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The radiologic evaluation of tinnitus

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Abstract Tinnitus ("ringing in the ears") is a prevalent symptom in the general population, and often brings patients to medical attention. Many causes of tinnitus are evident radiographically. The most frequently-encountered causes of tinnitus are discussed, and imaging recommendations are provided.

Keywords Tinnitus · Temporal Bone Imaging · MRI · CT

Introduction

Tinnitus is defined as any auditory sensation perceived in the absence of external stimuli [1]. Although the sensation is most frequently described as "ringing," patients may describe buzzing, hissing, whistling, humming, or cricket-like sounds [2]. In Western countries, approximately 12% of the population is affected by tinnitus [3–5]. However, the severity of symptoms varies widely: some patients are barely bothered by their tinnitus, while others are driven to suicide [6].

Most tinnitus patients are in their 7th or 8th decade of life, and men are more frequently affected than women [5]. Tinnitus is rarely reported in children [7]. Many patients have symptoms for years before seeking medical care [2]. It is rare that tinnitus reflects a serious underlying medical condition, but treatment is frequently unsuccessful [2].

Audiometric evaluation of patients may be sufficient to demonstrate a source of tinnitus (such as otosclerosis, Meniere's disease, or noise-induced hearing loss), but most patients with disabling symptoms will require radiologic evaluation [8, 9]. Even in carefully selected patient populations, however, many patients will have no radiologic abnormality to explain their tinnitus. The goal of imaging is to identify treatable causes of tinnitus; the workup is pursued with an understanding and expectation of its relatively low yield.

Many of the radiologic findings associated with tinnitus can be seen in asymptomatic individuals, or in patients with contralateral complaints. This reinforces the theory that the

primary abnormality responsible for tinnitus is abnormal brainstem processing of normal sounds [1]. It is critical to correlate radiographic findings with clinical symptomatology before suggesting that a radiologic finding is responsible for a patient's tinnitus.

Classification

Tinnitus may be classified on two axes: pulsatile versus continuous, and subjective versus objective [10]. Pulsatile tinnitus is a discrete, repetitive sound that accompanies the patient's pulse. Continuous (or non-pulsatile) tinnitus refers to all other rhythms, usually a constant, unrelenting noise.

Subjective tinnitus is appreciated only by the patient, and cannot be perceived by other observers. Objective tinnitus is the result of a sound that can be heard by the physician. Objective tinnitus may be quiet enough to be perceptible only with a stethoscope, or may be loud enough to be commented upon by the patient's friends and family.

Subjective tinnitus is almost never pulsatile, so some authors prefer to use the terms pulsatile and continuous only in the setting of objective tinnitus [8]. Subjective continuous tinnitus is the most frequently-encountered type of tinnitus, and the least likely to have a treatable cause [11, 12].

Imaging parameters

The choice of imaging modality is determined by the classification of the patient's tinnitus. Continuous tinnitus, when it has a radiographically identifiable cause, is most likely the result of a cerebellopontine angle (CPA) or internal auditory canal (IAC) tumor [13]. Brainstem pathology such as stroke or multiple sclerosis is also a consideration. Thus, contrast-enhanced magnetic resonance (MR) is the modality of choice [14]. This examination is often supplemented with MR arteriography (MRA) and MR venography (MRV) [15]. Although vascular abnormalities are more likely to cause *pulsatile* tinnitus, the addition of MR angiography provides a more thorough evaluation and decreases the likelihood of re-imaging.

The MR imaging protocol at our institutions consists of sequences dedicated to the brain and sequences dedicated to the temporal bones. For the brain, 5-mm T2-weighted and fluid-attenuation inversion recovery (FLAIR) transverse images, T1-weighted sagittal images, and contrast-enhanced fat-suppressed T1-weighted transverse images are used. For the temporal bones, 2- to 3-mm T1-weighted transverse images, contrast-enhanced fat-suppressed T1-weighted transverse and coronal images, and high-resolution thin-section T2-weighted transverse images [preferably, steady-state imaging such as fast imaging employing steady state acquisition (FIESTA) or continuous interference in steady state (CISS)] are used. Minimal interslice gap should be applied to all sequences.

Pulsatile tinnitus is most frequently the result of a vascular abnormality or vascular tumor. Because these lesions often involve the middle ear and otic capsule, where MR is less sensitive, computed tomography (CT) is the preferred modality. Intravenous contrast is used to better depict vascular structures.

When using volumetric CT scanners (64 detector rows), images are acquired in the transverse plane 6 min after the administration of contrast. This longer delay allows for more uniform penetration of any disrupted blood-brain barrier. Soft tissue reconstructions are performed at 3-mm slice thickness, and bone reconstructions are performed at 1-mm slice thickness. Bone kernels are also used for 1-mm coronal reformatted images. When using CT scanners with fewer than 16 detector rows, direct acquisition in both transverse and coronal planes is required. Additional images are obtained through the entire head on a larger field of view so that the entire calvarium and scalp are included in the study. Occasionally, vascular structures in these regions may be responsible for pulsatile tinnitus.

If atherosclerosis is suspected as a cause of pulsatile tinnitus [16], CT arteriography (CTA) of the neck and head should be considered. If compression of the jugular vein is a possible cause [17], a neck CT may be performed that extends to the upper mediastinum to include the entire course of the jugular veins. Because dural arterio-venous malformations (AVMs) can be radiographically occult on cross-sectional imaging, conventional catheter angiography is sometimes needed to exclude these lesions [18]. The decision process for choosing an imaging modality is summarized in Table 1.

Table 1 Recommendations for the radiographic evaluation of tinnitus. See text for specific protocols

Clinical scenario	Imaging modality
Continuous tinnitus	Contrast-enhanced MR of brain and temporal bones
Pulsatile tinnitus	Contrast-enhanced CT of head and temporal bones
Normal CT of head and temporal bones	Consider CTA for atherosclerosis, CT neck for jugular compression, or catheter angiogram for dural AVM

Diagnostic considerations

Pulsatile tinnitus

Vascular anomalies

There are several vascular structures that may be aberrant (having an unusual course), dehiscent (lacking a normal bony covering), or stenosed (of reduced caliber). These presumably cause tinnitus by producing turbulent blood flow, but the causative relationship between vascular anomalies and tinnitus has not been clearly established [19]. In particular, it remains unclear why an anomaly that has been present for the patient's entire life would cause the sudden onset of tinnitus. Nevertheless, these vascular structures are often implicated as a source of symptomatology.

Aberrant internal carotid artery An aberrant internal carotid artery (ICA) is a congenital anomaly in the course of the ICA that allows the artery to present as a middle ear mass [20]. The normal ICA enters the skull base through the carotid foramen and takes an antero-medial course through the petrous portion of the temporal bone. If the ICA fails to develop correctly during fetal life, then a collateral vessel, the inferior tympanic artery, enlarges to take its place. The enlarged inferior tympanic artery enters the skull through its own foramen (the inferior tympanic canniliculus), courses through the medial portion of the middle ear, and then rejoins the petrous ICA (Fig. 1).

The greatest danger from aberrant ICA is iatrogenic-if the aberrant artery is mistaken for a glomus tympanicum tumor, biopsy may be performed with potentially disastrous results [20]. Accurate imaging interpretation is critical to avoid this error.

Dehiscent internal carotid artery An aberrant ICA may or may not have a bony covering as it courses through the mesotympanum. If it lacks the bony covering, it is considered dehiscent. ICAs with a normal course may also be focally dehiscent and thus visible from the middle ear, but this is less common than dehiscence in the setting of an aberrant ICA [21].

Stenosed internal carotid artery Atherosclerosis of the ICA is almost ubiquitous in elderly patients. Tinnitus can result from severe compromise of any segment of the ICA, including the carotid bifurcation and the petrous ICA [16]. Calcified plaques are the hallmark of atherosclerosis (Fig. 2), but the disease is more precisely characterized with CTA. Treatment of the stenosis may obviate symptoms [22].

Aberrant anterior inferior cerebellar artery The anterior inferior cerebellar artery (AICA) arises from the mid-basilar artery. It normally loops into the CPA, and often enters the IAC (Fig. 3) [23]. Some authors believe that if branches of the AICA are adjacent to cranial nerves, pulsations from the artery will causes cranial nerve

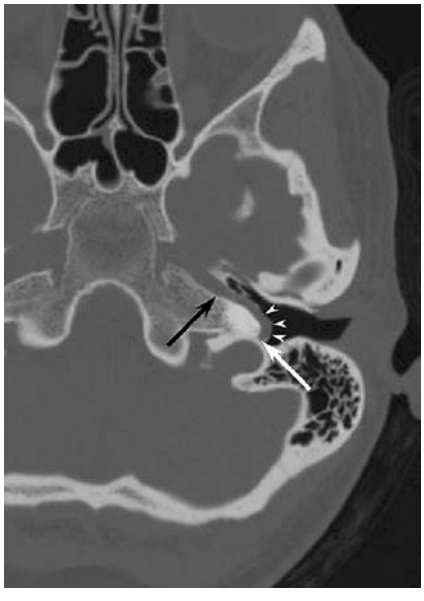

Fig. 1 Aberrant internal carotid artery. Axial bone-window CT demonstrates an abnormal course of the petrous ICA. The artery enters the middle ear through the inferior tympanic canniliculus (*white arrow*), just anterolateral to the jugular bulb. It courses through the mesotympanum (*arrowheads*) to recombine with a diminutive petrous internal carotid artery (*black arrow*) that then continues on its normal course

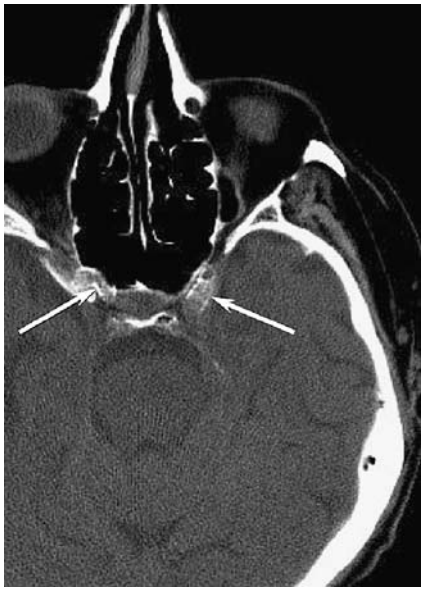

Fig. 2 Atherosclerosis. Axial unenhanced CT shows peripheral calcifications of the supraclinoid segments of both ICAs (*arrows*). Atherosclerotic disease, either intracranial or extracranial, may be a source of tinnitus

dysfunction [24]. This mechanism has been proposed to explain and treat trigeminal neuralgia, as well as tinnitus [25].

High-riding jugular bulb The jugular bulb is an expansion of the dural venous sinuses at the junction between the sigmoid sinus and the internal jugular vein. A high-riding jugular bulb (also known as an aberrant jugular bulb) is an extension of the jugular bulb superior to the level of the tympanic annulus [19]. This abnormality is often best observed in coronal reformatted images, where the relationship of the jugular bulb to the middle and inner ear contents can be best visualized (Fig. 4).

Dehiscent jugular bulb If the thin bone between the jugular bulb and the middle ear (the jugular plate) is absent, venous pulsations from the bulb may be transmitted to the ear. Occasionally, the vein may herniate slightly through the defect to form a small diverticulum. Dehiscent jugular bulb with or without jugular bulb diverticulum may be a cause of tinnitus. This abnormality is best identified on coronal reformatted images (Fig. 5).

Aberrant sigmoid sinus If the sigmoid sinus is displaced anteromedially from its normal course, it runs near the endolymphatic sac and the posterior semicircular canal and may transmit venous pulsations to the inner ear. Although MRV can show the aberrant course of the vessel, CT is more useful because it better delineates the relationship between the aberrant vessel and the otic capsule.

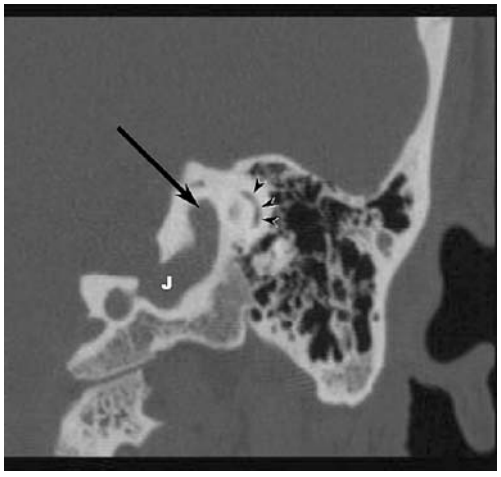

Fig. 4 Aberrant (high-riding) jugular bulb. Coronal bone-window CT demonstrates an extension (*arrow*) of the jugular bulb (*J*) superiorly into the petrous portion of the temporal bone. This high-riding bulb extends above the level of the posterior semicircular canal (*arrowheads*)

Stenosed dural sinus The intracranial dural venous sinuses vary in size in different patients. Often, paired sinuses are asymmetric within a single patient. The transverse sinuses are particularly prone to this asymmetry, but most patients have no related symptoms. However, if a patient has a focal stenosis of a dural venous sinus, turbulent flow may cause tinnitus [12, 26]. This abnormality can be assessed with MRV or CT venography (Fig. 6).

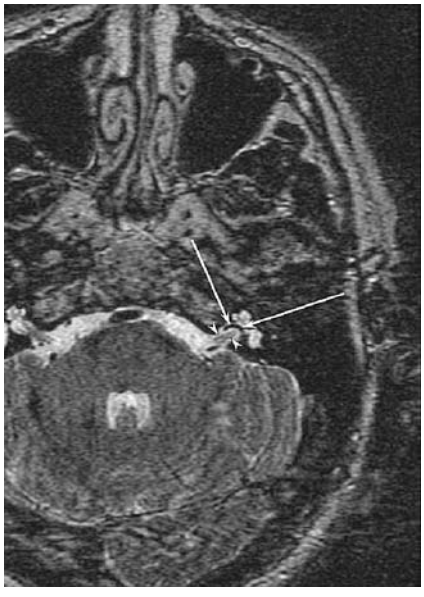

Fig. 3 Vascular loop. High-resolution axial T2-weighted MR image demonstrates a curvilinear signal void (*arrows*) deep in the IAC, near the base of the cochlea. This represents the anterior inferior cerebellar artery extending into the IAC. The artery can be distinguished from the nearby nerves (*arrowheads*) by the complete lack of signal in the artery, and its curved course

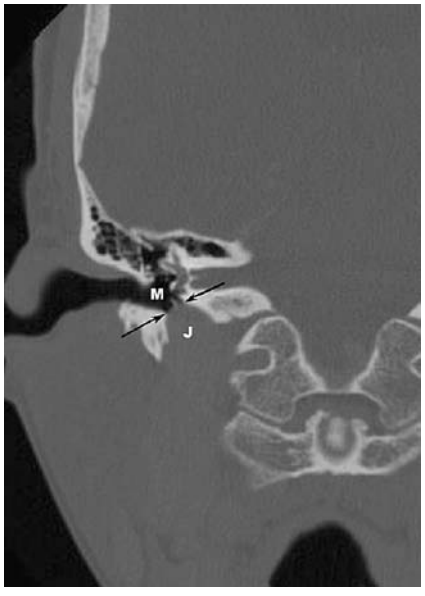

Fig. 5 Dehiscent jugular bulb. Coronal bone-window CT demonstrates absence of the normal thin bone (*arrows*) separating the jugular bulb (*J*) from the middle ear (*M*). Venous pulsations may be transmitted to the middle ear to cause tinnitus

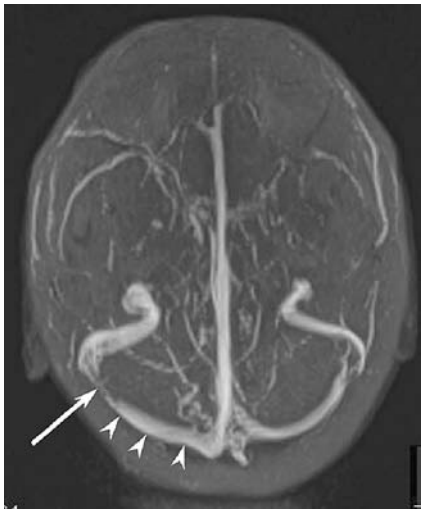

Fig. 6 Dural venous sinus stenosis. Supero-inferior maximum intensity projection of an MR venogram in a patient complaining of bilateral tinnitus demonstrates a focal narrowing (*arrow*) of the right transverse sinus (*arrowheads*) near the transition to the sigmoid sinus. A similar stenosis is seen on the left. A causative relationship between dural venous stenosis sinus stenosis and tinnitus has not been definitively proven

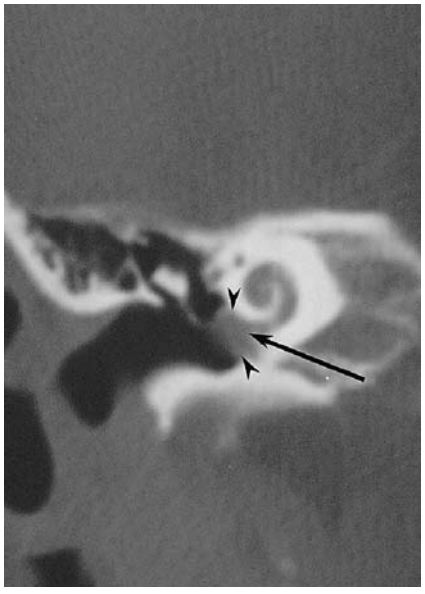

Fig. 7 Persistent stapedial artery. Coronal bone-window CT demonstrates a mass (*arrowheads*) in the medial mesotympanum. Adjacent images show the mass to be tubular in configuration. The abnormal structure divides and widens the crura of the stapes, and passes over the cochlear promontory (*arrow*)

Persistent stapedial artery The normal stapedial artery is a fetal collateral that runs through the middle ear between the crura of the stapes. The stapedial artery normally involutes, but a persistent stapedial artery may be seen as an isolated aberrant vessel in the middle ear, or may be seen in association with an aberrant internal carotid artery [27]. This abnormality is recognized by the unique relationship of the aberrant vessel to the stapes (Fig. 7). Because the persistent stapedial artery replaces the middle meningeal artery, the ipsilateral foramen spinosum is absent in these patients.

Other vascular pathology

Vascular malformation AVMs are abnormal communications between arteries and veins. They may be extracranial, dural, or parenchymal, and any of these varieties may be responsible for tinnitus [28, 29]. Extracranial AVMs are usually evident clinically, but may manifest as enlarged braches of the external carotid artery on skull base or neck CT. Parenchymal AVMs are characterized by tangles of enlarged or numerous arteries and veins around a central nidus within the brain itself. Smaller parenchymal AVMs may be inevident on cross-sectional imaging, and may only be seen as early venous filling on conventional angiography.

Dural AVMs, also called arterio-venous fistulas, are notoriously difficult to diagnose radiographically. Conventional angiography is the most sensitive modality [18], but even this technique may overlook small lesions. Dilated leptomeningeal, medullary, or extracranial vessels are the most frequently identified finding [30]. It is important to remember that dural AVMs are frequently fed by branches of the external carotid artery, and the occipital artery is often implicated.

Aneurysm Berry aneurysms of the Circle of Willis usually cause symptoms by rupturing or by mass effect. However, if the aneurysm is adjacent to the 8th cranial nerve, it may transmit pulsations to the nerve and cause pulsatile tinnitus [8].

Carotid artery dissection Internal carotid artery dissection usually causes pain, stroke, or Horner's syndrome. However, the turbulent flow that arises from a carotid dissection may also cause tinnitus in a manner similar to that of atherosclerotic disease [31]. CTA may demonstrate the lesion as an intimal flap within the lumen of the artery (Fig. 8). MR is more sensitive for dissection, but unenhanced fat-suppressed T1-weighted images are needed to demonstrate the thrombus in the false lumen. A crescentic focus of high signal on these images is characteristic of carotid dissection (Fig. 9).

Fibromuscular dysplasia Fibromuscular dysplasia (FMD) is an idiopathic inflammatory angiopathy affecting medium-sized vessels. After the renal arteries, the internal carotid arteries are the most common arteries to be affected. Patients most frequently present with intracranial ischemia, but tinnitus is the next most frequent manifestation [19]. Angiographic techniques such as CTA or MRA are needed to make the diagnosis. Although catheter

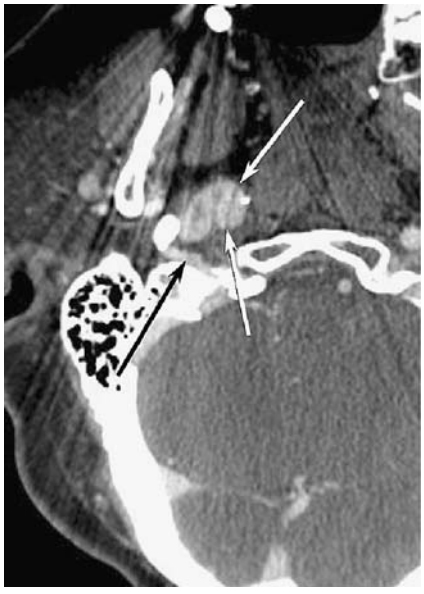

Fig. 8 Carotid artery dissection. Axial image from CT angiography demonstrates a curvilinear filling defect (*white arrows*) in the internal carotid artery. The filling defect represents an elevated intimal flap, and is distinguished from the dental streak artifact by its curved configuration. The internal jugular vein (*black arrow*) is displaced by the tortuous internal carotid artery

angiography is not required for diagnosis, angioplasty is the preferred treatment [32]. Radiographically, the affected arteries have a characteristic beaded appearance from multiple focal stenoses separated by regions of variable dilation (Fig. 10).

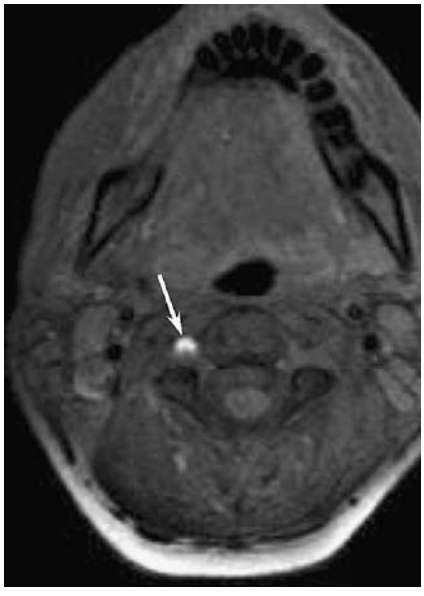

Fig. 9 Vertebral artery dissection. Axial fat-suppressed unenhanced T1-weighted image demonstrates a crescentic focus of high signal (*arrow*) adjacent to the right vertebral artery. This represents methemoglobin in the false lumen of a dissection

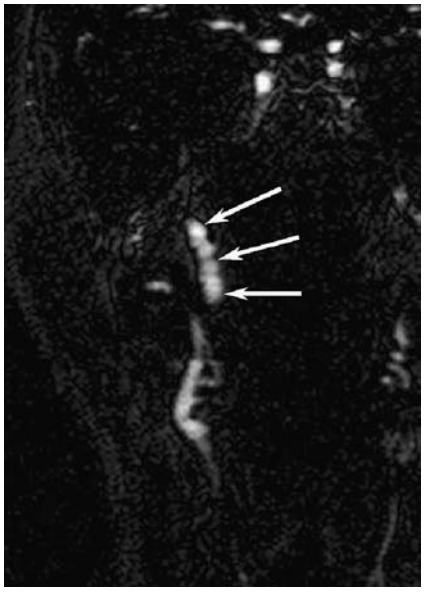

Fig. 10 Fibromuscular dysplasia. Coronal contrast-enhanced T1-weighted image from an MR arteriogram demonstrates a beaded appearance (*arrows*) to the cervical ICA. This appearance can be seen in both vasospasm and fibromuscular dysplasia

Venous hum A venous hum is objectively perceptible over the inferior portion of the jugular vein in approximately half of the normal adult population. Patients with increased venous flow, such as those with thyrotoxicosis or increased cardiac output from anemia, may experience this hum as tinnitus [17]. Alternatively, compression of the proximal jugular vein may causes turbulent flow resulting in tinnitus. Although cross-sectional imaging of the neck and upper chest infrequently identifies a treatable cause for venous hum, this may be considered in patients whose initial imaging is unrevealing (see Table 1).

Vascular neoplasms

Most neoplasms cause continuous, rather than pulsatile, tinnitus. However, highly vascular tumors may cause pulsatile tinnitus if they are located near the auditory apparatus.

Paraganglioma Paragangliomas (chemodectomas, glomus tumors) are benign vascular tumors that arise in predictable locations in the head and neck. Carotid body tumors and glomus vagale tumors do not cause tinnitus, but glomus tympanicum and glomus jugulare tumors may be responsible.

Glomus tympanicum tumors arise on the medial aspect of the middle ear, along the course of Jacobson's nerve (a branch of cranial nerve IX). Although the cochlear promontory is considered the most frequent site for this tumor (Fig. 11), it may arise anywhere in the medial

mesotympanum [33]. Because glomus tympanicum tumors are surrounded by bone and air, CT is the preferred imaging modality. Although the tumors are quite vascular, enhancement may not be subjectively evident in smaller tumors of the middle ear.

Glomus jugulare tumors arise in the jugular bulb. Although benign, these tumors aggressively erode the bone of the lateral jugular bulb and extend toward the hypotympanic recess of the middle ear (Fig. 12). If a glomus jugulare tumor is otoscopically visible within the middle ear, it is called a glomus jugulo-tympanicum tumor. On CT, the characteristic pattern of bone erosion is the most important clue to the diagnosis. On MR, large central flow voids interspersed with high-signal parenchyma provide a characteristic "salt and pepper" appearance (Fig. 13) [34]. The intense enhancement of the tumor may make it difficult to distinguish from the adjacent internal jugular vein on both CT and MR, and may make the inferior extent of tumor difficult to identify.

Ossifying hemangioma Ossifying hemangiomas of the facial nerve arise in two characteristic locations: the geniculate ganglion and the IAC [35]. Those tumors arising in the geniculate ganglion cause facial nerve paralysis; those arising in the IAC cause sensorineural hearing loss. Either location may, in rare instances, be associated with tinnitus. On MR, ossifying hemangioma appears as an enhancing mass along the expected course of the facial nerve (Fig. 14a). Unfortunately, this is a nonspecific finding that may easily be confused with schwannoma of the facial or vestibular nerves. CT demonstrates a characteristic stippled pattern of calcification that can suggest the correct diagnosis (Fig. 14b) [36].

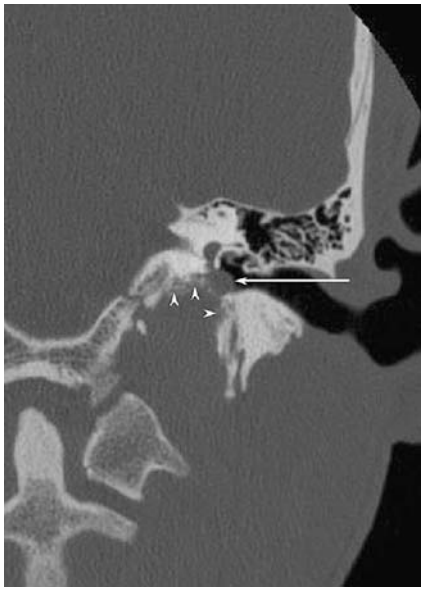

Fig. 12 Glomus jugulare tumor. Coronal bone-window CT demonstrates erosion of the lateral and superior borders of the jugular bulb (*arrowheads*). This tumor, which originated in the jugular bulb, has extended into the hypotympanic recess (*arrow*), and would be visible otoscopically

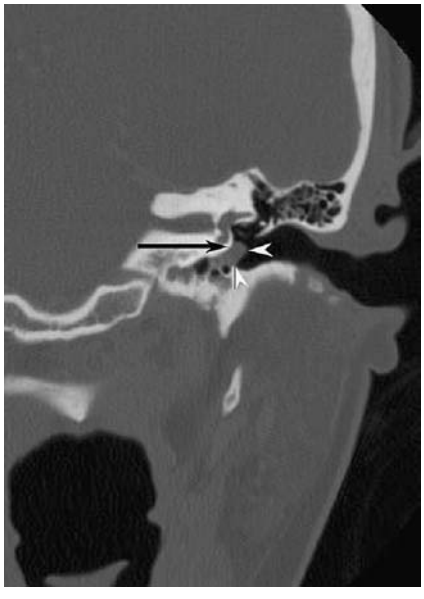

Fig. 11 Glomus tympanicum tumor. Coronal bone-window CT demonstrates a soft-tissue-density mass (*arrowheads*) in the medial mesotympanum, overlying the cochlear promontory (*arrow*). This is the expected location of Jacobson's nerve, from which glomus tympanicum tumors arise

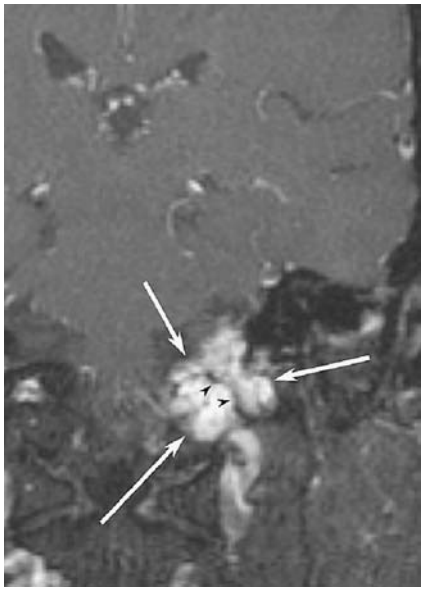

Fig. 13 Glomus jugulare tumor. Coronal contrast-enhanced fat-suppressed T1-weighted MR image demonstrates an intensely-enhancing mass (*arrows*) centered in the jugular bulb. Branching serpentine internal signal voids (*arrowheads*) provide the classic salt and pepper appearance of a paraganglioma

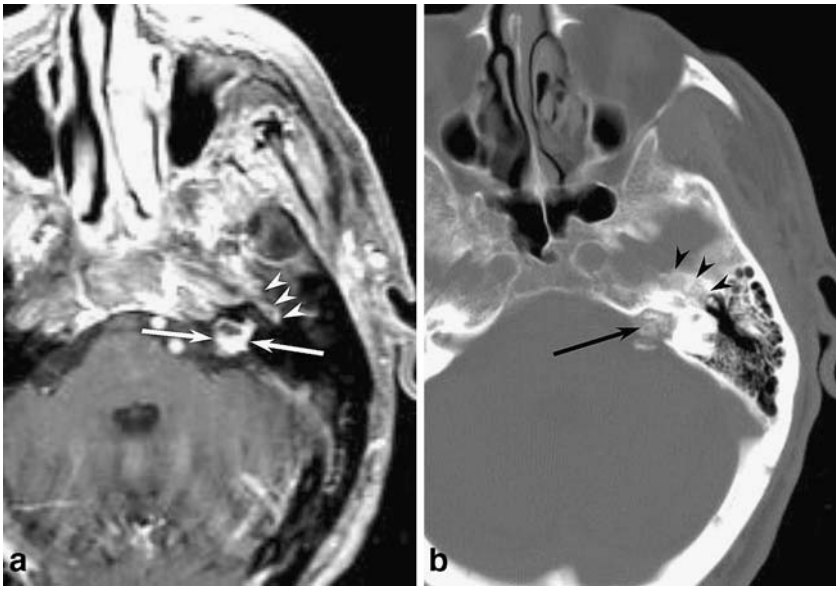

Fig. 14a,b Ossifying hemangioma of the facial nerve. **a** Axial fat-suppressed contrast-enhanced T1-weighted image demonstrates a heterogeneous focus of increased enhancement (*arrows*) at the porus acousticus. Additional abnormal enhancement is seen at the geniculate ganglion and along the tympanic segment of the facial nerve (*arrowheads*). This appearance is consistent with both schwannoma and hemangioma. **b** Axial bone-window CT at the same location demonstrates the typical stippled calcifications (*arrow*) of ossifying hemangioma. The same calcification pattern is seen at the geniculate ganglion and along the course of the tympanic segment of the facial nerve (*arrowheads*)

Cavernous hemangioma Cavernous hemangioma is a rare benign neoplasm of the middle ear that mimics a glomus tympanicum clinically, radiographically, and on otoscopic examination [19]. The diagnosis is often made only after resection of the tumor.

Osseous dysplasias

Some osseous dysplasias are associated with increased vascularity of the involved bone. If the petrous portion of the temporal bone is affected, this increased vascularity may transmit pulsations to the inner ear.

Otosclerosis Otosclerosis is an idiopathic infiltrative process of the petrous bone that characteristically causes mixed sensorineural and conductive hearing loss. Sometimes, tinnitus is a presenting complaint [37]. Temporal bone CT shows areas of lucency in the normally-dense petrous bone. The fenestral form of the disease shows abnormal density in the region of the fissula ante fenestram, just anterior to the oval window [38]. This can be a very subtle finding that requires careful attention and a suggestive clinical history (Fig. 15). The cochlear form of the disease shows a halo of lucency around the cochlea (Fig. 16) and may also show sclerosis of the oval window. Although MR may demonstrate high intraosseous T2 signal in the cochlear form of otosclerosis, subtle disease will be inconspicuous on MR; CT is the preferred modality.

Paget's disease Paget's disease most frequently affects the appendicular skeleton or the skull, but it may involve the temporal bone and cause tinnitus [19]. Paget's disease is evidenced by heterogeneously decreased attenuation on CT, usually throughout the temporal bone (Fig. 17). The abnormal vascular communications responsible for tinnitus in these patients may also cause congestive heart failure.

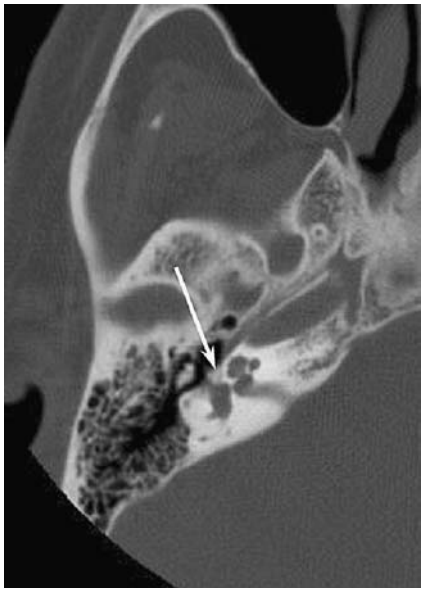

Fig. 15 Fenestral otosclerosis. Axial bone-window CT demonstrates a small focus of heterogeneously decreased attenuation (*arrow*) in the region of the fissula ante fenestram. The bone in this region should be just as dense as the rest of the otic capsule. The bone in the region of the fissula ante fenstram is also overgrown, which can cause conductive hearing loss if it impedes the motion of the stapes footplate

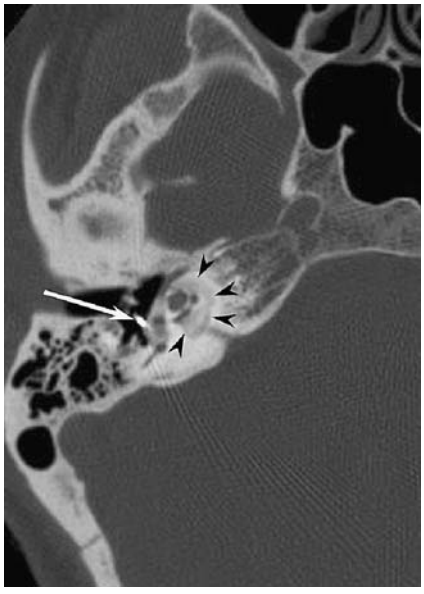

Fig. 16 Cochlear otosclerosis. Axial bone-window CT demonstrates a halo of lucent bone (*arrowheads*) around the cochlea. This patient has already received a stapes prosthesis (*arrow*), which is used to treat the fenestral form of otosclerosis. Fenestral otoclerosis is almost always present when cochlear otosclerosis is seen

Elevated intracranial pressure

In some series, benign intracranial hypertension is the most frequent identifiable cause of pulsatile tinnitus [39]. However, patients with elevated intracranial pressure more frequently present with headache and visual disturbances. Cross-sectional imaging may identify a cause for increased pressure, such as an obstructing mass lesion, but imaging is frequently negative and the diagnosis is made by evaluating the opening pressure at lumbar puncture. Findings sometimes associated with intracranial hypertension include flattening of the posterior globes (papilledema), empty sella, and settling of the cerebellar tonsils.

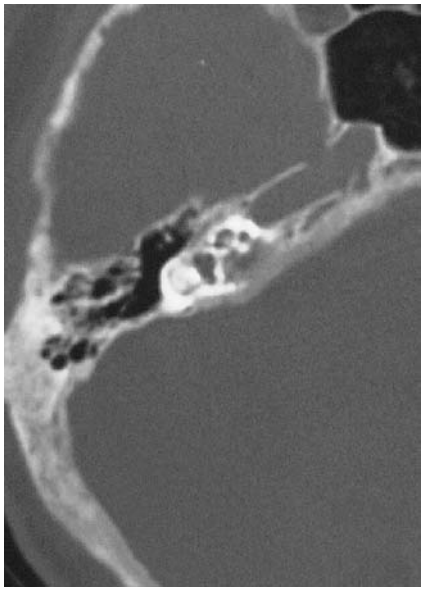

Fig. 17 Paget's disease of the temporal bone. Axial bone-window CT demonstrates abnormal lucency throughout the temporal bone, with relative sparing of the otic capsule

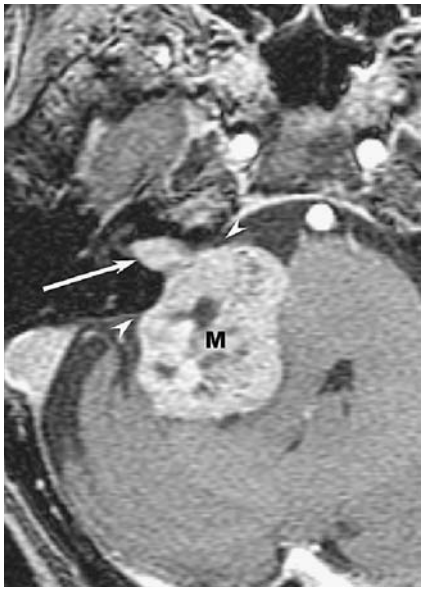

Fig. 18 Vestibular schwannoma. Axial contrast-enhanced fat-suppressed T1-weighted image demonstrates a heterogeneously-enhancing mass (*M*) in the cerebellopontine angle extending deep into the IAC (*arrow*). Note the acute angles formed between the tumor and the underlying bone (*arrowheads*). Acute angles, heterogeneous enhancement, and IAC extent are more characteristic of schwannoma than of meningioma, which is the major differential consideration

Continuous tinnitus

Neoplasm

Patients with continuous tinnitus are less likely to have an identifiable source than patients with pulsatile tinnitus. The most important diagnosis to exclude in the setting of continuous tinnitus is a mass of the CPA, and MR is ideal for this evaluation. Vestibular schwannomas are the most frequent example of CPA masses causing tinnitus (Fig. 18) [13]. Unfortunately, excision of the CPA tumor does not necessarily improve the tinnitus [40]. Presumably, tinnitus arises from damage to the 8th cranial nerve, as can be seen following microdecompression surgery [24].

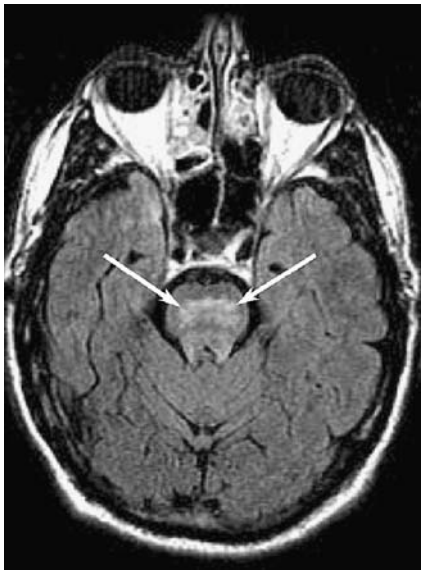

Fig. 19 Brainstem microvascular disease. Axial FLAIR MR image demonstrates increased signal (*arrows*) in the region of the pontine decussation. This is a classic location for ischemic microvascular disease

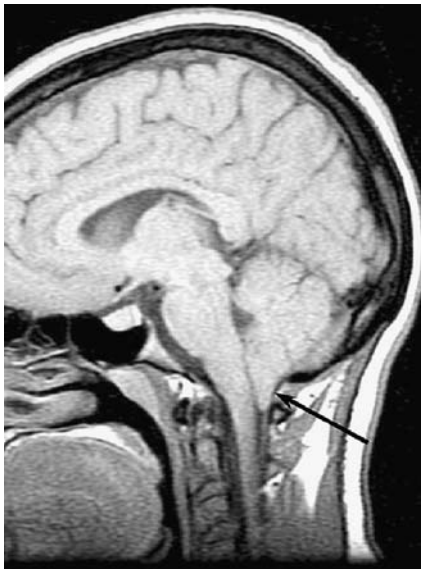

Fig. 20 Chiari I malformation. Sagittal T1-weighted MR image demonstrates herniation of the cerebellar tonsils (*arrow*) below the level of the foramen magnum. Tinnitus is among the many clinical manifestations of this abnormality

Temporomandibular joint

Diseases of the temporomandibular joint (TMJ) have a myriad presentation, and frequently mimic other diseases, such as sinusitis. TMJ degeneration is often overlooked as a potential cause of head and neck symptomatology. Patients with degenerative disease of the temporomandibular joint may have continuous tinnitus as a presenting complaint [41]. The mechanism for this relationship is not well understood. Degenerative TMJ disease is usually evident on CT of the face or temporal bone, but MR of the joint is more sensitive for disc disease [42].

Brainstem disease

Abnormal brainstem responses to auditory stimuli may be responsible for some forms of continuous tinnitus [1]. Thus, diseases that directly affect the brainstem can be a source of symptoms. In particular, advanced microvascular disease, as from hypertension, diabetes, or hypercholesterolemia, may be implicated (Fig. 19). Demyelinating diseases such as multiple sclerosis frequently affect the brainstem, and may cause tinnitus [43]. Brainstem strokes have also been implicated. MR imaging of the brainstem, particularly FLAIR sequences, is sensitive for brainstem pathology.

Chiari I malformation (ectopic cerebellar tonsils) has myriad manifestations, including tinnitus. Sagittal MR images are ideal for establishing this diagnosis (Fig. 20).

Diseases without radiologic manifestations

Many of the causes of tinnitus have no radiologic manifestations. Many drugs, most notably aspirin, ibuprofen, loop diuretics, and aminoglycosides, are known to cause tinnitus. Muscular tinnitus is another cause. It has two forms: middle ear tinnitus and palatal myoclonus, both of which are associated with rhythmic contractions of muscles around the skull base and may produce objective tinnitus [44]. A patulous Eustachian tube may produce a variety of extraneous sounds [8]. Valvular heart disease may be implicated in pulsatile tinnitus [8]. Sensorineural hearing loss, in and of itself, is a frequent source of tinnitus, particularly in elderly patients. Patients with conductive hearing loss due to middle ear infection or impacted cerumen may also complain of tinnitus [2].

Summary

Tinnitus is a prevalent symptom in the general population, and often brings patients to medical attention. Although some patients with tinnitus do not require imaging, many causes of tinnitus are evident radiographically. Patients with continuous tinnitus should undergo MR imaging of the brain, usually accompanied by MRA and MRV. Patients with pulsatile tinnitus should undergo contrast-enhanced CT of the temporal bones and brain.

References

1. Bauer CA (2004) Mechanisms of tinnitus generation. Curr Opin Otolaryngol Head Neck Surg 12:413–417
2. Lockwood AH, Salvi RJ, Burkard RF, Tinnitus (2002) N Engl J Med 347:904–910
3. Axelsson A, Ringdahl A (1989) Tinnitus-a study of its prevalence and characteristics. Br J Audiol 23:53–62
4. Hazell J (1990) Tinnitus and disability with ageing: adaptation and management. Acta Otolaryngol Suppl 476:202–208
5. Adams PF, Hendeshot GE, Marano MA (1999) Current estimates from the National Health Interview Survey, 1996. National Center for Health Statistics, Hyattsville
6. Luxon LM (1993) Tinnitus: its causes, diagnosis, and treatment. BMJ 306:1490–1491
7. Baguley DM, McFerran DJ (1999) Tinnitus in childhood. Int J Pediatr Otorhinolaryngol 49:99–105
8. Lockwood AH, Burkard RF, Salvi RJ (2004) Imaging tinnitus. In: Snow JB (ed) Tinnitus: theory and management. Decker, Hamilton, pp 255–264
9. Weissman JL (1997) Imaging of Meniere's disease. Otolaryngol Clin North Am 30:1105–1116
10. Heller AJ (2003) Classification and epidemiology of tinnitus. Otolaryngol Clin North Am 36:239–248
11. Shiley SG, Folmer RL, McMenomey SO (2005) Tinnitus and hyperacusis. In: Cummings CW (ed) Otolaryngology: head and neck surgery. Elsevier, Philadelphia, pp 2832–2847
12. Dietz RR, Davis WL, Harnsberger HR, Jacobs JM, Blatter DD (1994) MR imaging and MR angiography in the evaluation of pulsatile tinnitus. AJNR Am J Neuroradiol 15:879–889
13. Weissman JL (1996) Hearing loss. Radiology 199:593–611
14. Marsot-Dupuch K, Vignaud J, Mehdi M, Pharaboz C, Meyer B (1996) Magnetic resonance imaging assessment of labyrinthine pathology. Eur Radiol 6:621–630
15. Shin EJ, Lalwani AK, Dowd CF (2000) Role of angiography in the evaluation of patients with pulsatile tinnitus. Laryngoscope 110:1916–1920
16. Sismanis A, Stamm MA, Sobel M (1994) Objective tinnitus in patients with atherosclerotic carotid artery disease. Am J Otol 15:404–407
17. Nehru VI, al-Khaboori MJ, Kishore K (1993) Ligation of the internal jugular vein in venous hum tinnitus. J Laryngol Otol 107:1037–1038
18. Koenigsberg RA (1996) Spontaneous pulsatile tinnitus secondary to a dural malformation not visualized by magnetic resonance angiography. Clin Imaging 20:95–98
19. Levine SB, Snow JB Jr (1987) Pulsatile tinnitus. Laryngoscope 97:401–406
20. Branstetter BF (2004) Aberrant internal carotid artery. In: Harnsberger HR (ed) Diagnostic imaging: head and neck. Amirsys, Salt Lake City, pp 1.2.38–1.2.41
21. Lapayowker MS, Liebman EP, Ronis ML, Safer JN (1971) Presentation of the internal carotid artery as a tumor of the middle ear. Radiology 98:293–297
22. Emery DJ, Ferguson RD, Williams JS (1998) Pulsatile tinnitus cured by angioplasty and stenting of petrous carotid artery stenosis. Arch Otolaryngol Head Neck Surg 124:460–461
23. Nowe V, De Ridder D, Van de Heyning PH et al (2004) Does the location of a vascular loop in the cerebellopontine angle explain pulsatile and non-pulsatile tinnitus? Eur Radiol 14:2282–2289
24. Moller MB, Moller AR, Jannetta PJ, Jho HD (1993) Vascular decompression surgery for severe tinnitus: selection criteria and results. Laryngoscope 103:421–427
25. Brookes GB. Vascular-decompression surgery for severe tinnitus. Am J Otol 1996; 17:569–76
26. Russell EJ, De Michaelis BJ, Wiet R, Meyer J (1995) Objective pulse-synchronous "essential" tinnitus due to narrowing of the transverse dural venous sinus. Int Tinnitus J 1:127–137
27. Lo WW, Solti-Bohman LG, McElveen JT Jr (1985) Aberrant carotid artery: radiologic diagnosis with emphasis on high-resolution computed tomography. Radiographics 5:985–993
28. Shah SB, Lalwani AK, Dowd CF (1999) Transverse/sigmoid sinus dural arteriovenous fistulas presenting as pulsatile tinnitus. Laryngoscope 109:54–58
29. Roy D, Lavigne F, Raymond J (1993) Pulsatile tinnitus and dural arteriovenous fistula of the transverse sinus. J Otolaryngol 22:409–412
30. Kwon BJ, Han MH, Kang HS, Chang KH (2005) MR imaging findings of intracranial dural arteriovenous fistulas: relations with venous drainage patterns. AJNR Am J Neuroradiol 26:2500–2507
31. Vories A, Liening D (1998) Spontaneous dissection of the internal carotid artery presenting with pulsatile tinnitus. Am J Otolaryngol 19:213–215
32. Slovut DP, Olin JW (2005) Fibromuscular dysplasia. Curr Treat Options Cardiovasc Med 7:159–169
33. Weissman JL, Hirsch BE (1998) Beyond the promontory: the multifocal origin of glomus tympanicum tumors. AJNR Am J Neuroradiol 19:119–122
34. Olsen WL, Dillon WP, Kelly WM, Norman D, Brant-Zawadzki M, Newton TH (1987) MR imaging of paragangliomas. AJR Am J Roentgenol 148:201–204
35. Shelton C, Brackmann DE, Lo WW, Carberry JN (1991) Intratemporal facial nerve hemangiomas. Otolaryngol Head Neck Surg 104:116–121
36. Curtin HD, Jensen JE, Barnes L Jr, May M (1987) "Ossifying" hemangiomas of the temporal bone: evaluation with CT. Radiology 164:831–835
37. Sparano A, Leonetti JP, Marzo S, Kim H (2004) Effects of stapedectomy on tinnitus in patients with otosclerosis. Int Tinnitus J 10:73–77
38. Harnsberger HR (2004) Fenestral Fotosclerosis. In: Harnsberger HR (ed) Diagnostic imaging: head and neck. Amirsys, Salt Lake City, pp 1.2.138–1.2.141
39. Sismanis A (1998) Pulsatile tinnitus. A 15-year experience. Am J Otol 19:472–477
40. Baguley DM, Humphriss RL, Axon PR, Moffat DA (2005) Change in tinnitus handicap after translabyrinthine vestibular schwannoma excision. Otol Neurotol 26:1061–1063
41. Parker WS, Chole RA (1995) Tinnitus, vertigo, and temporomandibular disorders. Am J Orthod Dentofacial Orthop 107:153–158
42. Rao VM, Bacelar MT (2004) MR imaging of the temporomandibular joint. Neuroimaging Clin N Am 14:761–775
43. Weber PC, Adkins WY Jr (1997) The differential diagnosis of Meniere's disease. Otolaryngol Clin North Am 30:977–986
44. Howsam GD, Sharma A, Lambden SP, Fitzgerald J, Prinsley PR (2005) Bilateral objective tinnitus secondary to congenital middle-ear myoclonus. J Laryngol Otol 119:489–491