The Evolving MR Appearance of Structures in the Internal Auditory Canal after Removal of an Acoustic Neuroma

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PURPOSE: To identify patterns of enhancement in the internal auditory canal (IAC) on MR studies after removal of an acoustic neuroma, including changes in those patterns with time; to evaluate signal and enhancement of the labyrinth; to differentiate normal postoperative findings from those suggesting residual tumor; and to describe MR hallmarks of surgical approaches. **METHODS:** We reviewed the postoperative MR studies obtained in 36 patients who had had surgery for acoustic neuroma (101 images total). Four patterns of IAC enhancement were evaluated, as was labyrinthine signal intensity before and after contrast administration, changes in findings over time, and anatomic alterations caused by surgery. **RESULTS:** All patients had enhancement of the IAC on the first postoperative study. In 30 patients, IAC enhancement remained the same or decreased over time. Seventeen patients had hyperintense cochlear signal and 15 had cochlear enhancement that decreased with time. Effects of retrosigmoid craniotomy, a translabyrinthine surgical approach, and middle fossa craniotomy were recognizable. **CONCLUSION:** Linear enhancement in the IAC is probably normal after surgery. Nodular and masslike enhancement and any progressive enhancement may require close follow-up to monitor growth of residual tumor. Labyrinthine hyperintensity may reflect blood metabolites. An MR protocol is suggested for following up patients in the years after surgery.

Index terms: Magnetic resonance, postoperative; Neuroma; Temporal bone, neoplasms

AJNR Am J Neuroradiol 18:313–323, February 1997

Imaging studies are a crucial component of patient care after surgical removal of an acoustic neuroma (1, 2). Magnetic resonance (MR) imaging has become most surgeons' study of choice to follow up patients after surgery (2).

Postoperative enhancement in and around the internal auditory canal (IAC) is a frequent, if poorly understood, finding (3–7). Only a few authors have studied the changes in appearance that occur over time in the postoperative IAC (6, 8).

We evaluated consecutive postoperative MR examinations of patients in whom acoustic neuromas had been removed. Our goal was to identify patterns of enhancement in the IAC and changes in these patterns over time in an attempt to differentiate normal postoperative findings (requiring no intervention) from findings suggestive of residual tumor (for which closer follow-up or intervention might be important). In addition, the signal intensity and enhancement of the membranous labyrinth were assessed and the MR hallmarks of the various surgical approaches were identified.

Materials and Methods

Thirty-six patients who underwent removal of an acoustic neuroma between February 1987 and February 1995 had at least two postoperative MR studies available for

Received May 15, 1996; accepted after revision August 26.

Presented in part at the annual meeting of the American Society of Head and Neck Radiology, Los Angeles, Calif, April 1996, and at the annual meeting of the American Society of Neuroradiology, Seattle, Wash, June 1996.

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AJNR 18:313–323, Feb 1997 0195-6108/97/1802–0313

retrospective review. The subjects included 21 women and 15 men who were 17 to 67 years old at the time of surgery.

The number of follow-up studies ranged from two to five per patient (17 patients had two, 11 patients had three, six patients had four, and two patients had five studies), for a total of 101 MR studies in the 36 patients. The earliest follow-up MR examination was done 1 week after surgery. The longest follow-up examination was 8 years after surgery.

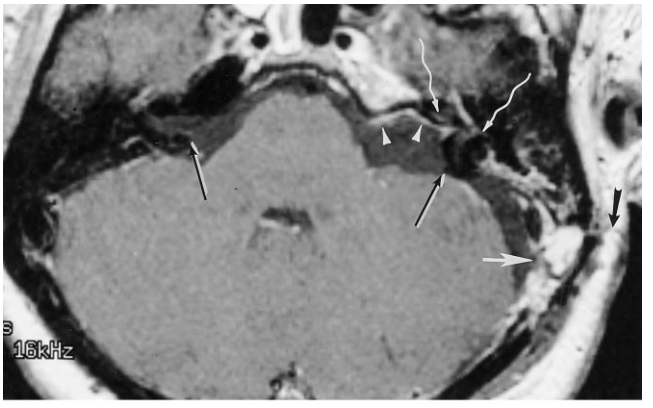
All but three of the MR studies included noncontrast T1-weighted axial images (short repetition time [TR]/short echo time [TE]) through the temporal bones. (One of the three patients had a noncontrast coronal T1-weighted study.) All 113 studies included contrast-enhanced axial T1-weighted images, and 90 studies included contrast-enhanced coronal T1-weighted images. All images were 3 mm thick. The largest intersection gap was 1 mm. Some of the studies included interleaved images with a 1-mm overlap between consecutive images (no intersection gap). Of the 17 MR examinations of patients with fat grafts in the surgical bed, only seven included fat-suppression sequences obtained after contrast administration. In 33 studies, high-resolution T2-weighted images (long TR/long TE/4 excitations, 256 × 512 matrix, 3-mm-thick sections with a 0.5-mm intersection gap) were obtained. The studies were reviewed by two experienced head and neck neuroradiologists.

On the noncontrast T1-weighted images (short TR/short TE), the signal intensity of the contents of the IAC on the side of the surgery was compared with the contralateral, normal IAC. After contrast administration, IAC enhancement was graded as faint or intense and characterized as having one of four patterns (Figs 1–4) as follows: linear, peripheral, and thin (pattern 1) (Fig 1); linear, peripheral, and thick (pattern 2) (Fig 4B); nodular, if a discrete nodule was present (pattern 3) (Fig 2); or masslike, if the entire canal was filled with enhancing tissue (pattern 4) (Fig 3). If more than one pattern was present (for example, intense, linear, thick, and nodular), the rating assigned was the greatest number (in this case, pattern 3, nodular).

The membranous labyrinth (lumen of the labyrinth) was divided into two components, vestibule/semicircular canals and cochlea. The signal of these (without contrast material) was compared with the signal of the contralateral (normal) labyrinth and judged as isointense, faintly hyperintense (signal intensity equal to that of brain by visual inspection), or intensely hyperintense (signal intensity greater than that of brain). After contrast administration, labyrinthine enhancement was graded as none, faint, or intense (Figs 5 and 6).

Because images were obtained at variable postoperative intervals, elapsed time after surgery was divided into four clinically relevant categories, with surgery considered time 0: 0 to 6 months, 7 to 12 months, 13 to 24 months, and more than 24 months. Ratings of signal intensity and enhancement were cross-tabulated with the four time categories and the Jonckheere-Terpstra (JT) test was used to test for row and column independence (9). Some of the cells in these tables contained sparse data, so the Statxact statistical package was then used to compute exact probabilities for the JT tests (10). For each patient, a two-level comparison was performed to evaluate two MR studies obtained at least 10 months apart (balanced analysis of variance [ANOVA]).

The calvaria, temporal bone, and subcutaneous soft tissues were scrutinized to discern the surgical approach. Other surgical changes, such as graft material, were also noted (Figs 1–7).



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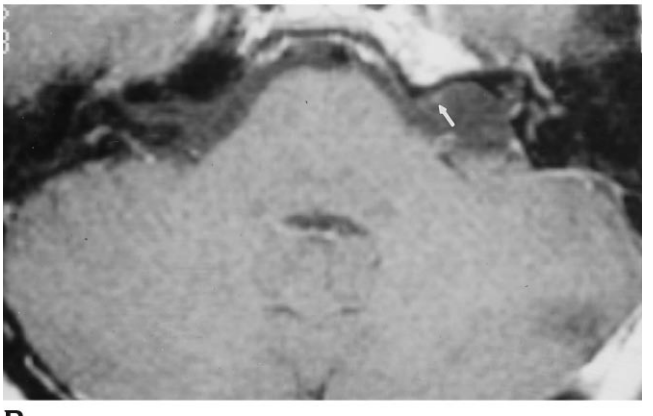

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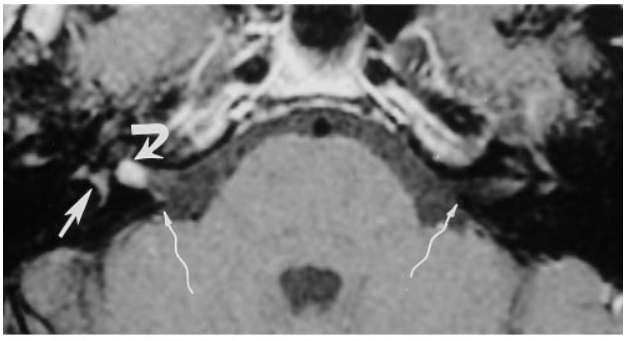
Fig 1. Thin, linear pattern of enhancement in the IAC that completely resolves with time; retrosigmoid craniotomy.

A, Axial contrast-enhanced T1-weighted image (60/23/2) shows thin, peripheral, linear enhancement within the IAC (*arrowheads*). The labyrinth was hyperintense before contrast administration, so the high signal on this image (*wavy arrows*) could represent inherent high signal or high signal plus enhancement. Evidence of a retrosigmoid approach to the tumor includes the subcutaneous scar (*black arrow*), occipital craniotomy (*straight white arrow*), and drilled posterior lip of the IAC, which is different from the configuration of the porus acousticus on the normal right side (*two-tone arrows*).

B, One year later, an axial T1-weighted image (583.3/23/3) shows essentially complete resolution of the enhancement within the IAC (*arrow*).



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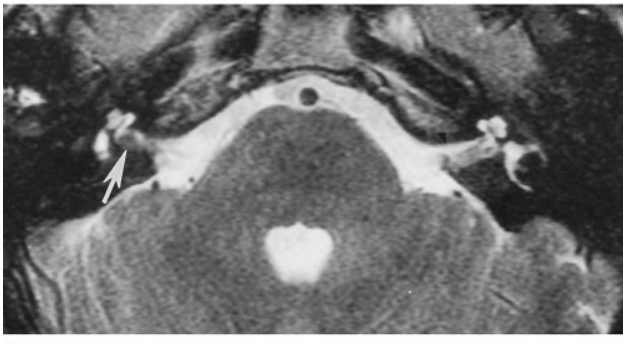

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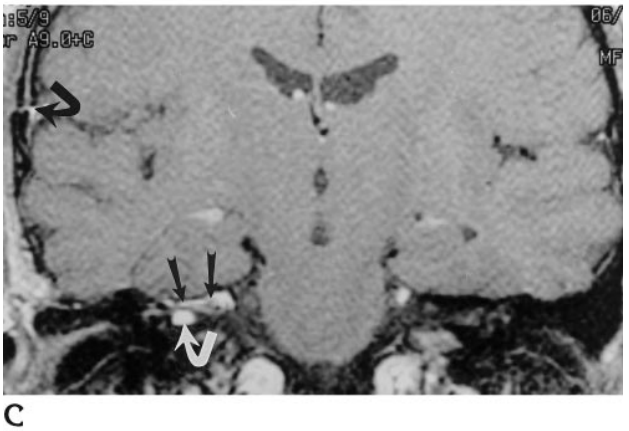

C

Fig 2. Nodular pattern of enhancement within the IAC and enhancing fascia graft above the IAC; middle fossa approach.

A, Axial contrast-enhanced T1-weighted image (783/23/2) shows nodular enhancement within the IAC (*curved arrow*). The vestibule is hyperintense (*straight arrow*). Because the signal was hyperintense before contrast administration, it is not possible to determine whether there is also enhancement. Note that the porus is intact; compare with the opposite side (*wavy arrows*).

B, Axial high-resolution T2-weighted image (4000/108/4; 512 × 256 matrix) shows low-signal soft tissue in the fundus of the IAC (*arrow*), which corresponds to the enhancing tissue seen in *A*. A cap of cerebrospinal fluid around the mass at the fundus is not apparent on the T1-weighted images. The mass could be scar or tumor.

C, Coronal noncontrast T1-weighted image (633/23/2) shows the nodular enhancement in the IAC (*curved white arrow*) and a temporal craniotomy (*curved black arrow*) of the middle fossa approach. Thick enhancement along the roof of the IAC (*straight arrows*) represents a fascia graft placed at surgery. This could easily be mistaken for tumor in the absence of information regarding the surgery.

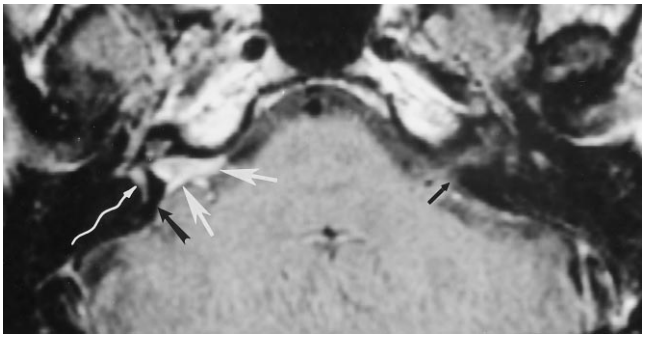

Fig 3. Masslike pattern of enhancement within the IAC; retrosigmoid craniotomy. Axial contrast-enhanced T1-weighted image (650/23/2) shows enhancing tissue filling the IAC (*straight white arrows*). The vestibule (*wavy arrow*) and the cochlea (unlabeled) were hyperintense before contrast administration, so it is not possible to determine on this image whether there is also enhancement. The posterior lip of the porus acusticus had been drilled away (*large black arrow*) as part of the retrosigmoid craniotomy. The left porus (*small black arrow*) is normal.

Results

Internal Auditory Canal

In the first 6 months after surgery, eight patients had faint high signal in the IAC before contrast administration. On studies performed 7 to 12 months after surgery, three patients had faint high signal. On studies performed 13 to 24 months after surgery, six patients had high signal; and on studies performed more than 24 months after surgery, seven patients had faint high signal (Fig 6A). No patient had intense high signal in the IAC at any time. The rest of the patients had obliterated IACs that could not be evaluated. There was no statistically significant correlation between IAC signal without contrast enhancement and time elapsed since surgery.

Within 0 to 6 months after surgery, 28 studies were obtained in 25 patients. Two studies showed faint thin linear enhancement, four studies showed intense thin linear enhancement (Fig 1), seven showed intense thick linear enhancement, seven showed intense nodular enhancement (Fig 2), four showed intense masslike enhancement (Fig 3), and in four enhancement could not be assessed (the IAC was obliterated or fat packing without fat suppression made assessment of enhancement impossible) (Fig 4) (Table 1).

All patients (in whom enhancement could be assessed) had enhancement of the IAC on the first postoperative study, regardless of the time elapsed since surgery (range, 1 week to 5

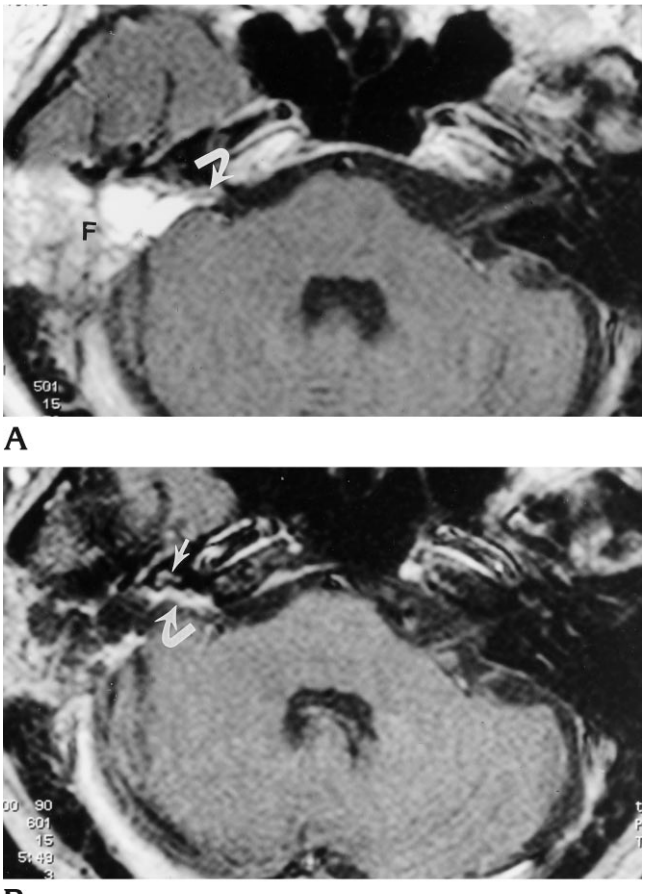

Fig 4. Importance of fat suppression on enhanced images after translabyrinthine resection.

A, Axial contrast-enhanced T1-weighted image (501/15/2) shows the large amount of hyperintense fat (*F*) placed in the mastoidectomy defect after a translabyrinthine resection. The fat makes it difficult to determine whether there is enhancement along the remaining anterior wall of the IAC (*arrow*).

B, On the axial contrast-enhanced T1-weighted image (601/15/3) with fat suppression from the same study, intense thick linear enhancement along the anterior wall of the IAC is easily identified (*curved arrow*). Note that portions of the fat graft enhance. The cochlea is present (*straight arrow*), but the vestibule, semicircular canals, and the posterior wall of the IAC have been drilled away.

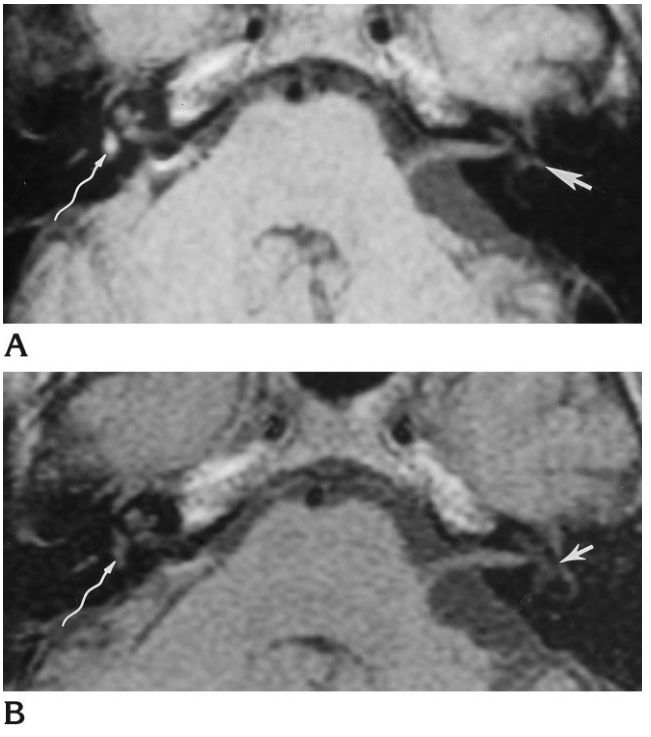

Fig 5. Hyperintense labyrinthine signal that fades with time; retrosigmoid craniotomy.

A, Axial noncontrast T1-weighted image (633/23/2) shows markedly hyperintense signal in the vestibule (*wavy arrow*). The left side is normal (*straight arrow*).

B, Axial noncontrast T1-weighted image (400/11/3) obtained 11 months later shows only faint hyperintense signal in the vestibule (*wavy arrow*) as compared with the opposite side (*straight arrow*).

years). Twenty-one patients had enhancement in the IAC that did not change over time (follow-up range, 4 months to 5 years 11 months): two patients maintained faint thin linear enhancement, two patients had intense thin linear enhancement (Fig 1), three had persistent intense thick linear enhancement, six had residual nodular enhancement (Fig 2), and eight had masslike enhancement (Fig 3).

Seven patients had IAC enhancement that decreased with time (follow-up range, 11 months to 3 years): three went from intense thick linear to intense thin linear, two went from intense thin linear to faint thin linear, one went from intense nodular to intense thin linear, and one went from faint thin linear to no enhancement. The balanced ANOVA revealed no statistically significant decrease in IAC enhancement between any two MR studies (of the same patient) obtained 10 months apart.

Five patients had IAC enhancement on an initial study that increased with time (follow-up range, 9 months to 5 years 11 months): three progressed from intense thick linear (Fig 4B) to intense nodular, one progressed from intense thin linear to intense thick linear, and one progressed from faint thin linear to faint thick linear enhancement.

Labyrinth

Seventeen patients had slightly or intensely high signal from the labyrinth before contrast administration on at least one postoperative

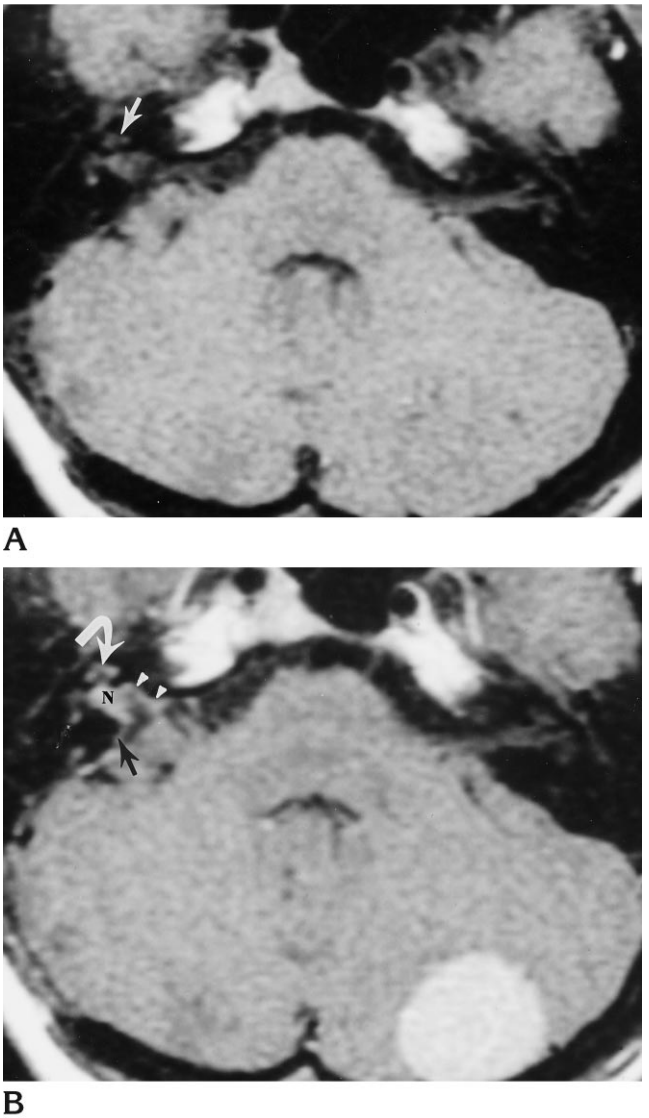

Fig 6. Labyrinthine enhancement; retrosigmoid craniotomy.

A, Axial noncontrast T1-weighted image (480/25/4) shows normal signal in the cochlea (*arrow*). The contents of the IAC are faintly hyperintense, suggesting tissue replacing some of the normal cerebrospinal fluid.

B, Axial contrast-enhanced T1-weighted image (480/25/4) from the same study shows intense enhancement of the cochlea (*curved arrow*). Note linear enhancement in the medial IAC (*arrowheads*) and nodular enhancement in the fundus (*N*). The porus has been drilled (*straight arrow*); compare with the normal left side. A meningioma also enhances (unlabeled).

study that diminished or resolved on a subsequent study (Fig 5). In the first 6 months, 16 patients had high signal in the cochlea; eight of these diminished or resolved. In the first 6 months, 12 patients had high signal in the vestibule that diminished or resolved. (Of these, four patients had high signal in both the cochlea and vestibule.) In seven patients, the vestibular signal could not be assessed because the vestibule had been drilled away. There was a statistically significant decrease in cochlear signal with time ($P < .05$) (Table 2). There was no statistically significant decrease in vestibular signal over time.

Three patients had initial studies less than 1 month after surgery. In two of these, the signal in the labyrinth increased between the initial scan and the subsequent scan.

In 15 patients, enhancement in the vestibule or cochlea on one postoperative study faded on a subsequent study (Fig 6). Two patients had vestibular enhancement on at least one postoperative study that diminished or resolved with time. (Statistical significance could not be assessed because the number was so small.) Thirteen patients had cochlear enhancement that diminished or resolved. There was a statistically significant inverse association between time elapsed since surgery and cochlear enhancement: enhancement was greater closer to surgery and faded with time ($P > .01$ for JT test; $P = .05$ for balanced ANOVA). No patients had enhancement that increased with time.

Seventeen patients had at least one postoperative study in which enhancement of the labyrinth after contrast administration could not be assessed, because the labyrinth was hyperintense before contrast administration (12 patients) (Fig 2A), because no noncontrast images were obtained (six patients), or because the vestibule had been drilled away (four patients) (Fig 7). (Some patients fell into both categories: a translabyrinthine resection obliterated the vestibule and created high signal in the cochlea.) On 15 studies in 11 patients, faint high signal became more hyperintense after contrast administration, which was interpreted as enhancement. In 19 patients, faint high signal before contrast administration did not change after contrast administration; this was considered no enhancement.

Surgical Approach

The three surgical approaches used were the retrosigmoid, translabyrinthine, and middle fossa. All three were correctly identified on the studies.

Thirty patients had retrosigmoid craniotomies (Figs 1, 3, and 6). A scar in the subcutaneous fat and an occipital craniotomy were the hallmarks of this approach (Fig 1A). The pos-

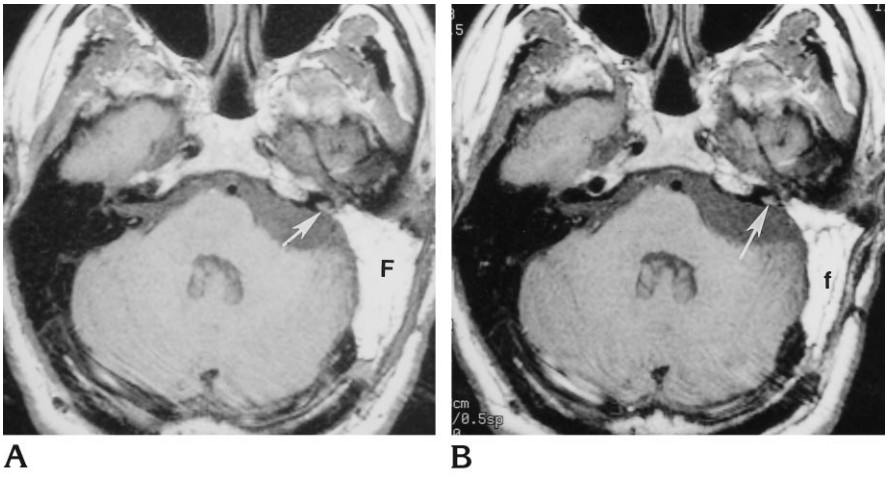

Fig 7. Sequential studies showing hyperintense cochlea unchanging over time and atrophy of the fat graft after translabyrinthine approach.

A, Axial noncontrast T1-weighted image (750/23/2) shows a bulky fat graft (*F*) filling the mastoidectomy defect. The cochlea (*arrow*) is hyperintense.

B, Axial T1-weighted image (750/23/2) 11 months later, again without contrast material, shows loss of bulk of the fat graft (*f*) and infiltration of fat by linear low signal, presumably fibrous tissue. The cochlear signal remains high (*arrow*).

TABLE 1: Signal from internal auditory canal seen after administration of contrast material by time and enhancement pattern

	Time Since Surgery, m				
	6 or Less	7 to 12	13 to 24	More than 24	**Total**
Faint enhancement					
Thin or thick linear	3	2	5	2	**12**
Nodular or masslike	0	0	0	0	**0**
Intense enhancement					
Thin or thick linear	11	9	5	6	**31**
Nodular or masslike	11	7	12	17	**47**
Total	**25**	**18**	**22**	**25**	**90**

TABLE 2: Signal from cochlea seen without contrast enhancement by time

	Time Since Surgery, m				
	6 or Less	7 to 12	13 to 24	More than 24	**Total**
Normal signal	10	10	13	15	**48**
	(20.84)	(20.83)	(27.08)	(31.25)	**[51.61]**
Faint high signal	13	8	10	10	**41**
	(31.71)	(19.51)	(24.39)	(24.39)	**[44.09]**
Intense high signal	3	0	1	0	**4**
	(75.00)	(0.00)	(25.00)	(20.00)	**[4.30]**
Total	**26**	**18**	**24**	**25**	**93**
	[27.96]	**[19.35]**	**[25.81]**	**[26.88]**	

Note.—Numbers in parentheses are row percentages; numbers in brackets are percentage of total.

terior wall of the IAC was drilled (Figs 1A, 3, and 6B), but the cochlea, vestibule, and semicircular canals were always intact.

In nine patients, a translabyrinthine approach had been used (Figs 4 and 7). The mastoid, vestibule, and posterior wall of the IAC had been drilled away and the cavity packed with fat (Figs 4A and 7A and B). The cochlea remained, and could be evaluated for signal and enhancement on T1-weighted images (Figs 4B and 7A and B), but was seen best on the high-resolution T2-weighted images. The large amount of fat filling the mastoidectomy was the clue to a translabyrinthine approach. The fat atrophied with time: it lost bulk, and was infiltrated by lower-signal tissue that did not enhance, presumably representing development of fibrous tissue (Fig 7A and B). The fat grafts in these patients made it very difficult and sometimes impossible to identify enhancement along the remaining anterior wall of the IAC after contrast administration (Fig 4A) when fat suppression was not used (Fig 4B).

In one patient the tumor was removed through the middle cranial fossa (Fig 2). This approach created a subcutaneous scar above the external auditory canal and a craniotomy through the temporal squamosa (Fig 2C). In-

tense linear enhancement along the roof of the IAC represented a fascia graft the surgeon had placed to close the defect after the bone was drilled and the tumor removed (Fig 2C).

Discussion

Acoustic neuromas are benign tumors that grow slowly (2, 11). Because they can enlarge to encroach upon brain stem, cerebellum, and the fifth through 11th cranial nerves, surgical removal is the treatment of choice for patients who are in good health. These are histologically benign neoplasms, so tumor growth after surgery is presumed to represent residual tumor and not (as with a malignant tumor) recurrence (2, 7). Tumor resection that appears complete to gross inspection may leave microscopic foci (2); sometimes, small nodules are deliberately left to preserve facial nerve function (7). With modern surgical techniques, the reported rate of inadvertent subtotal resection is only 0.5% to 1.0% (2).

Documenting growth of residual tumor is difficult when assessment is based solely on symptoms. Hearing-preservation surgery is not always possible, so the surgeon cannot rely on subjective accounts of hearing loss or changes in the audiogram to monitor growth of residual tumor. Surgery may leave patients less sensitive to vertigo and tinnitus, which might otherwise signal tumor growth (2). MR examination thus becomes a crucial component of postoperative management (1).

Intracranial enhancement after various neurosurgical procedures has been described in normal (uncomplicated) MR studies (3–5) in up to 100% of postoperative patients studied (5). Disruption of the blood-brain barrier (3), chemical meningitis from subarachnoid hemorrhage (4), development of granulation tissue (3), and inflammation around resorbable materials such as absorbable gelatin powder and sponge (Gelfoam, Upjohn, Kalamazoo, Mich) and oxidized regenerated cellulose (Surgicel, Johnson & Johnson, Arlington, Tex) (5), and bone wax may all contribute to the enhancement. Earlier studies in a small number of patients described normal postoperative enhancement within the IAC (3, 5). However, the concern is always that this normal enhancement in fact reflects tumor. One group of investigators frankly admitted that it is difficult to distinguish postoperative changes from residual tumor (7).

In the current study, we identified four patterns of enhancement in the IAC (Figs 1–4). Thick or thin linear enhancement in the IAC almost certainly does not represent tumor, and so can be considered normal (Fig 1). Linear enhancement probably represents a combination of factors that can be explained as the response of the dura to surgical manipulation and enhancement of the remaining nerves in the IAC. Nerve enhancement most likely reflects disruption of the blood-nerve barrier (12). Findings in which the enhancement diminishes or completely resolves with time would seem to confirm these hypotheses (Fig 1).

Thick linear enhancement (Fig 4) can be difficult to differentiate from nodular enhancement (Fig 6B). It is possible that thick linear enhancement and nodular enhancement are two points along a continuum. The classification system used here may have artificially divided continuous data into discrete categories. In addition, during the normal course of healing, the thickened leptomeninges lining the IAC may retract at varying rates, creating a nodular appearance that could mimic recurrent tumor.

In three patients, there was progression of enhancement from thick linear to nodular. In two patients, enhancement progressed from thin linear to thick linear. It is quite possible that these progressions do not represent recurrent tumor. Rather, they may reflect normal variations in appearance with healing and, possibly, subtle variations of the plane of scanning through the IAC from one follow-up study to the next. In addition, thick linear enhancement can mimic masslike enhancement on axial images. Coronal images may better show the linear nature of the enhancement. All these observations underscore the need for continued postoperative scanning. Enhancement patterns that continue to evolve are more likely to suggest tumor than is enhancement that remains stable or resolves.

Masslike enhancement is difficult to interpret (Fig 3). This could be either a very thick version of the thick, linear pattern or it could really be a mass within the canal. Because this pattern was seen on initial postoperative studies, it seems more likely that this is not tumor, as the surgeon can see the canal contents at the time of surgery. Small tumor nodules may intentionally be left if they are adherent to the facial nerve (7), but a mass filling the IAC would be removed. The fundus is not directly accessible from the

retrosigmoid approach, so nodular enhancing tissue in the fundus on postoperative studies merits special attention in these patients. The lack of a statistically significant decrease in IAC enhancement over time suggests that continued enhancement is likely to be an expected (normal) finding that, by itself, need not raise the concern of residual tumor.

High-resolution T2-weighted images showed soft tissue that was hypointense relative to cerebrospinal fluid and corresponded to the enhancing soft tissue on T1-weighted images after contrast administration (Fig 2B). However, even these T2-weighted images could not differentiate tumor from scar. The signal intensity of the contents of the IAC before contrast administration did not prove to be a useful observation. Few patients had hyperintense signal before contrast administration, and of those who did, the signal did not change appreciably with time. It is therefore not clear what the high signal represents. In one small study of 10 patients (12), the authors claimed that this finding reflects hematoma in the preserved nerves running in the IAC, but the MR studies were obtained within the first month after surgery and no subsequent images were described. In the current study, the lack of interval change of the IAC signal, over years of follow-up in some patients, makes hematoma an unlikely explanation. However, the evolution of hematoma within cranial nerves is not known.

High signal from the labyrinth (especially the cochlea) seems to be part of the normal (uncomplicated) postoperative course. Most likely this represents a blood metabolite, such as extracellular methemoglobin (13). High signal developed in two patients after an immediate postoperative scan (less than 1 month after surgery) on which signal was normal. This would tend to substantiate this hypothesis, although the number of patients is small. Although methemoglobin appears sooner in brain, for instance, we are unaware of studies that have evaluated the turnover of blood products in the endolymph or perilymph. Conceivably, metabolism of blood or turnover of blood-tainted fluids is quite slow in the labyrinth. This could account for the slow and protracted course of the appearance and resolution of the hyperintense signal.

Several mechanisms can explain the presence of blood in the labyrinth after acoustic neuroma surgery. Traction on and transection of the cochlear and vestibular nerves at surgery could cause bleeding into the membranous labyrinth. Interruption of arterial supply (labyrinthine infarction or stroke) is another possible explanation. The labyrinthine artery is an end artery that arises from the anterior inferior cerebellar artery within the IAC. The labyrinthine artery is small, and may be damaged or sacrificed at surgery. Bleeding from cochlear concussion (trauma to the labyrinth induced by drilling nearby bone) is, however, unlikely to cause the high signal. Cochlear concussion is associated with hearing loss (14), but patients whose mastoids are drilled for other reasons (cholesteatomas) do not experience hearing loss after surgery. Therefore, drilling probably does not cause cochlear concussion.

It is somewhat surprising that high signal tends to persist in the cochlea even after fading in the vestibule. Although more often called acoustic neuromas, the overwhelming majority of these tumors are really vestibular schwannomas. They arise from the superior or inferior division of the vestibular branch of the eighth cranial nerve. One might have predicted, therefore, that there is greater traction on or damage to the vestibular nerve than the cochlear nerve at surgery, and more subsequent signal abnormality in the vestibule. It seems likely that blood in the vestibule is also dispersed within the semicircular canals, and this larger volume of distribution might yield a lower concentration of blood and so make the signal less intense.

Enhancement of the membranous labyrinth after surgery may be understood by considering other settings in which labyrinthine enhancement occurs. Infection, such as syphilis and inflammation, causes enhancement (15, 16). Blood is a chemical irritant that causes dural inflammation and enhancement (3); even a small amount of blood (too small to create a signal change perceptible on MR images) may irritate the membranous labyrinth by a similar mechanism.

We encountered two situations in which it was not possible to determine whether the labyrinth enhanced. The first was in those patients whose T1-weighted images obtained before contrast administration showed a hyperintense labyrinth: it was often difficult to determine if the labyrinth also enhanced after contrast administration. (Rarely, a labyrinth was faintly hyperintense before contrast administration, in which case enhancement could be detected if the signal became even more hyperintense after con-

trast administration.) The second situation was that in which patients were scanned during a temporary change in scanning protocol: postoperative temporal bone MR studies were obtained entirely after intravenous administration of contrast material. This created a subset of patients for whom unenhanced T1-weighted images were not available. A hyperintense labyrinth on these postcontrast images could therefore represent either intrinsic high signal or enhancement. Conceivably, one portion of the labyrinth (eg, the cochlea) could have been hyperintense before contrast administration and another portion (eg, the vestibule) could have enhanced after contrast administration. In these patients, the hyperintense portion might also enhance but its enhancement would be undetectable. Since the clinical significance of high signal and enhancement in this setting is unknown, the ability to distinguish between the two is of (as yet) unknown value.

The subjective impression of the radiologists who reviewed the studies was that filming techniques could affect the apparent hyperintensity of the labyrinth. The use of narrow windows and low levels could make signal appear greater than does wider windows and higher levels. Quite possibly, these variations in filming contributed to (or created) two subsets of patients. In one group, high signal developed transiently in the labyrinth on a single postoperative study months or years after surgery, which then resolved on subsequent studies. In the other group, patients had a single study with normal signal from the labyrinth, preceded and followed by studies showing hyperintense signal. Conceivably, these variations in technique could even skew the results on a single study, creating the impression of hyperintense signal where none exists, or of normal signal where faintly hyperintense signal is found. Because the study was retrospective, there was no way to control for these variables.

Familiarity with the indications for each surgical procedure and with the anatomic alterations of each makes it easier to interpret the postoperative studies. A translabyrinthine approach to the IAC is a transtemporal labyrinthectomy that spares the cochlea (17) (Figs 4 and 7). This approach provides complete exposure of the IAC, including the fundus, which facilitates tumor removal but destroys any residual hearing. Absence of the vestibule and semicircular canals and preservation of the cochlea could be appreciated on all pulse sequences. To obliterate the mastoidectomy cavity and prevent cerebrospinal fluid leaks, the surgeon places a fat graft (abdominal fat) in the mastoidectomy (17) (Figs 4 and 7) and Surgicel, temporalis muscle, or both in the eustachian tube and aditus ad antrum (17). The hyperintense graft makes it difficult and sometimes impossible to evaluate the remaining IAC (Fig 4A) and to identify enhancement in the surgical bed unless fat suppression is added (Fig 4B). Surgeons know that the fat graft normally shrinks with time (18). The MR studies show this (Fig 7A and B) and also suggest that, over time, the fat is infiltrated by fibrous tissue (scar).

The retrosigmoid suboccipital approach leaves the labyrinth intact (17) (Figs 1, 3, and 6). To reach the tumor, the surgeon drills the posterior wall of the IAC (Figs 1A, 3, and 6B). In carefully selected patients, this operation can preserve hearing (17). However, to preserve hearing by avoiding damage to the posterior semicircular canal, the surgeon does not drill the most lateral portion of the IAC. Therefore, the final tumor removal is done without direct visualization of the fundus of the IAC. It is, therefore, especially important to scrutinize the fundus closely on postoperative MR studies in patients in whom a retrosigmoid approach was used. Nodular or masslike enhancement here (Fig 6B) merits close follow-up.

The middle fossa temporal craniotomy provides complete exposure of the fundus (17) (Fig 2). In carefully selected patients with small tumors and normal or nearly normal hearing, this hearing-preservation approach may be ideal. It is important to know whether the surgeon has placed fascia, muscle, or both along the roof of the IAC; otherwise, the thick enhancing tissue may be mistaken for tumor (Fig 2C).

Although it would have been interesting, it was not possible to correlate the MR findings (especially cochlear signal and enhancement) with audiometric changes. Many of our patients had either lost all useful hearing before surgery or the size and location of their tumor and the status of the mastoid and middle ear did not allow a hearing-preservation procedure. Moreover, even after hearing-preservation surgery, patients may lose hearing for other reasons, such as endolymphatic hydrops. Postoperative audiograms were not available for most patients. A worthwhile investigation for the future

is one that correlates the signal intensity and enhancement of the membranous labyrinth with hearing in those patients in whom hearing preservation was attempted. Surgery may also leave patients less sensitive to symptoms other than hearing loss (tinnitus, vertigo) that would otherwise signal tumor recurrence or growth (2).

The current study has some important limitations. The design is retrospective. The paucity of findings in some categories (for example, change in vestibular signal with time) limit statistical significance. Some data are incomplete; for example, the absence of fat suppression made assessment of IAC enhancement impossible, and the absence of noncontrast images made assessment of vestibular enhancement impossible. And overlapping data were recorded in patients in whom, for example, it was impossible to determine labyrinthine enhancement for not one but two reasons (ie, high signal in the cochlea before contrast administration and obliteration of the vestibule at surgery). In addition, the postoperative studies were obtained at varying intervals, making it difficult to compare findings in patients at a single point in time and to compare findings in patients over time. Because there is currently no uniformly accepted protocol for obtaining postoperative MR studies, the interval between surgery and the initial examination and the intervals between subsequent studies varied widely. Therefore, the data were analyzed for changes between two studies in the same patient and for changes over time among all patients.

Tests of statistical significance of MR imaging for assessing patients after acoustic neuroma surgery are limited by lack of an appropriate control population. There is no surgical proof for any of the MR observations. None of these patients has had tumor recurrence large enough to mandate a return to the operating room. This is not surprising, as acoustic neuromas are slow-growing benign neoplasms (11). After removal, regrowth is rare (2) and residual tumor tends to grow slowly (8).

The combination of the evolution of postoperative findings identified in our patients and the known indolent pattern of recurrence of acoustic neuromas led us to develop a postoperative imaging algorithm. We believe it is useful to obtain an MR study of the temporal bones and brain, both before and after administration of contrast material, within 6 months after surgery but no sooner than 1 month after surgery. This examination then serves as the baseline study. If IAC enhancement is linear, imaging 1 year after the baseline study can be followed by a third postoperative MR examination 3 years later if the studies continue to show a linear pattern of enhancement and the patient remains clinically stable. Nodular or masslike enhancement merits closer follow-up to exclude recurrent tumor that could grow unchecked to compromise surrounding structures. If the enhancement progresses (becomes more nodular, more masslike, or thicker), continued follow-up at intervals of 6 months (or less) is appropriate to determine whether surgery or other treatment is indicated. If the enhancement regresses, the interval should be increased to 1 year.

Conclusion

Peripheral linear enhancement in the IAC, enhancement that fades over time, hyperintense signal from the membranous labyrinth before contrast administration, and enhancement of the labyrinth all seem to be normal or uncomplicated MR findings seen after the removal of an acoustic neuroma. Normal findings are those that are unlikely to reflect abnormalities requiring surgical intervention. Nodular or masslike enhancement in the IAC is of greater concern, is more likely to represent residual tumor, and merits closer follow-up. Familiarity with the normal or expected postoperative findings and the evolving appearance of the surgical site facilitates recognition of significant disease.

Acknowledgments

We are grateful to Sabrina L. Jennings for continuous secretarial expertise and to Rubin Sheng for 11th hour statistical assistance.

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