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Pictorial essay

Magnetic resonance imaging assessment of labyrinthine pathology

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Abstract. Membranous labyrinth pathologies are quite rare. They were until recently difficult to demonstrate by imaging technics, CT being the modality of choice. Our purpose was to stress the interest of MR examination for investigating patients complaining of vertigo, tinnitus, and profound sensorineural hearing loss. Normal anatomy as well as the main pathologically encountered changes are illustrated.

Key words: Hearing loss – Temporal bone – Magnetic resonance – Ear – Neoplasms – Cochlear implants

Introduction

Magnetic resonance imaging (MRI) is a new technique for examining the internal ear. Thin T2 sections as well as pre- and postcontrast T1-weighted images (T1WI) are mandatory for analyzing the status of the labyrinthine fluid, the cerebrospinal fluid (CSF), and the nerves and vessels within the internal auditory canal (IAC). Although CT remains the best imaging method to analyze the bony structures of the temporal bone, MR may be the first or sole examination for investigating patients complaining of vertigo, tinnitus and sensorineural hearing loss (SNHL). Radiologists must therefore be familiar with the normal anatomy as well as the pathological changes of the membranous labyrinth (ML) depicted by MR [1], screening not only for intracanalicular or labyrinthine tumors, but also for any modification of shape, signal, or enhancement of ML. Magnetic resonance cannot differentiate the different structures of ML (e.g., the membranes of the otic capsule as well as the endo- and perilymphatic fluid).

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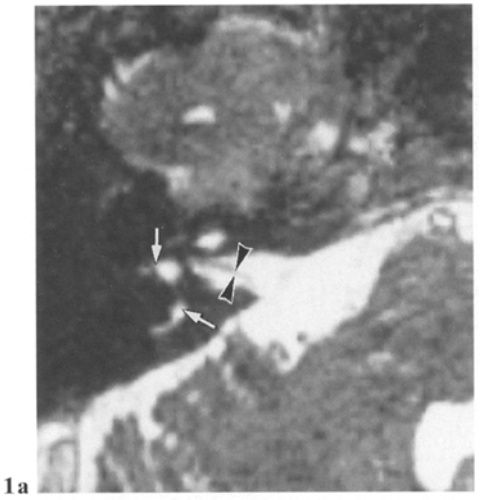
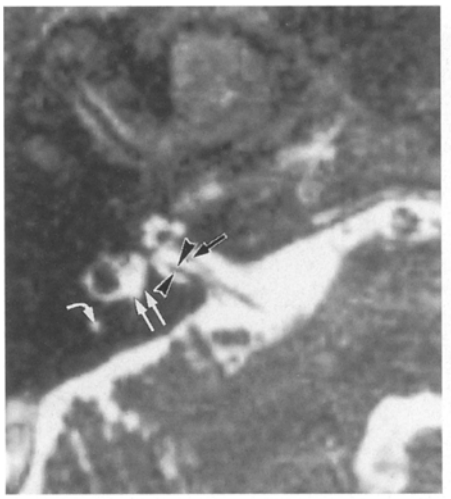
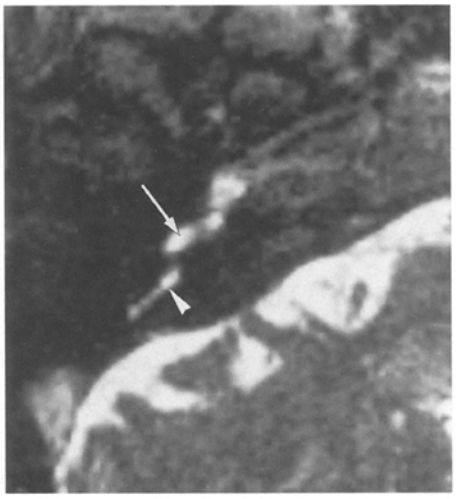
How to perform the inner ear examination

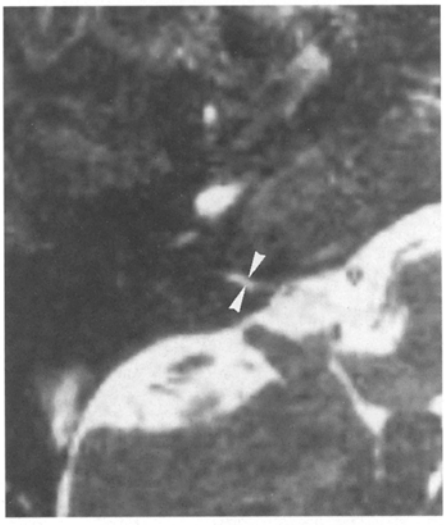
Magnetic resonance delineation of the anatomy of the ML requires high resolution (HR) sequences with a good SNR ratio using contiguous thin T2WI and T1WI sections. The following MR protocol for the examination of the inner ear is carried out:

1. Contiguous thin T2WI MRI of the inner ear. This sequence can be performed using a 3DFT CISS (constructive interference in steady state) sequence [2–4] with contiguous slices of 1 mm thickness, producing a precise imaging of the whole inner ear. The parameters used are as follows: TR/TE/matrix 20/8/256 × 256; field of view (FOV) 180–176 mm; acquisition time 2 × 2 min 46 s; flip angle 50°; slab 32. Modification of the CISS sequence parameters allows contiguous sections of 0.7 mm (TR/TE/matrix 15/21/256 × 256; FOV 170 mm; flip angle 65°; slab 22.4 mm; 32 partitions; acquisition time 2 × 4 min 30 s). A 512 rectangular matrix allows a better spatial resolution, but with an increase in acquisition (2 × 3 min 52 s) and reconstruction time (35 min). The 3D T2WI fast spin echo (SE; TR/TE/matrix 4000/150/256/512; FOV 200 mm; 1 NEX) can produce similar high-resolution contiguous images of 0.7 mm thickness, using either a head coil or a 3-inch phased array coil. On T2WI the endo/perilymphatic (E/P) fluid is delineated as high-intensity signal structures contrasting with the low signal of the surrounding bone (Fig. 1). Both CISS sequence and 3D fast SE sequence allow a multiplanar reformation (MPR) of the temporal bone (Fig. 2) and a 3D reconstruction of the inner ear (Fig. 3).
2. High-resolution pre- and postgadolinium SE T1WI using a rectangular matrix of 512 with a slice thickness of 3 mm and FOV 200/250 mm. Delineation of the temporal bone anatomy needs the use of the smallest FOV possible. A 512 matrix sequence using an intermediate FOV improves the temporal bone study resolution enough to accept the decrease in SNR. These sequences have a better spatial resolution than SE sequences of same thickness performed with a 256 asymmetrical matrix and a smaller FOV. Brain MR examination (axial









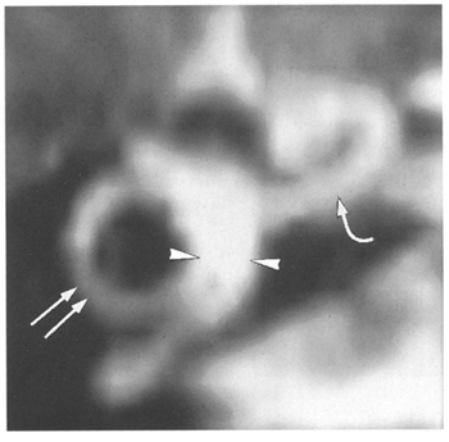
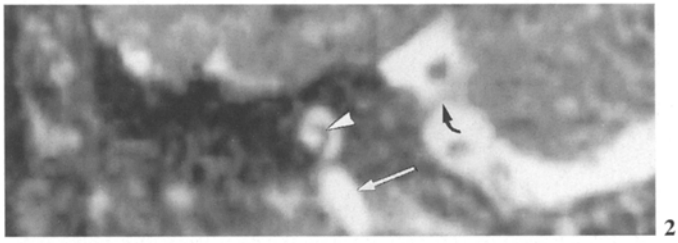


Fig. 1a–d. **a** Normal anatomy of the membranous labyrinth depicted in high-resolution T2W gradient-echo (GE) images. The upper slice shows the ampulla of the lateral and superior semicircular canals *(arrows)* and the facial nerve within the internal auditory canal (IAC; *arrowheads*). **b** 2 mm below, the acoustic branch *(arrow)* and the inferior vestibular branch *(arrowheads)* of the vestibulocochlear nerves are illustrated. The vestibule *(double arrows)* and the posterior semicircular canal *(curved arrow)* are illustrated. **c** 2 mm below, the basal turn of the cochlea *(arrow)* and the posterior semicircular canal *(arrowhead)* are depicted. **d** 2 mm below, lumen of the cochlear aqueduct *(arrowheads)*



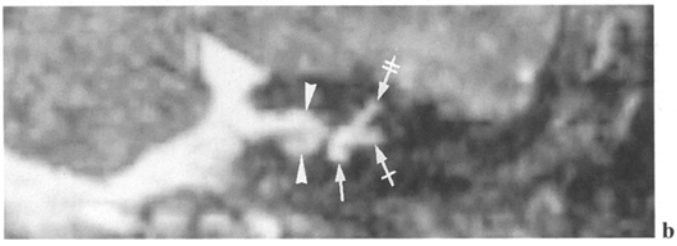


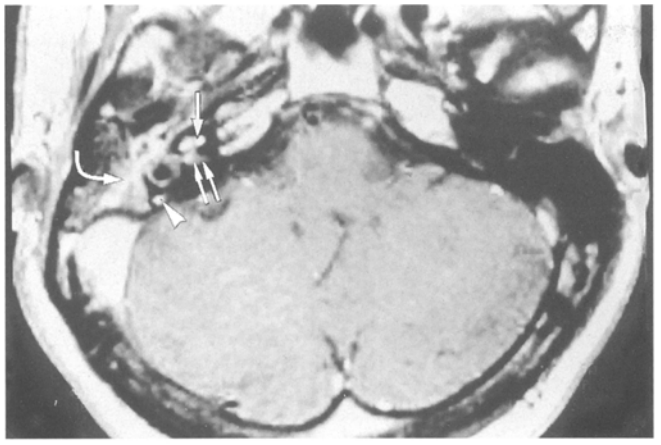
Fig. 2a, b. Reformatted coronal sections of the temporal bone. **a** Anteriorly, above the ICA *(arrow)* the spiral lamina *(arrowhead)* may be detected within the right cochlea. **b** Medially, vessels and nerves *(curved arrow)* are seen within the cerebellopontine angle. The left IAC *(arrowheads)*, vestibule *(arrow)*, the lateral (*cross-arrow*), and the superior (*double cross-arrow*) semicircular canals are well depicted in a more posterior section

Fig. 3. Three-dimensional reconstruction of the inner ear depicts anteriorly the cochlea and its basal turn *(curved arrow)*; posteriorly, the vestibule *(arrowheads)* and the superior *(arrow)*, lateral, and posterior (*double arrows*) semicircular canals

T2WI; pre- and postgadolinium SE T1WI) is always simultaneously performed to look for a brainstem process (e.g., demyelinating disease, cervicocranial malformation) as clinical symptoms are not specific enough for a precise localization of the investigated pathologies.

Inner ear pathology

At present, some inner ear pathology can be delineated only by MR [5]. The modifications of the signal of the E/P fluid in pre- and postgadolinium T1WI and T2WI may detect ML fibrosis as well as inflammatory or space-occupying lesions before they can be seen by CT



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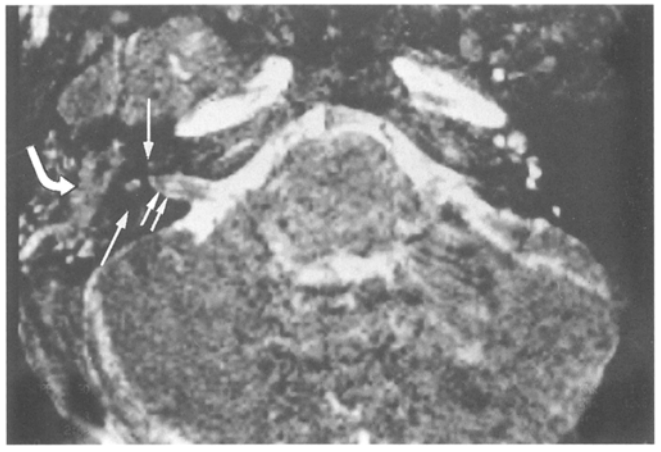

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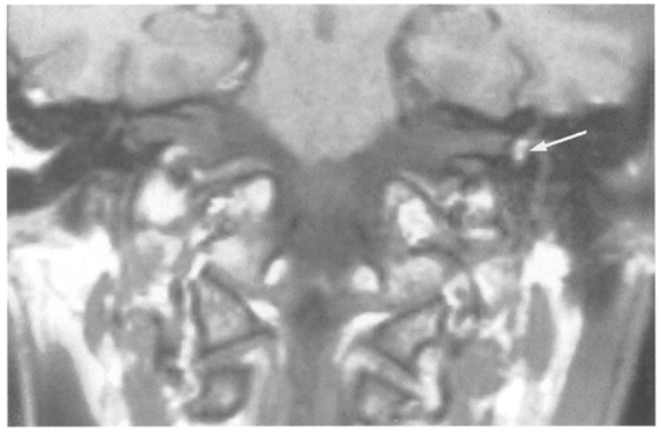

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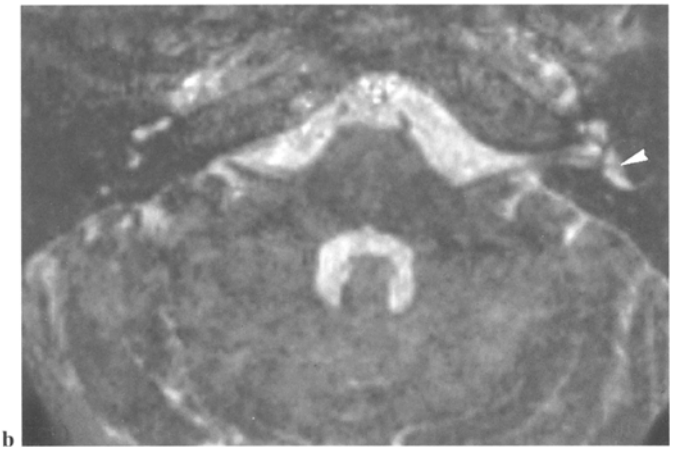

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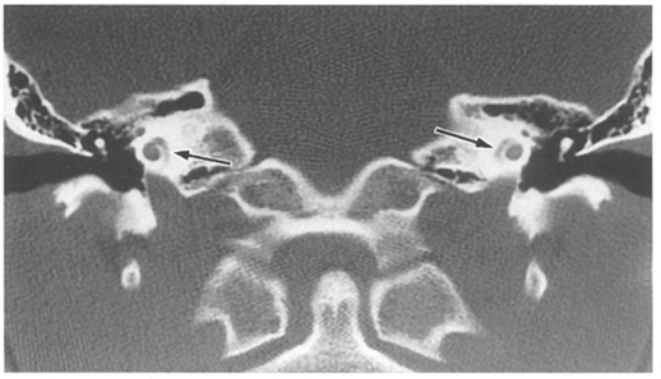

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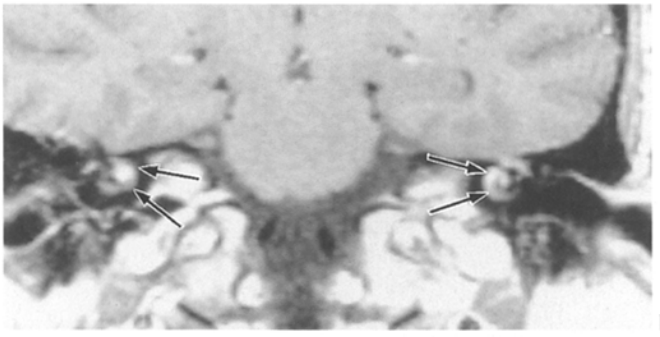

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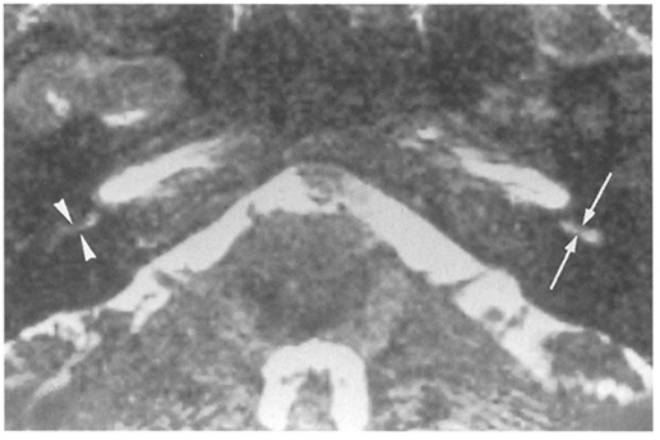

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Fig. 4a, b. Inflammatory bacterial labyrinthitis. Axial enhanced T1WI (a) and T2WI (b) MR images of an 18-year-old man with a history of sudden vertigo followed by acute deafness. **a** The cochlea *(arrow)* and the posterior semicircular canal are intensely enhanced *(arrowhead)*. Note the spread of the enhancement through the IAC (double *arrows*). Enhancement of the middle ear and the mastoid cells *(curved arrow)*. **b** The obliteration of the membranous labyrinth is attested by an almost complete loss of the normal high signal intensity (SI; *arrows*). Intermediate SI of the middle ear and mastoid cells *(curved arrow)*; normal aspect of the acousticovestibular nerves (*double arrows*)

Fig. 5a, b. An HIV-positive patient presenting with a cytomegalovirus infection, referred for acute deafness and vertigo. Coronal enhanced **a** T1WI and **b** axial T2WI MR show an enhancement of the vestibule (**a**; *arrows*) with a normal high SI on T2WI (**b**; *arrowhead*): viral labyrinthitis

Fig. 6. **a** Coronal CT scan, **b** enhanced T1WI MR, and **c** axial T2WI MR of a cochlear implant candidate explored 4 months after a bacterial meningitis. Partial calcifications of both basal turns are depicted by CT (**a**; *arrows*). The enhanced MR image reveals a persistent bilateral cochlear enhancement (**b**; *arrows*). **c** Significant decrease in the SI of the right cochlea basal turn *(arrowheads)* on T2WI regarding the signal of the normal contralateral basal turn as reference: postmeningitis sclerosing labyrinthitis with a remaining inflammatory process

[6]. Detection and exact topography of ML fibrosis are especially important to delineate if a cochlear implantation is planned. Moreover, MRI can depict the morphology of labyrinthine structures and of the IAC in congenital anomalies.

Inflammatory labyrinthitis

Membranous labyrinth inflammation of bacterial, viral, or autoimmune origin can be detected only by MR [7], leading to a suggestive contrast enhancement of the ML structures and sometimes to a T2 signal abnormality.Thus, ML enhancement should be carefully investigated in patients referred for sudden vertigo and deafness (Fig. 4). This enhancement seems to be correlated with a severely impaired labyrinthine function because mild labyrinthitis may show no detectable enhancement.

In bacterial labyrinthitis, two associated signs are mandatory to look for; a cochlear aqueduct enhancement (suggesting a bacterial spread from meninges) and a decrease in the normal SI (SI) of ML fluid on T2WI. Decrease of the normal high SI from labyrinthine fluid in T2WI suggests the presence of an inflammatory tissue filling the labyrinth [9].

In viral labyrinthitis, postgadolinium enhancement is usually observed without T2WI changes. The enhancement is probably due to a breakdown of the hemato-perilymphatic barrier (Fig. 5) without intralabyrinthine inflammatory tissue [10, 11].

In autoimmune labyrinthitis (Cogan syndrome) E/P fluid obliteration is depicted by CT and MR. Hyperintensity observed on precontrast T1WI probably represents high protein fluid concentration because it can persist a long time without any change.

In all cases ML contrast enhancement may remain a long time, even if a partial ossified labyrinth is depicted by CT; suggesting a long-lasting inflammatory process (Fig. 6).

Sclerosing and ossifying labyrinthitis

When the ML is destroyed by a suppurative process, a trauma, or by a vascular occlusion, bony healing may partially or totally obliterate the fluid spaces. After a trauma or after postchronic middle ear infection suppurative labyrinthitis, the phenomenon may be focal involving only a part of the labyrinth (e.g., the vestibule, the semicircular canal), sparing the remainder of the inner ear.

The normal E/P fluid disappears when the ML is filled by sclerosing tissue or bone. Thin T2WI can detect and localize ML fibrosis with a better sensitivity than CT. The normal high SI of the ML disappears (Fig. 7) in locations where fibrosis is present before any calcification exists. This is an important finding in cochlear implant candidates, because a patent basal turn is mandatory for a successful intracochlear device [15].

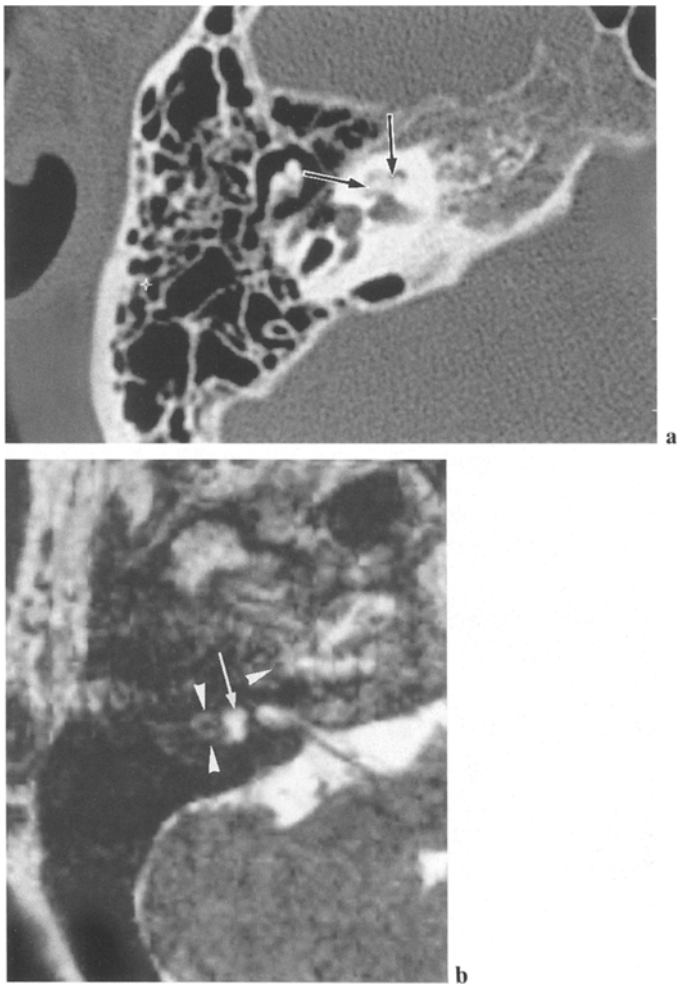


Fig. 7. a CT shows cochlear calcifications *(arrows)*. **b** T2WI MR shows a complete absence of the SI of the anterior labyrinthine structures and of the lateral and the posterior semicircular canals *(arrowheads)* sparing the lumen of the vestibule *(arrow)*: postbacterial calcified labyrinthitis

Hemorrhage

Intralabyrinthine hemorrhage may be spontaneous, traumatic, iatrogenic, or may result from temporal bone tumor invasion of the labyrinth. Only MRI may detect blood breakdown products (e.g., methemoglobin) as a signal increase on T1WI within the ML [11, 13]. For these reasons the precontrast T1WI must always be performed.

Trauma

In the initial evaluation MR is complementary to CT being able to detect methemoglobin within the labyrinth. This is an important finding when no temporal bone fracture can be depicted by CT to appreciate the severity and the prognosis of posttraumatic SNHL and for medicolegal purposes [13].

In long-standing labyrinthine trauma [14], especially in the evaluation of cochlear implant candidates

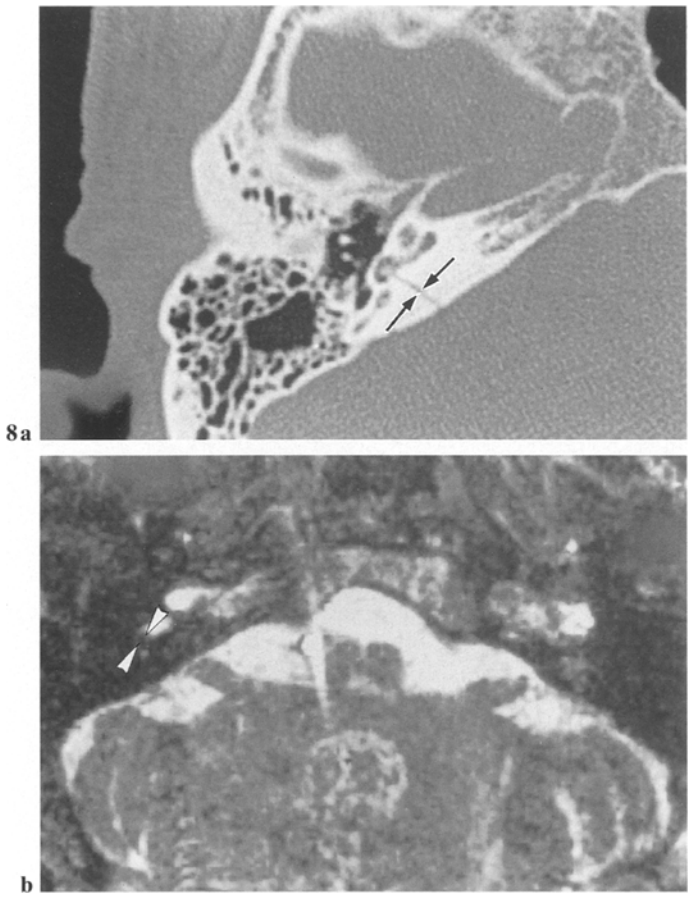


Fig. 8 a, b. Bilateral profound posttraumatic sensorineural hearing loss (SNHL). **a** Axial CT scan reveals a fracture line *(arrows)* passing through the posterior part of the basal turn of the cochlea. **b** T2WI MR shows a focal round hypointense mass located on the pathway of the fracture line just in front of the round window *(arrowheads)*: posttraumatic nodular fibrosis

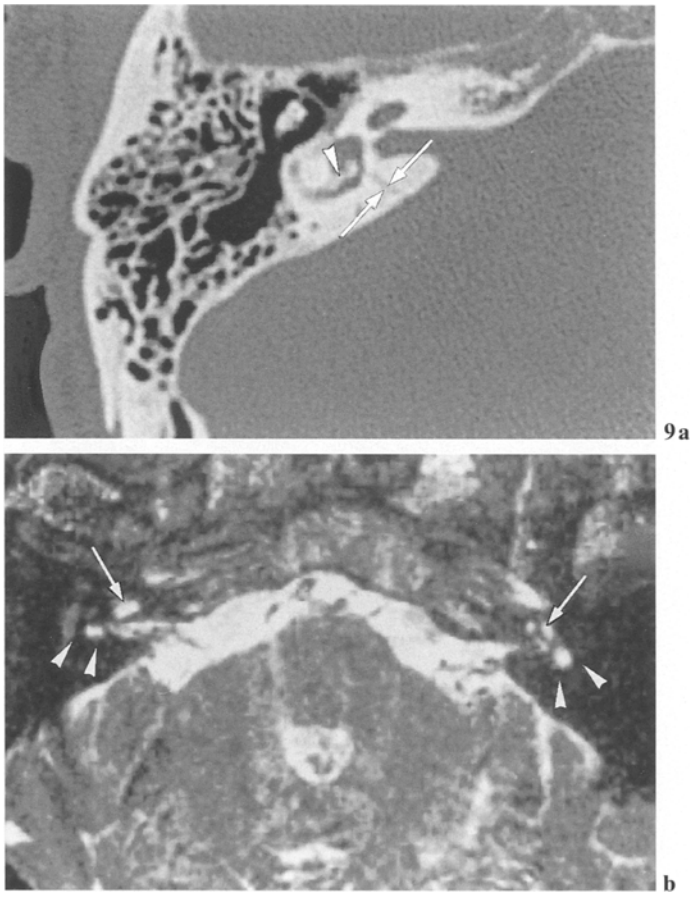


Fig. 9 a, b. Evaluation of old bilateral temporal bone fractures in a cochlear implant candidate. **a** CT depicts a fracture line passing through the right vestibule *(arrows)* with a small bony impaction within the lumen *(arrowhead)*. **b** T2WI MR shows a complete loss of the normal SI affecting the posterior labyrinths *(arrowheads)*. The preserved high SI of the anterior labyrinth *(arrows)* allows a successfull intracochlear device

(Fig. 8), MR may detect the extension of trauma-related sclerosis, essentially located along the fracture pathway, sparing the other parts of the labyrinth. This stresses the fact that anatomical barriers limit the spread of intralabyrinthic bleeding (Fig. 9) and explains the remaining permeability of the basal turn in some traumatized patients.

Otosclerosis

Otosclerosis is a common mostly bilateral (80 %) disorder [16]. This disease leads to a resorption of the endochondral layer of the otic capsule accompanied by the deposition of vascular spongy new bone, preferentially located in the area of the fissula antefenestram. These deposits are well depicted by high-resolution CT (HRCT), but are not seen on MR images. On rare occasions (< 10 %) of extensive cochlear capsule foci, a profound SNHL, or even a sudden hearing loss, are the sole manifestations of the disease. More rarely, dystrophic foci located along the vestibular aqueduct, the cochlear aqueduct, or the IAC are considered to be the cause of certain clinical complaints such as vertigo or tinnitus. Therefore, although HRCT is the best method for the assessment of cochlear capsule dysplasia, MRI can be the first-choice evaluation in these rare cases of acute deafness, profound SNHL, or vertigo revealing an otosclerosis. The suggestive MRI anomalies are the presence of nodular areas or a linear band of iso-SI on T1WI (Fig. 10), enhancing after gadolinium injection. The uptake of contrast is nodular or diffuse, often weak, requiring a strict comparison of pre- and post-T1WI. They should be realized in the same conditions with identical window settings, without new calibration for the enhanced T1WI. This enhancement should be carefully searched in certain anatomical areas such as the oval window, the round window, the cochlear and vestibular walls, and the fundus. It may affect the whole cochlear capsule or imitate a pseudo-intracanalicular mass. This enhancement caused by the hypervascularity of the dystrophy is probably greater in the active phase of the disease than in the chronic phase.

Moreover, otosclerotic foci may partially narrow or even obstruct the E/P fluid. This is well depicted by 3D

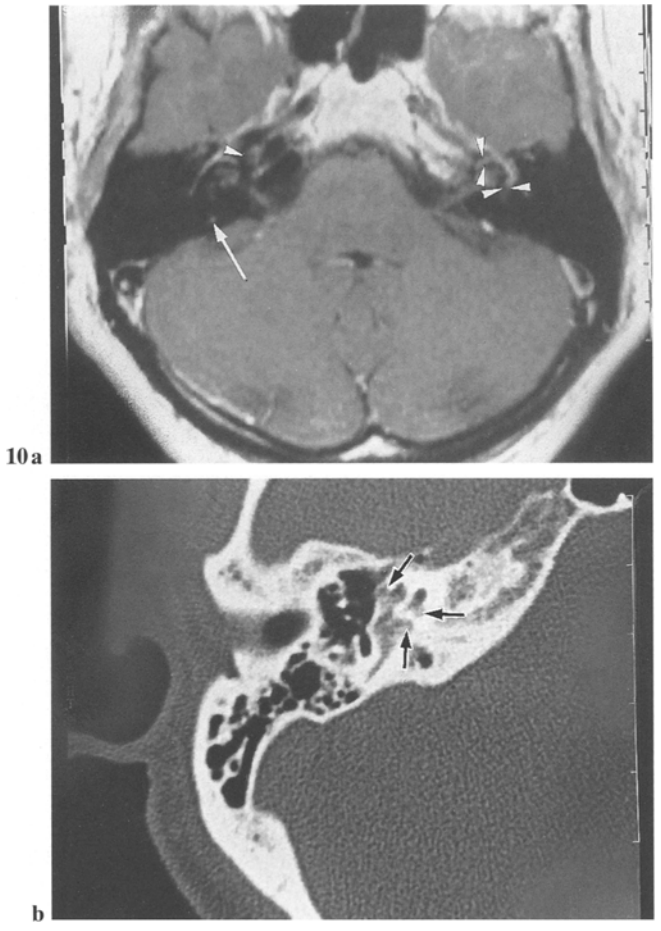


Fig. 10a, b. Patient referred before cochlear implant surgery. **a** Enhanced T1WI MR shows a bilateral weak enhancement of the otic wall *(arrowhead)* medial to the facial nerve. Enhancement of the right endolymphatic sac *(arrow)* and of the fundus of the IAC *(curved arrow)* is depicted. **b** CT scan depicts pericochlear hypodense areas *(arrows)*: active cochlear otospongiosis

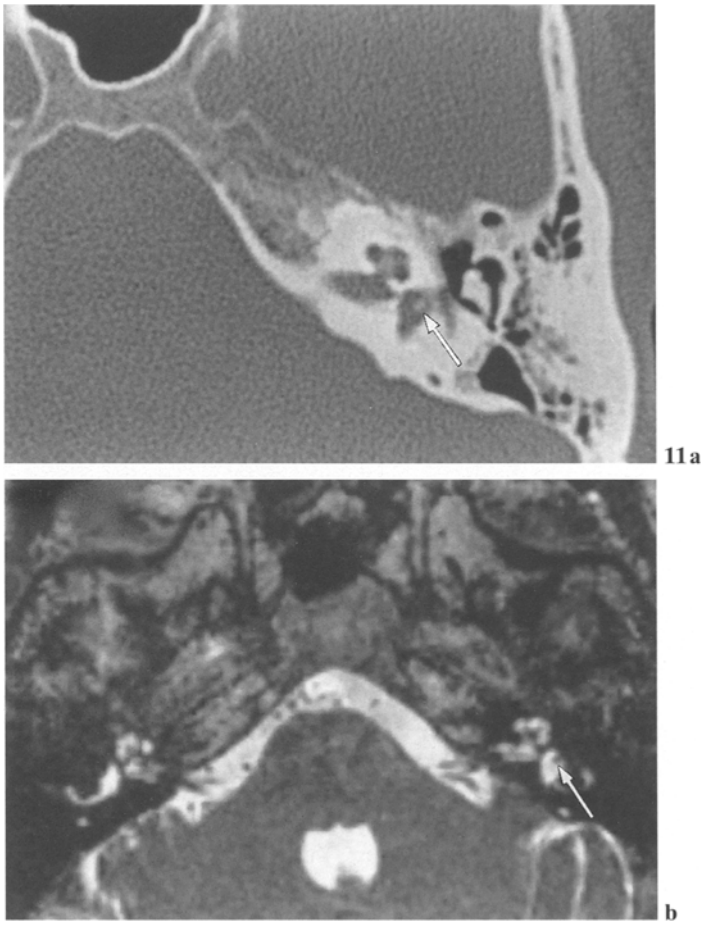


Fig. 11a, b. Vertigo post stap edectomy. Both **a** CT and **b** thin T2WI MR depict the intravestibuler location of the medial part of the device *(arrow)*. The normality of the endo/perilabyrinthic fluid is attested only by MR

reconstructions of the labyrinth. On T2WI the lesions are often not detectable being of iso- or of faintly hyper SI. These foci cause an abnormal blurred shape of ML on T1WI and T2WI, and on 3D reconstructions.

Therefore, MR is a new technique to be used in otospongiosis/sclerosis when a cochlear implantation is planned, especially in patients presenting with periostal bone thickening around the round window, to appreciate the remaining lumen of the basal turn. The active phase of the disease is suggested if a contrast enhancement of the otic walls exists.

In postsurgery follow-up, MR may demonstrate the intravestibular displacement of the prothesis as well as the modifications of the ML fluid referred to as serofibrinoid or purulent labyrinthitis (Fig. 11).

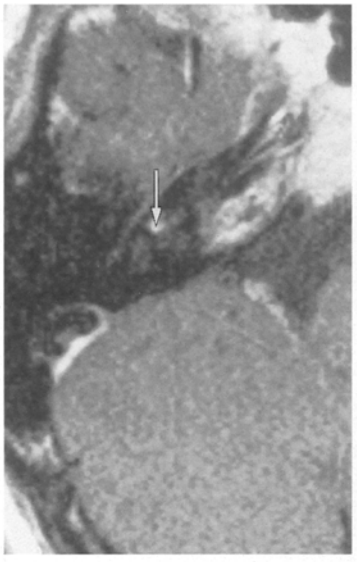
Tumors

Intralabyrinthine tumors are extremely rare [17]. In an autopsy series of 490 temporal bones, Leonard and Talbot found only 1 case of a vestibular tumor [18]. Most of the latter were discovered incidentally during surgery of endolymphatic hydrops or by autopsy. Until now, the poor spatial resolution of CT and the unspecific complaints of the patient referred for long-standing history of vertigo, dizziness, or SNHL explain the late and difficult diagnosis.

The most frequently encountered tumors are schwannomas. They originate from the vestibular nerve

Fig. 12a, b. Patient referred for a progressive SNHL affecting the low frequency tones. **a** Axial enhanced T1WI; **b** axial T2WI MR: nodular enhancing mass of the posterior part of the cochlea (**a**; *arrow*), **b** hypointense in T2WI *(arrowhead)* located at the origin of the cochlear nerve: cochlear slow expansive process

Fig. 13a–c. **a** Pre- and **b** postcontrast axial T1WI. **c** T2WI MR of a 40-year-old woman complaining of tinnitus occurring after a period of vertigo 14 years before. **a** Mild spontaneous high intermediate SI of the vestibule *(arrows)* intensely enhanced *(arrows)*. **b** Enhancement affecting the vestibule *(arrow)*, the lateral semicircular canal *(arrowhead)*. **c** T2WI reveals a complete decrease of the signal of posterior membranous labyrinth (ML) fluid *(arrows)*, and a mass of intermediate signal filling the lumen of the vestibule *(arrowheads)*: vestibular mass spreading to the ampulle of the lateral semicircular canal



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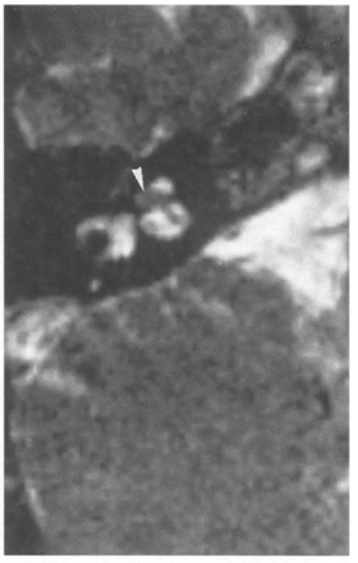

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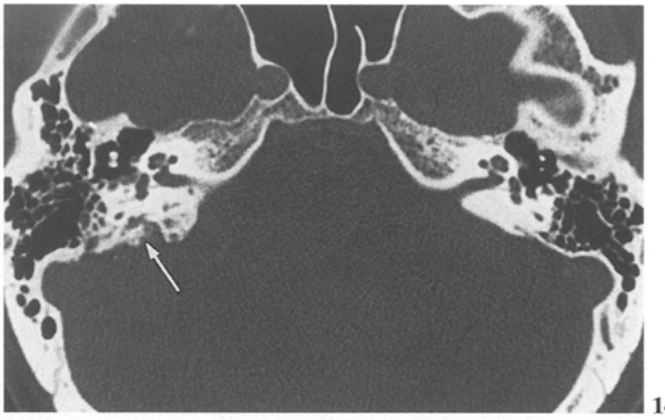

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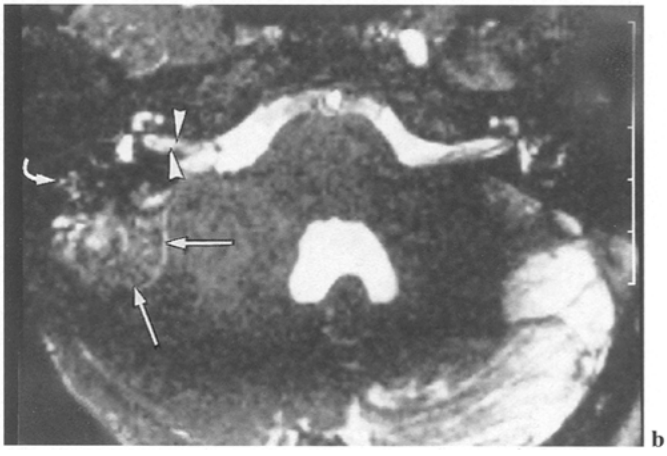

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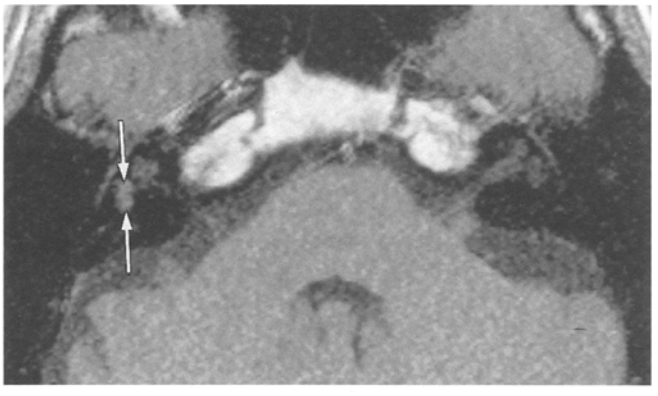

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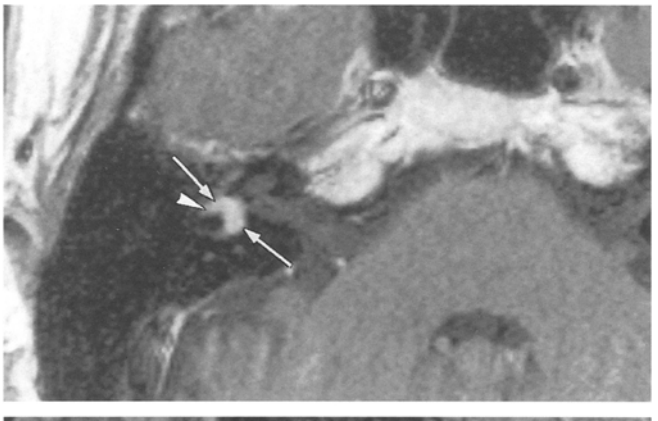

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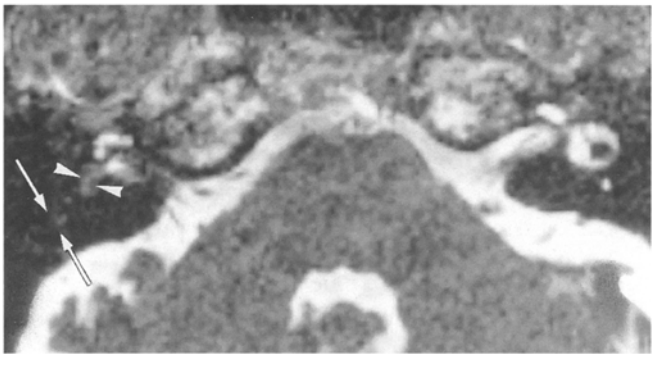

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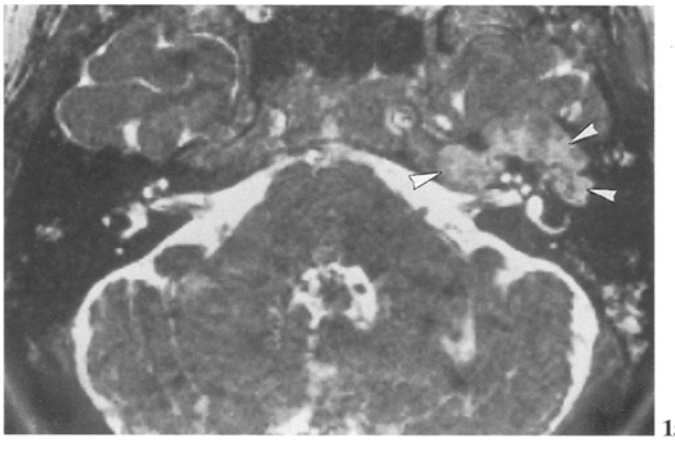

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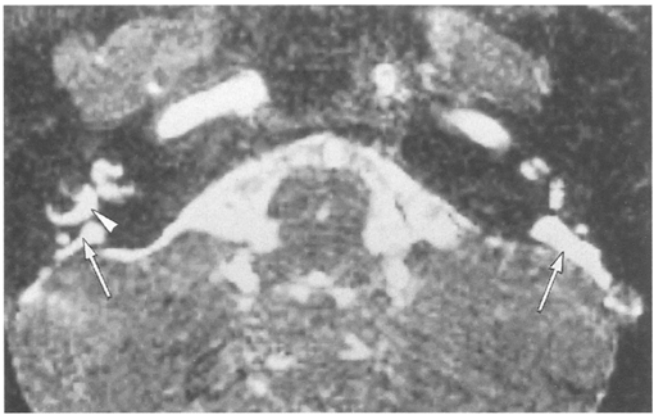
Fig. 14a, b. A 31-year-old woman suffering from Von Hippel-Lindau disease complaining of invading tinnitus and vertigo. **a** Axial CT scan shows a right bony lytic mass of the endocranial posteromedial part of the temporal bone *(arrow)*. **b** T2WI shows a large exophytic mass of intermediate signal spreading medially into the cerebellopontine angle (CPA; *arrows*), laterally into the mastoid and the middle ear *(curved arrow)*, and anteriorly into the IAC *(arrowheads)*: endolymphatic sac tumor

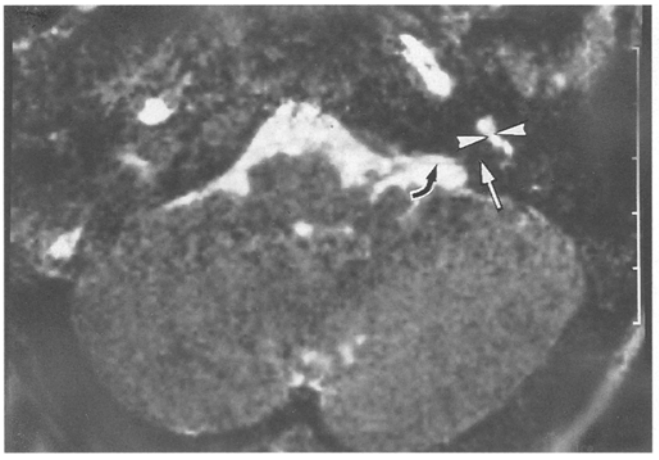
Fig. 15. Postsurgery cholesteatoma survey. Thin T2WI MR reveals a large intermediate signal mass *(arrowheads)* originating from the middle ear surrounding the anterior part of the labyrinth



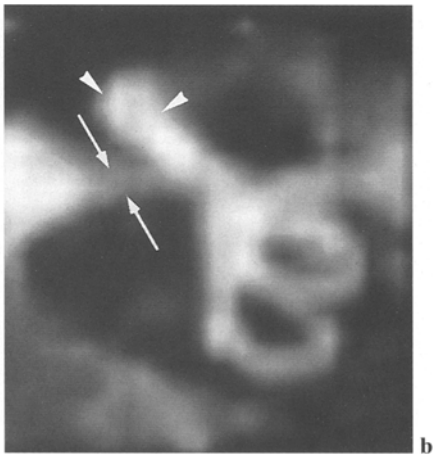
16

Fig. 16. A 37-year-old man referred for a bilateral profound SNHL. Thin T2WI MRI depicts bilaterally a large endolymphatic duct *(arrows)*. Associated right vestibular dilatation *(arrowhead)*: enlargement of the endolymphatic duct and of the vestibule

Fig. 17a, b. Young patient referred before cochlear implant surgery. **a** Thin T2WI MR and **b** 3D reconstruction MR depict the abnormal shape of the cochlea presenting only one cystic basal turn *(arrowheads)*. Note the extreme narrowing of the IAC *(arrows)* and the asymmetry of temporal bone. Left acousticovestibular nerve illustrated within the CPA *(curved arrows)*: bilateral Mondini malformation



17a



b

fibers of any of the cristae or maculae within the vestibule or the fibers of the cochlear nerve within the turns of the cochlea. Tumor spread within the internal auditory canal seems extremely rare [18]. The CT evaluation is negative because its contrast resolution is inadequate to depict these small tumors. Magnetic resonance imaging may show E/P fluid signal alterations suggestive of a space-occupying lesion. The affected labyrinthine part usually displays a slightly elevated SI on noncontrast short TR/TE images, enhancing after administration of gadolinium. The diagnosis of intralabyrinthine mass is clearly suggested by thin T2WI sections, however. They depict a round, space-occupying lesion of low SI surrounded by the normal high SI of E/P fluid.

In the cochlea they are located near the base, attached to the modiolar wall (Fig. 12) [19]. The spread into different parts of the cochlear turns explains the affected frequency of hearing loss.

In the vestibule the whole lumen can be obliterated [20]. The lesion may reach the ampulla of the semicircular canals, simulating an inflammatory process in post-gadolinium SE T1 images (Fig. 13). Clinical findings associated with a focal T2 signal decrease and a long-standing history of complaints exclude this latter diagnosis. In fact, the tumor extension affecting both the vestibula and the ampulla of the lateral semicircular canal is due to the anatomical status of the superior vestibular nerve, the superior vestibular branch supplying the utricule, and the superior and lateral semicircular canals.

Labyrinthine schwannomas may extend laterally into the middle ear through the oval window and/or the round window. Medially, it can extend into the IAC through the lateral porus acousticus. Extension of a cochlear mass into the vestibule and vice versa may be seen. The follow-up of the spontaneous evolution of some non-operated-on suggestive lesions shows a very slow growing rate.

Other intralabyrinthine tumors have been reported such as lipoma, although there is usually no fat within the labyrinth [21].

Recently, endolymphatic sac tumors [22] have been reported with a high frequency in Von Hippel Lindau disease. They appear as a lytic bony mass of the posteromedial part of the temporal bone presenting a geographic "moth-eaten patternO (Fig. 14) [23]. These slow-growing tumors involve the mastoid and the middle ear, the IAC, and the labyrinth. Extension into the cerebello pontine angle (CPA) is frequently encountered. In MR the lesion presents a heterogeneous signal on T1WI and T2WI, with massive tumoral enhancement. Serpiginous bands of signal voids suggest a hypervascular mass. Frequently, areas of high SI on T1WI and T2WI suggest intratumoral hemorrhage. Thin T2WI delineate the extension of the tumor into the mastoid and show its location around the aperture of the endolymphatic sac.

The labyrinth can be affected by tumors originating from surrounding structures; medially from IAC tumors

and laterally from tumors growing into the middle ear. This should be stressed prior to any surgical procedure when a patient is referred for a recurrent cholesteatoma (Fig. 15) or in IAC tumor evaluation.

Congenital malformations

These cause unilateral or bilateral hearing loss occurring alone or associated with vertigo and tinnitus. They affect the vestibular system more frequently (63 %) than the cochlea. Dilatation of the semicircular canals and large vestibular aqueduct [24, 25] are commonly encountered, whereas large vestibule, vestibule agenesis, and cochlear malformations are rarely depicted [26]. These anomalies are usually well depicted by HRCT and now by HR MRI T2WI. Partial or diffuse increase or decrease in the size of ML structures are seen correlated to shape anomalies.

In large vestibular aqueduct (Fig. 17), only MRI identifies the endolymphatic sac and duct dilatation within the vestibular aqueduct [26]. It presents as a fluid-filled cystic dilatation of high SI on T2WI of tubular shape, running parallel to the inner border of the endocranial part of the temporal bone. 3D ML reconstructions delineate the largest diameter of the endolymphatic sac and duct, the isthmus, and the junction with the saccule. The increase is usually more pronounced beyond the isthmus only affecting the endolymphatic sac (70 %). Associated vestibular dilatation (10 %) or cochlear dysplasia are less frequent. Endolymphatic sac enlargement is bilateral in 20 % of the cases studied [27]. Endolymphatic sac and duct dilatation is encountered in sensorineural hearing loss, although some authors have depicted such an abnormality in Meniere's disease.

In cochlear malformations, MR depicts the shape of the dilated portions of the ML and the status of the cochlear turns. A turn may be absent or cystic (Fig. 17), may communicate freely with the IAC, or may be associated with a dilatation of the subarachnoid spaces along the labyrinthine portion of the acousticofacial nerves suggesting a Gusher syndrome.

Moreover, MR of congenital ML malformations gives additional informations about the size of the IAC, the status of acousticovestibular nerves, and on eventual associated brain malformations. In cases of asymptomatic disease until adult life (e.g., large vestibular aqueduct syndrome), MRI may be the first-choice examination. Thus, such pathologies should always be carefully ruled out in patients complaining of progressive hearing loss, vertigo, or tinnitus and in cochlear implant candidates.

Cochlear implant

Computed tomography remains the first exam to perform in order to have a precise map of the inner ear and temporal bone anatomy as well as to depict any abnormality of the round window or calcification of the membranous labyrinth. Temporal bone MR exam adds useful information before surgery because only thin T2 sequences may depict focal or diffuse obliteration of the membranous labyrinth fluid space before calcification with a high degree of sensitivity (100 % in our experience). Moreover, MR plays a role in planning surgery, because a remaining postmeningitis labyrinthitis enhancement seems to predict a rapid evolution towards labyrinthine calcification. In malformative inner ear, CT and MR give complementary information especially for predicting Gusher's syndrome.

In conclusion, temporal bone MR may depict inner ear pathologies if thin sections on T1W and T2W sequences with a good SIN ratio and spatial resolution are performed. Both pre- and postgadolinium T1WI and thin T2WI are necessary to depict membranous labyrinth fluid space enhancement and obliteration. This is especially important when a cochlear implant surgery is planned. Therefore, management of vertigo, tinnitus, or of sensorineural hearing loss needs a careful screening of inner ear abnormality (Tables 1–3) in addition to brain examination. Magnetic resonance should be the first examination in patients presenting such symptoms. If MR is normal and if otospongiosis is suspected, will a CT scancomplete the inner ear examination to detect otospongiotic foci.

Table 1. Labyrinthine MR signal abnormalities

	High signal T1	Low signal T2	Postgadolinium enhancement	Bony abnormality
Hemorrhage	+	Subacute (–) Old (+)		–
Protein	+	+	–	–
Viral labyrinth.	–	–	+	–
Bacterial labyrinth.	–	Acute (–) Old (+)	++	+/–
Sclerosis/calcifications	–	++	+/– –	+/–
Autoimmune labyrinth.	+	+	+	–
Tumors	–	+	+	–
Otospongiosis	–	–	–/+	+ Low T1
Fibrous dysplasia		Decrease in size		

Labyrinth: Labyrinthitis

Table 2. Management of a sensorineural hearing loss. CISS: constructive interference in steady state; FSE: fast spin echo; IAC: internal auditory canal

Thin T2WI (CISS, FSE)
IAC abnormalities (soft tissue mass, neuritis)
Inner ear tumors
Thin pre- and post-T1WI
Labyrinthitis
Subacute hemorrhage
Bony abnormalities (otospongiosis, fibrous dysplasia)
Coronal T2WI
Brainstem abnormalities

Table 3. Management of vertigo

Sagittal SE T1WI
Cervicocranial junction abnormalities
Quadrigeminal cisterna abnormalities
Brain coronal or axial SE T2WI
Brain, brain stem, temporal area signal abnormalities
Thin axial/coronal T2 (CISS, FSE)
Vestibulo-acoustico nerve tumors
(schwannoma, hemangioma, metastasis)
Neuritis
Endolymphatic sac tumors
Vascular loop
Congenital inner ear abnormalities (e.g., large vestibular sac)
Thin pre- and postgadolinium SE T1WI
Vestibulo-acoustico nerve status Neuritis,
meninges abnormalities
Endolymphatic sac
Membranous labyrinth
Bony labyrinth (otospongiosis, fibrous dysplasia)

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