preservation rates fall from 98% in sporadic vestibular schwannoma cases treated with 57.6 Gray given in 1.8 Gray fractions to 64% in NF-2 patients treated with the same regimen. Chan and colleagues [9] saw no differences in results between their NF-2 and sporadic cases in regard to tumor control or hearing preservation rates, using a fractionated schedule. The Stanford, California CyberKnife (Accuray, Sunnyvale, California) group obtained hearing preservation rates of 67% at 2 years, using a hypofractionated technique of 21 Gray delivered in three fractions of 7 Gray, with 90% tumor control at a mean follow-up of 26 months. Nine percent developed trigeminal nerve injury [24].

Radiographic follow-up

MRI scans should be obtained at regular intervals following therapy. Loss of central enhancement is a common finding, usually associated with enlargement and capsular thickening. A group at UCLA reported the loss of central tumor enhancement in two thirds of their patients at a median of 6 months following stereotactic radiotherapy [9]. The increase in tumor size was less than 2 mm. Radiographic enlargement occurring after 2 years, or growth 3 mm or greater, is indicative of tumor regrowth. Resection can be performed after radiation therapy; however, some authors feel it is more difficult than resection as initial treatment. Conversely, radiation therapy following resection has a higher complication rate as well.

Clinical follow-up

Most patients undergoing treatment for acoustic neuromas tolerate their treatments well, and tumor growth is controlled. At the doses currently used, the majority of patients do not develop new symptoms. A minority of patients experience an improvement in symptoms, and a minority experience worsening symptoms.

Hydrocephalus

Hydrocephalus in the absence of progressive tumor growth has been described as occurring in 3% to 11% of patients in both SRS and FSRT series [26,27]. The hydrocephalus occurs at a median of 1 year. Hydrocephalus is believed to be the result of tumor necrosis, with proteinaceous debris blocking cerebrospinal fluid (CSF) flow. The development of hydrocephalus is more common following treatment of larger (>25 mm) tumors. The hydrocephalus sometimes resolves spontaneously [10]. Shunting may be required if hydrocephalus becomes symptomatic.

Tinnitus

Pittsburgh radiosurgery experience describes symptoms of tinnitus resolving in approximately one half of patients, whereas the UCLA FSRT experience with stereotactic fractionated radiotherapy provided improvement in 6 of 50 patients, whereas two patients experienced worsening [9]. Karpinos and colleagues [27], in comparing microsurgery and radiosurgery, found more tinnitus at long-term follow-up in patients undergoing radiosurgery. In their radiosurgery group, 26% reported increase in tinnitus, whereas 10% reported decreased tinnitus.

Vertigo

Karpinos and colleagues [27] noted no significant difference in experiencing postprocedural imbalance between microsurgery and Gamma Knife radiosurgery: 22% worsened, whereas 14% improved. Niranjan and colleagues [28] describe episodic vertigo continuing following radiosurgery in 3 of 11 patients presenting with this symptom. Balance disturbances worsened in 17% of patients treated in Hokkaido using a low-dose, fractionated stereotactic radiation scheme [16].

Malignant transformation

Malignant transformation can occur, but the risk is estimated to be very rare. Bari and colleagues [29], in a literature review of malignancy in vestibular schwannomas, describe malignant degeneration in both radiated and unirradiated eighth-nerve tumors. In their literature review,

they describe the incidence of malignant transformation as being very low [29]. Most series are not sufficiently mature to have along-term follow-up in this regard. In the 20-year experience published by Maire [30], one case of malignant transformation is described in the cohort of 45 patients. Isolated case reports have been cited by others as well.

Discussion

In patients presenting with large tumors or tumors causing pontine compression, surgery is necessary. Otherwise, patients who have acoustic schwannomas should be given the option of having either SRS or FSRT. The major advantage of stereotactic radiation over surgery is the ability to achieve tumor control while minimizing morbidity and cost. In an outcomes analysis of treatment of small (<3 cm) acoustic neuromas, Pollack, and colleagues [31] concluded that SRS was more effective in preserving hearing compared with microsurgery. Microsurgery is associated with a greater rate of facial and trigeminal neuropathy in the immediate postoperative period, as well as with long-term follow-up. Both surgery and radiosurgery had similar effects on other preoperative symptoms such as tinnitus or imbalance. Patient satisfaction was higher in the radiosurgery group. Quality of life studies favor the use of radiation over microsurgery.

The initial hearing preservation data favor fractionation for those patients who have functional hearing and larger tumors, but there has been no randomized trail between the two techniques. Comparative studies [18,26] in Philadelphia and Amsterdam describe early advantages with FSRT for hearing preservation, but favor SRS for NF-2. Many studies show equivalency in effect. Longer follow-up is necessary, because further hearing decline with time might occur in both groups. Doses to critical structures need to be further studied in an attempt to steer high-dose deposition away. The advantage of SRS or the hypofractionated techniques is convenience, because tumor control for smaller tumors is equivalent.

St. Thomas brain and spinal cord tumor center experience

The same radiosurgical team, comprising a neurosurgeon, a radiation oncologist, and a medical physicist, accounts for over 30 patients treated for acoustic neuroma who have at least 6 months follow-up in the authors' linac-based program. Patients admitted to our radiosurgical program follow the scrutiny of our interdisciplinary brain tumor board for enrolment. We have developed a treatment algorithm that places patients indicated for treatment into one of three categories:

- Surgery: occipital, translabrynthine, or middle fossa approach
- Stereotactic radiosurgery/radiotherapy (SRS/SRT): radiosurgery is here defined as single treatment, whereas radiotherapy refers to our capability to fractionate stereotactic doses (Novalis Shaped Beam Surgery TM, BrainLAB, Feldkirchen, Germany).
- Fourth pathway: intra-operative recognition that aggressive dissection might be mitigated with SRS as back-up after tumor debulking and mass effect reduction goals have been attained

The basis of which treatment is offered often depends upon patient predilection. Because our center has vast experience, with surgical extirpation as well as SRS/SRT, our patients are afforded the opportunity to extensively discuss all treatment modalities.

Surgery is recommended when patient's age, health, and tumor mass effects and size predispose. Brainstem and cerebellar mass effect correlated to tumor size greater than 3.0 cm, and resulted in placement in the surgical extirpation treatment arm of our algorithm. Medical contraindications—age and strong patient predilection—mitigate the surgical algorithm. When tumor size is less than 3.0 cm and there is a paucity of mass effect, either radiographically or symptomatically, our algorithm favors SRS/SRT.

Discussion of SRS/SRT includes the procedure and expected outcomes based on our experience, as well as, the existing literature. We cite SRS/SRT greater than 80% tumor control rates documented in existing literature with less than 5% complication rates of facial, trigeminal, or cranial nerve palsy. Hearing preservation is an ongoing concern, and we are currently audiometrically surveying our patients beginning at 1-year intervals, comparing to pretreatment baseline. Current hearing preservation rates reported range 85% to 95%. No hearing loss has been documented to date in our patient cohort.

Materials and methods

Between June 2003 and September 2006, 30 patients were treated by one of the radiosurgical

teams at our institution, composed, as noted, of one neurosurgeon, one of two radiation oncologists, and one medical physicist. Patients are pre-evaluated for SRS/SRT treatment by neurosurgical examination as well as neuro-otologic assessments. Patients are clinically evaluated per cranial nerve involvement, specifically facial nerve function and vestibular and trigeminal nerve function, and audiometrically tested at baseline. Follow-up neurosurgical evaluation of cranial nerve function at 6 weeks post-SRT and at 3-month intervals for the first year. Six-month clinical follow-up ensues thereafter. MRI surveillance is obtained at the 6-week post-SRT appointment, and at 6-month intervals thereafter up to 2 years. There is 1-year MRI follow-up thereafter until 5-year post-treatment. MRIs are evaluated according to tumor volume, axial anterior-posterior dimensions at the porous, medial-lateral dimensions at porous, and coronal rostral-caudal dimension at porous. Signal characteristics of MRI are also noted—contrast degradation and necrotic or cystic breakdown of tumor consistency on MRI.

SRS/SRT was performed using Novalis Shaped Beam Surgery TM. SRS refers to a single-dose treatment plan, whereas treatment planning using Novalis affords fractionation capability. We term this modality treatment as SRT to distinguish this capability. We are currently evaluating our cranial nerve data to corroborate previous experience that fractionation affords greater cranial nerve preservation rates, or to determine that it does not. We are also determining evaluation of specific cochlear dosing as it pertains to fractionation, in order to consider its impact on hearing preservation in SRS/SRT.

All patients were treated at a single institution using the Novalis Shaped Beam Surgery TM 6-Mev linac. Initial imaging consisted of 1-mm thick slice MRI scans, with T1-weighted images with gadolinium contrast enhancement. The BrainLAB-compatible MRI is obtained and entered into the Novalis work station. Meticulous contouring shaping is performed by the neurosurgeon. Consideration of the cerebello-pontine angle, brainstem, skull base, cranial nerve, and surrounding neuroanatomy was incorporated into the treatment planning to ensure high conformality and selectivity. CT scans were then performed while the patient was immobilized in a BrainLAB mask. These image sets were fused for treatment planning. All target volumes were defined jointly by the attending neurosurgeon and radiation oncologist. The plan was then exported to the radiation oncologist and medical physicist for dosimetry.

Target doses were prescribed to the 905 isodose volume using a single isocenter with multileaf collimation. Doses range from 30 Gray given in 10 fractions to 45 to 54 Gray delivered in 20 to 35 fractions, depending on brainstem limitations (45 Gray) and the preference of the radiation oncologist. Patients treated with shorter fractionation schemes usually lived a longer distance from our institution, or already had some decrease in hearing. No patients were treated in a single fraction in our institution. Maintenance dose steroid is administered through treatment; typified by medrol dose pack, or three-times-a-day dosing if fractionation lasts longer than five sessions.

Results

As noted, patients have undergone surveillance MRI and clinical evaluation by the treating neurosurgeon.

Magnetic resonance imaging response

Of the 30 patients treated, all but one had either tumor control or signal degradation. Tumor control is typified by tumor volume dimensions as outlined above under Materials and methods. As previously anecdotally reported, we too have appreciated small millimetric variability at the initial 6-week post-SRT MRI. The millimetric expansion at this MRI interval is thought to reflect early SRT effects, and subsides on subsequent MRI surveillance. Tumor volumes either maintained control or demonstrated signal degradation in ongoing MRI surveillance. Degradation is typified by contrast diminishment or fading, or actual necrotic cavitations on MRI. (Fig. 1A, B) has two MRIs showing a typical sequence. One patient went on to millimetric expansion of tumor beyond the 6-week post-SRT tolerance. This patient will be considered for retreatment after ongoing MRI surveillance, because no clinical sequelae have ensued.

Cranial nerve preservation

We are currently evaluating hearing audiometric data of our patients at 1-year follow-up versus baseline pre-SRT testing. As previously reported, we expect very high hearing preservation rates, at least through the current follow-up time frame. We have not as yet documented hearing degradation in our experience through close clinical follow-up. We have no reported facial nerve or

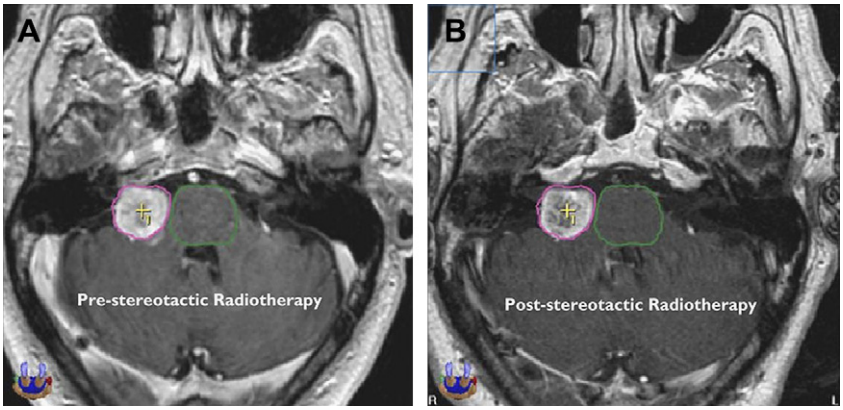

Fig. 1. Demonstrating degradation of contrast signal and contraction of size of acoustic neuroma. (*A*) Pre-stereotactic radiotherapy. (*B*) Post-stereotactic radiotherapy.

trigeminal nerve clinical sequelae following SRT. We have no incidence of brainstem symptomotology following SRT. We have not experienced exacerbations of ataxia, dysphagia, paresthesia, or vertigo in our patients post-SRT. In our clinical review of our SRT experience, we have appreciated cases of chronic symptom abatement in respect to ataxia, paresthesia, and vertigo. We have not documented abatement of symptomatic tinnitus post-SRT.

Conclusions

The management options of acoustic neuroma include observation, microsurgery, and SRS/SRT. We also include consideration of what has been termed "the fourth pathway" of SRS/SRT after microsurgical debulking, to acknowledge tumors that are otherwise too large or mass effectively symptomatic for SRS/SRT alone. Surgical risks are minimized by the planned postoperative use of SRS/SRT. We have developed a treatment algorithm to guide our patients through these treatment options (Figs. 2 and 3).

The goal of SRS/SRT is permanent control of tumor growth, while preserving neurologic function. Tumors greater than 3.0 cm, exhibiting mass effect, and harboring acute symptomatic evolution are not thought to be appropriate for SRT. The dose distribution required for larger tumors as well as the abatement of mass effects is not appropriate for the time frame of SRT to address. For tumor sizes less than 3.0 cm and a paucity of radiographic mass effects, SRT affords high rates of tumor control and neurologic preservation. MRI surveillance demonstrates the realization of

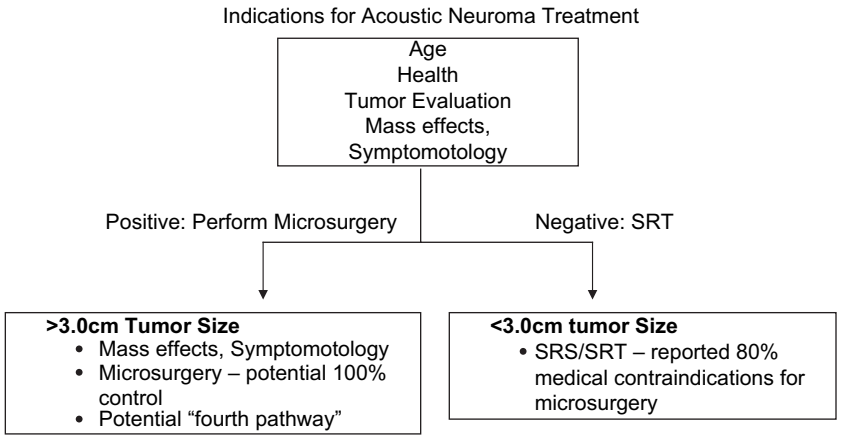

Fig. 2. Indications for acoustic neuroma treatment.

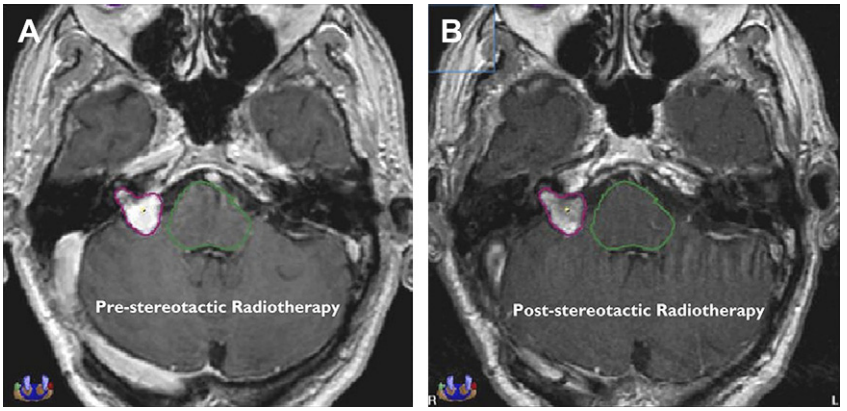

Fig. 3. Demonstrating degradation of contrast signal and contraction of size of acoustic neuroma. (*A*) Pre-stereotactic radiotherapy. (*B*) Post-stereotactic radiotherapy.

the goal of tumor control as its growth has been inactivated by SRT. Close clinical follow-up has not demonstrated neurologic deterioration. We have appreciated symptom abatement in the post-SRT clinical assessments. Symptoms of vertigo, ataxia, and paresthesia have abated in a number of our cases. We differentiate acutely progressive mass effect symptoms as microsurgical indicators, but appreciate chronic symptomotology as appropriate to consider SRT indicators. SRT is not only confined to asymptomatic lesions.

Our experience has evolved an algorithm considering tumor size (less than 3.0 cm), location, or paucity of mass effect (eg, intra- versus extracanalicular) and symptom chronicity to favor SRS/SRT in treating acoustic neuromas. The dramatic achievement in tumor control, preservation of neurologic function, and even abatement of chronic symptoms are countered to advances in microsurgery. Microsurgery is relegated to larger tumors (greater than 3.0 cm), significant radiographic extracanalicular mass effect, and acute symptoms the natural history of which SRS/SRT would not address.

Summary

SRT is appealing as a modality to obviate surgery related morbidity, which remains an issue despite microsurgical advances. In the authors' experience, SRT prevents acoustic neuroma growth, preserves neural function, and is not associated with treatment-related complications. We offer SRT as a first-line treatment choice in patients harboring small to medium sized tumors, even when chronically symptomatic. SRT will be offered more frequently as a primary option as tumors are more frequently identified at smaller sizes.

Appendix

A physicist's perspective: comparisons of Gamma Knife circular collimated stereotactic radiotherapy and accelerator-based, micro-multileaf, collimated stereotactic radiotherapy

The treatment of acoustic neuromas has been performed with stereotactic radiotherapy for quite some time. The benefits of a noninvasive procedure reduce the risks inherent with surgery, and also limit damage to normal tissues that might be injured with a surgical approach. Initially, such therapy was limited to SRS. This denoted a single fraction of radiation delivered to the patient, whose head was fixated with a frame capable of three-dimensional target placement. A frame was necessary so that a consistent coordinate system could be mapped to the patient's skull. For the Gamma Knife patient, this frame was applied by the neurosurgeon the morning of the procedure. After framing, the patient would then be sent to MRI for imaging; the Leksell frame used by the Gamma Knife being compatible with MRI. After imaging with a spoiled gradient (SPGR)-type sequence such that the voxel sizes of the scan were equivalent in all dimensions, the patient would then wait in a holding area while the physicist transferred the images into the planning computer.

Although all planning was performed on the MRI, the actual dose calculations were performed

by taking an approximation of the shape of the patient's skull and entering those data into the computer. The physicist would then review a wire-frame model of the interpolated skull radii measurements, and then the neurosurgeon would commence the planning process. This was performed by placing several isocenters of discrete sizes to cover the acoustic neuroma with the 50% isoshell. The collimators available were 18 mm, 14 mm, 8 mm, and 4 mm. A three-dimensional plan could be easily performed on a regularly shaped spherical acoustic neuroma that had a diameter similar to that of the collimators described. When the acoustic neuroma was irregularly shaped and large, however, treatment planning became a challenge because of sphere packing. The normalization of the plan was constantly updated to reflect the maximum dose location and magnitude as more isocenters were placed. Locating isocenters too close to one another would significantly renormalize the plan. This could cause a complex plan to fall apart, and then time would need to be taken to space out the corresponding isocenters and regain a reasonable dose distribution.

Another issue with large lesions related to the number of isocenters needed to cover the target was penumbra enlargement. A single isocenter has a very sharp penumbra, and therefore is exceptionally good at tumor conformality; however, because more and more isocenters are required to cover a large or irregularly shaped lesion, the penumbra of the lesser isoshell values broadens considerably. Also, dose heterogeneity is a commonplace side effect of dose sphere-generated plans. Some say this heterogeneity is of value for malignant tumors, but might be questionable for benign lesions.

Finally, it is difficult to calculate a verification of the treatment times of a Gamma Knife plan independently of the treatment planning computer. Linac-based stereotactic therapy has its own benefits and liabilities. The commissioning of such a system is very difficult because of the increased number of degrees of freedom of the unit. Once completed, the treatment delivery can be every bit as accurate as a Gamma Knife. Treatments can be mimicked with frame fixation and stereotactic cones to replicate a Gamma Knife dose distribution, with the same liabilities denoted above. The true benefit lies in being able to mask the patient in a way such that the documented maximum deviation should not exceed 1.5 mm and, in practice, is approximately 1 mm, this leads to the delivery of stereotactic radiotherapy or SRT.

The ability to fractionate the treatment gives the advantage of dose escalation for increased control, without increased probability of normal tissue damage. The masking system also allows several steps of the treatment planning process to be completed without the presence of the patient.

All of the steps of therapy are not required to be completed in a single day, meaning the patient does not need to spend considerable time at the center awaiting treatment. An additional advantage can be the inclusion of a miniature multileaf collimator (MMLC). With this apparatus, 3-mm leaves can shape to any desired target, and have penumbra that averages 2.5 mm, from 2-cm to 10-cm field sizes. The main advantage is the ability to treat the lesion with a single isocenter of 9 to 12 static beams, conforming to the target shape, and spaced out in a Gamma Knife-like fashion to equally distribute the radiation entry areas. The patient benefits from a much shorter treatment time with such an accelerator-based system, and a decreased margin for positional error because of a single isocenter placement rather than multiple placements. A typical isoshell that covers the tumor nicely is approximately 90% rather than 50%, and lends itself to a more homogeneous distribution. The ability to perform hand-calculation second checks of the accelerator beams also gives more confidence in the treatment delivery. The treatment planning systems use the CT scan of the patient for dose calculation, thus giving the ability to use heterogeneous dose calculations to account for bone and air density differences as well.

For truly difficult lesions, intensity modulated radiotherapy (IMRT) is also an option. Benefits of a linac-based system are fractionated therapy, improved dose calculation, decreased planning time for difficult lesions, and, in most instances, decreased treatment times for the patient.

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Guiding Patients Through the Choices for Treating Vestibular Schwannomas: Balancing Options and Ensuring Informed Consent

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Counseling patients who are diagnosed with vestibular schwannomas (VS), formerly known as acoustic neuromas, can be challenging. These benign neoplasms, which originate from either the superior or inferior vestibular nerves, have an average growth rate of 1 mm^3 per year. Larger tumors can cause brainstem compression with few noticeable symptoms, whereas smaller VS can cause vertigo, tinnitus, and hearing loss.

Patients are confronted by the difficult task of choosing a treatment method based on the advice of caregivers, currently available literature, and Internet-based information sources. Patients often visit the Internet either before or during their decision period, which can be helpful or even more confusing for them as they weigh their options. The health care provider has the responsibility to explain, in understandable language, to the patient or legal representative governing the patient's care the proposed treatment options, risks and complications associated with each form of treatment, and alternatives to treatment, including no therapy. The medical record must contain evidence of the patient's informed consent, with the exception of emergency situations in which a delay in intervention could compromise outcomes in a life or limb-threatening situation. Aside from cases with brainstem compression and hydrocephalus, patients should be encouraged to gather information before making a treatment decision. For the physicians managing these patients, information should be delivered in a balanced way to ensure patient understanding of their options leading to adequate informed consent.

Options for treatment include radiation therapy, surgical excision, and observation with serial MRI. Discerning treatment advantages from a particular modality is made difficult because of nonstandardized definitions of tumor control and hearing preservation and varied posttreatment intervals presented in the medical literature. Surgical techniques and radiotherapy dosage paradigms have evolved considerably over the past two decades. Currently, no randomized, prospective clinical trial has compared the three treatment options and there are no clearly accepted, evidence-based, best practices for managing VS.

The treatment of VS requires a multidisciplinary team not only to deliver the chosen therapy but also to assist in the decision-making process. At our center, a nurse familiar with VS treatment coordinates appointments with a neurotologist, a neurosurgeon, and a radiotherapist. Patients are encouraged to take the amount of time necessary to make a decision with full understanding of potential risks and benefits. The amount of time necessary to come to a decision depends on the needs of a particular patient.

This article evaluates the English language literature, dating back to 1994, to present long-term results for tumor control and complication rates for the treatment of VS, excluding cases of

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neurofibromatosis type II. The goal is to provide a guide to otolaryngologists who provide initial counseling for patients with newly diagnosed VS.

Stereotactic radiation therapy

Radiation therapy was initially used as an adjunct to surgery in patients with incompletely resected VS. In a study reported from University of California San Francisco, Wallner and colleagues [1] demonstrated that conventional fractionated radiation therapy to more than 45 Gy significantly reduced regrowth from 46% to 6% in incompletely resected VS. In this series, 31 patients treated between 1945 and 1983 were followed from 2.6 to 40.7 years. The authors concluded that postoperative radiation therapy reduced the "local recurrence rate" of VSs that were incompletely excised or only biopsied and demonstrated the effectiveness of radiation therapy in the treatment of acoustic neuromas.

Stereotactic radiosurgery, first developed in 1951 by Leksell [2], is a method of delivering a highly conformal single dose of ionizing radiation with submillimeter accuracy to an intracranial target. The goal of a single high-dose delivery was to cause tumor necrosis and control of growth as an alternative to surgery for patients who were suboptimal candidates for excision. The first stereotactic radiosurgery treatment for VS was performed in 1969 with the gamma knife (GK), also developed by Leksell [3]. The GK is a highly specialized radiation delivery system that uses 201 radioactive cobalt 60 sources to deliver high-dose radiation accurately to tumor masses. A stereotactic frame is fixed to the skull and attached to the treatment table to provide rigid immobilization of the patient's head and ensure accurate localization of the radiation dose.

Thousands of patients have been treated with the GK. Initially, doses were high, and although tumor control was excellent, toxicity was significant [4–6]. Several refinements, including dose reduction, improved target definition, and treatment accuracy, have provided excellent tumor control while minimizing toxicities of treatment [7,8]. Lower radiation doses were shown to be effective in controlling tumor growth, whereas tumor shrinkage could take several years to document radiographically.

The linear accelerator (LINAC) also can be adapted to perform stereotactic radiosurgery [9]. Multiple beam positions or arcs are used to create a conformal dose distribution around the target. Although the tumor margin doses are similar to the GK, LINAC radiosurgery typically uses fewer isocenters and the dose to the tumor is more homogeneous. Similar to the GK, a non-relocatable invasive head frame is required for patient immobilization during treatment.

Fractionated stereotactic radiation therapy is the most recently developed technique for delivering high-dose and localized treatment. Unlike with GK or LINAC, a noninvasive relocatable head frame or thermoplastic mask is used for fractionated stereotactic radiation therapy. This head frame increases patient comfort but may result in less dose conformality when compared with other radiosurgery techniques. Subscribers of this technique believe that fractionation takes advantage of radiobiologic principles to reduce late toxicity while maintaining tumor control [10,11]. The fractionated treatment regimens range from doses given over several days to standard fractionation given over 4 to 5 weeks, similar to the scheme originally used by Wallner.

Data regarding the outcomes GK, LINAC, and FSRT are reviewed in the next section. Because few centers in the world offer proton beam radiation therapy to treat VS, studies that used proton-based treatments were excluded. All pertinent papers published from 1994 to the present that assess local control rates with a median follow-up of at least 2 years were reported. Outcomes included tumor control, hearing preservation, facial neuropathy, and trigeminal neuralgia. In most patients, tumors were sporadic and had a maximum diameter of $\leq$3 cm.

The goal of radiation therapy is to arrest tumor growth. Local control rate in radiotherapy studies can be defined as the percentage of tumors that do not increase in size on follow-up imaging. Many researchers define the local control rate as the percentage of tumors that do not require salvage therapy. This determination could overestimate the control rate because some tumors may have progressed but are not symptomatic enough to require further treatment. All three techniques seem to achieve excellent local control with a range of 87% to 100% (Tables 1–3) [8,10,12–45].

Up to 50% of radiated tumors developed central necrosis that results in transient increase in tumor volume (23% of cases) [12]. This phenomenon was observed up to 4 years after treatment and took from 6 months to 5 years to disappear. If tumor progression was defined as tumor growth, then some patients may have

Table 1
Gamma knife radiosurgery outcomes (median marginal dose 12–14 Gy)

Author (year)	Number of patients	Local control rate	Hearing preservation rate	Facial neuropathy rate	Trigeminal neuropathy rate	Other complications
Flickinger, 2004 [14]	313	98.6% @ 6 y	78%@ 6 y	0% @ 6 y	4.4% @ 6 y	
Andrews, 2001 [10]	63	98%	33%	2%	5%	hc 2.9% vertigo 1.4% ataxia 1.4%
Massager, 2006 [15]	82	98%	65%			
Ottaviani, 2002 [16]	30	87%	73%	3% temp	16% mild symptoms	
Wackym, 2004 [17]	29	94%		0%	3% temp	
Prasad, 2000 [12]	153	92%	75% for tumors <1 cc, 57% for tumors >1 cc	2.3%	1.7% perm, 2.5% temp	
Chung, 2005 [18]	195	95% @ 7 y	60%	1.4% temp	1.1%	hc 3.5%
Wowra, 2005 [19]	111	95% @ 6 y		2.7%	11.5%	
Lundsford, 2005 [8]	829	97% @10 y	78.6%	0%	4.4%	ventriculoperitoneal shunt 0.8%, tinnitus 0.2%, peritumoral cyst 3.6%, no radiation-induced malignancy
Petit, 2001 [20]	45	96%	88%	4% temp	0%	
Hasegawa, 2005 [13]	317	92% @10 y	68%	1% temp	2%	hc 4.1%, malignant transformation 0.3%
Pollock, 2006 [21]	46	100%	63%	4%	2.1%	hc 4%
Mysreth, 2005 [22]	103	89.2%		5.2%		hc 3.9%
Paek, 2005 [23]	25	100%	46%	0%	5%	
Litvack, 2003 [24]	134	97.7%	61.7%	2.2% temp	5.8% temp	hc 3.0%
Hirato, 1996 [25]	29	93%	59%	10.3% temp	3% temp	hc 6.8%
Muacevic, 2004 [26]	219	97%	49%	0.5%	5% temp	
Iwai, 2003 [27]	51	96%	56%	0%	4%	hc 8%
Kwon, 1998 [28]	88	95%	67%	4% temp, 4% perm	3.4% temp	hc 3.5%

Hearing preservation rate is the percentage of patients who maintained or gained useful hearing (Gardner-Robertson class 1-2) after treatment.
Abbreviations: perm, permanent; hc, hydrocephalus; temp, temporary.

Table 2
LINAC stereotactic radiosurgery outcomes (median marginal dose 12–14 Gy)

Author (year)	Number of patients	Local control rate	Hearing preservation rate	Facial neuropathy rate	Trigeminal neuropathy rate	Other complications
Friedman, 2006 [7]	390	90% @ 5 y, only 1% required surgery		4.4% (0.7% w/dose ≤12.5 Gy)	3.7% (0.7% w/dose ≤12.5 Gy)	
Chung, 2004 [29]	45	100%	all patients deaf before tx	4.4% perm	7.5% perm	hc 4.4%
Combs, 2006 [30]	26	91% @ 10 y	55%	5% perm, 15% temp	8%	1 patient with radionecrosis of cerebellum
Meijer, 2003 [31]	49	100%	75%	7%	8%	hc 2%
Speigelman, 2001 [32]	44	98%	71%	2.4% temp, 8% perm	18%	
Foote, 2001 [33]	149	95.4%		11.8% temp	9.5% temp	
Lee, 2001 [34]	42	100%	82%	2.5% perm	2.5% perm	hc 7.5%

Hearing preservation rate is the percentage of patients who maintained or gained useful hearing (Gardner-Robertson class 1-2) after treatment.
Abbreviations: hc, hydrocephalus; perm, permanent; temp, temporary.

undergone surgery for salvage treatment unnecessarily [13].

Lundsford and colleagues [8] and Hasegawa and colleagues [13] reported 10-year local control rates of more than 90% in separate series of patients treated with GK. The median follow-up period in Hasegawa's study was 7.8 years. Partial or complete radiographic response to treatment occurred in 62% of radiated tumors, and tumors <15 cm^3 had a better progression-free survival than tumors >15 cm^3 (96% versus 57%, $P < .001$). Tumors not compressing the brainstem or obstructing the fourth ventricle had a better progression-free survival (97% versus 74%, $P < .008$). Tumor progression occurred within 3 years from the time of treatment in most cases. Forster and colleagues [46] documented local control rates for tumors >3 cm, 2 to 3 cm, and <2 cm at 33%, 86%, and 89%, respectively. Similar findings were reported by Kondziolka and colleagues [4].

Friedman and colleagues [7] reviewed the outcomes of 390 patients treated with LINAC radiosurgery for VS. With a median follow-up of 40 months and a median dose of 12.5 Gy, the 5- and 10-year local control rates were 90% with only 1% of patients requiring surgery for treatment failure during the follow-up period. With a median follow-up period of 48.5 months, Combs and colleagues [30] also reported a local control rate of 91% at 10 years.

The longest follow-up periods for the FSRT studies are from Combs and colleagues [35], Sawamura and colleagues [40], and Chan and colleagues [42]. The median follow-up periods in these studies averaged 48 months, and the 5-year local control rates in all three studies are more than 90% (see Table 3).

Unlike with use of GK, tumor volume or size has not been shown to be of prognostic value in predicting response to treatment with LINAC radiosurgery or fractionated stereotactic radiotherapy for acoustic neuromas. Different fractionation regimens were used depending on tumor size [11,37].

Hearing preservation has not been documented according to a consistent standard (Table 4), and no randomized studies regarding tumor control and hearing preservation have been reported to date. The use of pure tone audiometry and discrimination testing before and at a standardized interval after treatment would be optimal. In most of the radiotherapy papers reviewed, the statistic that is most often reported is the percentage of patients who maintained or gained

Table 3
Fractionated stereotactic radiation therapy outcomes

Author (year)	Number of patients	Dose	Local control rate	Hearing preservation rate	Facial neuropathy rate	Trigeminal neuropathy rate	Other complications
Andrews, 2001 [10]	46	50 Gy/25 fx	97%	81%	2%	7%	hc 3.6%, vertigo 1.7%, ataxia 3.6%
Combs, 2005 [35]	106	57.6 Gy/32 fx	93%	98%	2.3%	3.4%	
Chung, 2004 [29]	27	45 Gy/25 fx	100%	57%	4% temp	7% temp	hc 4%
Shirato, 1999 [36]	33	36–44 Gy/20–22 fx	98%	53%	5% temp	12% temp	
Williams/Shokek, 2004 [11,37]	375	25 Gy/5 fx (<3 cm diam), 30 Gy/10 fx (>3 cm diam), 40 Gy/20 fx	97%	59%	1.5% temp 0.5% perm	1.2% temp	
Meijer, 2003 [31]	80	20–25 Gy/5 fx	94% @ 5 y	61%	3%	2%	
Fuss, 2000 [38]	42	57.6 Gy/32 fx	97.5% @ 5 y	85%	0%	4.8%	
Selch, 2004 [39]	48	54 Gy/30 fx	100%	91.4%	2.8%	3.8%	Tinnitus 5%, hc 0%, ataxia 2%
Sawamura, 2003 [40]	101	40–50 Gy/20–25 fx	97%	71	4% temp	14%	hc 11% disequilibrium 16.8%
Poen, 1999 [41]	31	21 Gy/3 fx/24 h	97%	77%	3%	16%	
Chan, 2005 [42]	70	54 Gy/30 fx	92%	84%	1%	4%	
Chang, 2005 [43]	61	21 Gy/3 fx/24 h	98%	74%	0% perm	0%	
Lederman, 1997 [44]	38	20 Gy/4 fx or 20 Gy/5 fx	100%	93.5% (does not distinguish useful from no useful hearing)	2.6% temp	0%	
Ishihara, 2004 [45]	38	15–20.5 Gy/1–3 fx	94%	93%	2.6% temp	2.6%	

Hearing preservation rate is the percentage of patients who maintained or gained useful hearing (Gardner-Robertson class 1-2) after treatment.
Abbreviations: fx, fraction; hc, hydrocephalus; perm, permanent; temp, temporary.

Table 4
Hearing classification scales

AAO-HNS classification		
Class	Pure tone average (0.5, 1, 2, 3 kHz measured in dB HL)	Speech discrimination score (%)
A	0–30	70–100
B	31–50	50–100
C	>50	50–100
D	Any	<50
Gardner-Robertson Classification		
Class	Pure Tone/Speech Reception Threshold (dB HL)	Speech Discrimination Score (%)
1	0–30	70–100
2	31–50	50–69
3	51–90	5–49
4	>90	1–4
Word Recognition Scores		
Class	Word Recognition Score (%)	
I	70–100	
II	50–69	
III	1–50	
IV	0%	

Data from Meyer TA, Canty PA, Wilkinson EP, et al. Small acoustic neuromas: surgical outcomes versus observation or radiation. Otol Neurotol 2006;27(3):380–92.

useful hearing (Gardner-Robertson classes 1 and 2). Intervals for testing hearing after treatment are not clear, and in some studies residual hearing was defined by whether patients could talk on the telephone on the side of the affected ear. Hearing loss is rare, tends to occur over 6 to 24 months, and can continue to decline for years after treatment. Prasad [12] reported a useful hearing preservation rate of 58% with a median follow-up of 4.2 years. In most patients, hearing decline occurred after 2 years and continued up to 8 years after treatment, with a hearing preservation rate of 75% for tumors $<1\ cm^3$ compared with 57% for tumors $>1\ cm^3$. Massager [15] demonstrated better useful hearing preservation rates in tumors with intracanalicular volumes $<100\ mm^3$ compared with larger tumors (82.6% versus 44.8%, $P = .045$).

Improved hearing outcome has been demonstrated at doses $\leq$13 Gy. In an early paper from the University of Pittsburg, Kondziolka and colleagues [4] reported a "useful" hearing preservation rate of 46% at an average marginal dose of 16 Gy. Later studies documented hearing preservation in up to 68% to 78% of cases treated in the range of 12 to 13 Gy [13,14]. Because there were no differences in the local control rates with the lower doses, it is common practice to prescribe 12 to 13 Gy to the tumor margin when using stereotactic radiosurgery to control VS growth and maximize hearing preservation.

Others attempt to minimize toxicity and improve hearing preservation rates by fractionating the dose. This practice is based on a radiobiologic principle that there is a direct relationship between late normal tissue damage and dose per treatment delivered to the tissue. Andrews and colleagues [10] performed a prospective, nonrandomized study to compare the outcomes of treatment for acoustic neuromas with GK radiation to FSRT. The GK dose was 12 Gy and the FSRT dose was 50 Gy in 25 fractions given daily. They found no difference in local control rates, but the hearing preservation rate was 33% for GK treatment compared with 81% for fractionated stereotactic radiotherapy. These results should be viewed with caution because they had relatively short mean follow-up of less than 3 years. Although most of the FSRT studies reported excellent hearing preservation rates, they have shorter follow-up than other radiosurgery series. The different fractionation regimens make it difficult to make any conclusions regarding dose and hearing preservation.

Facial neuropathy is a potential complication with all three treatment options for VS. Kondziolka and colleagues [4] performed a multivariate analysis of 162 patients treated with GK and found that tumor volume and dose of radiation

to the tumor margin were associated with the risk of neuropathy ($P < .001$). With an average marginal dose of 16 Gy, the overall rate of facial neuropathy was 15%. In subsequent reports, when 12 to 13 Gy were prescribed to the margin, the facial neuropathy rate dropped to 0%. Friedman and colleagues [7] presented a similar finding when they lowered their LINAC radiosurgery dose to 12.5 Gy (4.4% versus 0.7%). As seen in Tables 1 and 2, the risk of facial neuropathy with radiosurgery is minimal and often temporary with a dose of 12 to 13 Gy. Similar rates of facial neuropathy are reported with FSRT despite variable doses and fractionation regimens (see Table 3). Unfortunately, no consistent facial nerve function scale was used for reporting.

After performing a multivariate analysis, Kondziolka and colleagues [4] documented that tumor volume and radiation dosage to the tumor margin were associated with the risk of trigeminal neuropathy ($P < .001$). With average marginal doses of 16 Gy, the overall rate of trigeminal neuropathy was 16% compared with 4.4% at 12 to 13 Gy. There was no evidence of trigeminal nerve damage for intracanalicular tumors. Friedman and colleagues [7] noted a similar finding when they lowered their LINAC radiosurgery dose to 12.5 Gy (3.7% versus 0.7%).

Meijer and colleagues [31] reported the outcomes of treatment with LINAC compared with FSRT for their patients with VS. They found a statistically significant increased incidence of trigeminal neuropathy in patients treated with FSRT (8%) versus LINAC radiosurgery (2%) ($P = .048$).

Other potential side effects from radiation therapy include vertigo, tinnitus, ataxia, headache, hydrocephalus, cyst formation, radiation-induced edema or necrosis, intratumoral bleeding, and malignant transformation (see Tables 1–3). The reporting of these toxicities has not been consistent. The rate of hydrocephalus ranges from 0 to 11% [10,29,39]. Sawamura and colleagues [40] reported the highest incidence of hydrocephalus, with 11% of 101 patients treated with FSRT 40 to 50 Gy in 20 to 25 daily fractions manifesting communicating hydrocephalus that requires ventriculoperitoneal shunting. The hydrocephalus resolved in all patients with shunt placement and was assumed to be caused by cerebrospinal fluid (CSF) malabsorption associated with VS.

Delayed malignant transformation or radiation-induced malignancy may occur with radiation therapy for acoustic neuromas. Malignant transformation occurred in one patient 51 months after radiotherapy treatment. The malignant transformation rate in this study was 0.3% among patients who were followed longer than 5 years after GK [13]. Two other case reports of malignant transformation have been reported with GK [47,48]. One case was reported of a patient who developed a glioblastoma multiforme adjacent to an acoustic neuroma that was treated with GK 7.5 years earlier [49]. Another patient treated with FSRT developed a low-grade malignant nerve sheath tumor 216 months after initial treatment [50]. Delayed malignant transformation or radiation-induced malignancies are rare. Long-term, yearly follow-up of these patients provides a more accurate assessment of incidence.

Recent papers compared the outcomes of microsurgery to stereotactic radiosurgery to attempt to determine which treatment is better for sporadic VS ≤ 3 cm. Myrseth and colleagues [22] reported a retrospective study of 189 consecutive patients—86 treated by microsurgery and 103 by GK. The mean follow-up period was 5.9 years. In addition to local control and cranial nerve preservation, they also evaluated quality of life through standardized questionnaires. The overall local control rates for microsurgery and GK were 89.2% versus 94.2%, which was not statistically significant. Facial nerve function (House-Brackmann grade 1-2) was preserved in 79.8% of the microsurgery group and in 94.8% of the GK group ($P = .0026$). The middle fossa approach was not used in these patient cohorts. Overall, the quality-of-life scores were significantly lower in the microsurgery group compared with the GK group. The authors concluded that these results favored GK as the treatment of choice for this group of patients.

Pollock and colleagues [21] presented a prospective cohort study of 82 patients with unilateral, unoperated VS <3 cm in greatest dimension undergoing surgical resection ($n = 36$) or GK ($n = 46$). Other than age (patients undergoing microsurgery were younger), all other pretreatment characteristics were matched in the two treatment groups. The mean follow-up period was 42 months. There was no difference in the local control rate between microsurgery and GK (100% versus 96%, $P = .50$). They found that facial nerve preservation (96% versus 75%, $P < .01$) and serviceable hearing rates (63% versus 5%, $P < .001$) were better in the GK group than microsurgery group. With regard to quality of life, patients who underwent microsurgery

had a decline in physical functioning and bodily pain scores, whereas patients who had GK had lower Dizziness Handicap Inventory scores. All of these differences in quality of life were statistically significant. Although the authors concluded that radiosurgery should be considered the best treatment for this group of patients, they commented on the necessity of longer follow-up.

Microsurgery

Three principal approaches are used to surgically remove VS. The translabyrinthine (TL) route is used for tumors with either no hearing or in cases with little chance of preserving hearing. TL typically would be chosen in patients with less than 50% speech discrimination and 50 dB speech reception threshold, in intracanalicular tumors extending to the lateral margin of the internal auditory canal, or in tumors >3 cm in largest dimension. Two approaches, the middle cranial fossa (MCF) and retrosigmoid (RS), are used in cases with potential hearing preservation. MCF is ideal for intracanalicular tumors with minimal extension into the cerebellopontine angle. The RS approaches the cerebellopontine angle with better visualization of the cranial nerves, the pons, and brainstem. Anatomic limitations may foster recidivism with the hearing preservation approaches. The falciform crest obscures the inferior portion of the lateral fundus when using the MCF. Inferior vestibular nerve tumors may extend into this region to an inaccessible space of 1.82 to 2.33 mm [51,52]. The RS approach leaves up to the lateral 3 mm of the internal auditory canal underexposed. The addition of operative endoscopy has proved useful in accessing the lateral 30% of the internal auditory canal [53]. Larger tumors with significant brainstem compression may be accessed via an RS route in the setting of poor hearing because of the better visibility of brainstem structures. Prognostic factors that favor hearing preservation include a small tumor, greater than 70% speech discrimination and a 30dB speech reception threshold, auditory brainstem response with a normal wave V amplitude and latency, and no medical contraindications for surgery [54]. The choice of approach also depends on the preferences of the surgical team.

The most commonly reported major complications from microsurgery include hearing loss, facial nerve dysfunction, balance abnormalities, cerebrospinal fluid leakage, headache, meningitis, and stroke. Harsha and Backous [55] performed a Medline search for all articles pertaining to VS surgery published from 1994 to 2004. They refined the search to include only English-language articles that reported hearing outcome using either the AAO-HNS hearing or Gardner-Robinson hearing outcome scales, facial nerve outcomes using the AAO-HNS (House-Brackmann) facial nerve grading scale for facial nerve outcome, and complication rates based on approach used. Of 1132 articles evaluated, 31 met the criteria for inclusion and only 14 compared one or more outcome measure for more than one approach from the same institution. Two additional articles, which specifically addressed hearing preservation after middle fossa surgery, have been published subsequent to this article (Table 5) [56–69].

Overall, hearing outcome was serviceable (AAO-HNS class A or B/Gardner-Robinson class 1 or 2) in 618 of 2034 (30%) of patients treated with a hearing preservation approach. Results were considerably better with MCF (523/1017 [51%]) than with RS (95/304 [31%]). In six papers, MCF was compared with RS in the same institution. Forty-eight percent preserved serviceable hearing with MCF compared with 31% with RS [55]. The two most recent articles that addressed hearing preservation and the MCF approach reported high levels of maintained hearing in the serviceable range. Arts and colleagues [67] reviewed 73 consecutive patients excised via MCF. Of the 27 with class A hearing preoperatively, 62% remained class A, 18% deteriorated to class B, and 20% fell to class C. Overall, of the 62 patients presenting with class A or B hearing before surgery, 73% remained in class A or B. No patients improved their hearing class with surgery [66].

Meyer and colleagues [68] reported on 162 consecutive VS resected by the MCF approach. Class A or B/Class 1 or 2 hearing was preserved in 66 of 162 (41%) patients overall. Eight patients improved to class 1 hearing with surgical removal of their VS. Of the 113 patients with word recognition scores (WRS) more than 70% preoperatively, 56 (50%) maintained WRS of more than 70% after excision. Tumor size ranged between 0.2 and 2.5 cm in largest diameter. Smaller tumors (0.2–1.0 cm) had 59% maintain WRS more than 70% and 9% improved to more than 70% when falling below preoperatively. For tumors that measured 1.1 to 1.4 cm, 39% maintained at least 70% WRS and 3 additional patients (9%) improved to more than 70% postoperatively. Only

Table 5
Hearing outcomes using AAO-HNS/Gardner-Robinson classifications

Postoperative hearing classification [Number (%)]				
Author (ref)	A/1	B/2	C/3	D/4
Retrosigmoid approach				
Colletti, 2003 [56]	2 (8%)	3 (32%)	4 (16%)	11 (44%)
Staecker, 2000 [57]	6 (40%)	1 (7%)	1 (7%)	7 (46%)
Hecht, 1997 [58]	4 (10%)	5 (12%)	4 (10%)	29 (68%)
Arriaga, 1997 [59]	8 (31%)	6 (23%)	1 (4%)	11 (42%)
Sanna, 2004 [60]	8 (18%)	7 (16%)	4 (9%)	29 (66%)
Magnan, 2002 [61]	17 (15%)	18 (16%)	23 (19%)	61 (51%)
Lassaletta, 2003 [62]	0 (0%)	5 (17%)	1 (3%)	23 (79%)
Total	45 (15%)	50 (16%)	38 (13%)	171 (56%)
Middle Cranial Fossa Approach				
Colletti, 2003 [56]	3 (12%)	10 (40%)	3 (12%)	9 (36%)
Staecker, 2000 [57]	5 (33%)	3 (20%)	2 (13%)	5 (33%)
Hecht, 1997 [58]	4 (22%)	3 (17%)	1 (5%)	10 (56%)
Arriaga, 1997 [59]	15 (44%)	8 (24%)	2 (6%)	9 (26%)
Sanna, 2004 [60]	4 (7%)	15 (26%)	10 (17%)	30 (50%)
Slattery, 1997 [63]	35 (25%)	39 (27%)	5 (4%)	64 (45%)
Brackmann, 2000 [64]	109 (33%)	87 (26%)	16 (5%)	121 (36%)
Weber, 1996 [65]	5 (10%)	13 (27%)	3 (6%)	28 (57%)
Satar, 2002 [66]	30 (22%)	42 (31%)	18 (13%)	64 (34%)
Arts, 2006 [67]	21 (29%)	24 (33%)	6 (8%)	22 (30%)
Meyer, 2006 [68]	37 (23%)	29 (18%)	4 (2.5%)	83 (51%)
Total	268 (26%)	255 (25%)	64 (6%)	430 (42%)

33% of patients with tumors between 1.5 and 2.5 cm maintained WRS more than 70%. In the hands of the same surgical team, statistically significant improvement was noted in cases in which near field eighth nerve compound action potential monitoring was used. With tumors <1.4 cm, MCF done in experienced hands provided significant hearing preservation not previously reported. This article suggested changing the hearing classification to report only WRS because it more accurately reflects rehabilitation potential for "residual" hearing.

Hearing preservation rates reported in Table 5 address the number and percent of patients with serviceable hearing when compared with all patients treated by approach. This rate does not take into account the number of patients who maintained or improved their hearing status after operative tumor removal. In the two studies that specifically examined MCF for intracanalicular tumors, the results are much more favorable. Long-term (>5 years) hearing preservation in this group of patients should be compared with patients who received radiotherapy at matched intervals.

The AAO-HNS (House-Brackmann) facial nerve grading scale score, determined at the 6- to 12-month visit, is the gold standard for reporting facial nerve outcomes after VS treatment. In the review by Harsha and Backous [55], overall grade I/II function was 82%, with 92% of RS, 89% of MCF, and 73% of TL having a good outcome. Only one center reported results of all three approaches within the same institution. Grade I/II function was maintained as follows: RS (91%), MCF (88%), and TL (77%). Centers with more than 100 cases with a single approach were analyzed separately and good function (AAO-HNS I/II) was maintained as follows: RS (97%), MCF (93%), and TL (78%). These results were not analyzed separately for tumor size, which may have contributed to the choice of surgical approach. Meyer and colleagues [68] reported grade I/II function in 97% of 162 patients (86% grade I) operated via the MCF, whereas Arts and colleagues [67] maintained 96% grade I/II function (85% grade I).

No specific data were presented with standardized methodology to assess postoperative vestibular and other balance dysfunction.

CSF leakage is the most common complication after VS resection. Typically, CSF leaks occur 2 to 3 days postoperatively (early) and at 10 to 14 days after surgery (late). Pooling the data from

19 studies revealed an incidence of 8% (360/4297) for CSF leakage. Leakage rate stratified by approach was: 6% MCF (67/1038), 11% RS (42/380), and 8% TL (253/2881) [55]. Slattery and colleagues [70,71] reported 1697 patients from the House Ear Clinic and found a higher incidence of CSF leakage in the RS group (15%) compared with TL (11%) and MCF (6%). Selesnick [72] performed a meta-analysis and found the lowest CSF leakage rate in the TL group (9.5%) when compared with the RSA and MCF groups (both 10.6%). Overall, the CSF leak rate is low for each of the three approaches and may vary according to the preferences and experiences of individual surgeons and skull base teams.

Headache is common in the first few days to weeks after VS resection. Prolonged postoperative headache—beyond 3 months—occurs in roughly 10% of cases. Bone dust in contact with the CSF and meninges causing secondary aseptic meningitis, entrapment of the occipital nerve in neck musculature and scar tissue, scarring of the neck muscles to the dura, and migraines are considered the four mechanisms for prolonged headaches [54]. No standardized reporting scheme has been developed for postoperative headache, but pooled data from six articles revealed clinically significant headache in 21% of RS, 8% MCF, and 3% TL [55]. Staecker [57] found more than double the headache rate in RS (47%) when compared with MCF (20%).

The rate of bacterial meningitis was 2% to 3% for all three approaches [55]. Meningitis peaked at 3 to 5 days postoperatively and was most commonly caused by *Staphylococcus aureus*. Aseptic meningitis can occur in up to 20% of patients and responds to intravenous or oral steroids. Seizure, hydrocephalus, and stroke rates caused by surgery are rare and occur in less than 2% of cases.

Observation with serial neuroimaging

An often undervalued treatment modality for VS is observation with serial neuroimaging using MRI scanning at regular intervals. A report from Saudi Arabia retrospectively reviewed 205 patients with intracanalicular tumors for an average of 40 months (range 12–180 months). No growth was found in 66.3% of patients, 23.9% showed slow growth, and 4% had rapid growth. Six patients (3%) had tumor regression [73]. Charabi and colleagues [74] reported on 127 tumors in 123 patients followed from 1973 to 1993 in Denmark. At a mean follow-up of 3.8 years, tumor growth was found in 82% of tumors, no growth in 12%, and regression in 8%. Growth patterns were variable and unpredictable. Leeuwen and colleagues [75] found no correlation among tumor size, symptoms, and patient age in 164 patients treated and over a period of 13 years in Holland. Yoshimoto [76] reviewed 1340 patients from a Medline search of 26 studies. The overall growth of VS during a mean follow-up period of 38 months was 46%. Growth averaged 1.2 mm/y. Raut and colleagues [77] reviewed conservative management of 72 patients in the United Kingdom. Growth was seen in 38.9%, no growth in 41.7%, and regression in 19.4% after a mean follow-up of 80 months. Pure tone and speech discrimination scores deteriorated regardless of tumor growth. In Aarhus County, Denmark, 162 VSs were diagnosed and 64 patients opted for conservative management. Twenty-three percent of tumors grew more than 1 mm/y, 55% did not grow, and 22% regressed. The observation period extended to 15 years (180 months) [78]. Unfortunately, no common predictors or ''red flags'' could be extracted from these studies to predict growth patterns for VS.

Battaglia and colleagues [79] reviewed 164 patients treated with radiation therapy from 1986 to 2004 at the Southern California Permanente Group and compared their long-term growth rates with the results of a meta-analysis of five studies of cases managed conservatively. They reported an average growth rate of 0.7 mm/y. They determined that small intracanalicular tumors grew more slowly, if at all, when compared with tumors in the cerebellopontine angle. They concluded that to determine whether radiotherapy alters tumor growth rate, a direct comparison of small, medium, and large tumors is necessary.

The option of observation with serial MRI scanning is a valid form of managing VS. Severity of symptoms does not correlate with lesion size, and determination of efficacy of radiotherapy in small tumors VS the natural history of intracanalicular lesions is unclear. If no brainstem compression or hydrocephalus is present, patients should be offered an observation course with an understanding that no clear risk factors for growth have been defined. Our center considers observation and interval symptom review, physical examination, and neuroimaging a form of treatment and document it as such.

Summary

Patients diagnosed with VS are faced with a difficult decision-making process based on

medical literature weakened by inconsistent reporting of results. Radiotherapy protocols and surgical techniques have evolved in the past 30 years. Most reporting is retrospective and does not adhere to consistent and validated standards for hearing preservation, facial nerve function, or balance disturbance.

Confusion surrounding recidivism versus recurrence of VS and no clear and validated definition of tumor control further clouds the interpretation of results. The lack of randomized, blinded clinical trials has retarded the development of evidence-based clinical best practices for recommending optimal treatment for patients dictated by such factors as tumor size, location, and hearing level. Patients often review data on the Internet, which is not peer reviewed and can complicate preoperative counseling.

The mandate for establishing and documenting informed consent in a format that is timely and understandable to individual patients remains. A trend is developing in which neurosurgeons and neurotologists are acquiring certification in GK and other forms of radiosurgery. These surgeons provide a balanced informational process because they are involved in either option selected by a patient. In other centers, a multidisciplinary team of surgeons and radiotherapists individually consults with patients within their own area of expertise to present options to patients and foster appropriate informed consent. Whichever the approach taken to explain options to patients, one physician should remain the gatekeeper of the patient's care. Answering additional questions and ensuring the opportunity for interval clinical, audiometric, and radiographic follow-up remains the responsibility of the care team. The choice to follow surveillance is a patient choice.

Standardized quantification of tumor size (volumetric or greatest dimension), consistent adherence to the AAO-HNS facial nerve reporting scale, consistent acceptance of one reporting standard for hearing and agreed-upon intervals for testing, and follow-up for longer intervals improve data and foster outcomes-based best practices for reporting results and advising patients for treatment.

Currently, in patients without significant brainstem compression, hydrocephalus, or ataxia, a conservative approach with annual MRI scans and physical examinations remains an acceptable option. Although the literature is not clear, larger tumors with extension into the cerebellopontine angle seem to grow more quickly and intracanalicular VSs grow more slowly. The interval for imaging may vary depending on tumor characteristics in individual patients.

Stereotactic radiosurgery is a valid option for tumors <3 cm in largest dimension. Tumors >3 cm are poorer candidates for radiotherapy and should be considered for surgical removal unless the patient is medically unstable. An intracanalicular VS that is <1.4 cm in greatest dimension with CSF between the lateral margin of the tumor and the labyrinth on T2-weighted MRI imaging, has a normal wave V on auditory brainstem response, has more than 70% WRS, and is in a patient with good health can be excised via MCF with hearing preservation approaching 70% and good facial nerve function in more than 96% of cases.

Longer term follow-up determines the longevity of hearing preservation in surgical and radiotherapy patients and catalogs late complications possible after both. Most surgical complications occur within the first weeks after resection, whereas radiotherapy complications may occur many years after treatment is complete. The difficult cases are found in patients with VS <3 cm. All three options are potentially useful, and unbiased provision of information to help them decide on a path of treatment is mandatory for proper informed consent.

At our institution, we use a multidisciplinary team that includes a neurotologist, neurosurgeon, and radiotherapist to explain the risks and benefits of each option for treatment. We give the patient a packet with selected articles, and a nurse-coordinator follows up to be sure all questions are relayed to the appropriate physician and answered. The process is designed to allow the patient a balanced presentation of information to foster a timely decision that ensures adequate informed consent.

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