

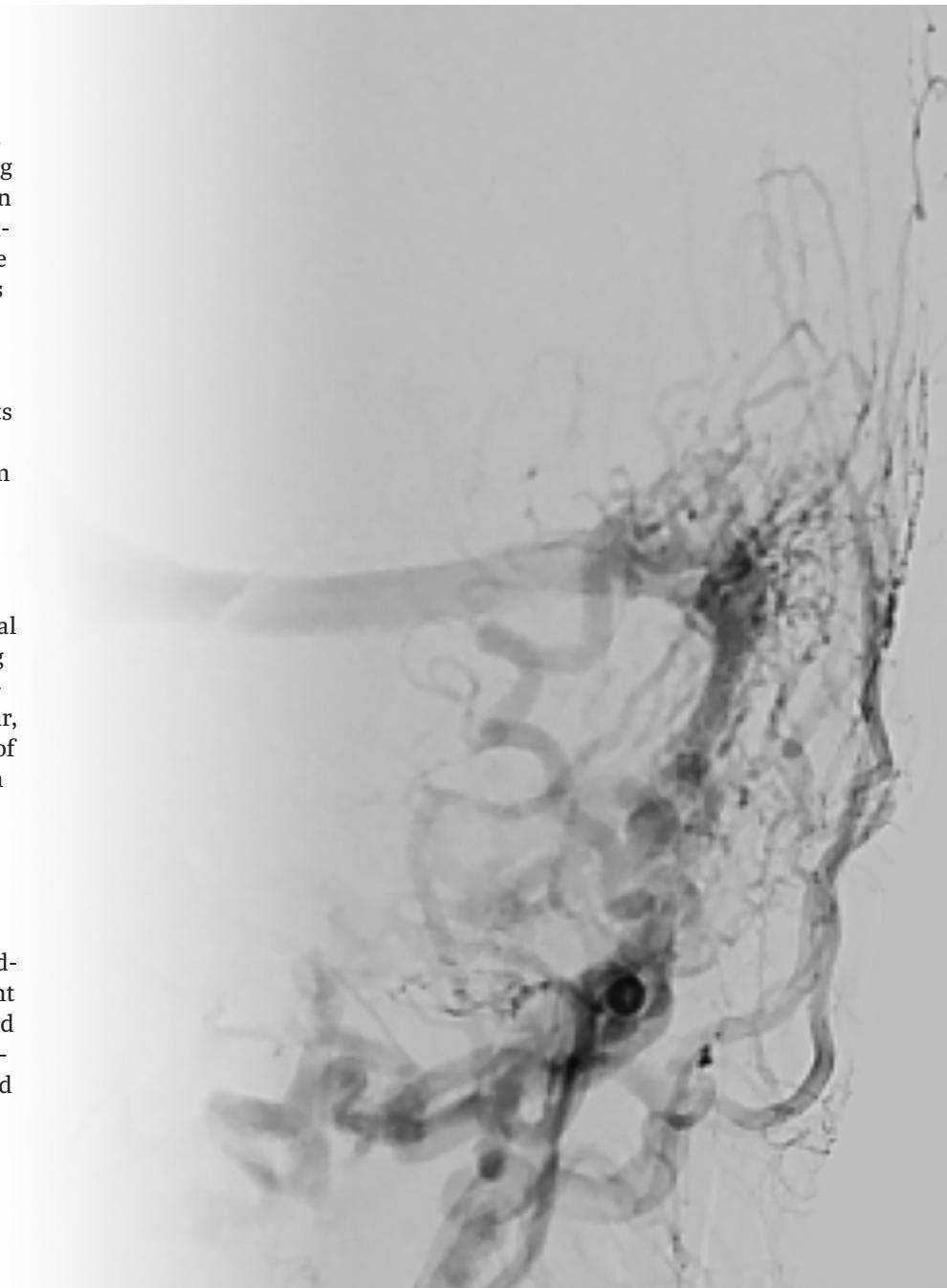
Imaging of Pulsatile Tinnitus

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Tinnitus is a common complaint that approximately three-fourths of adults will experience at some point in their life. While for many it is a mild nuisance, for some it can be debilitating, affecting cognition and quality of life, increasing stress, and leading to anxiety, depression, and in severe circumstances even suicide. Pulsatile tinnitus refers to the perception of a heartbeat-like sound without external stimulus. Although less common than nonpulsatile tinnitus, pulsatile tinnitus raises concern for underlying disease that can have a high risk of causing the patient harm if undiagnosed, and most of these patients will have positive findings at imaging. While these findings are often subtle, identifying them can have a meaningful impact on the patient's quality of life. The literature on pulsatile tinnitus is changing rapidly with improved imaging techniques and novel minimally invasive treatment options. A careful history and physical examination together with appropriate imaging are therefore critical in identifying the underlying cause. With emerging surgical, endovascular, and supportive technologies, the vast majority of patients with bothersome pulsatile tinnitus can be cured or have their symptoms ameliorated. The objective of this narrative review is to present a comprehensive analysis of the currently available literature on pulsatile tinnitus, with a focus on understanding its pathophysiologic mechanisms, diagnostic pathways, imaging findings, and the spectrum of available management strategies and ultimately to propose a structured framework that aids radiologists as well as clinicians in identifying an underlying diagnosis and guiding management of these patients.

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Supplemental Material**Test Your Knowledge****TEACHING POINTS**

- Most patients with pulsatile tinnitus will have an identifiable and often treatable cause of tinnitus at imaging. A careful review of the history and physical examination results will improve the diagnostic yield of imaging.
- MR angiography or MR venography is the most appropriate initial test for patients presenting with pulsatile tinnitus and an audible bruit or alteration of tinnitus with head position or internal jugular vein compression.
- A clinical history of high-pitched tone in the presence of a bruit and absence of hearing loss should increase the radiologist's suspicion for the presence of an arteriovenous fistula or malformation.
- A low humming or whooshing sound is more likely to be associated with a venous cause, while a higher-pitched tinnitus is more likely to be associated with an arterial cause.
- Temporal bone CT is the most appropriate initial imaging study for the patient presenting with both pulsatile tinnitus and conductive hearing loss. Vascular imaging may still be warranted to exclude dural arteriovenous fistula or arteriovenous malformation.

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Abbreviations: DSA = digital subtraction angiography, IAC = internal auditory canal, ICA = internal carotid artery, IIH = idiopathic intracranial hypertension, MIP = maximum intensity projection, 3D = three-dimensional

Introduction

Tinnitus is the internal perception of a sound without an external auditory stimulus. Up to 75% of adults may experience either isolated or repeated chronic tinnitus (1). Globally, tinnitus is estimated to affect up to 740 million people, with approximately 120 million having severe tinnitus (2). There is scant evidence evaluating the total burden of tinnitus on patients' personal lives and the health care system (3). Tinnitus can affect cognition; decrease quality of life; contribute to stress, depression, and anxiety; and when severe and persistent, can even lead to suicide in extreme cases (4–6).

Tinnitus that is rhythmic and synchronous with a patient's heartbeat is characterized as *pulsatile tinnitus*. Patients often describe their perceived sound as rhythmic whooshing. Pulsatile tinnitus can be further characterized as subjective when heard only by the patient or objective when it can be heard by auscultation (7). Patients with pulsatile tinnitus experience anxiety and depression at higher rates than patients who have tinnitus of either kind (6,8). There is a wide gamut of pathologic conditions that represent the possible causes of pulsatile tinnitus; in more than 75% of patients with pulsatile tinnitus, a specific causative abnormality can be found at imaging (9–13).

Evaluation for every patient with tinnitus starts with a careful history and physical examination. For those with pulsatile tinnitus, this should include auscultation of the neck, ear, and surrounding structures (14,15). Given the high rate of positive imaging findings in the setting of pulsatile tinnitus, the radiologist should make every effort to obtain information regarding the clinical scenario. The radiologist should strive to identify within the medical record the patient's symptomatic side. The history and physical examination results should

be reviewed and noted for exacerbating and alleviating factors that will help guide review of the imaging findings.

High-pitched tinnitus with objective components, such as an audible bruit, is more likely due to an arterial or arteriovenous junction cause. Some neoplasms may cause objective tinnitus, for example paragangliomas. Tinnitus due to a venous cause is more likely to be low pitched. Pulsatile tinnitus that is altered with head positioning, standing, or lying down will support a venous cause. A venous cause is also supported by alteration of pulsatile tinnitus with jugular vein compression.

The presence of hypercoagulability may suggest the presence of a venous cause, while trauma or neck manipulation would support a possible arterial cause, for example an internal carotid or vertebral artery dissection. Conductive hearing loss itself can lead to pulsatile tinnitus and is more common with bony structural causes, neoplastic causes, or middle ear masses. These are all details that can help clue the radiologist into possible causes and can help guide the radiologist's eye to the underlying pathologic condition, which can sometimes be subtle.

Once the history is clarified, appropriate imaging can be recommended, and the imaging findings can be reviewed systematically to evaluate for potential underlying treatable disease (Fig 1). Most patients with pulsatile tinnitus will have an identifiable and often treatable cause of tinnitus at imaging (9–13). A careful review of the history and physical examination results will improve the diagnostic yield of imaging. Shared decision making regarding the extent of imaging workup and treatment options should be undertaken with the patient, considering the patient's perceived disability, the risk features of the specific underlying pathologic condition, and the patient's broader health picture.

This review provides a framework for imaging evaluation in the setting of pulsatile tinnitus. The goal of this review is to help the diagnostic radiologist establish a systematic approach for evaluating imaging studies with appropriate clinical history to increase sensitivity in detecting potential causes of pulsatile tinnitus. In addition to reviewing the common causes of pulsatile tinnitus, findings across different modalities are synthesized with an emphasis on cross-sectional imaging, and appropriate recommendations are proposed. Common imaging findings, normal variants, and common misses and misinterpretations or pitfalls and artifacts are reviewed to help increase the radiologist's accuracy in interpreting imaging studies performed for the purposes of evaluating pulsatile tinnitus.

Pulsatile tinnitus can be attributed to any cause of altered conduction or blood flow within the temporal bone, either from altered arterial or venous vascular flow, an abnormal arteriovenous connection, a hypervasculär mass in the temporal bone, thinning of the temporal bone cortex, or loss of normal ossicular conduction (16). This review provides a systematic framework for image interpretation, considering the patient's history and analysis of vascular structures, soft tissues and bones for mass lesions, and the temporal bone for any bony abnormalities that can result in pulsatile tinnitus.

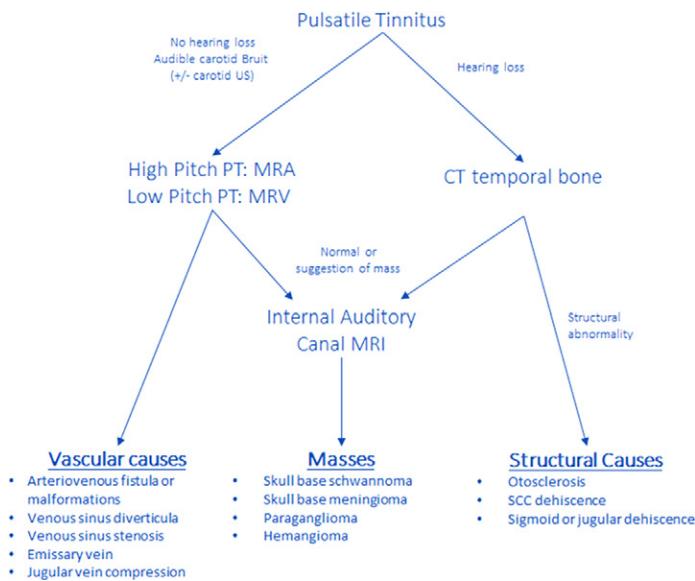


Figure 1. Summary of the algorithm for diagnosing pulsatile tinnitus (PT). MRA = MR angiography, MRV = MR venography, SCC = superior semicircular canal.

Vascular Causes

The most recognized causes of pulsatile tinnitus are vascular. Any pathologic condition that alters the flow of blood in or around the petrous bone can cause pulsatile tinnitus. These can be classified into arterial, arteriovenous junction, or venous causes (17–19). While pulsatile tinnitus is most commonly caused by alterations in venous flow, the most clinically concerning lesions are arteriovenous fistulas.

Imaging Considerations

In a patient with pulsatile tinnitus with absence of conductive hearing loss, the initial workup should include MR angiography or MR venography to assess for venous or arterial causes of pulsatile tinnitus, depending on the supporting clinical feature. If these are unavailable or a patient prefers to not undergo MRI, CT angiography or CT venography may be ordered. MR angiography or MR venography is the most appropriate initial test for patients presenting with pulsatile tinnitus and an audible bruit or alteration of tinnitus with head position or internal jugular vein compression (Tables 1, 2) (20–23).

Although we do not routinely perform dynamic (four-dimensional) MR angiography or CT angiography at our institution, these are emerging techniques that can be employed to better study the flow dynamics of contrast material and can increase sensitivity for detection of shunting lesions, although at the cost of increased imaging time and radiation (1). Even at single-phase imaging, assessment of the attenuation of veins compared with that of adjacent arteries can sometimes reveal shunting from an arteriovenous fistula or malformation (Fig S1). An advantage of dynamic MR angiography or CT angiography is temporal resolution of contrast material physiology without the inherent risks associated with conventional digital subtraction angiography (DSA). However, both the temporal and spatial resolution are less than those of DSA, and patients with positive results at conventional imaging ultimately benefit from DSA for confirmation and treatment planning.

Table 1: Vascular Imaging and Search Patterns Based on Clinical History

High-pitched PT with bruit: MR angiography	Carotid artery course and caliber for dissection, atherosclerosis, stenosis, or fibromuscular dysplasia
	Superior and inferior petrosal vein enhancement
	Transverse and sigmoid sinus enhancement
Low-pitched PT improved with jugular compression or head turning: MR venography	Transverse and sigmoid sinus caliber
	Transverse and sigmoid sinus filling defects
	Jugular bulb course and caliber
	Internal jugular vein caliber

Note.—PT = pulsatile tinnitus.

Table 2: Vascular Causes of Pulsatile Tinnitus

Arterial	Carotid atherosclerotic stenosis
	Carotid dissection
	Fibromuscular dysplasia
	Carotid aneurysm
	Aberrant carotid artery course and persistent stapedial artery
Arteriovenous junction	Dural arteriovenous fistula
	Arteriovenous malformation
Venous	Venous sinus stenosis
	Venous sinus diverticulum
	Mastoid or condylar emissary vein
	Extracranial jugular vein compression

When venous imaging is performed in a patient with a clinical history supporting a vascular cause, attention should be given to the transverse and sigmoid sinuses, jugular bulb, and internal jugular vein to assess for the presence of transverse sinus stenosis, sigmoid sinus or jugular bulb diverticula, a high-riding jugular bulb, or compression of the internal jugular vein. Mastoid, occipital, or condylar emissary veins can also be noted. Although emissary veins are commonly seen incidentally and represent normal anatomic variants, they can be potential causes of pulsatile tinnitus, particularly in the presence of a stenosis (18,19,21,24–26). Normal-variant dominant marginal or occipital sinus drainage is worth noting, as stenosis of these can also cause pulsatile tinnitus (27). Occasionally, patients can reduce the pulsatile tinnitus from mastoid emissary veins with pressure behind the ear. This is not as commonly tested as jugular vein compression and may be suggested if this is found. If an audible cervical bruit is present, duplex US evaluation of the cervical vasculature may be helpful as well (28).

At MR angiography, the course, caliber, extent of atherosclerosis, and morphology of the carotid artery should be

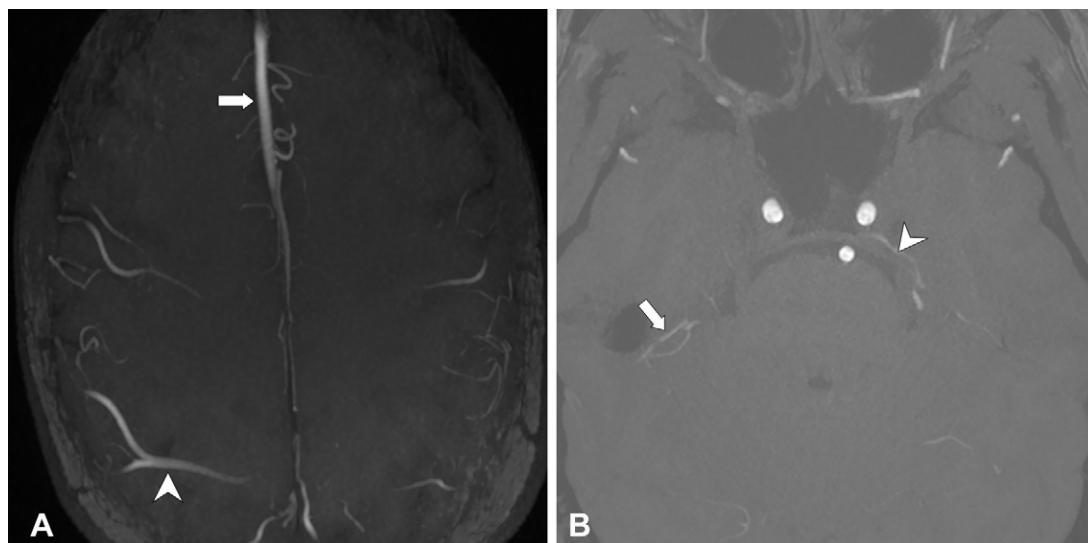


Figure 2. Artifacts on three-dimensional (3D) time-of-flight maximum intensity projection (MIP) MR angiograms in a 34-year-old patient with left-sided intermittent tinnitus. **(A)** Axial MIP image shows that flow in the superior sagittal sinus (arrow) and cortical veins (arrowhead) near the skull vertex is normal. It should not be misconstrued as shunting unless there is asymmetry. **(B)** Axial MIP image at the skull base shows flow-related enhancement in the right superior petrosal sinus (arrow) and along the left free margin of the tentorium (arrowhead). These findings were misinterpreted as concerning for shunting, and the patient was referred for conventional DSA, which confirmed absence of shunting. These findings can be mentioned, particularly if there is asymmetry and the finding is ipsilateral to the symptoms.

carefully evaluated. Fibromuscular dysplasia, atherosclerosis, or an aberrant course should be commented on, as they may account for the pulsatile tinnitus (28,29). MR angiography has greater sensitivity and specificity in detection of shunting lesions and should be the first-line modality for imaging of pulsatile tinnitus when a vascular cause is suspected (9,23,28,30–32). Although CT angiography is less sensitive, this is sometimes the first imaging study ordered. We evaluate every CT angiography study by first assessing the quality of the contrast material bolus and assessing the peak of enhancement to help guide interpretation, with particular attention to any asymmetric or early venous enhancement, as this may represent a shunting lesion.

While MR angiography has greater sensitivity and specificity for arteriovenous junction lesions, it is also more prone to artifacts. Retrograde flow can result in high signal intensity in the left sigmoid sinus or the superior or inferior petrosal sinus and can be mistaken for a shunting lesion (Fig 2) (33). The specificity of this finding can be improved by modifying MRI or MR angiography protocols for evaluating pulsatile tinnitus to include arterial spin labeling (ASL) (9,23), which can confirm shunting if the suspected vein is also seen to have high signal intensity at ASL. If ASL is not available or performed and a vein is seen with high signal intensity at three-dimensional (3D) time-of-flight MR angiography, the authors advocate having a low threshold for mentioning the finding, particularly if it corresponds to the side of pulsatile tinnitus.

Flow reversal can also be seen in cortical veins and the anterior superior sagittal sinus near the vertex, usually with fainter signal intensity than arteries at the same level. Vascular loops of the anterior inferior cerebellar artery entering the internal auditory canal (IAC) do not warrant mention. These

are found incidentally in many patients with no correlating symptoms and should be considered normal anatomic variants (34). If a vascular cause is suspected, invasive diagnostic angiography may allow confirmation of the suspected finding and provide insight into potential treatment options and remains the standard of reference (35).

Arteriovenous Junction Causes

Up to 20% of patients undergoing workup for pulsatile tinnitus may have a dural arteriovenous fistula, an acquired abnormal connection between a dural arterial supply and adjacent vein (26,36,37), often in the setting of a preceding trauma. Arteriovenous malformations near or involving the skull base may also manifest with pulsatile tinnitus (Fig 3) (1). A clinical history of high-pitched tone in the presence of a bruit and absence of hearing loss should increase the radiologist's suspicion for the presence of an arteriovenous fistula or malformation. All cases of pulsatile tinnitus should have a high index of suspicion for arteriovenous junction entities, as these can represent dangerous lesions that are at risk for rupture, resulting in significant morbidity and even in death (15,23,26).

If a dural arteriovenous fistula is identified, its location, the presence of cortical vein drainage, and the presence of venous ectasia should be described to assist with classification schemes stratifying the risk of hemorrhage (38). The direction of venous drainage can be elucidated with invasive angiography (Figs 4, S2). For arteriovenous malformations, the size of the nidus, the location relative to eloquent brain regions, and superficial or deep venous drainage should be noted to assist with grading schemes that help predict operative risk and guide management options (39). In patients with high

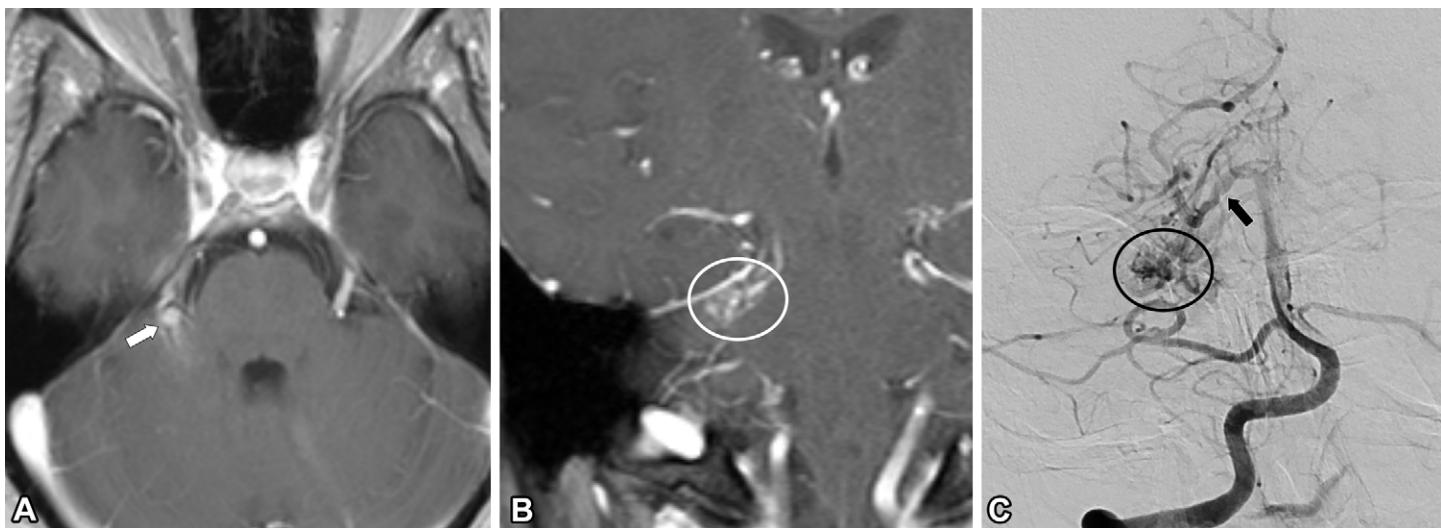


Figure 3. Arteriovenous malformation of the right middle cerebellar peduncle in a 34-year-old patient who underwent MRI with an IAC protocol for workup of right-sided pulsatile tinnitus. **(A)** Axial contrast-enhanced T1-weighted image shows an area of enhancement (arrow) in the right middle cerebellar peduncle. **(B)** Coronal postcontrast MR image shows a tangle of vessels (oval). The patient underwent DSA. **(C)** Frontal angiogram obtained with a right vertebral artery injection shows a cerebellar arteriovenous malformation (oval) with an early-draining deep cerebral vein (arrow). The patient elected radiation therapy.

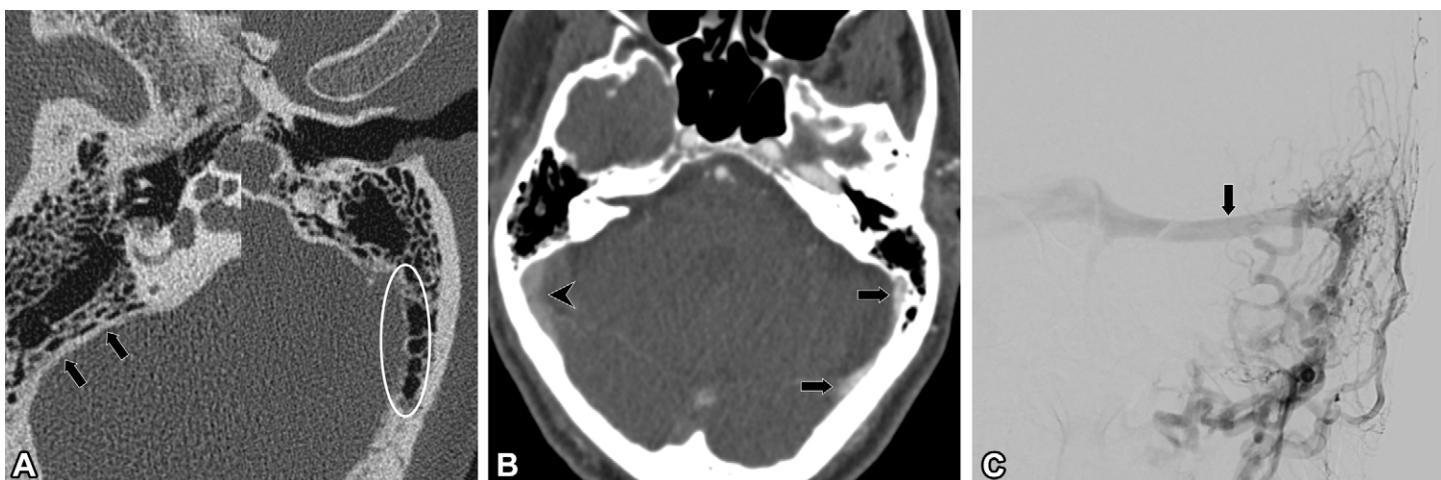


Figure 4. Left dural arteriovenous fistula in an 80-year-old patient with left-sided pulsatile tinnitus. **(A)** CT image of the temporal bone shows subtle irregularity of the left sigmoid sinus wall (oval). The normal right sigmoid sinus wall is shown for comparison (arrows). **(B)** Axial image from subsequent CT angiography shows asymmetric enhancement of the left transverse and sigmoid sinuses (arrows) relative to the right transverse and sigmoid sinuses (arrowhead). **(C)** DSA image shows a dural arteriovenous fistula with retrograde drainage into the left transverse sinus (arrow). Coil embolization was performed from a transvenous approach (Fig S2), and the patient's symptoms resolved.

pretest probability of a vascular lesion or positive findings at noninvasive imaging, conventional angiography can both be diagnostic and present therapeutic options. When diagnostic angiography is being performed for evaluating pulsatile tinnitus from suspected intracranial hypertension, a fluoroscopic lumbar puncture can be performed concurrently to measure opening pressures. Venous sinus pressure measurements are often obtained to guide therapeutic interventions (18,40,41).

Venous Causes

More commonly, pulsatile tinnitus is caused by alterations in venous flow, with lesions located anywhere from the transverse sinus to any point of the internal jugular vein through the mediastinum (12,14,16).

Obtaining a clinical history and reviewing the clinician's physical examination findings are critical before review of imaging findings. Idiopathic intracranial hypertension (IIH), formerly referred to as *pseudotumor cerebri*, is much more common in women of childbearing age with obesity (20,42–44). Common imaging findings with IIH include an enlarged partially empty sella, protrusion of the cerebellar tonsils into the foramen magnum (Fig S3), or arachnoid pits in the middle cranial fossa (20,21,42,45). The presence of papilledema should decrease the radiologist's threshold for commenting on arachnoid granulations within or any irregularity involving the venous sinuses. Large arachnoid granulations or any venous sinus stenosis warrants mention even in the absence of suspected IIH (Fig 5) (41). Pulsatile tinnitus alleviated by



Figure 5. Transverse venous sinus stenosis from a large arachnoid granulation without IIH in a 54-year-old patient with left-sided pulsatile tinnitus. **(A, B)** Axial post-contrast T1-weighted (**A**) and T2-weighted (**B**) MR images of the IAC show a distal left transverse sinus arachnoid granulation (arrow). **(C)** On an axial image from CT angiography performed before MRI, the arachnoid granulation (arrow) can be seen but was not commented on, prompting the MRI. The patient underwent conventional DSA. **(D)** Angiogram obtained with left internal carotid artery (ICA) injection in the venous phase shows the arachnoid granulation (arrow). Most of the left hemisphere drains across the right transverse sinus and the inferior anastomotic vein of Labbé into the sigmoid sinus. Although the patient underwent subsequent stent placement in the transverse sinus, the pulsatile tinnitus was only minimally improved.

ternal jugular vein also have condylar collaterals (Fig 6) (47). These findings should be interpreted with caution to avoid overcalling, particularly in the asymptomatic patient, but the authors advocate mentioning a possible vascular Eagle syndrome if the history and physical examination results support an ipsilateral pulsatile tinnitus that is not otherwise explained by other findings and is augmented with head rotation.

Arterial Causes

Common arterial causes of pulsatile tinnitus include atherosclerotic carotid disease, internal carotid artery (ICA) dissection, ICA aneurysm (Fig 7) (48), and fibromuscular dysplasia (49). Other more rare causes of pulsatile tinnitus include an aberrant carotid artery or a persistent stapedial artery (50,51). Just as with arteriovenous junction abnormalities, evaluation of the arterial vascular tree should be undertaken with careful consideration for aneurysms, particularly intradural aneurysms, which can rupture and cause significant patient harm (15,26).

A history of atherosclerotic disease, hypertension, dyslipidemia, or smoking are risk factors associated with arterial causes of tinnitus (18,52). If a carotid bruit is audible, atherosclerosis or other arterial causes may be the culprit (52). Additionally, some of the characteristics of pulsatile tinnitus can also be informative in distinguishing arterial from venous causes. A low humming or whooshing sound is more likely to be associated with a venous cause, while a higher-pitched tinnitus is more likely to be associated with an arterial cause (15,53).

Treatment Considerations

Treatment of pulsatile tinnitus caused by an arteriovenous fistula or malformation depends on the severity of the

compression of the ipsilateral jugular vein increases the likelihood of a venous finding (19).

Of the venous causes of pulsatile tinnitus, the most common is venous sinus stenosis, and this is often seen in combination with IIH. Venous sinus stenosis in the absence of IIH can also manifest with pulsatile tinnitus (26,41). Up to two-thirds of patients with IIH may report pulsatile tinnitus (20,42–44). Pulsatile tinnitus in the setting of intracranial hypertension can be from narrowing of the transverse sinus from intrinsic or extrinsic causes (21), or from prominent emissary veins (24,25). Other common causes of venous pulsatile tinnitus include jugular bulb anomalies including a high-riding jugular bulb, venous sinus diverticulum, or less commonly compression of the internal jugular vein anywhere along its course, for example compression of the internal jugular vein between the C1 transverse process and styloid process or the posterior belly of the digastric muscle (18,19,46).

Jugular bulb anomalies are often seen in association with venous sinus stenosis (41). Internal jugular vein compression by the styloid process and digastric muscle is commonly seen incidentally in asymptomatic patients, with severe stenosis in nearly one-fourth of patients undergoing CT angiography for any cause (47). Patients who have severe compression of the in-

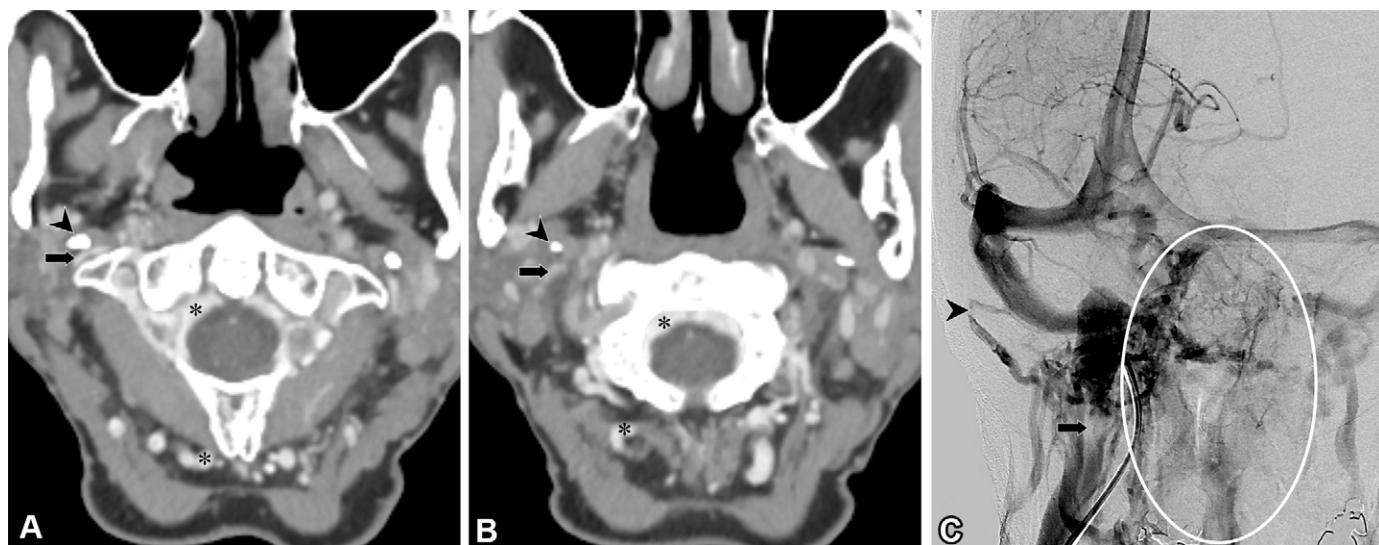


Figure 6. Vascular Eagle syndrome in a 66-year-old patient with right pulsatile tinnitus. **(A, B)** Axial CT angiograms show effacement of the internal jugular vein (arrow) between the styloid process (arrowhead) and C1 transverse process. The vertebral venous plexus and deep cervical veins (*) are of higher attenuation than both internal jugular veins. The patient underwent DSA. **(C)** Right internal carotid angiogram in the venous phase of enhancement shows effacement of the internal jugular vein at the level of the C1 transverse process (arrow). Most of the contrast material is seen draining across condylar collaterals, a prominent mastoid emissary vein (arrowhead), and the vertebral venous plexus (oval), consistent with vascular Eagle syndrome. The patient was counseled and opted for conservative management.

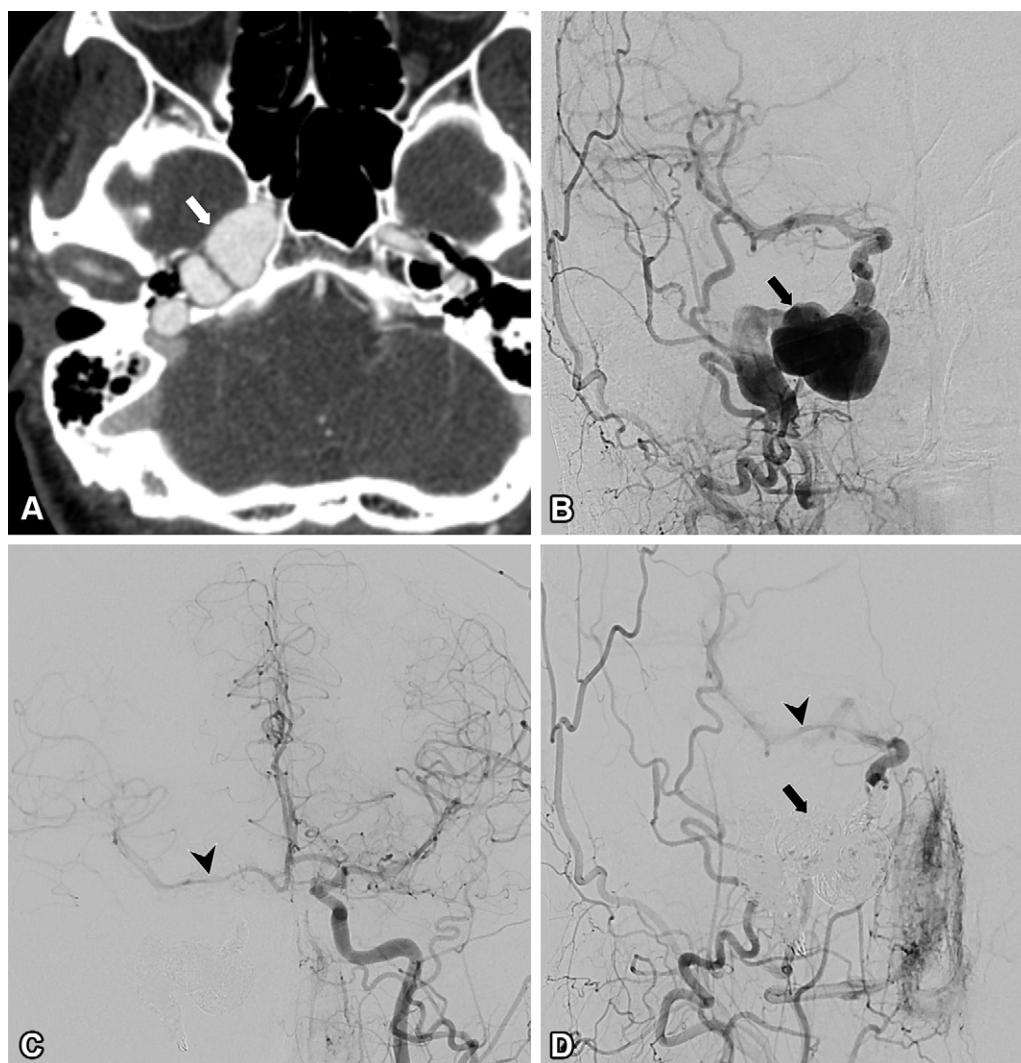


Figure 7. Left petrous ICA aneurysm in a 33-year-old woman with a 3-month history of progressive constant right pulsatile tinnitus. **(A)** Axial CT angiogram through the horizontal petrous segment of the right ICA shows a large petrous aneurysm (arrow). This eroded into the middle ear cavity, pneumatized petrous apex, and mastoid air cells (not shown). **(B)** Initial frontal DSA image obtained with right common carotid artery injection shows the petrous aneurysm (arrow). **(C)** Angiogram after coil embolization of the aneurysm shows some filling of the right middle cerebral artery (arrowhead). **(D)** DSA image obtained with left common carotid artery injection after coil embolization of the aneurysm (arrow) shows collateral filling of the right anterior and middle cerebral arteries (arrowhead). The patient's pulsatile tinnitus resolved after embolization of the right ICA.

Table 3: Osseous Causes of Pulsatile Tinnitus

Any cause of conductive hearing loss
Ossicular chain disruption or erosions
Otosclerosis
Semicircular canal wall dehiscence
Sigmoid sinus wall and jugular bulb defects and diverticula
Styloid process elongation

symptoms, the patient's perceived disability, the operative risk stratification of the lesion, and the accessibility of the arterial supply or venous drainage. Peripheral fistulas with external carotid arterial supply and direct sinus drainage are more amenable to endovascular management (54). Hemorrhagic risk stratification of fistulas is performed using the location and direction of venous drainage to identify a fistula at higher risk of rupture. Fistulas demonstrating cortical or deep venous drainage with retrograde flow or venous ectasia are at greater risk of rupture (38). Clipping remains viable as either primary or adjunctive treatment of fistulas with arterial supply from the ICA or with deep venous drainage (55).

Treatment of arteriovenous malformations is more nuanced and warrants a multimodality and multidisciplinary approach to determine appropriate management. The goal of noninvasive and invasive imaging is to help elucidate risk features associated with surgical resection. A nidus involving eloquent brain regions—for example, motor or sensory cortex, brainstem, or deep gray nuclei and internal capsule—limits operability, hence involvement of eloquent brain should be commented on. A larger nidus and deep cerebral venous drainage are also higher-risk features worth mentioning (39). Conservative monitoring, surgical resection, and adjunctive or primary radiation therapy or endovascular embolization are all potential treatment strategies (Fig S4) (56), depending on the risk features of the arteriovenous malformation, the patient's baseline functional status, and the patient's comorbidities.

In the setting of pulsatile tinnitus from transverse sinus stenosis, stent placement in the transverse sinus can be performed to relieve the tinnitus and often helps decrease intracranial pressure as well (57,58). Stent placement can relieve tinnitus in patients with venous stenosis even in the absence of a venous pressure gradient or elevated intracranial pressures (40). Emissary vein embolization with or without stent placement in the transverse sinus stenosis is also effective (Fig S5) (59).

In summary, emerging treatments include stent placement in the transverse and sigmoid sinuses and venous diverticula and emissary vein embolization, which can effectively cure pulsatile tinnitus related to specific pathologic conditions. Embolization of arteriovenous fistulas can be curative. Arteriovenous malformations require a multimodal approach with endovascular, radiation, and operative management strategies. Successful treatment of atherosclerotic arterial causes of pulsatile tinnitus with stent placement (60) and endarterectomy (61) has also been reported.

Osseous Causes

Imaging Considerations

In a patient with pulsatile tinnitus and conductive hearing loss, CT of the temporal bone is the study of choice for evaluating the inner ear structures, middle ear, and mastoid portion of the temporal bone. CT of the temporal bone may also be informative in the patient with pulsatile tinnitus in whom no identifiable cause is identified at vascular studies such as MR angiography or CT angiography and no mass lesion is seen at MRI (62). Temporal bone CT is the most appropriate initial imaging study for the patient presenting with both pulsatile tinnitus and conductive hearing loss. Vascular imaging may still be warranted to exclude dural arteriovenous fistula or arteriovenous malformation. Note that any cause of conductive hearing loss can result in pulsatile tinnitus, while sensorineural hearing loss is associated with nonpulsatile tinnitus.

Anatomic Considerations

The temporal bone has five parts, of which the petrous, tympanic, and mastoid parts compose the external auditory canal, middle ear cavity, and inner ear structures. The middle ear cavity contains the ossicular chain, composed of the malleus, incus, and stapes. Sound vibrates the tympanic membrane; the vibrations are transmitted through the malleus, incus, and stapes to the fluid-filled cochlea via the oval window. The round window is a flexible window at the other end of the cochlear duct and allows decompression of these vibrations.

Besides the cochlea, the inner ear is composed of the three semicircular canals and vestibule contained within the membranous labyrinth; the surrounding dense bone is the otic capsule and osseous labyrinth. The roof of the middle ear and mastoid is composed of the tegmen tympani and tegmen mastoideum, respectively. The mastoid air cells and eustachian canal function to regulate the middle ear cavity pressures (63). The sigmoid sinus wall consists of a thin cortical bone running between the sigmoid sinus wall and the adjacent mastoid air cells and middle ear cavity and is referred to as the sigmoid plate.

Temporal Bone Causes

Any pathologic condition that alters the bony structures of the middle or inner ear, the tegmen, or the bone covering the blood vessels running through the area of the mastoid can contribute to pulsatile tinnitus (Table 3).

Careful attention should be paid to areas of thinning or dehiscence in this area in any patient undergoing evaluation of pulsatile tinnitus who undergoes temporal bone CT (64–66). Many patients with IIH may also have findings of a sigmoid sinus dehiscence or diverticulum in addition to transverse sinus stenosis and emissary veins (45). Owing to the overall prevalence of pulsatile tinnitus in the setting of IIH and transverse sinus stenosis, and the strong association of venous sinus stenosis with sigmoid sinus wall defects (41), the most common osseous defect causing pulsatile tinnitus is an abnormality of the sigmoid plate.

Thinning or dehiscence of the sigmoid sinus wall can be subtle and is easily missed (Fig 8). Sigmoid sinus wall

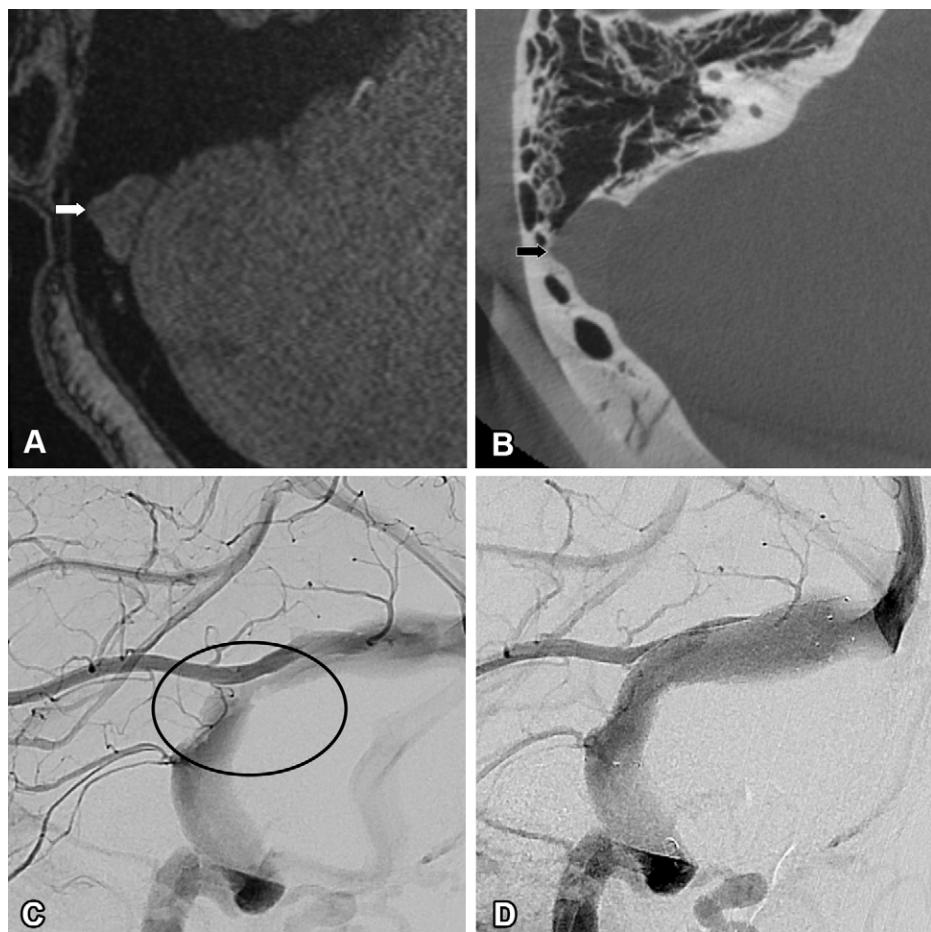


Figure 8. Left sigmoid sinus wall defect in a 29-year-old woman with bilateral tinnitus, subjective hearing loss with a normal audiogram, and imbalance. **(A)** Axial image from MR angiography performed to evaluate for a potential vascular lesion is retrospectively notable for subtle protrusion of the right sigmoid sinus wall (arrow). Conventional DSA was performed. **(B)** Axial image from 14-second flat-panel rotational CT of the temporal bone performed during DSA shows the sigmoid sinus wall defect (arrow). **(C)** Oblique angiogram obtained with right ICA injection in the venous phase shows irregularities of the transverse-sigmoid sinus junction (oval) with mild stenosis. The patient opted for stent placement. **(D)** Angiogram after stent placement shows no residual luminal irregularity. The patient experienced immediate resolution of her symptoms after the procedure.



Figure 9. Middle ear effusion in a 70-year-old patient with a several-week history of pulsatile tinnitus. **(A)** Axial CT image of the temporal bone shows opacification of the right middle ear cavity (circle). **(B)** Coronal CT image shows opacification of the mesotympanum (circle) with relative sparing of the epitympanum (arrow). The patient was subsequently referred to otolaryngology and diagnosed with otitis media and a middle ear effusion. This did not resolve with antibiotics, and the patient ultimately underwent myringotomy.

dehiscence is often incidental and is seen in up to 1% of patients undergoing temporal bone CT for an indication other than pulsatile tinnitus (65); in the authors' experience, this may be more common and can be seen at thin-cut high-resolution CT angiography, for example (Fig S3). Therefore, the presence of sigmoid sinus wall defects should be interpreted with caution, particularly when there is no history of pulsatile tinnitus. When the indication for temporal bone CT is pulsatile tinnitus, the radiologist should have a low threshold to mention thinning or dehiscence of the sigmoid sinus wall.

A sigmoid sinus diverticulum, jugular bulb diverticulum, or high-riding jugular bulb suspected at temporal bone CT should prompt either CT venography or conventional DSA for further evaluation of the venous sinus for any stenosis (67). The styloid process should be evaluated and noted if it is elongated and no other finding is apparent, as compression of the internal jugular vein may manifest with pulsatile tinnitus (46,47).

Any cause of conductive hearing loss can result in pulsatile tinnitus (62). A middle ear effusion (Fig 9) or any mass of the middle ear cavity can impede conduction and manifest with

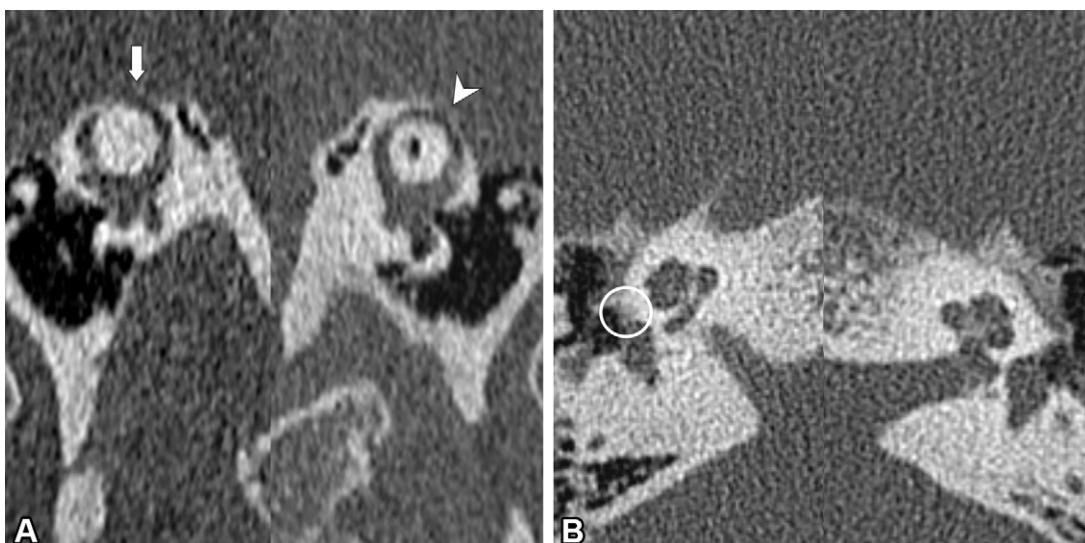


Figure 10. Superior semicircular canal dehiscence and fenestral otosclerosis in a 42-year-old woman with right-sided conductive hearing loss and bilateral tinnitus after recovery from an upper respiratory infection. **(A)** CT image of the temporal bone in the Pöschl plane shows dehiscence of the right superior semicircular canal (arrow) and thinning of the left superior semicircular canal (arrowhead). **(B)** Axial CT image of the temporal bone shows subtle lucency in the right otic capsule (circle) along the anterior aspect of the right oval window in the region of the fissula ante fenestram, consistent with fenestral otosclerosis. The patient opted for conservative management with use of hearing aids, which improved her symptoms.

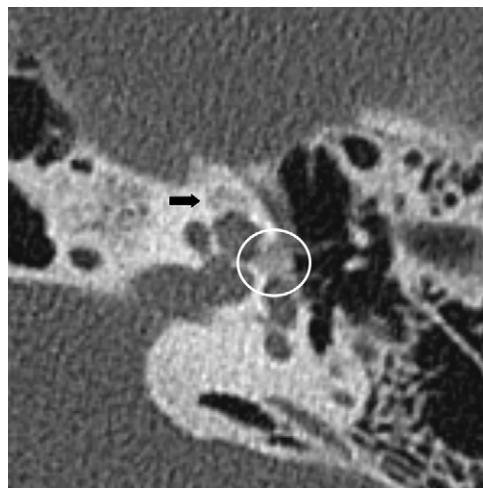


Figure 11. Fenestral and retrofenestral otosclerosis in a 35-year-old patient with a family history of otosclerosis who presented with progressive left-sided conductive hearing loss and pulsatile tinnitus. Axial CT image of the temporal bone shows lucency of the otic capsule adjacent to the oval window and stapes footplate in the region of the fissula ante fenestram (circle) and lucency around the cochlea remote from the stapes footplate (arrow), consistent with fenestral and retrofenestral otosclerosis.

tinnitus. After evaluation of the ossicular chain and middle ear cavity, the otic capsule surrounding the inner ear structures should be carefully evaluated to assess for any demineralization around the cochlea or the covering of the semicircular canals or for sclerosis of the labyrinth structures (62).

Less commonly, pulsatile tinnitus may be caused by otosclerosis, Paget disease, an enlarged vestibular aqueduct, temporomandibular joint disorder, or any third window disorder (11). Third window disorders are conditions where there is an osseous defect in the bony labyrinth, resulting in a third opening besides the oval and round windows. Superior semicircular canal dehiscence is the most common of these (Fig 10) (62). Besides pulsatile tinnitus and hearing loss, patients with semicircular canal dehiscence may also have a history of vertigo induced by loud sounds or pressure changes (Tullio phenomenon), ear fullness, and autophony.

Otosclerosis is a rare entity of abnormal bone demineralization and growth and generally manifests with conductive hearing loss in the 2nd or 3rd decade of life (11,62). The most common location of this is abnormal bone turnover just anterior to the oval window and the stapes footplate involving a small cleft known as the *fissula ante fenestram*, and this is termed *stapedial* or *fenestral* otosclerosis. Otosclerosis of the otic capsule beyond or separate from the oval window and stapes footplate is termed *retrofenestral* and may lead to sensorineural hearing loss. This often coexists with the stapedial or fenestral form of the disease (Fig 11). Clinically, otosclerosis may be present even in the absence of radiologic findings.

Treatment Considerations

Stent placement or coil embolization in a sigmoid sinus or jugular bulb defect or diverticulum can effectively cure pulsatile tinnitus (Fig 12) (68–71). Transmastoid reconstruction of the

pulsatile tinnitus. These are often apparent clinically and with audiometric testing. Similarly, disruption of the ossicular chain or erosion of the ossicles can result in hearing loss and pulsatile tinnitus. A cholesteatoma (discussed later) may opacify the middle ear cavity and in some cases erode the scutum and ossicles, manifesting with conductive hearing loss and pulsatile



Figure 12. Jugular bulb diverticulum in a 58-year-old patient with left-sided pulsatile tinnitus. **(A)** Oblique sagittal MIP from phase-contrast MR venography shows a left jugular bulb diverticulum (arrow). **(B)** Oblique image from conventional DSA of the jugular bulb in the venous phase shows stent-assisted coil embolization of the jugular bulb (arrow). The patient's symptoms resolved after the procedure.

Table 4: Neoplastic Causes of Pulsatile Tinnitus

Any vascular tumor of the petrous bone
Paragangliomas of the head and neck
Skull base or petrous meningiomas
Skull base low-flow venous malformations (hemangiomas)
Skull base and inner ear schwannomas*

* Schwannomas more commonly manifest with deficits or paresthesia associated with the affected nerve.

sigmoid sinus wall (Fig S6), jugular bulb, or arcuate eminence covering the superior semicircular canal is also effective for treatment of skull base defects causing tinnitus (71,72). Hearing aids, sound generators, ossicular reconstruction, or stapedectomy may be helpful in treatment of ossicular erosion, third window disorders, and otosclerosis, depending on the specific pathologic condition.

Neoplasms and Other Masses

Hypervascular masses involving the petrous, mastoid, or tympanic part of the temporal bone can manifest with pulsatile tinnitus (Table 4). The most common middle ear mass causing pulsatile tinnitus is a paraganglioma, and most paragangliomas of the middle ear manifest with pulsatile tinnitus (1,11,73). A paraganglioma of the head and neck is most often an asymptomatic palpable mass. Pulsatile tinnitus is present in most patients with a tympanic paraganglioma (previously known as glomus tympanicum tumor) and may be a presenting symptom of a jugular paraganglioma. At audiometric testing, half of patients with a tympanic paraganglioma will have conductive hearing loss at presentation; a tympanic paraganglioma is often visible as a red, pulsating, vascular middle ear mass and will often be imaged with temporal bone CT, revealing a soft-tissue mass lateral to the cochlear promontory (74).

A vagal, carotid body, or jugular bulb paraganglioma can have an audible bruit. An enlarging jugular paraganglioma can also manifest with hearing loss or involvement of the lower cranial nerves and hypoglossal canal (73). At temporal

bone CT, a jugular paraganglioma will manifest as a permeative lesion of the skull base often originating around the jugular bulb and can be misinterpreted as opacified mastoid air cells. As the tumor enlarges, it can extend into the middle ear cavity and can erode the caroticojugular spine, between the carotid canal and jugular fossa.

At T2-weighted MRI, these appear T2 hyperintense, sometimes with a salt-and-pepper appearance due to their extensive vascularity. They demonstrate marrow replacement on T1-weighted images with avid contrast enhancement (Fig 13). When a paraganglioma of the head and neck is identified, gallium 68 tetraazacyclododecane tetraacetic acid–octreotide (DOTATATE) or copper 64-DOTATATE scanning is helpful for staging (Fig 13) and provides increased sensitivity for detecting additional sites of involvement (75).

A middle ear cholesteatoma, essentially an epidermoid cyst of the middle ear cavity, can manifest with hearing loss, chronic or recurrent infections, and occasionally pulsatile tinnitus. These characteristically begin in the lateral epitympanic Prussak space and can be seen as opacification of the space (Fig 14) (62). As these grow, they may blunt the scutum, erode middle ear ossicles, and invade adjacent spaces. Cholesteatomas characteristically restrict diffusion. Treatment of cholesteatoma is surgical excision in combination with ossicular reconstruction or prosthesis if warranted (76).

Other masses that may manifest with nonpulsatile tinnitus include schwannomas of the skull base or IAC or meningiomas of the petrous bone. A schwannoma of any of the nerves near the mastoid or tympanic temporal bone may rarely cause pulsatile tinnitus (1,77–79). An IAC schwannoma will manifest more often with sensorineural hearing loss than with pulsatile tinnitus. These manifest as an enhancing mass of the IAC and can extend into the cerebellopontine angle, cochlea, or IAC fundus. Intralabyrinthine schwannomas are smaller and harder-to-detect schwannomas that also manifest with sensorineural hearing loss. The cochlea, vestibule, and semicircular canals should be closely examined with T1-weighted fat-suppressed sequences for contrast enhancement and with high-resolution 3D imaging for loss of the normal fluid signal intensity (15).

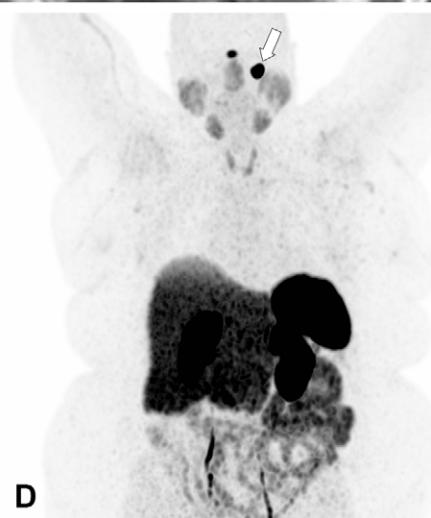
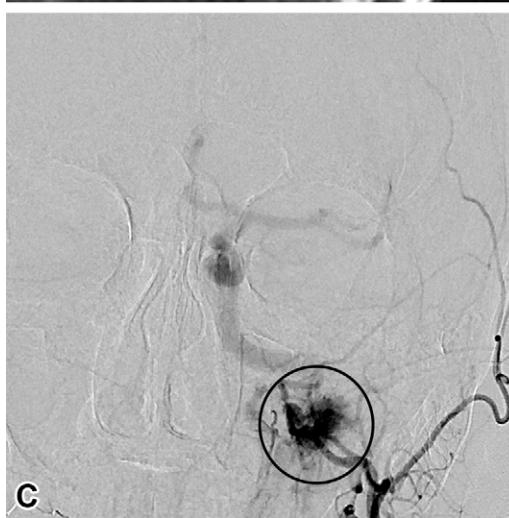
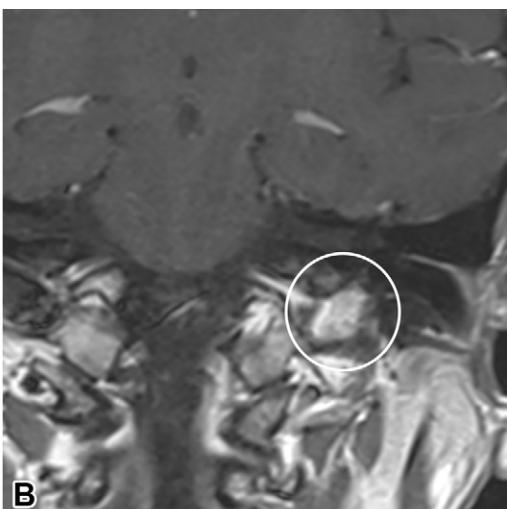
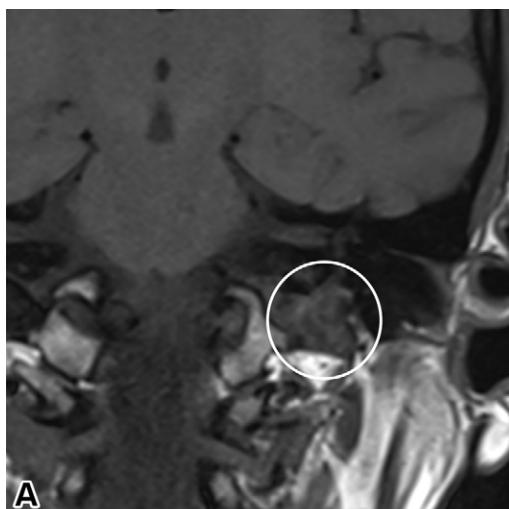


Figure 13. Paraganglioma of the jugular bulb in a 61-year-old patient with left-sided pulsatile tinnitus and normal hearing. An MRI study of the IAC was interpreted as normal. (A, B) Coronal precontrast (A) and postcontrast (B) T1-weighted images show a T1-hypointense lesion (circle in A) near the jugular foramen with avid postcontrast enhancement (circle in B). The patient presented for angiography. (C) Frontal DSA image obtained with selective external carotid artery injection shows avid enhancement (circle) corresponding to the enhancing mass at MRI, with arterial supply from the ascending pharyngeal and occipital arteries. (D) Copper 64 tetraazacyclododecane tetraacetic acid-octreotate (DOTATATE) staging scan shows the same lesion (arrow) but no other lesions. The remainder of the scan shows normal physiologic uptake of DOTATATE in the pituitary gland, salivary glands, thyroid gland, liver, spleen, kidneys, and bowel. The patient opted for conservative management with routine surveillance imaging.

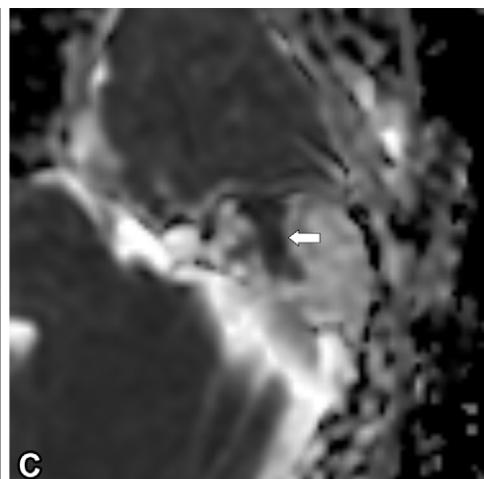
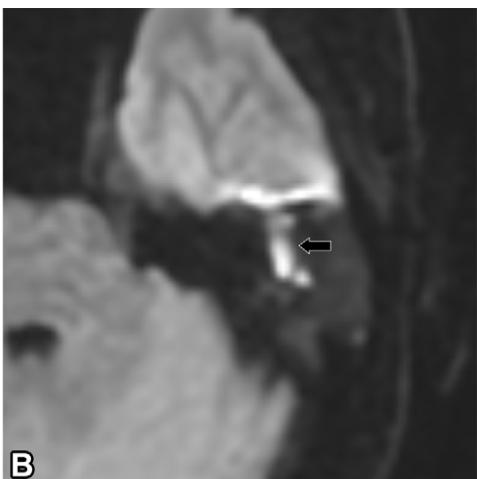
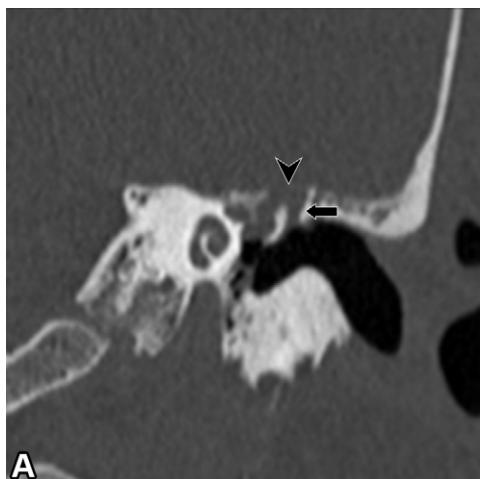


Figure 14. Cholesteatoma in a 43-year-old patient with left-sided hearing loss. (A) Coronal CT image of the temporal bone shows opacification of the epitympanum including the Prussak space (arrow) and dehiscence of the tegmen tympani (arrowhead). (B, C) Axial diffusion-weighted (B) and apparent diffusion coefficient (C) images from subsequent MRI show restricted diffusion in the middle ear (arrow). The patient underwent mastoidectomy, resection, and skull base resurfacing. Pathologic analysis demonstrated a cholesteatoma.

Facial nerve schwannoma can manifest with facial nerve palsy or spasm. The intratemporal course of the facial nerve from the canalicular, labyrinthine, tympanic, and mastoid segments should be carefully evaluated for the caliber of the

facial nerve canal and for the presence of any abnormal enhancement. Trigeminal schwannomas often manifest with facial pain and numbness. A hypoglossal schwannoma will rarely present with tinnitus.

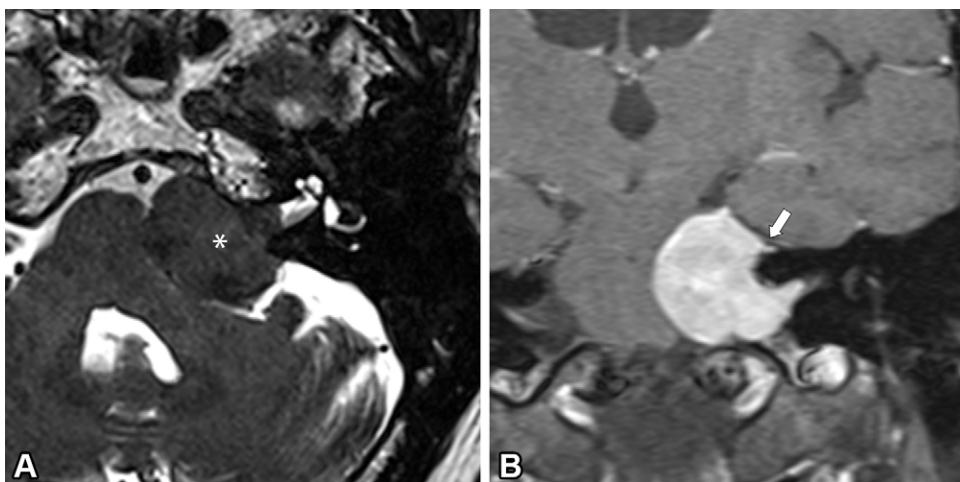


Figure 15. Cerebellopontine angle meningioma in a 66-year-old patient with left-sided pulsatile tinnitus and hearing loss. **(A)** Axial image from 3D T2-weighted sampling perfection with application-optimized contrast using different flip angle evolution (SPACE) MRI shows a large mass (*) at the left cerebellopontine angle protruding into the IAC. **(B)** Coronal postcontrast T1-weighted image shows avid enhancement of the lesion with a dural tail (arrow). The patient underwent resection; results of pathologic analysis were consistent with a meningioma.

A meningioma of the petrous bone or an intraosseous low-flow venous malformation (formerly known as a hemangioma) of the skull base can manifest with pulsatile tinnitus. A meningioma of the petrous bone will be isointense or hyperintense to gray matter on T2-weighted images and will avidly enhance (Fig 15). A dural tail is characteristic, and there may be adjacent hyperostosis. A low-flow venous malformation will be T2 hyperintense and demonstrate avid enhancement and most often has reassuring intrinsic T1 hyperintensity from fat signal intensity.

Treatment Considerations

While most paragangliomas are considered benign slow-growing neoplasms, rapidly enlarging and symptomatic paragangliomas can be treated with a multimodal approach. Surgical resection can be effective in curing pulsatile tinnitus, and this is preceded by endovascular embolization to reduce the risk of intraoperative hemorrhage (80). Radiation therapy is also safe and effective (81). For patients with advanced inoperable or metastatic disease, lutetium 177 polypeptide therapy is a recent effective treatment option (75). To date, the effects of embolization, external radiation therapy, or polypeptide therapy on treatment of pulsatile tinnitus have not been described. Schwannomas and meningiomas may be observed, resected, or irradiated, depending on their size and growth and the patient's preference.

Conclusion

Pulsatile tinnitus can be a severely disabling condition with a broad range of causes. Many imaging findings can be subtle, but most patients with pulsatile tinnitus will have an identifiable and often treatable cause of tinnitus at imaging (9–13). Careful consideration of the presenting history and physical examination results coupled with imaging findings can elucidate these treatable causes. Causes of pulsatile tinnitus can be broadly classified into a framework of vascular, bony structural, and neoplastic categories. In patients with conductive hearing loss, temporal bone CT or brain MRI with attention to the IAC can reveal a bony structural or neoplastic process (Fig 1). Any cause of conductive hearing loss can also lead to pulsatile tinnitus.

In patients with absence of conductive hearing loss, the initial workup should include MR angiography or venography or CT angiography or venography to assess for venous or arterial causes of pulsatile tinnitus. When a noninvasive imaging study is positive or pretest suspicion for a vascular cause is high and no finding is present at noninvasive imaging, conventional DSA and flat-panel rotational CT are helpful for confirmation and therapeutic decision making. In patients with mild symptoms or an underlying benign cause, monitoring or conservative measures are prudent and can be helpful in alleviating the stress associated with pulsatile tinnitus. In patients with debilitating symptoms or an aggressive pathologic condition, established and emerging treatment options guided toward specific causes can often be curative of pulsatile tinnitus.

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