Imaging of the Mastoid, Middle Ear, and Internal Auditory Canal After Surgery: What Every Radiologist Should Know

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KEYWORDS

• Postoperative imaging • Mastoidectomy
• Retrosigmoid approach • Translabyrinthine approach
• Tympanoplasty • Ossiculoplasty

Interpreting CT or MR imaging examinations performed on patients with a history of middle ear (ME), mastoid, or neurotologic surgery can be challenging. This is greatly simplified by knowing the surgical procedures used and the expected postoperative appearance. These are reviewed and illustrated. Knowing the normal postoperative appearance is the key to recognizing signs of recurrent disease.

MASTOID AND MIDDLE EAR

The goal of surgery in the ME and mastoid is the elimination of disease and, if possible, the preservation or restoration of normal function. The following approaches and procedures may be performed singularly or in combination.

Transcanal Approach

This approach is used for disease processes that can be adequately visualized in the external auditory canal (EAC) with the entire surgery performed through the EAC. Areas accessible include the EAC; central tympanic membrane (TM); the central ME; ossicles (to include the stapes); tympanic sinus; and portions of the facial recess and hypotympanum. Visualization of the anterior portions of the EAC near the TM and protympanum is limited. It is the preferred approach for EAC stenosis, exostosis, osteomas, myringitis, repair of central perforations, placement of pressure equalization tubes, and so forth. There may be little evidence of postoperative change in the EAC. A widened appearance to the EAC may be evident from drilling of bone at the level of the isthmus. Advantages include limited surgical dissection so postoperative pain is diminished. As a result, patients normally return to their presurgical functional status within 1 to 2 days.[1]

Postauricular Approach

An incision is made posterior to the ear, which is reflected anteriorly. The mastoid cortex and subjacent air cells are drilled away providing exposure to the ME and mastoid. It is the primary approach for many neurotologic procedures, such as the translabyrinthine, retrosigmoid, or retrolabyrinthine surgery. It provides the best exposure to the EAC and facilitates tympanoplasty when the transcanal approach cannot give the necessary visualization. It does require the use of a mastoid dressing that can be removed the next day. The

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postoperative recovery period is 4 to 5 days longer than that of the transcanal approach.[1]

Canaloplasty

This is a procedure enlarging the EAC facilitating immediate or future surgery by the transcanal approach. Postoperative change on imaging may be minimal in cases of EAC stenoses or could be extensive in patients with EAC atresia. The most common finding on CT is a "box-like" appearance to the EAC from bone drilling at the isthmus level and loss of bone anteriorly deep in the EAC secondary to "blue lining" giving the appearance of an EAC–temporomandibular joint "fistula" (**Fig. 1**).

Myringoplasty

Myringoplasty, also known as "type 1 tympanoplasty," refers to surgery performed on the eardrum. The aim of myringoplasty is to restore the normal functions of the TM.[2] The procedure may be simple, such as patching small perforations, or more complex to include lasering of chronic myringitis, removal of tympanosclerotic plaques, or reconstructing the entire TM with fascial grafts. The TM can be quite variable in appearance on CT, appearing so thin as to be barely perceptible or thickened focally and diffusely (**Figs. 1** and **2**). Blunting of the anterior angle is common, this "acute" angle formed by the TM and the adjacent anterior wall of the EAC. The posterior angle is more obtuse and less commonly blunted. The presence of a pressure equalization tube is common and should be noted.

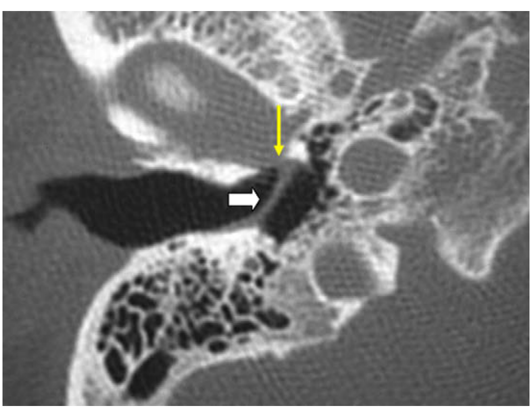

Fig. 1. Axial CT image from a patient following transcanal surgery. Note the bone defect in the anterior wall of the EAC (*yellow arrow*) just lateral to the annulus created during surgery. The TM (*white arrow*) is diffusely thickened either from scarring, grafting, or myringitis.

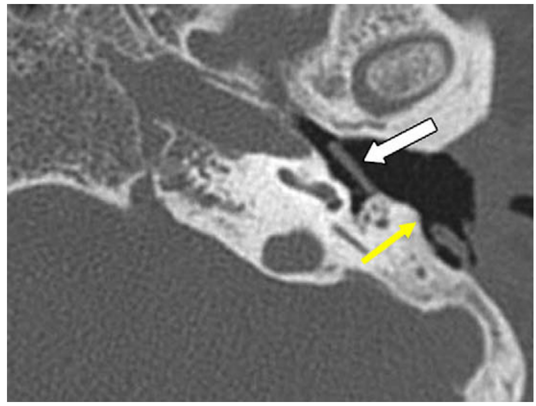

Fig. 2. Axial CT image in a patient after mastoidectomy with resection of the posterior canal wall (*yellow arrow*). There is nonspecific debris in the mastoid. Note the plate-like thickening of the TM (*white arrow*) resulting from a cartilage graft. Polymeric silicone deep to a TM placed to prevent collapse of the TM with subsequent scarring against the cochlear promontory results in a similar appearance.

Tympanoplasty

Although tympanoplasty and myringoplasty appear synonymous, the term "tympanoplasty" includes surgery involving the TM, ossicles, and associated ME disease.[3]

Ossiculoplasty

Surgery is performed to reconstruct the continuity of the ossicular connection between the TM and graft and the oval window. This reconstruction may involve repositioning of ossicles, usually the incus. The incus may be reshaped for proper fitting extending from the drum or the malleus to the stapes superstructure or footplate. Various forms of ossicular prosthesis are available extending from the TM and malleus handle to the stapes superstructure (partial ossicular prosthesis) or from eardrum and malleus handle to the stapes footplate (total ossicular prosthesis). The materials used are numerous and may be used in combination to prostheses made from calcified particles or hydroxyapatite; metal (eg, titanium); or plastic (plastipore). Stacked cartilage and bone cement may also be used. Specific prostheses are not discussed here because they are numerous. It is best to become familiar with the prosthesis used locally by one's surgeons.[4,5]

Stapedectomy

Stapedectomy specifically refers to complete removal of the stapes and footplate. There is significant variation, however, in "stapes surgery" in that a portion of the stapes superstructure may remain along with the footplate. The arch only may be removed and the footplate mobilized. A stapedotomy refers to a hole made in an immobile footplate through which prosthesis is placed.

There are five variations of tympanoplasty: (1) myringoplasty, ossicles intact; (2) TM perforation with diseased malleus or incus, most commonly the long process of the incus; (3) intact mobile stapes with destruction of malleus and incus; (4) destruction of ossicles (including stapes arch) with mobile footplate; and (5) destruction of ossicles as in type 4 with fixed footplate.

There are two important points regarding tympanoplasty-ossiculoplasty. First, when reading CT examinations, look for continuity of the ossicular chain–prosthesis from the TM and graft to the stapes or footplate (**Figs. 3** and **4**). Off-axis reformatted images are often valuable for this purpose.[6,7] The ossicular reconstructions used are quite variable and not always "straight line" or centered on the footplate (**Fig. 5**). The prosthesis may extend just deep to the footplate but should not extend deep into the vestibule (**Fig. 6**). It is important to remember that the apparent penetration below the footplate estimated by CT may be greater than the actual penetration anatomically.[8] Secondly, audiometry trumps imaging. That is to say, if there is significant conductive hearing loss, even with a "satisfactory" imaging appearance the patient likely needs revision surgery. Displacement of the prosthesis by only 0.1 to 0.2 mm is sufficient to cause conductive hearing loss and is difficult to detect by CT, particularly if there is artifact from metal.

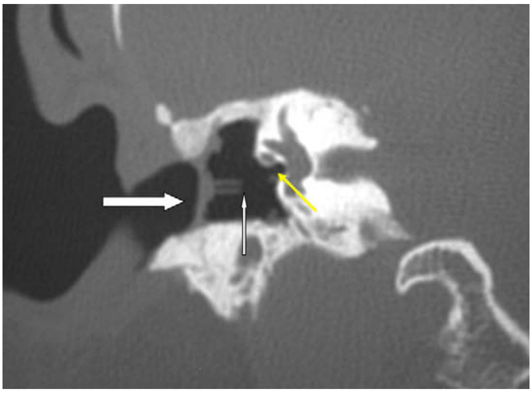

Fig. 4. Coronal CT image following mastoidectomy and tympanoplasty. Note the thickened lateralized TM/graft (*thick white arrow*) with the caudal portion in particular lateralized. The piston-type partial ossicular prosthesis (*thin white arrow*) has remained with the TM and has been pulled laterally away from the oval window/stapes (*yellow arrow*).

Mastoidectomy

The term "mastoidectomy" is a misnomer because the mastoid is not removed in its entirety. The term refers to removal of the mastoid cortex and portions of the mastoid that are diseased to include aircells, the mastoid tip, the sinodural angle, and tegmen.[9] Anterior and superiorly, the surgery may extend to the epitympanum and the anterior attic region with the geniculate ganglion the anterior most landmark. The size of the mastoidectomy defect is quite variable depending on the degree of mastoid pneumatization, extent of disease, and procedures performed. The smallest defects are seen in patients with sclerotic

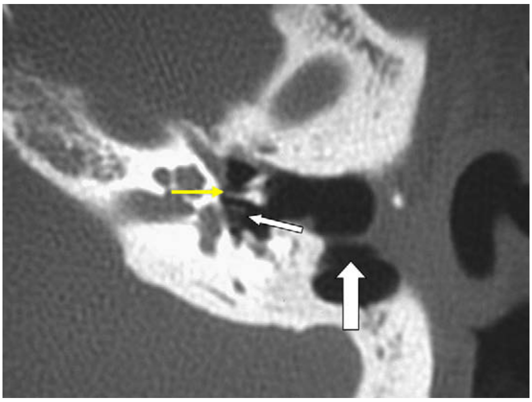

Fig. 3. Axial CT in a patient following open mastoidectomy and tympanoplasty. Note that the incus interposition (*yellow arrow*) no longer articulates with the stapes capitulum (*thin white arrow*). There is a weblike closure of the posterior canal defect from scarring (*thick white arrow*).

Fig. 5. Axial CT following tympanoplasty. Note continuity of the ossicular prosthesis from the malleus to the stapes footplate. Note the concavity in the prosthesis (*white arrow*) where it articulates with the malleus "handle," the medial end positioned posteriorly on the footplate (*yellow arrow*).

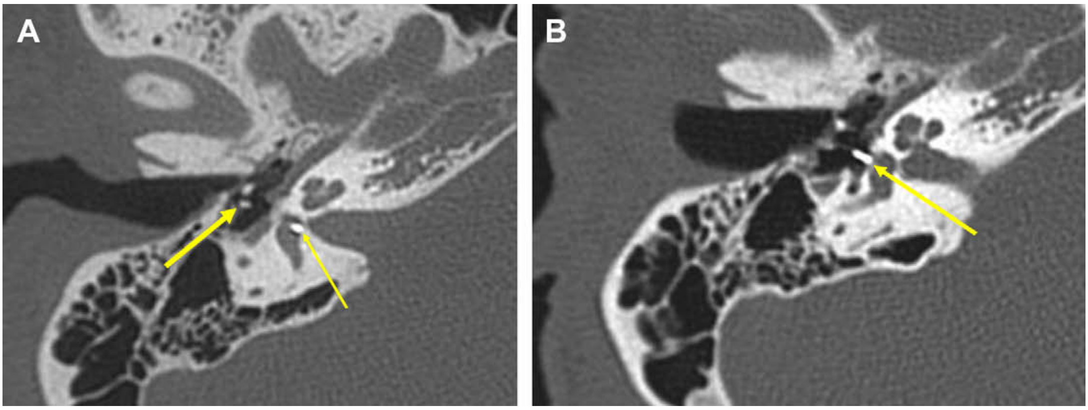

Fig. 6. (*A*) Axial CT image obtained following stapedectomy for otosclerosis. There was postoperative hearing loss and dizziness. Note loss of continuity between the long process of the incus (*thick arrow*) and the prostheses, which has fallen deep into the vestibule (*thin arrow*). (*B*) Image from the same patient following revision shows the prostheses now in good position extending from the long process of the incus to the stapes footplate (*arrow*).

mastoids and no mastoid disease, the surgery performed for placement of an endolymphatic shunt. Large defects are seen in patients with well-pneumatized mastoids with extensive disease requiring complete air cell exenteration.

Radiologists should make note of a very sclerotic mastoid, especially those with almost no aeration of the mastoid antrum. In these patients the transmastoid approach can be dangerous because it is difficult to delineate the lateral semicircular canal and second genu of the facial nerve, both of which may be injured at the time of surgery. On postoperative scans in these patients it is common to see small surgical defects in bone over the inferior temporal region above the tegmen, created during attempts to enter the mastoid.

The posterior wall of the EAC may be left intact or resected. When intact, this is commonly referred to as a “closed” mastoidectomy or mastoidectomy with intact posterior canal wall (Fig. 7). If the posterior canal wall is removed, this is referred to as mastoidectomy with takedown of the posterior canal wall or “open” mastoidectomy (see Fig 3; Fig. 8). On rare occasions, the posterior canal wall may be removed, allowing wider access to the ME and mastoid, then replaced or reconstructed. The choice of performing an intact canal wall versus canal wall down mastoidectomy is dependent on whether future mastoid surgery is anticipated because these procedures are usually performed in patients with cholesteama. With the canal wall down mastoidectomy and meatoplasty, additional surgery is required only in 10% of patients.[10] With the intact canal wall mastoidectomy, a “second look” mastoidectomy is required because the recurrence of cholesteatoma is approximately 20% to 30%, even if the surgeon believes all cholesteatoma was removed grossly during the initial operation. A common site for recurrent cholesteatoma is around the oval window because complete surgical removal of cholesteatoma around the facial nerve or that adherent to the mobile stapes footplate can result in significant facial nerve paralysis or significant sensorineural hearing loss, tinnitus, and dizziness with vertigo.

Facial Recess Approach

This is performed with an intact canal wall mastoidectomy with an opening made just medial to the chorda tympanic nerve and just lateral to the mastoid segment of the facial nerve. This is used

Fig. 7. Axial CT following mastoidectomy with intact posterior canal wall (*bone anterior to white arrow*). Note the polymeric silicone (*white arrow*) extending through the facial recess into the mesotympanum. The facial recess approach is lateral to the mastoid segment of the facial nerve (*yellow arrow*) and medial to the chorda tympani.

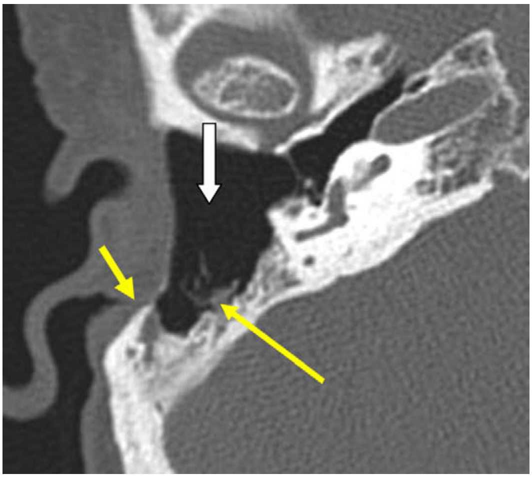

Fig. 8. Axial CT after open tympanomastoidectomy. Note absence of the posterior canal wall (*white arrow*). Debris or residual inflammatory disease is commonly seen within residual air cells or adjacent to irregular bony margins with "overhanging" edges (*yellow arrows*). Note that the remainder of the mastoid is well aerated and free of disease.

for cochlear implant surgery and also as a means to removal of disease in the facial recess (see **Fig. 7**).

Atticotomy

An atticotomy refers to removal of the lateral wall of the epitympanum. This is not typically described separately if the patient is posttympanoplasty or mastoidectomy because the lateral wall may be removed as part of the mastoid procedure. On occasion, however, atticotomy is performed alone and this should be noted. The mastoid cortex and posterior canal wall appear normal. An atticotomy is best visualized on coronal images.

Postoperative Imaging

Patients are not typically imaged acutely unless complications are suspected. Immediately after surgery, it is normal to see soft tissue swelling, absorbable gelatin sponge, mastoid cell opacification, and fluid levels (**Fig. 9**). For intracranial complications, such as stroke, dural sinus thrombosis, abscess, or cerebritis, MR imaging, MR angiography, and MR venography are preferred. CT has a limited role but may be useful if there is clinical concern for cerebrospinal fluid (CSF) leak, facial nerve injury, or labyrinthine fistula.

In the more typical case, patients are imaged months or years after surgery because of recurrent hearing loss, inflammatory disease, or cholesteatoma. When imaging is normal, there is good

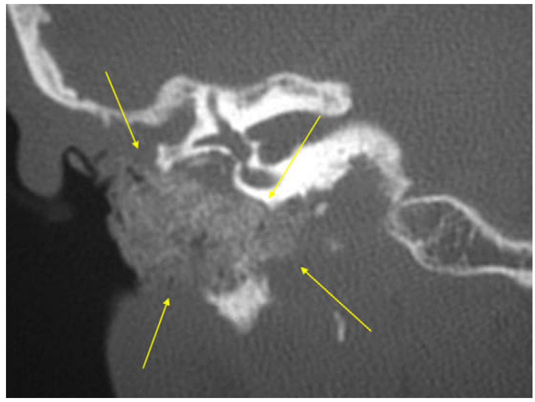

Fig. 9. Coronal CT image shortly after surgery shows the appearance of absorbable gelatin sponge packing in the middle ear and mastoid (*material between arrows*).

aeration of the ME and mastoid without fluid. The EAC has normal margins without soft tissue thickening. The TM appears thin and intact. The ossicles or prostheses are intact and in continuity. The otic capsule appears normal without erosion, fistula, or pneumolabyrinth.

Postoperative CT imaging after mastoidectomy is very challenging with regard to differentiating acute disease versus chronic, nonactive disease. Mild soft tissue thickening with otherwise good aeration is commonly seen and likely represents scar or granulation tissue. More extensive thickening, especially when accompanied by fluid or complete opacification of the ME and mastoid, is more likely to represent active disease. It is common to see soft tissue opacification of residual mastoid air cells and around the margins of the mastoid defect, especially if the margins are irregular with "overhanging" edges (see **Fig. 8**; **Fig. 10**).[11] Patients with eustachian tube dysfunction often have retained fluid. Because of this, it is important to recognize that "mastoiditis" is a clinical diagnosis supported by imaging findings.

Recurrent cholesteatoma may be difficult to diagnose with certainty by CT, especially so when it is surrounded by inflammatory disease or fluid. It is important to know that after surgery, cholesteatoma may recur in the EAC, TM, ME, or mastoid. Progressive bone erosion should always raise concern but this requires a baseline postoperative examination (rarely indicated) to differentiate from preoperative erosion or operative drilling. A focal round soft tissue mass is suspicious but uncommon. On occasion, a mass has irregular air-filled interstices from shedding of keratin debris. Rarely, the cholesteatoma contents are shed or removed with only the peripheral

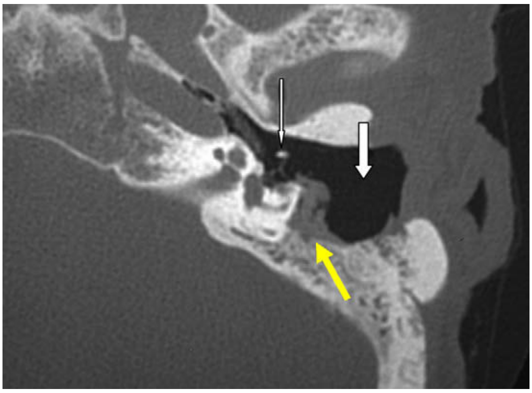

Fig. 10. Axial CT following open tympanomastoidectomy. Note absence of the posterior canal wall (*thick white arrow*). Debris in the mastoid (*yellow arrow*) is indeterminate and could reflect cholesteatoma or inflammatory debris. The open cavity facilitates direct inspection for clarification. The malleus "handle" remains (*thin white arrow*) and there is a type 3 tympanoplasty, the TM articulating directly with the stapes capitulum.

matrix remaining, a so-called "thin wall" cholesteatoma.

If a mass is noted on the postoperative CT, differential considerations other than cholesteatoma include encephalocele-meningocele and cholesterol granuloma (see Fig. 10; Fig. 11). It is important to inspect the tegmen to be sure it is intact (Fig. 12). MR imaging without and with contrast is useful in differentiating encephalocele, inflammatory disease, cholesteatoma, and cholesterol granuloma but is infrequently obtained because it seldom alters clinical management.

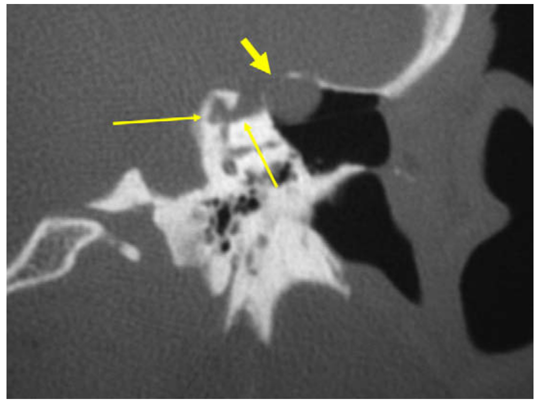

Fig. 12. Coronal CT image in patient following mastoid surgery for cholesteatoma. The patient experienced hearing loss and dizziness. Note the bone erosion involving the posterior semicircular canal (*thin arrows*). There is a contiguous round mass along the caudal margin of the dehiscent tegmen (*thick arrow*). The appearance of the round mass is consistent with cholesteatoma, cholesterol granuloma, meningocele, or encephalocele.

Hazy ill-defined new bone formation is occasionally seen about the margins of the mastoid and ME. When seen, this should suggest active inflammation (Fig. 13).

Tympanosclerosis refers to fibrous tissue, calcification, or new bone formation in response to chronic otitis media (Fig. 14).[11] It is important to note the presence and extent of tympanosclerosis. It is a cause of conductive hearing loss poorly visualized by the surgeon. Tympanosclerotic fixation of the malleus and incus occurs in the lateral

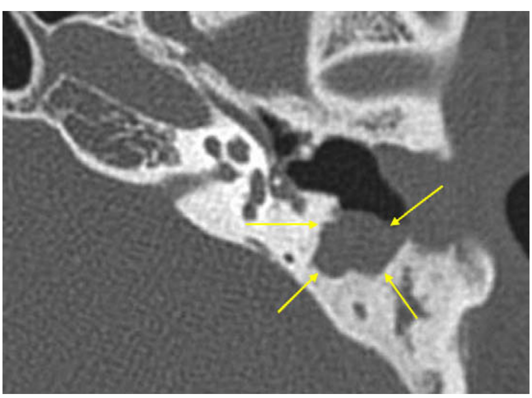

Fig. 11. Axial CT in patient following open mastoid surgery. The TM is collapsed and the middle ear cleft opaque from inflammatory disease. Note the soft tissue mass with rounded margins (*yellow arrows*). The appearance along the medial margin raised concern for bone erosion from cholesteatoma. Surgery, however, revealed cholesterol granuloma.

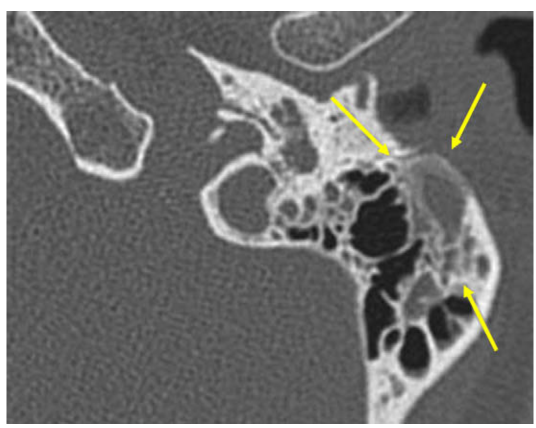

Fig. 13. Axial CT image acquired from a patient experiencing otalgia. Note the opacification of air cells and the partial obliteration of air cells by new bone formation (*area surrounded by arrows*). New bone formation strongly suggests persistent inflammation (periostitis).

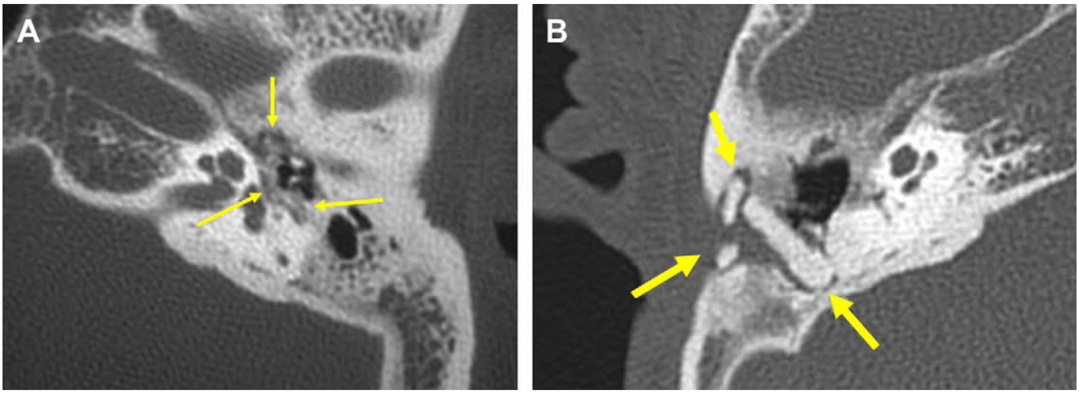

Fig. 14. (*A*) Axial CT demonstrates mixed soft tissue and amorphous calcific change in the middle ear surrounding the ossicles and filling the tympanic sinus (*arrows*). Tympanosclerosis with ossicular fixation proved by subsequent surgery. (*B*) Tympanosclerosis should not be confused with attempts to obliterate a mastoid cavity with bone (*arrows*) or hydroxyapatite.

epitympanum or involves the stapes footplate. The scarring obliterates normal ME landmarks that the surgeon relies on for orientation. This increases morbidity and results in longer operative times.

INTERNAL AUDITORY CANAL AND CEREBELLOPONTINE ANGLE

Acoustic schwannomas (AS) are the most commonly encountered extra-axial internal auditory canal (IAC)–cerebellopontine angle (CPA) tumors followed in frequency by meningiomas. These lesions along with epidermoids account for over 90% of CPA tumors. Over 60 other tumors account for the other 10%. Tumors may present as a purely intracanalicular lesion (AS, meningioma, facial neuroma, hemangioma, lipoma, and so forth); within the CPA; or in both regions.[12,13]

There are three options available to the clinician once a tumor is diagnosed: (1) observation, (2) radiotherapy, or (3) surgery. This article does not discuss radiotherapy or imaging algorithms for observation. There are three standard approaches to the IAC-CPA region with variations described in the literature. The approach used depends more on the expertise and training of the surgeon rather than the size or location of the lesion.

Retrosigmoid Approach

The retrosigmoid approach is a commonly used technique used for removal of tumors in the IAC and CPA. It is a team approach. The neurosurgeon performs the craniotomy with cerebellar retraction and removal of the intracranial portion of the tumor with the neurotologist performing any required drilling around the IAC with subsequent tumor removal in the IAC. Advantages are that tumors of any size can be removed. There is excellent exposure to the CPA, lower cranial nerves, brainstem, and cerebellum. CN7 and CN8 are easily identified. After drilling of the IAC and removal of the tumor in the temporal bone–IAC, the most difficult part of the surgery is the removal of the tumor around the porous of the IAC because the facial nerve is often splayed and adherent to the capsule of the tumor.[14,15]

Monitoring of the facial nerve (CN7) significantly reduces the risk of facial paresis and paralysis. The use of intraoperative auditory nerve monitoring also reduces the risk of losing the hearing in small acoustic tumors, usually those confined in the IAC.[16,17]

A weakness of this approach is that the lateral end of the IAC cannot be exposed without potentially damaging hearing. The lateral 2 to 3 mm of the IAC cannot be drilled out without drilling into the posterior semicircular canal or into the vestibule, both of which could result in significant sensorineural hearing loss. This is not a concern if there is profound hearing loss preoperatively. If hearing is to be spared, then the lateral 2 to 3 mm of the IAC is not drilled and tumor is left in this area. If the labyrinth is violated, preservation of the lateral semicircular canal is most critical. Careful removal of the superior and posterior semicircular canals can be performed without significant loss of hearing over 80% of the time (transcrusal approach).[18,19]

Translabyrinthine Approach

This approach is used if there is no useful hearing on the side of the tumor and there is no concern for hearing preservation. Following an extended mastoidectomy the labyrinth is drilled away with removal of all three of the semicircular canals and opening into the vestibule. The facial nerve can be followed directly from the second genu in the mastoid to the geniculate and points the way into the IAC. This approach was developed by Drs. William House and William Hitselberger for identification of the facial nerve.[20] When developed, there was no intraoperative facial nerve monitoring available. The bone over the sigmoid sinus is removed or islanded to allow placement of a retractor and better exposure in the CPA. Subsequently, bone of the IAC is removed with tumor in the IAC and CPA exposed and resected. The size of the defect depends on the exposure required. The cochlea is commonly spared.

This approach can be used for tumors of all sizes, exposure facilitated by extensive drilling of bone lateral to the tumor. Wide access nearly to the midline can be achieved, extending anteriorly to the carotid and posteriorly to the sinodural plate. After tumor removal, the medial defect is covered with fascia and then filled laterally with fat. There is complete hearing loss after surgery. Advantages include early identification of CN7 lateral to the tumor (in the case of AS) with low incidence of tumor recurrence.[21] The incidence of headache is low with the incidence of CSF leak similar to that of the posterior fossa approach (about 5%). Disadvantages include lack of visualization of the brainstem until the tumor is resected and limited familiarity of the approach to neurosurgeons should complications arise.

Middle Cranial Fossa Approach

Following a subtemporal craniotomy, the dura along the floor of the middle cranial fossa is elevated along with the temporal lobe extradurally. The dura covering the middle cranial fossa floor is elevated medially to the petrous ridge, usually

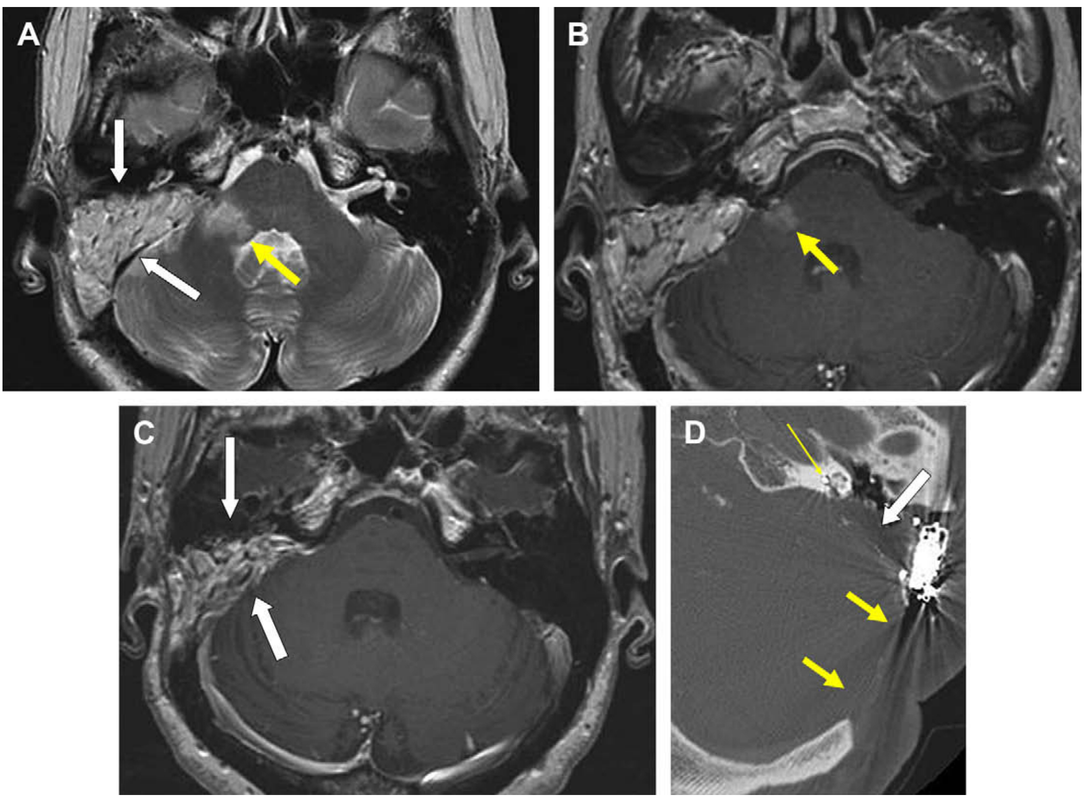

Fig. 15. (*A*) Axial T2-weighted image following translabyrinthine resection of AS. Note the mixed fat and soft tissue graft (*white arrows*) filling the surgical defect, triangular in configuration, apex toward the IAC. There is increased T2 signal in the right cerebellar peduncle from ischemia (*yellow arrow*). (*B*) Axial T1-weighted image at the same level following contrast administration demonstrates enhancement in the right cerebellar peduncle (*arrow*). Findings were consistent with a small infarct postoperatively. (*C*) Axial postcontrast T1-weighted image in same patient 6 months later shows a diminishing volume of graft material in the surgical defect (*arrows*). (*D*) Axial CT in a different patient following combined retrosigmoid (*thick yellow arrows*) and translabyrinthine surgery (*white arrow*) with subsequent cochlear implantation (*thin yellow arrow*). Note the wide exposure of the cerebellopontine angle that can be achieved extending anteriorly toward the clivus.

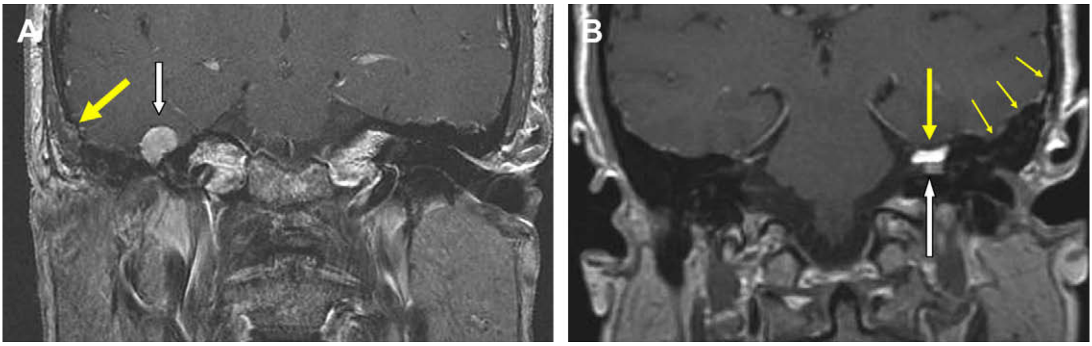

Fig. 16. (*A*) Coronal T1-weighted postcontrast image demonstrates subtle findings consistent with middle cranial fossa surgery. There is a small calvarial defect (*yellow arrow*) with moderate atrophy of the temporalis muscle. There is minimal change to the floor of the middle cranial fossa. Note the enhancing mass (*white arrow*), a recurrent facial nerve tumor at the geniculate ganglion level. (*B*) Coronal postcontrast T1-weighted image in a patient after middle cranial fossa surgery for acoustic schwannoma. Note fat filling the defect in the roof of the IAC (*large yellow arrow*). Postoperative change along the floor of the middle cranial fossa is minimal (*small yellow arrows*). The craniotomy defect is small and there is temporalis muscle atrophy. Note enhancement in the IAC (*white arrow*) from residual tumor (proved subsequent to this examination by interval growth).

from a posterior to anterior manner with the superficial petrosal sinus as the medial landmark.

The anterior limit is the middle meningeal artery. Care is taken during dural elevation from posterior to anterior looking for dehiscence of the greater superficial petrosal nerve (40%–50%) or the superior semicircular canal (5% or more). This could result in facial nerve injury or hearing loss.[1,22] The location of the IAC can be found by locating the arcuate eminence (beneath which is the superior semicircular canal) or by locating the superior petrosal nerve, which leads to the geniculate ganglion of the facial nerve. The IAC superiorly is drilled from medial to lateral with special care laterally so as not to violate the superior semicircular canal, labyrinthine portion of the facial nerve, and the basal turn of the cochlea.[23] These structures are the lateral limits of the exposure and are within 2 to 3 mm of each other. The labyrinthine portion of the facial nerve comes laterally from the lateral end of the IAC to the geniculate ganglion at a 45-degree angle, is not covered by dura, and is quite small. This is the portion of the facial nerve most commonly injured during middle fossa removal of an acoustic tumor.

Advantages include better hearing preservation, low incidence of CSF leak and headache, excellent exposure to the lateral portion of the IAC, and less

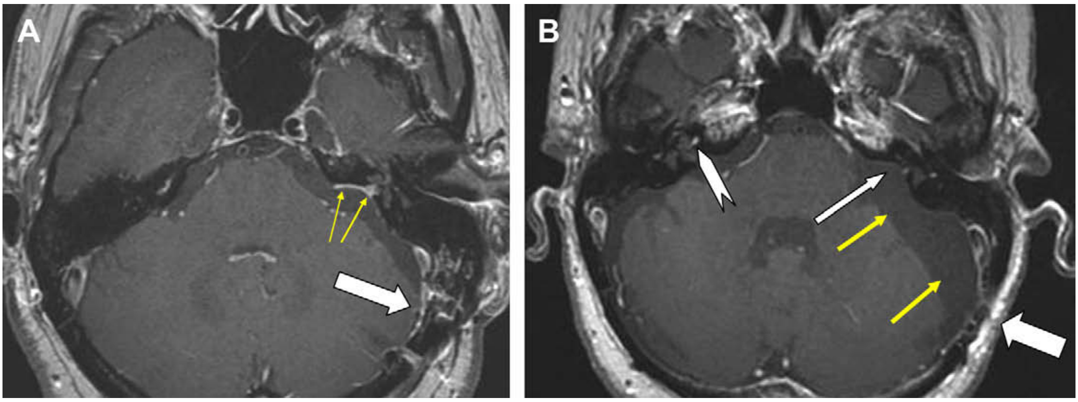

Fig. 17. (*A*) Axial postcontrast T1-weighted image demonstrates typical postoperative change following AS surgery by the retrosigmoid (RS) approach. Note the craniotomy defect (*white arrow*), prominence of CSF along the left lateral margin of the cerebellum, and fine linear enhancement along the medial margin of the widened porous acousticus (*yellow arrows*). (*B*) Axial postcontrast T1-weighted image in a different patient following AS resection by RS approach demonstrates a craniotomy defect (*thick white arrow*), mild cerebellar encephalomalacia (*yellow arrows*), and no appreciable residual enhancement in or about the widened IAC (*thin white arrow*). Incidental note is made of a cochlear schwannoma on the right (*white chevron*).

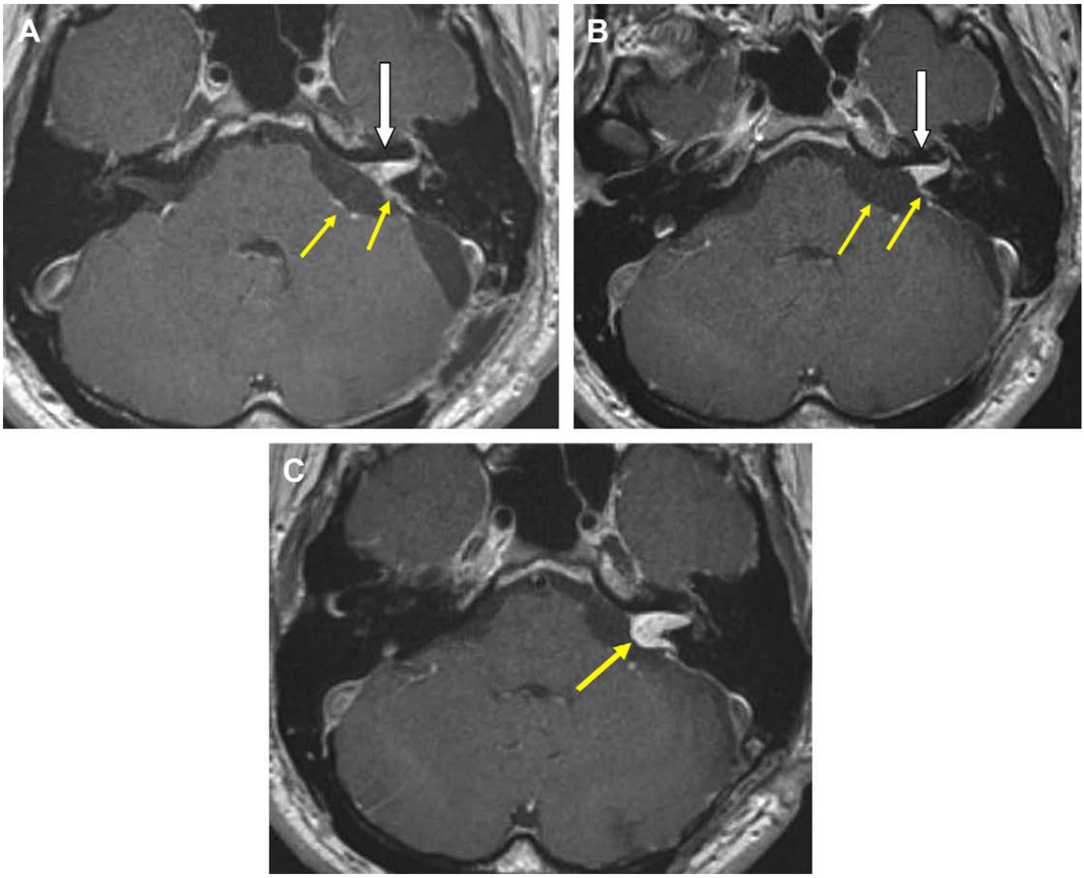

Fig. 18. (*A*) Axial postcontrast T1-weighted image 6 months following surgery (RS approach for AS) demonstrates solid enhancing tissue filling the IAC (*white arrow*). The appearance is indeterminate regarding postoperative change, graft, or tumor. Note thinner linear enhancement along the brainstem and posterior to the widened porous (*yellow arrows*). (*B*) Same patient but 12 months following surgery. The linear enhancement has diminished (*yellow arrows*). The enhancing tissue filling the IAC (*white arrow*) appears slightly smaller, the medial margin now concave (previously flat). Findings were interpreted as postoperative change without tumor. (*C*) Same patient 24 months after surgery. Note that the tissue filling the IAC now has a convex medial margin and is beginning to bulge into the CPA (*arrow*). Findings consistent with progressive residual tumor.

meningeal irritation. The dura does not need to be opened because this protects the temporal lobe from further injury. Disadvantages are a higher incidence of CN7 injury, occasional memory disturbances and aphasia, and seizures.[24–26]

Because of limited access to the CPA, the middle cranial fossa approach is usually used for AS less than 2 cm in size, preferably those confined to the IAC. This approach may be combined with the RS approach for large tumors and is useful for lesions involving the cephalad portion of the petrous apex or junction of CN7 with the greater superficial petrosal nerve and clival region.

Complications

Complications related to AS resection are uncommon but include hemorrhage, stroke,

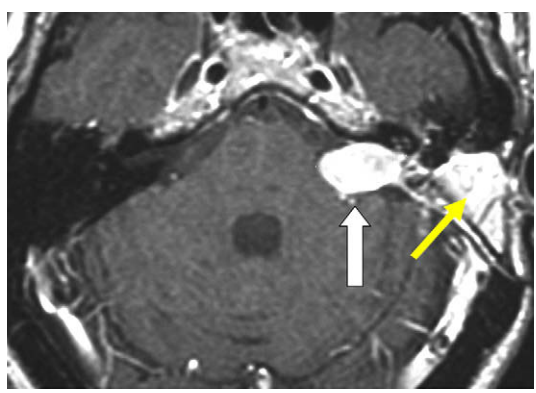

Fig. 19. Axial postcontrast T1-weighted image obtained 4 years after TL resection of AS. Note the recurrent tumor with early brainstem compression (*white arrow*). Fat fills the translabyrinthine defect (*yellow arrow*).

vascular injury, infection and meningitis, brainstem and cranial nerve injury, CSF leak, and death.[27–30] The incidence of cranial nerve injury has diminished with the availability of real-time monitoring. CN7 in particular can be difficult to identify secondary to displacement and effacement, adhesions, or involvement by the tumor.

CSF leaks may occur with all three approaches, especially if a combined approach is used. Air cells opened during the craniotomy or drilling around the IAC need to be sealed by bone wax or fascia.[31] Leaks may resolve with conservative measures, such as elevation of the head and lumbar drains. Occasionally surgery is required, especially if the patient experiences meningitis. Over a 2- to 3-month basis, in patients with CSF leak, meningitis occurs 50% of the time.[32] Perioperative meningeal irritation is common and managed conservatively.[26]

Postoperative Imaging

The need for postoperative imaging is dictated by the tumor pathology and completeness of resection. For benign lesions, a baseline postoperative examination 6 to 12 months after surgery is useful. Some advocate follow-up imaging (for AS) 5 years after surgery, assuming the patient is asymptomatic. Unfortunately, because the ear may be "dead," the first sign of recurrence could be brainstem compression. If imaged 1 to 2 years after surgery it may be difficult to differentiate postoperative change from recurrent or residual disease without a baseline examination. This commonly leads to unnecessary multiple additional follow-up examinations to clarify and creates significant anxiety for patients.

The baseline examination after uncomplicated surgery may be confined to the posterior fossa (unless otherwise indicated). For these

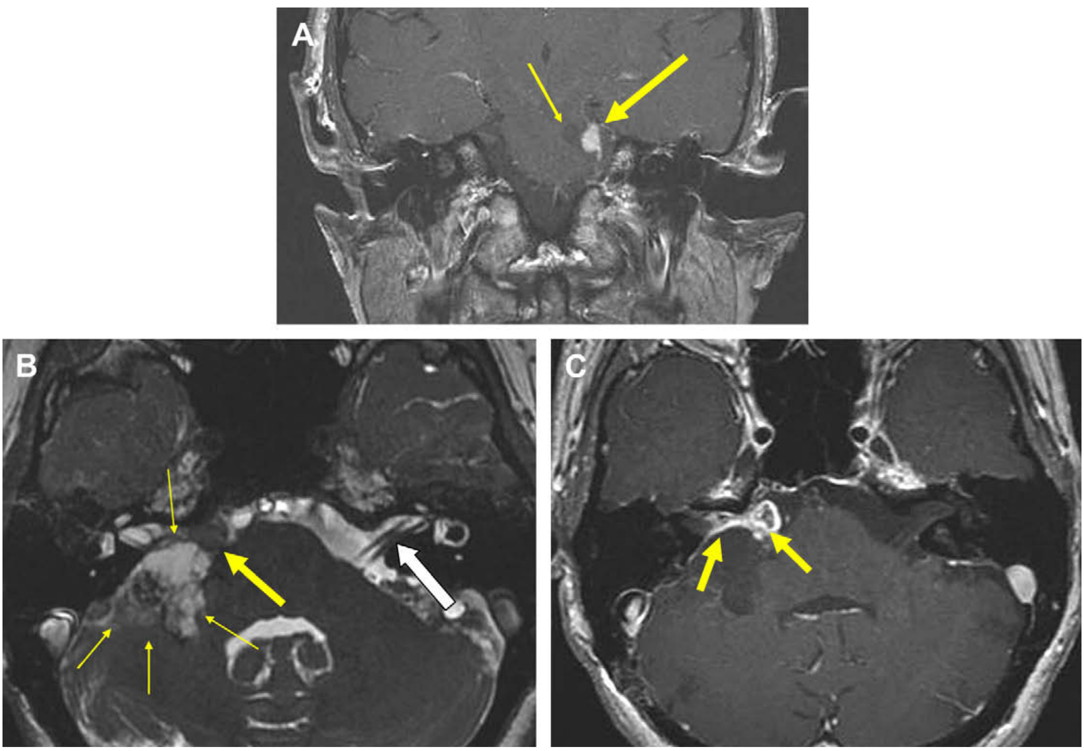

Fig. 20. (*A*) Coronal postcontrast T1-weighted image in patient following RS surgery for AN removal. There is an enhancing mass (*thick yellow arrow*) adherent to the prepontine segment of the trigeminal nerve. Note brainstem encephalomalacia adjacent (*thin arrow*). The tumor was left intentionally to spare CN5 and had been stable in size for 36 months. (*B*) Axial T2* FIESTA image during a 6-month baseline postoperative examination. Tumor was left adherent to CN7 at the time of surgery to spare the nerve. Note the cerebellar encephalomalacia, hemorrhagic residua, and stranding of soft tissue interspersed with CSF along the right cerebellar margin (*thin yellow arrows*). There is soft tissue anteriorly (*thick yellow arrow*) with poor definition of CN7 and CN8. Note the normal appearance of CN7 and CN8 on the left (*white arrow*). (*C*) Axial T1 postcontrast image at the same level and time as *B*. There is enhancement of residual tumor (*arrows*) extending from the brainstem to the widened porous. CN7 cannot be clearly identified.

examinations a suggested protocol is axial 2-mm T1-weighted images precontrast and postcontrast along with thin T2* weighted images (FIESTA, CISS, and so forth). Diffusion sequences are useful if the original tumor was an epidermoid. Precontrast images are useful for definition of fat and hemorrhage. Fat saturation sequences are useful but uneven fat saturation is common and pathology may be missed. On subsequent examinations, only the T2* and postcontrast T1 images need to be obtained. These limited examinations save a considerable amount of money over the patient's lifetime as compared with complete brain and IAC examinations.

CT is less useful for following patients after AS surgery but excludes large tumors in patients where MR imaging is contraindicated. For bone-destroying tumors, subtle destruction of additional bone demonstrated by CT is more sensitive for detection of disease progression than is change of tumor size detected by MR imaging.

Following translabyrinthine surgery, there is a roughly triangular defect in the mastoid extending anteriorly through the labyrinth, the apex directed toward the IAC.[33] Portions of the labyrinth may remain anteriorly. Signal in the residual labyrinth may be altered by blood, fibrosis, or ossification. On MR imaging, the fat graft filling the defect is the predominant feature early on but may resorb or atrophy over time (**Fig. 15**). Fascia closing the dural defect is typically poorly visualized.

Following a middle fossa approach, postoperative changes may be minimal and missed on axial images if the craniotomy is not noted.[33] Coronal imaging facilitates visualization of the surgical approach and postoperative alterations to the temporal lobe and roof of the IAC. There is a small volume of tissue filling the defect in the roof of the IAC, which may partially protrude into the IAC (**Fig. 16**). The IAC-CPA may look normal. Dural thickening or hyperemia may be seen in the portion of the dura elevated along the approach. The CPA may appear widened, especially if the tumor was large and the brainstem compressed.

Following the RS approach, the craniotomy defect may be very subtle and is often the only sign of surgery. The cerebellum commonly demonstrates variable encephalomalacia from retraction-infarction but may appear normal.[34] The CPA appears wide if the tumor was large and the brainstem compressed. If the posterior wall of the IAC was drilled, there is a widened appearance to the porous acousticus. Alteration of labyrinthine signal (blood, fibrosis, ossification, granulation tissue) indicates transgression of the semicircular canals, most commonly the posterior canal.

With all three approaches scar, fascia, and dura may be rather inapparent or enhance in a linear or "whorled" fashion, the extent depending on the tumor size, dural repair, and so forth (**Fig. 17**).[35–37] This is commonly confined to the region of the IAC but may be found anywhere in the CPA. The prominence of enhancement decreases over time and then stabilizes after 12 to 24 months. Round or globular enhancement should be viewed

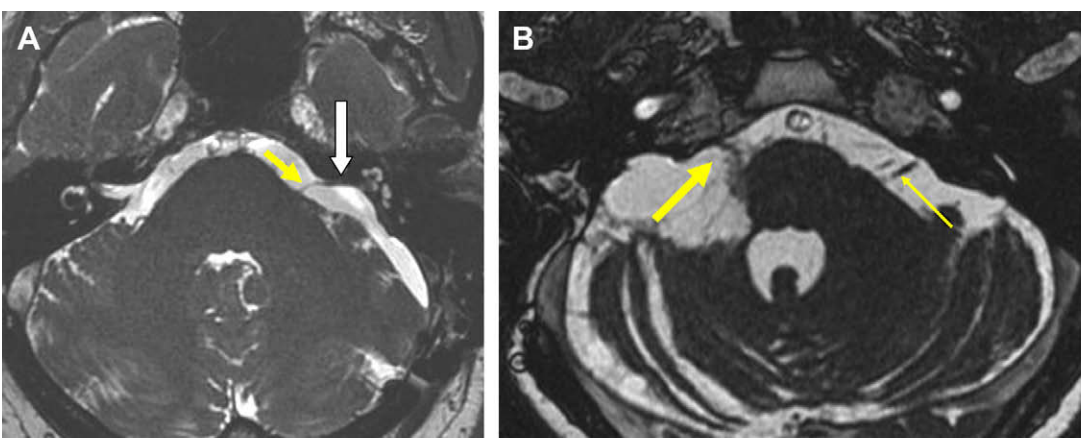

Fig. 21. (*A*) Axial T2* FIESTA image in patient following RS surgery for AN shows a stretched, anteriorly displaced facial nerve (*yellow arrow*). The nerve is adherent to the anterior wall of the IAC (*white arrow*) and cannot be visualized as a separate structure from the anterior wall of the porous acousticus to the fundus. (*B*) Axial T2* FIESTA image in patient following RS surgery for AN demonstrates a shaggy irregular linear structure that was interpreted as being CN7 (*thick arrow*). It is adherent to the lateral wall of the CPA anterior to the porous and anterior wall of the IAC. It could not be visualized as a separate structure. Note the normal appearing CN7 and CN8 on the left (*thin arrow*).

with suspicion for residual or recurrent disease (Figs. 18 and 19).[38] If muscle was used to fill the defect, this may mimic enhancing tumor and should be correlated clinically.

The tumor may be incompletely removed for clinical reasons, such as involvement of cranial nerves or critical vascular structures (Fig. 20). With "near total" resection, the residual tumor may be devitalized by laser or cautery. These patients are followed with more frequent imaging following the 6-month baseline examination for evaluation of the growth potential (if any) of the residua. On rare occasions patients have progressive disease after both surgery and radiotherapy.

The appearance of CN7 after surgery is quite variable (authors observations). With small tumors, CN7 appears normal and can be traced from the brainstem to the fundus of the IAC. With larger tumors, CN7 is stretched and displaced, typically anteriorly and either superiorly or inferiorly. In the CPA, the nerve has a bowed appearance, concave posteriorly (Fig. 21). It may appear relatively normal in thickness or thickened either from adhesions or residual tumor (subtotal resection). After removing a large tumor, it is unusual for the nerve to be traceable in the IAC from the porous to the fundus, this likely related to adhesions and adherence to the anterior wall. This makes evaluation of CN7 difficult in those patients who have postoperative paralysis. That is to say, lack of definitive visualization of CN7 is common and does not necessarily indicate injury or transection.

Finally, if the CPA appears small with the brainstem deviated toward the side of surgery, this suggests CN7 has been repaired or grafted. This is likely from postoperative scarring related to packing placed around the graft to protect the nerve or graft anastomosis.

SUMMARY

Interpreting CT and MR images in patients who have undergone surgery in the ME, mastoid, or CPA-IAC can be challenging. Understanding the surgical options and normal postoperative appearance in these regions facilitates rendering an interpretation that is both succinct and accurate.

REFERENCES

1. Jackler RK, Brackman DE. Neurotology. 2nd edition. Philadelphia: Elsevier/Mosby; 2005. p. 727–82.
2. Fisch U. Myringoplasty . Tympanoplasty, mastoidectomy and stapes surgery. New York: Thieme; 1994. 10–40.
3. Fisch U. Tympanoplasty . Tympanoplasty, mastoidectomy and stapes surgery. New York: Thieme; 1994. 2–7.
4. Fisch U. Ossiculoplasty . Tympanoplasty, mastoidectomy and stapes surgery. New York: Thieme; 1994. 44–116.
5. Stone JA, Mukherji SK, Jewett BS, et al. CT evaluation of prosthetic ossicular reconstruction procedures. Radiographics 2000;20:593–605.
6. Lane JI, Witte RJ, Driscoll CL, et al. Imaging microscopy of the middle and inner ear: CT microscopy. Clin Anat 2004;17(8):607–12.
7. Fatterpekar GM, Doshi AH, Dugar M, et al. Role of 3D CT in the evaluation of the temporal bone. Radiographics 2006;26:117–32.
8. Hahn Y, Diaz R, Hartman J, et al. Assessing stapes piston position using computed tomography: a cadaveric study. Otol Neurotol 2009;30:223–30.
9. Fisch U. Mastoidectomy . Tympanoplasty, mastoidectomy and stapes surgery. New York: Thieme; 1994. 146–197.
10. Brackman DE, Shelton C, Arriaga MA. Otologic surgery. Philadelphia: WB Saunders; 1994. p. 211–41.
11. Schuknecht HF. Pathology of the ear. 2nd edition. Malvern (PA): Lea and Febiger; 1993. p. 563–74, p. 210–11.
12. Eisenman D, Voight EP, Selesnick SH. Unusual tumors of the internal auditory canal and cerebellopontine angle. In: Jackler RK, Driscoll CLW, editors. Tumors of the ear and temporal bone. Philadelphia: Lippincott Williams and Wilkins; 2000. p. 236–75.
13. Driscoll CLW. Vestibular schwannoma (acoustic neuroma). In: Jackler RK, Driscoll CLW, editors. Tumors of the ear and temporal bone. Philadelphia: Lippincott Williams and Wilkins; 2000. p. 172–218.
14. Mazzoni A, Calabrese V, Danesi G. A modified retrosigmoid approach for direct exposure of the fundus of the internal auditory canal for hearing preservation in acoustic neuroma surgery. Am J Otol 2000; 21(1):98–109.
15. Bennett M, Haynes D. Surgical approaches and complications in the removal of vestibular schwannomas. Otolaryngol Clin North Am 2007;40: 589–609.
16. Moriyama T, Fukushima T, Asaoka K, et al. Hearing preservation in acoustic neuroma surgery: importance of adhesions between the cochlear nerve and the tumor. J Neurosurg 2002;97(2):337–40.
17. Yingling CD, Gardi JN. Intraoperative monitoring of facial and cochlear nerves during acoustic neuroma surgery. Otolaryngol Clin North Am 1992;25(2):413–48.
18. Horgan MA, Delashaw JB, Schwartz MS, et al. Transcrusal approach to the petroclival region with hearing preservation. J Neurosurg 2001;94:660–6.
19. Kaylie DM, Horgan MA, Delashaw JB, et al. Hearing preservation with the transcrural approach to the petroclival region. Otol Neurotol 2004;25:594–8.

20. House WF, Luetje CM. Acoustic tumors: management. vol 2. Baltimore (MD): Univ. Park Press; 1979. p. 43–88.
21. Schmerber S, Palombi O, Boubagra K, et al. Long-term control of vestibular schwannoma after a translabyrinthine complete removal. [Comparative study. Journal article]. Neurosurgery 2005;57(4):693–8 [discussion: 693–8].
22. House WF, Luetje CM. Acoustic tumors: management. vol 2. Baltimore (MD): Univ. Park Press; 1979. p. 15–42.
23. Driscoll CL, Jackler RK, Pitts LH, et al. Is the entire fundus of the internal auditory canal visible during the middle fossa approach for acoustic neuroma? Am J Otol 2000;21(3):382–8.
24. Schick B, Greess H, Gill S, et al. Magnetic resonance imaging and neuropsychological testing after middle fossa vestibular schwannoma surgery. Otol Neurotol 2008;29(1):39–45.
25. Bryce GE, Nedzelski JM, Rowed DW, et al. Cerebrospinal fluid leaks and meningitis in acoustic neuroma surgery. Otolaryngol Head Neck Surg 1991;104(1):81–7.
26. Ross D, Rosegay H, Pons V. Differentiation of aseptic and bacterial meningitis in postoperative neurosurgical patients. J Neurosurg 1988;69(5):669–74.
27. Hegarty JL, Jackler RK, Rigby PL, et al. Distal anterior inferior cerebellar artery syndrome after acoustic neuroma surgery. Otol Neurotol 2002;23(4):560–71.
28. Mass SC, Wiet RJ, Dinces E. Complications of the translabyrinthine approach for the removal of acoustic neuromas. Arch Otolaryngol Head Neck Surg 1999;125(7):801–4.
29. Ajolloveyan M, Doust B, Atlas MD, et al. Pneumocephalus after acoustic neuroma surgery. Am J Otol 1998;19(6):824–7.
30. Falcioni M, Romano G, Aggarwal N, et al. Cerebrospinal fluid leak after retrosigmoid excision of vestibular schwannomas. Otol Neurotol 2008;29:384–6.
31. Gal TJ, Bartels LJ. Use of bone wax in the prevention of cerebrospinal fluid fistula in acoustic neuroma surgery. Laryngoscope 1999;109(1):167–9.
32. Brodie HA, Thompson TC. Management of complications from 820 temporal bone fractures. Am J Otol 1997;18:188–97.
33. Brors D, Schafers M, Bodmer D, et al. Postoperative magnetic resonance imaging findings after transtemporal and translabyrinthine vestibular schwannoma resection. Laryngoscope 2003;113:420–6.
34. Bordure P, O'Donoghue GM, Jaspan, et al. Cerebellar encephalomalacia after removal of acoustic tumor. Otolaryngol Head Neck Surg 1999;121(1):144–9.
35. Weissman JL, Hirsch BE, Fukui MD, et al. The evolving MR appearance of structures in the internal auditory canal after removal of an acoustic neuroma. AJNR Am J Neuroradiol 1997;18(2):313–23.
36. Umezu H, Seki Y. Postoperative magnetic resonance imaging after acoustic neuroma surgery: influence of packing material in the drilled internal auditory canal on assessment of residual tumor. Neurol Med Chir (Tokyo) 1999;39(2):141–7.
37. Bennett ML, Jackson CG, Kaufmann R, et al. Postoperative imaging of vestibular schwannomas. Otolaryngol Head Neck Surg 2008;138:667–71.
38. El-Kashlan HK, Zeitoun H, Arts HA, et al. Recurrence of acoustic neuroma after incomplete resection. Am J Otol 2000;21(3):389–92.