

**American College of Radiology
ACR Appropriateness Criteria®
Tinnitus**

Variant: 1 Pulsatile tinnitus, unilateral or bilateral; No retrotympenic lesion on otoscopy. Initial imaging.

Procedure	Appropriateness Category	Relative Radiation Level
MRA head with IV contrast	Usually Appropriate	○
MRI head and internal auditory canal without and with IV contrast	Usually Appropriate	○
CTA head and neck with IV contrast	Usually Appropriate	☼☼☼
CTA head with IV contrast	Usually Appropriate	☼☼☼
MRA head without and with IV contrast	May Be Appropriate (Disagreement)	○
MRA head without IV contrast	May Be Appropriate	○
MRI head and internal auditory canal without IV contrast	May Be Appropriate	○
MRV head with IV contrast	May Be Appropriate	○
CT temporal bone without IV contrast	May Be Appropriate (Disagreement)	☼☼☼
CTV head with IV contrast	May Be Appropriate	☼☼☼
US duplex Doppler carotid artery	Usually Not Appropriate	○
US duplex Doppler transcranial	Usually Not Appropriate	○
US head	Usually Not Appropriate	○
Arteriography cervicocerebral	Usually Not Appropriate	☼☼☼
MRI head and internal auditory canal with IV contrast	Usually Not Appropriate	○
MRV head without and with IV contrast	Usually Not Appropriate	○
MRV head without IV contrast	Usually Not Appropriate	○
CT head with IV contrast	Usually Not Appropriate	☼☼☼
CT head without and with IV contrast	Usually Not Appropriate	☼☼☼
CT head without IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without and with IV contrast	Usually Not Appropriate	☼☼☼

Variant: 2 Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

Procedure	Appropriateness Category	Relative Radiation Level
CT temporal bone without IV contrast	Usually Appropriate	☼☼☼
CTA head and neck with IV contrast	May Be Appropriate	☼☼☼
CTA head with IV contrast	May Be Appropriate	☼☼☼
CTV head with IV contrast	May Be Appropriate	☼☼☼
US duplex Doppler carotid artery	Usually Not Appropriate	○
US duplex Doppler transcranial	Usually Not Appropriate	○
US head	Usually Not Appropriate	○
Arteriography cervicocerebral	Usually Not Appropriate	☼☼☼
MRA head with IV contrast	Usually Not Appropriate	○
MRA head without and with IV contrast	Usually Not Appropriate	○

MRA head without IV contrast	Usually Not Appropriate	○
MRI head and internal auditory canal with IV contrast	Usually Not Appropriate	○
MRI head and internal auditory canal without and with IV contrast	Usually Not Appropriate	○
MRI head and internal auditory canal without IV contrast	Usually Not Appropriate	○
MRV head with IV contrast	Usually Not Appropriate	○
MRV head without and with IV contrast	Usually Not Appropriate	○
MRV head without IV contrast	Usually Not Appropriate	○
CT head with IV contrast	Usually Not Appropriate	☠☠☠
CT head without and with IV contrast	Usually Not Appropriate	☠☠☠
CT head without IV contrast	Usually Not Appropriate	☠☠☠
CT temporal bone with IV contrast	Usually Not Appropriate	☠☠☠
CT temporal bone without and with IV contrast	Usually Not Appropriate	☠☠☠

Variant: 3 Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

Procedure	Appropriateness Category	Relative Radiation Level
MRI head and internal auditory canal without and with IV contrast	Usually Appropriate	○
MRI head and internal auditory canal without IV contrast	May Be Appropriate	○
US duplex Doppler carotid artery	Usually Not Appropriate	○
US duplex Doppler transcranial	Usually Not Appropriate	○
US head	Usually Not Appropriate	○
Arteriography cervicocerebral	Usually Not Appropriate	☠☠☠
MRA head with IV contrast	Usually Not Appropriate	○
MRA head without and with IV contrast	Usually Not Appropriate	○
MRA head without IV contrast	Usually Not Appropriate	○
MRI head and internal auditory canal with IV contrast	Usually Not Appropriate	○
MRV head with IV contrast	Usually Not Appropriate	○
MRV head without and with IV contrast	Usually Not Appropriate	○
MRV head without IV contrast	Usually Not Appropriate	○
CT head with IV contrast	Usually Not Appropriate	☠☠☠
CT head without and with IV contrast	Usually Not Appropriate	☠☠☠
CT head without IV contrast	Usually Not Appropriate	☠☠☠
CT temporal bone with IV contrast	Usually Not Appropriate	☠☠☠
CT temporal bone without and with IV contrast	Usually Not Appropriate	☠☠☠
CT temporal bone without IV contrast	Usually Not Appropriate	☠☠☠
CTA head and neck with IV contrast	Usually Not Appropriate	☠☠☠
CTA head with IV contrast	Usually Not Appropriate	☠☠☠
CTV head with IV contrast	Usually Not Appropriate	☠☠☠

Variant: 4 Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

Procedure	Appropriateness Category	Relative Radiation Level
US duplex Doppler carotid artery	Usually Not Appropriate	○
US duplex Doppler transcranial	Usually Not Appropriate	○

US head	Usually Not Appropriate	○
Arteriography cervicocerebral	Usually Not Appropriate	☼☼☼
MRA head with IV contrast	Usually Not Appropriate	○
MRA head without and with IV contrast	Usually Not Appropriate	○
MRA head without IV contrast	Usually Not Appropriate	○
MRI head and internal auditory canal with IV contrast	Usually Not Appropriate	○
MRI head and internal auditory canal without and with IV contrast	Usually Not Appropriate	○
MRI head and internal auditory canal without IV contrast	Usually Not Appropriate	○
MRV head with IV contrast	Usually Not Appropriate	○
MRV head without and with IV contrast	Usually Not Appropriate	○
MRV head without IV contrast	Usually Not Appropriate	○
CT head with IV contrast	Usually Not Appropriate	☼☼☼
CT head without and with IV contrast	Usually Not Appropriate	☼☼☼
CT head without IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without and with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without IV contrast	Usually Not Appropriate	☼☼☼
CTA head and neck with IV contrast	Usually Not Appropriate	☼☼☼
CTA head with IV contrast	Usually Not Appropriate	☼☼☼
CTV head with IV contrast	Usually Not Appropriate	☼☼☼

Panel Members

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Summary of Literature Review

Introduction/Background

Tinnitus is a symptom defined by the perception of sound in the absence of external stimuli. It affects more than 50 million Americans, equally among men and women, and is most commonly noted in patients between 40 and 70 years of age [1]. Tinnitus may range from innocuous to devastating, with significant negative impacts on psychosocial well-being and quality of life. In a survey conducted in 2008, approximately 10% of the United States adult population experienced endorsed at least 1 episode of tinnitus lasting more than 5 minutes in the preceding year [2].

Patients describe tinnitus as hearing various sounds ranging from hissing, buzzing, ringing, pulsations, and clicking to a roaring kind of noise. There are various etiologies and subtypes [3], and tinnitus can be classified in a number of ways such as primary or secondary, subjective or objective, and pulsatile tinnitus (PT) or nonpulsatile tinnitus (NPT). Primary tinnitus is idiopathic, whereas secondary tinnitus is associated with an underlying cause. Subjective tinnitus is perceived by the patient only and comprises 70% to 80% of cases [4]. Objective tinnitus can be detected on

examination by the clinician. Tinnitus may be pulsatile or nonpulsatile depending on the quality of the noise perceived by the patient. PT relates to rhythmic noise with the patient's heartbeat or pulse and can be either subjective or objective. PT can be pulse-synchronous or pulse-asynchronous as seen in palatal or tympanic myoclonus. NPT relates to a continuous nonsynchronized sound, which is almost always subjective and is the more common variant [5]. It is important to classify tinnitus as pulsatile or nonpulsatile to optimize the imaging workup, given different diagnostic considerations for each. Pulsatile and objective types of tinnitus are more likely to have an underlying identifiable cause, and imaging is usually advised.

It is important to know the otoscopy examination findings in patients with PT because it guides the imaging algorithm. In the setting of PT, the possibility of a vascular retrotympenic lesion leads to a different set of differential diagnostic considerations compared to the absence thereof and therefore influences the decision for which imaging studies to pursue and in what order. However, the detection of retrotympenic lesions can be difficult without appropriate equipment.

The American Academy of Otolaryngology and Head and Neck Surgery Foundation (AAO-HNS) guidelines recommend targeted history and clinical examination as the initial evaluation and determination as to whether the tinnitus is bothersome or not, before any imaging. The guidelines also recommend a prompt and comprehensive audiological examination in patients with hearing problems or with unilateral persistent tinnitus. The guidelines make a strong recommendation against any imaging studies of the head and neck for the subset of patients in whom tinnitus does not localize to 1 ear, is nonpulsatile, and is not associated with focal neurological abnormalities or an asymmetric hearing loss. The guidelines also suggest that some patients with severe anxiety, depression, or psychological disturbances may need prompt identification and intervention [6,7].

The type of tinnitus and associated symptoms often determine the choice of imaging studies and their appropriateness. Tinnitus often coexists with other symptoms such as hearing loss, vertigo, previous head trauma, and neurological deficits. The ACR Appropriateness Criteria[®] topics for "[Hearing Loss and/or Vertigo](#)" [8], "[Head Trauma](#)" [9], and "[Cerebrovascular Diseases-Aneurysm, Vascular Malformation, and Subarachnoid Hemorrhage](#)" [10] should be used to guide imaging in those settings.

Special Imaging Considerations

CT angiography (CTA) can be performed to include a mixed arterial and venous phase through the head and neck in selected clinical scenarios. In these cases, imaging can be performed after peak arterial timing and can be obtained approximately 20 to 25 seconds after injection of intravenous (IV) contrast material. This allows for a single test with lower total radiation dose when compared to separate CTA and CT venography (CTV) examinations. Overlapping of vessels is not problematic, and it shows not only both arterial and venous anatomy and pathology adequately in this balanced phase but also the bony details of the temporal bone when reconstructed in thin bone window settings [1,11,12].

MR brain, when performed for temporal bone/skull base evaluation of tinnitus, is usually performed using specialized internal auditory canal (IAC) protocols, which include thin-section, heavily T2-weighted sequences to evaluate for vascular loops and small vestibular schwannomas. Volumetric postcontrast T1-weighted images are also increasingly becoming a part of this protocol, which has the added benefit of assessment of the transverse and sigmoid sinuses.

Functional MRI in patients with tinnitus is still a research tool and is not used in routine applications [13,14]. Venous arterial spin-labeling technique has a high sensitivity and specificity for the presence of a dural arteriovenous fistula (dAVF), and the addition of spin-labeling increases confidence in the diagnosis of this entity on MRI [15].

Time-resolved gadolinium-enhanced MR angiography (MRA) is a technique which combines many factors like T1 shortening effect of gadolinium, digital subtraction technique, dynamic imaging, and parallel imaging to provide high temporal resolution MRA images. Use of intelligent k-space sampling technique can further improve the spatial and temporal resolution. The rapid acquisition of multiple 3-D volumes with high temporal and spatial resolution renders excellent depiction of the vessels to detect dAVFs [16-18].

Initial Imaging Definition

Initial imaging is defined as imaging at the beginning of the care episode for the medical condition defined by the variant. More than one procedure can be considered usually appropriate in the initial imaging evaluation when:

- There are procedures that are equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient's care)

OR

- There are complementary procedures (ie, more than one procedure is ordered as a set or simultaneously wherein each procedure provides unique clinical information to effectively manage the patient's care).

Discussion of Procedures by Variant

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympenic lesion on otoscopy. Initial imaging.

PT has many causes, and no single imaging study is appropriate for all patients. A diagnostic algorithm based on a detailed history and clinical evaluation should progress from less invasive to more invasive imaging studies. The history and clinical assessment have implications on the choice of imaging modality [12,19]. For example, an otoscopic examination is extremely useful to help guide optimal imaging evaluation of PT.

PT results from an abnormal ability to perceive one's own vascular flow and therefore often have vascular etiologies. Etiologies for PT in patients without a vascular retrotympenic lesion on otoscopy include arterial lesions in the neck like carotid atherosclerosis, dissection, fibromuscular dysplasia of the carotids, and intracranial etiologies such as arteriovenous malformation (AVM), dAVF, and vascular tumors such as glomus jugulare that do not extend to the middle ear [20]. Venous causes of PT are transverse sinus stenosis, at times in the setting of idiopathic intracranial hypertension (IIH), sigmoid sinus diverticulum, persistent petrosquamosal sinus, and prominent mastoid or condylar emissary veins [4,21-24]. Bony abnormalities such as superior semicircular canal dehiscence (SSCD), sigmoid sinus wall dehiscence [25], high jugular bulb, and Paget disease can also cause PT. Sigmoid sinus wall dehiscence and sigmoid sinus diverticulum are collectively called sigmoid sinus wall abnormalities (SSWA) and are increasingly recognized causes of PT

[19,25-27].

Structural and anatomic causes of PT can be found in up to 44% to 91% of patients; however, in some studies, many patients remained idiopathic even after extensive workup [28,29].

The history and physical examination may also suggest PT due to systemic causes such as pregnancy-related hemodynamic changes, anemia, thyrotoxicosis, or due to mechanical causes like palatal or tympanic myoclonus, Eustachian tube contractions, or temporomandibular joint problems [30-34].

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.
Initial imaging.

A. Arteriography cervicocerebral

Arteriography is an invasive test, which can diagnose many conditions causing PT, and is used when noninvasive tests like CTA and MRA are inconclusive and there is persistent clinical suspicion for a vascular lesion. It is particularly useful for detecting dAVFs and other lesions with arteriovenous shunts such as AVM and carotid cavernous sinus fistulas [35]. These lesions may cause PT, and arteriography has a higher sensitivity and specificity than MRA or CTA, which are used for screening. Therefore, arteriography is still useful if CTA/MRA is negative but dAVF remains suspected to be the cause of PT [23,29]. It is also useful for preoperative planning and embolization for paragangliomas [36]. A study found an incidence of 24% for arteriovenous shunts in patients who were referred for digital subtraction angiography (DSA) for evaluation of PT. Other retrospective studies showed a prevalence of 2% to 27% for arteriovenous shunts in patients with PT undergoing DSA [35]. Cervical artery dissection (carotid or vertebral) can also cause PT in 8% to 10% of patients with PT and can be diagnosed by CTA, MRA, or catheter arteriography. Catheter arteriography is rarely required for this indication; however, because of the excellent accuracy of CTA and MRA [37,38].

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.
Initial imaging.

B. CT head with IV contrast

There is no relevant literature to support the use of CT head with IV contrast for evaluation of PT when otoscopy does not show a vascular retrotympanic lesion.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.
Initial imaging.

C. CT head without and with IV contrast

There is no relevant literature to support the use of CT head without and with IV contrast for evaluation of PT when otoscopy does not show a vascular retrotympanic lesion.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.
Initial imaging.

D. CT head without IV contrast

There is no relevant literature to support the use of CT head without IV contrast for evaluation of PT when otoscopy does not show a vascular retrotympanic lesion.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.
Initial imaging.

E. CT temporal bone with IV contrast

There is no relevant literature to support the use of CT temporal bone with IV contrast for evaluation of PT when otoscopy does not show a vascular retrotympanic lesion. IV contrast is not needed in the assessment of semicircular canal dehiscence, enlarged vestibular aqueduct, SSWA, jugular bulb dehiscence, or glomus jugulare paraganglioma. This may detect transverse sinus stenosis and some cases of dAVFs but cannot assess the neck vessels.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

F. CT temporal bone without and with IV contrast

There is no relevant literature to support the use of CT temporal bone without and with IV contrast for evaluation of PT when otoscopy does not show a vascular retrotympanic lesion.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

G. CT temporal bone without IV contrast

CT temporal bone without IV contrast does not show the arterial abnormalities in the neck, or intracranial vascular abnormalities such as dAVF, AVM, and transverse sinus stenosis, which are potential causes in this setting. High-resolution CT (HRCT) of the temporal bone can detect some other conditions that cause PT but do not always present as a vascular retrotympanic mass, such as otospongiosis, Paget disease, sigmoid sinus diverticulum and sigmoid sinus dehiscence, high-riding jugular bulb, and SSCD [39]. SSCD is a known cause of PT and other symptoms of peripheral vestibulopathy and is readily diagnosed on CT temporal bone [40,41]. SSWAs are known causes of PT and may be the most common identifiable causes in this group [25]. They are much more frequently seen on CT temporal bone in patients with PT compared with the general population and are a common and treatable cause of PT [19,26,27,42]. It can be readily diagnosed with either CT temporal bone or CTA/CTV of the head [19]. One study found that SSWA was seen in all patients with venous PT in addition to other venous abnormalities [43]. IV contrast is not necessary. CT temporal bone has a smaller field-of-view and a higher resolution to evaluate the temporal bone compared with CTA.

Labyrinthine sequestrum is a very rare infective condition in which there is destruction of the inner ear structures and can cause tinnitus in addition to other symptoms. On CT, one may see erosion of the bony labyrinth. On MRI, there may be abnormal enhancement of the labyrinthine contents on the postcontrast images [44].

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

H. CTA head and neck with IV contrast

CTA is an excellent tool when a patient has PT and intracranial or cervical vascular pathologies are suspected [45]. It can diagnose intracranial vascular pathology such as dAVFs and has a combined sensitivity and specificity of ³90% and provides a convenient low-risk test [29,46]. CTA can also detect other pathologies such as AVM, glomus tumors, and the reconstructed high-resolution thin-section bony window images provide osseous details to merit its use as a first-choice imaging technique [29,47]. CTA can also diagnose glomus jugulare, which can cause PT, and was found to be more accurate than MRI in 1 study [48].

Slight modification from usual CTA bolus timing can allow for a balanced phase examination facilitating detection of both arterial and venous etiologies of PT in the head and neck. Therefore,

it is the imaging modality of choice in patients with PT without a suspected retrotympenic lesion because it can show both arterial and venous pathologies in the head, skull base, and neck, in addition to the bony details of the temporal bone. Temporal bone anatomy is also well seen on thin bone window reconstructions, which cannot be assessed on MRA/MRI. Bony reconstructions from CTA thus can show SSWA such as diverticulum and wall dehiscence due to bony contour abnormalities [27]. However, 1 study reported a cause of PT being found only in 68% to 72% of patients with PT despite an extensive workup with CTA, MRI, and ultrasound (US) [12].

Fibromuscular dysplasia of the carotids, atherosclerotic disease, vascular dissection, and carotid aneurysms in the neck can also cause PT, which can be accurately diagnosed by CTA head and neck [1,23,28,46,49].

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympenic lesion on otoscopy.

Initial imaging.

I. CTA head with IV contrast

CTA is an excellent tool when a patient has PT and intracranial vascular pathologies are suspected. Intracranial vascular pathology such as dAVFs can show asymmetrically prominent arterial feeding vessels, transosseous vascular channels, increased number and size of extracranial vessels, and shaggy appearance of the tentorium and draining dural sinus in cases of dAVF, and can be diagnosed by CTA [46]. CTA has a combined sensitivity and specificity of ³90% to detect dAVF and provides a convenient low-risk test to detect dAVF as a cause of PT [29]. However, CTA has limitations because of the static nature of the examination and has less sensitivity than DSA, and this study had a small sample size of 7 patients. CTA can also detect other pathologies such as AVM, and the reconstructed thin-section bony window images provide osseous details [29,47]. CTA can also diagnose glomus jugulare, although contrast is not necessary in its diagnosis [48].

Slight modification from usual CTA bolus timing can allow for a balanced phase examination facilitating detection of both arterial and venous etiologies of PT in the head and neck. Therefore, it is the imaging modality of choice in patients with PT without a suspected retrotympenic lesion because it can show both arterial and venous pathologies in the head, skull base, and neck, in addition to the bony details of the temporal bone. Temporal bone anatomy is also well seen on thin bone window reconstructions, which cannot be assessed on MRA/MRI. Bony reconstructions from CTA thus can show SSWA such as diverticulum and wall dehiscence due to bony contour abnormalities [27]. However, 1 study reported a cause of PT being found only in 68% to 72% of patients with PT despite an extensive workup with CTA, MRI, and US [12].

CTA head does not cover the neck and therefore will not be able to detect the vascular abnormalities in the neck that can cause PT.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympenic lesion on otoscopy.

Initial imaging.

J. CTV head with IV contrast

CTV can readily show various venous causes of PT such as transverse sinus stenosis and SSWA such as dehiscence and diverticulum. Transverse sinus stenosis, sigmoid sinus diverticulum, and sigmoid sinus dehiscence have been proposed as potential causes for PT [50]. Thin-section bone window reconstructions from the CTV are used to detect wall dehiscence, whereas diverticulum is seen on both CTV and bone window reconstruction images. The SSWAs are seen because of bony contour abnormalities, and contrast offers confirmatory value in diverticulum but is not imperative. SSWAs

are much more frequently seen on CT temporal bones among patients with PT compared to the general population [26]. SSWAs are a known cause of vascular PT, and 4% to 32% of PT patients may have it, and they can be successfully treated by wall reconstruction or endovascular stenting [51-55].

Some studies have shown that the degree of transverse sinus stenosis seen on CTV correlates well with transstenotic pressure gradient measured on catheter manometry [26,43,56]. Transverse sinus stenosis could be a cause or result of IIH or SSWA [26,56]. Approximately 60% of the patients of IIH experience tinnitus. Stent placement in transverse sinus stenosis for disabling cases of PT can be curative [57]. Abrupt change in the luminal caliber of the transverse sinus is proposed as a possible etiology for PT and can be seen on CTV of the head. Unilateral dominant size of the venous system is a normal variant in the general population but is equivocal as a causal factor for PT [45]. Venous causes of PT are more frequent than arterial causes [11]. CTV cannot detect arterial abnormalities, whereas a CTA/CTV can detect both arterial and venous abnormalities.

VARIANT 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

K. MRA head with IV contrast

MRA head with IV contrast can diagnose dAVF, AVM, and larger glomus jugulare tumors, which can cause PT. A study on 54 patients with PT found excellent accuracy of combined MRA and MRI head without and with IV contrast to detect various pathologies causing PT such as dAVF, AVM, and paragangliomas [58]. IV contrast improves the sensitivity to detect dAVF, AVM, and vascular tumors like glomus jugulare. Time-resolved gadolinium-enhanced MRA is a useful technique in showing vessels with high temporal and spatial resolution to detect dAVFs. It is a reliable technique in the screening and surveillance of DAVF [16,18].

Catheter angiography has better sensitivity and may still be considered if MR studies are negative and dAVF is still suspected [1,23,29]. MRA head cannot identify bony lesions like jugular fossa dehiscence, SSWA, SSCD, intracranial mass lesions, and venous causes of PT. It is usually combined with MRI head with and without contrast for a better evaluation of intracranial pathologies which can cause PT.

VARIANT 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

L. MRA head without and with IV contrast

MRA head without and with IV contrast may be used to detect dAVF, AVM, larger glomus jugulare tumors, carotid dissection, and carotid cavernous fistula for evaluation of PT when otoscopy does not show a vascular retrotympanic lesion. Catheter angiography has better sensitivity and may still be considered if MR studies are negative and dAVF is still suspected [1,23,29]. MRA head cannot identify bony lesions like jugular fossa dehiscence, SSWA, SSCD, intracranial mass lesions, and venous causes of PT. It is usually combined with MRI head without and with contrast for a better evaluation of intracranial pathologies that can cause PT.

VARIANT 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

M. MRA head without IV contrast

MRA head without IV contrast can diagnose AVM and aberrant ICA but has less sensitivity to diagnose dAVF, smaller AVM, and small glomus tumors. MRA would not be able to diagnose bony

lesions like jugular fossa dehiscence, SSWA, and SSCD, intracranial mass lesions, and venous causes of PT. MRA head without IV contrast is usually combined with MRI head with and without IV contrast for a better evaluation of intracranial pathologies, which can cause PT [58]. Source images of 3-D time-of-flight MRA and DSA can detect moderate to high-flow dAVF; however, it is not as sensitive as catheter angiography [59]. Catheter angiography has better sensitivity and may still be considered if MR studies are negative and dAVF is still suspected [1,23,29].

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

N. MRI head and internal auditory canal with IV contrast

There is no relevant literature to support the use of MRI head and IAC with IV contrast for evaluation of PT.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

O. MRI head and internal auditory canal without and with IV contrast

Vascular loops in contact with cranial nerve (CN) VIII are a normal variant and may be seen in up to one-third of normal patients. However, they may contribute to otological symptoms due to neurovascular compression. Patients with PT are 80 times more likely to have vascular loops in contact with CN VIII than patients without PT [60]. Heavily T2-weighted thin-section sequences of MRI head can detect the neurovascular loops in patients with PT [61,62]. Typewriter tinnitus is described as paroxysmal attacks of staccato sounds, thought to be caused by neurovascular compression of the cochlear nerve, and responds well to carbamazepine.

MRI can be helpful for detection and evaluation of the extension of the glomus jugulare in the internal jugular vein. Mass lesions in the IAC and posterior fossa such as schwannoma, meningioma, and endolymphatic sac tumor, are readily diagnosed by MRI. Vestibular schwannomas cause NPT more commonly than PT. Other masses can rarely cause tinnitus [49,63,64].

Postgadolinium 3-D images through the brain show detailed anatomy of the transverse and sigmoid dural venous sinuses and can be used to detect the transverse sinus stenosis. The sigmoid sinus diverticulum can also be seen on these images.

MRI 4-D flow can detect abnormal flow patterns in the transverse sinuses in patients with venous PT [65]. It was able to detect increased blood flow and blood velocity in patients who had markers of venous PT such as SSWA and transverse sinus stenosis [43].

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

P. MRI head and internal auditory canal without IV contrast

Vascular loops in contact with CN VIII are a normal variant and may be seen in up to one-third of normal patients. However, they may contribute to otological symptoms due to neurovascular compression. Patients with PT are 80 times more likely to have vascular loops in contact with CN VIII than patients without PT [60]. Heavily T2-weighted thin-section sequences of MRI head can detect the neurovascular loops in patients with PT [61,62]. Typewriter tinnitus is described as paroxysmal attacks of staccato sounds, thought to be caused by neurovascular compression of the cochlear nerve, and responds well to carbamazepine. MRI without IV contrast can also detect some

larger mass lesions like glomus jugulare and AVM that can cause PT. However, absence of contrast would limit its ability to detect smaller masses and AVM.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

Q. MRV head with IV contrast

MRV head with IV contrast is a robust tool for detecting stenosis in the transverse sinuses, which could lead to tinnitus in association with IIH. Dural venous sinus stenosis could be from an intrinsic or extrinsic cause [66,67]. The source images can potentially detect a prominent sigmoid sinus diverticulum. However, MRV does not provide bony details to evaluate other pathologies that can cause PT such as sigmoid wall dehiscence and jugular foramen dehiscence.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

R. MRV head without and with IV contrast

MRV head with IV contrast is a robust tool to detect stenosis in the transverse sinuses, which could lead to tinnitus in association with IIH as described in the previous section. However, MRV does not provide bony details to evaluate other pathologies that can cause PT such as sigmoid wall dehiscence and jugular foramen dehiscence. There is no added benefit in simultaneously performing MRV head without IV contrast also.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

S. MRV head without IV contrast

MRV head without IV contrast can detect transverse sinus stenosis, but flow-related artifacts can limit optimal evaluation of the transverse sinus stenosis and sigmoid sinus diverticulum [56]. Arachnoid granulations can mimic transverse sinus stenosis.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

T. US duplex Doppler carotid artery

US duplex Doppler carotid artery is useful to detect atherosclerotic narrowing of the carotids, which is a cause of PT. Atherosclerosis can lead to arterial stiffness, which is also associated with tinnitus [68]. Duplex US may be performed when there is a bruit in the neck and carotid stenosis is suspected. However, most other conditions which cause PT cannot be evaluated by US. US can also detect parameters of low flow resistance, high-flow velocity and high-flow volume in external carotid arteries in patients with suspected dAVF as a cause of PT [69]. However, sensitivity is lower than CTA, MRA, and conventional angiography.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

U. US duplex Doppler transcranial

There is no relevant literature to support the use of US duplex Doppler transcranial for evaluation of PT when otoscopy does not show a vascular retrotympanic lesion.

Variant 1: Pulsatile tinnitus, unilateral or bilateral; No retrotympanic lesion on otoscopy.

Initial imaging.

V. US head

There is no relevant literature to support the use of US head for evaluation of PT when otoscopy

does not show a vascular retrotymppanic lesion.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotymppanic lesion on otoscopy. Initial imaging.

PT has many causes, and no single imaging study is appropriate for all patients. A diagnostic algorithm based on detailed history and clinical evaluation should progress from less invasive to more invasive imaging studies, and a targeted history and clinical assessment can have implications on the choice of imaging modality [12,19]. Otoscopic examination is extremely useful to guide the correct imaging in evaluation of the PT. When a vascular retrotymppanic lesion is seen, the main differential diagnoses are glomus tympanicum, glomus jugulotympanicum, and vascular variants like aberrant ICA, dehiscent jugular foramen, and persistent stapedial artery (PSA). Otosclerosis can also appear as a pinkish retrotymppanic lesion (Schwartz sign). It is extremely important to diagnose the vascular variants because inadvertent biopsy can have devastating complications. High-resolution thin-section temporal bone CT (HRCT) without IV contrast has excellent accuracy to make these diagnoses by carefully evaluating the contour of the bone and air spaces. IV contrast is not necessary to diagnose these conditions. HRCT of the temporal bone would also show some other conditions that cause PT but do not present as a vascular retrotymppanic lesion. These conditions include high-riding jugular bulb, jugular bulb diverticulum, SSWA, SSCD, and Paget disease.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotymppanic lesion on otoscopy. Initial imaging.

A. Arteriography cervicocerebral

There is no relevant literature to support the use of cervicocerebral arteriography for evaluation of PT when otoscopy shows a vascular retrotymppanic lesion.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotymppanic lesion on otoscopy. Initial imaging.

B. CT head with IV contrast

There is no relevant literature to support the use of CT head with IV contrast for evaluation of PT.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotymppanic lesion on otoscopy. Initial imaging.

C. CT head without and with IV contrast

There is no relevant literature to support the use of CT head without and with IV contrast for evaluation of PT.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotymppanic lesion on otoscopy. Initial imaging.

D. CT head without IV contrast

There is no relevant literature to support the use of CT head without IV contrast for evaluation of PT.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotymppanic lesion on otoscopy. Initial imaging.

E. CT temporal bone with IV contrast

There is no relevant literature to support the use of CT temporal bone with IV contrast for evaluation of PT when otoscopy shows a vascular retrotymppanic lesion. CT temporal bone without contrast is adequate as described in the next section, and IV contrast does not offer any significant

benefits in those indications. Changes in contour of air and bone in the temporal bone study are the key features in making the diagnosis.

Variation 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

F. CT temporal bone without and with IV contrast

There is no relevant literature to support the use of CT temporal bone without and with IV contrast for evaluation of PT.

Variation 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

G. CT temporal bone without IV contrast

CT temporal bone without IV contrast is useful as a first-line imaging modality when a vascular retrotympenic lesion is seen on otoscopic examination. Retrotympenic lesion may be because of a retrotympenic mass such as a glomus tumor (paraganglioma), or vascular variants. It is imperative to distinguish between the two to avoid unnecessary biopsies, which can have devastating complications. CT temporal bone without contrast can show masses like glomus tympanicum, otic capsule lesions such as otosclerosis, or vascular variants like aberrant or lateralized internal carotid artery (ICA), PSA, and dehiscent jugular foramen [1,36].

HRCT of the temporal bone can also detect some other conditions that cause PT but do not always present as a vascular retrotympenic mass, such as otospongiosis, Paget disease, sigmoid sinus diverticulum and sigmoid sinus dehiscence, high-riding jugular bulb, and SSCD [39].

Variation 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

H. CTA head and neck with IV contrast

CTA is an excellent tool when a patient has PT, and intracranial or cervical vascular pathologies are suspected [45]. It can diagnose intracranial vascular pathology such as dAVFs and has a combined sensitivity and specificity of 90% and provides a convenient low-risk test [29,46]. CTA can also detect other pathologies such as AVM, glomus tumors, aberrant carotid, and PSA and can provide osseous details such as bony dehiscence from the thin bone reconstructions [29,47]. Thin-section bone reconstructions from the source images of the CTA provide enough resolution and details to detect bony abnormalities, vascular variants, and glomus tympanicum in patients with vascular retrotympenic lesions. Contrast is not necessary to make the diagnosis of aberrant ICA, PSA, dehiscent jugular foramen, or glomus tympanicum because changes in bony and air contour in the temporal bone are sufficient to make these diagnoses. Although CTA can detect a lot of the pathologies that can be the cause of PT when there is a retrotympenic vascular lesion, CT of temporal bone can accomplish the same, and perhaps better, because of a smaller field-of-view and higher resolution, and therefore that is preferred over the CTA.

Variation 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

I. CTA head with IV contrast

CTA is an excellent tool when a patient has PT, and intracranial or cervical vascular pathologies are suspected [45]. It can diagnose intracranial vascular pathology such as dAVFs and has a combined sensitivity and specificity of 90% and provides a convenient low risk test [29,46]. CTA can also detect other pathologies such as AVM, glomus tumors, aberrant carotid, and PSA and can provide

osseous details such as bony dehiscence from the thin bone reconstructions to merit its use as a first-choice imaging technique [29,47]. Thin-section bone reconstructions from the source images of the CTA provide enough resolution and details to detect bony abnormalities, vascular variants, and glomus tympanicum in patients with vascular retrotympenic lesions. Contrast is not necessary to make diagnosis of aberrant ICA, PSA, dehiscent jugular foramen, or glomus tympanicum because changes in bony and air contour in the temporal bone are sufficient to make a diagnosis. If a confirmatory study is needed for a suspected glomus tympanicum, then MRI with contrast is preferred over CT with contrast or CTA.

Although CTA can detect a lot of the pathologies that can be the cause of PT when there is a retrotympenic vascular lesion, CT of temporal bone can accomplish the same, and perhaps better, because of a smaller field-of-view and higher resolution, and therefore that is preferred over the CTA.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

J. CTV head with IV contrast

Thin-section bone reconstructions from the source images of the CTV provide enough resolution and details to detect bony abnormalities, vascular variants, and glomus tympanicum, which present as vascular retrotympenic lesions. Contrast is not necessary to make diagnosis of aberrant ICA, PSA, high-riding jugular bulb, dehiscent jugular foramen, or glomus tympanicum, because changes in bony and air contour in the temporal bone are sufficient to make a diagnosis. Although CTV can be used for a lot of the pathologies that can be the cause of PT when there is a retrotympenic vascular lesion, CT of temporal bone can accomplish the same, and perhaps better, because of a smaller field-of-view and higher resolution, and therefore that is preferred over the CTV.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

K. MRA head with IV contrast

There is no relevant literature to support the use of MRA head with IV contrast for evaluation of PT when otoscopy shows a vascular retrotympenic lesion. Aberrant ICA can be diagnosed by MRA head with IV contrast. However, it is not a good test to diagnose other conditions like glomus tympanicum, PSA, glomus jugulotympanicum, or dehiscent jugular foramen, which can present as vascular retrotympenic lesions.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

L. MRA head without and with IV contrast

There is no relevant literature to support the use of MRA head without and with IV contrast for evaluation of PT when otoscopy shows a vascular retrotympenic lesion.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

M. MRA head without IV contrast

There is no relevant literature to support the use of MRA head without IV contrast for evaluation of PT when otoscopy shows a vascular retrotympenic lesion. Aberrant ICA can be diagnosed by MRA head without IV contrast. However, it is not a good test to diagnose other conditions like glomus tympanicum, PSA, glomus jugulotympanicum, or dehiscent jugular foramen, which can present as vascular retrotympenic lesions.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

N. MRI head and internal auditory canal with IV contrast

There is no relevant literature to support the use of MRI head and IAC with IV contrast for evaluation of PT when a retrotympenic lesion is suspected on otoscopy.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

O. MRI head and internal auditory canal without and with IV contrast

The usual etiologies for vascular retrotympenic lesions are glomus tympanicum, glomus jugulotympanicum, aberrant ICA, PSA, and dehiscent jugular bulb. Small glomus tympanicum and glomus jugulotympanicum may be difficult to see on the MRI head and are easily seen on the CT temporal bone. There is no relevant literature to support the use of MRI head for detection of vascular variants that may present as vascular retrotympenic lesions.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

P. MRI head and internal auditory canal without IV contrast

The usual etiologies for vascular retrotympenic lesions are glomus tympanicum, glomus jugulotympanicum, aberrant ICA, PSA, and dehiscent jugular bulb. Tiny glomus tympanicum may be difficult to see on the MRI head and are easily seen on the CT temporal bone. There is no relevant literature to support the use of MRI head for detection of vascular variants that may present as vascular retrotympenic lesions.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

Q. MRV head with IV contrast

There is no relevant literature to support the use of MRV head with IV contrast for evaluation of PT when a retrotympenic lesion is suspected on otoscopy.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

R. MRV head without and with IV contrast

There is no relevant literature to support the use of MRV head without and with IV contrast for evaluation of PT when a retrotympenic lesion is suspected on otoscopy.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

S. MRV head without IV contrast

There is no relevant literature to support the use of MRV head without contrast for evaluation of PT when a retrotympenic lesion is suspected on otoscopy.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

T. US duplex Doppler carotid artery

There is no relevant literature to support the use of US duplex doppler carotid artery for evaluation of PT when a retrotympenic lesion is suspected on otoscopy.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympenic lesion on otoscopy. Initial imaging.

U. US duplex Doppler transcranial

There is no relevant literature to support the use of US duplex doppler transcranial for evaluation of PT when otoscopy shows a vascular retrotympanic lesion.

Variant 2: Pulsatile tinnitus, unilateral or bilateral; suspected retrotympanic lesion on otoscopy. Initial imaging.

V. US head

There is no relevant literature to support the use of US head for evaluation of PT when otoscopy shows a vascular retrotympanic lesion.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

NPT is described as a continuous sound that is not synchronous with the patient's heartbeat. It is almost always subjective and is more prevalent than PT. Unilateral NPT can be idiopathic or have a defined etiology. Thorough history, physical examination, and audiometry are helpful for elucidating a cause. NPT can be caused by cerumen impaction, chronic otitis media, tympanic membrane perforation, and cholesteatoma, which are conditions that can be seen on otoscopic examination. Middle ear adenomatous tumors are rare tumors that can cause NPT. They show contrast enhancement on MRI (and possibly CT especially if large enough) but no vascular blush on angiography [70]. History can point to prior chronic noise exposure or acoustic trauma as a cause. Neurological conditions like brainstem infarction, multiple sclerosis, and cerebellopontine angle tumors can also cause unilateral NPT [34]. Tinnitus often coexists with many other symptoms such as hearing loss, vertigo, previous head trauma, and neurological deficits. If there are such concomitant symptoms, then imaging should be guided by the ACR Appropriateness Criteria[®] topics for "[Hearing Loss and/or Vertigo](#)" [8], "[Head Trauma](#)" [9], and "[Cerebrovascular Diseases- Aneurysm, Vascular Malformation, and Subarachnoid Hemorrhage](#)" [10]. Patients with unilateral or asymmetrical NPT with additional neurological, otological, and head and neck symptoms are more likely to have an underlying causative pathology than patients with no additional symptoms or patients with bilateral tinnitus [71]. Imaging should be considered to exclude a retrocochlear lesion in cases of unilateral NPT [72]. Patients with temporomandibular joint derangement have increased incidence of tinnitus, but the underlying mechanism is unclear [73]. IIH can cause both PT and NPT, and venous stenting can provide relief in some patients, but its indiscriminate use is controversial [66,74].

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

A. Arteriography cervicocerebral

There is no relevant literature to support the use of conventional arteriography for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

B. CT head with IV contrast

There is no relevant literature to support the use of CT head with IV contrast for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

C. CT head without and with IV contrast

There is no relevant literature to support the use of CT head without and with IV contrast for evaluation of unilateral NPT.

Variation 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

D. CT head without IV contrast

There is no relevant literature to support the use of CT head without IV contrast for evaluation of unilateral NPT.

Variation 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

E. CT temporal bone with IV contrast

CT temporal bone with IV contrast may be considered in a few patients if they need imaging to evaluate for retrocochlear pathology and MRI cannot be performed. Larger mass lesions such as vestibular schwannomas and meningiomas can be seen. However, small lesions, especially when located in the IAC rather than the cerebellopontine angle cistern, can be missed on CT because of the limited contrast resolution.

Middle ear adenomatous tumors are rare tumors in the middle ear that can cause NPT. They show significant contrast enhancement on CT and MRI but no vascular blush on angiography [70]. Otoscopic examination reveals a retrotympenic mass.

Variation 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

F. CT temporal bone without and with IV contrast

There is no relevant literature to support the use of CT temporal bone without and with IV contrast for evaluation of unilateral NPT.

Variation 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

G. CT temporal bone without IV contrast

CT can diagnose cochlear nerve aperture stenosis, which is associated with cochlear nerve hypoplasia, a condition in which patients can present with sensorineural hearing loss and tinnitus [75]. CT temporal bone is considered if chronic inflammation in the middle ear is suspected to differentiate otitis media from cholesteatoma [76]. It can also be considered for abnormalities of the vestibular aqueduct.

Variation 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

H. CTA head and neck with IV contrast

There is no relevant literature to support the use of CTA head and neck with IV contrast for evaluation of unilateral NPT.

Variation 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

I. CTA head with IV contrast

There is no relevant literature to support the use of CTA head with IV contrast for routine evaluation of unilateral NPT.

Variation 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no

trauma. Initial imaging.

J. CTV head with IV contrast

There is no relevant literature to support the use of CTV head with IV contrast for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

K. MRA head with IV contrast

There is no relevant literature to support the use of MRA head with IV contrast for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

L. MRA head without and with IV contrast

There is no relevant literature to support the use of MRA head without and with IV contrast for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

M. MRA head without IV contrast

There is no relevant literature to support the use of MRA head without IV contrast for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

N. MRI head and internal auditory canal with IV contrast

There is no relevant literature to support the use of MRI head and IAC with IV contrast for evaluation of unilateral NPT. The study should be performed without and with IV contrast as described in the next section.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

O. MRI head and internal auditory canal without and with IV contrast

MRI head and IAC without and with IV contrast can be considered for retrocochlear processes such as vestibular schwannoma or other mass lesions in patients with NPT [71,77]. Tinnitus may occur among patients with vestibular schwannomas (63%-75%) and intralabyrinthine schwannomas [78]. These are readily diagnosed by MRI. Rarely, meningiomas in or around the IAC and cerebellopontine angle cistern can also cause tinnitus and are accurately diagnosed on MRI [79,80]. Vestibular schwannomas cause NPT more commonly than PT [49]. Mass lesions in the IAC and posterior fossa such as schwannomas, meningiomas, and endolymphatic sac tumors are readily diagnosed by MRI. A study of 218 patients with NPT found that 91.8% of patients had an unremarkable MRI [81].

Spontaneous intracranial hypotension can cause tinnitus, probably due to venous engorgement in the IAC [82,83]. Endolymphatic hydrops has an association with Meniere disease, which presents with episodic hearing loss, vertigo, and tinnitus [84-87] and requires special sequences for diagnosis, including heavily T2-weighted 3-D FLAIR (fluid-attenuated inversion recovery) and 3-D real inversion recovery performed 4 hours after injection of the IV contrast [88], best interpreted alongside a routine 3-D heavily T2-weighted sequence.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

P. MRI head and internal auditory canal without IV contrast

CISS (Constructive Interference Steady State), FIESTA (Fast Imaging Employing Steady-state Acquisition), SPACE (sampling perfection with application optimized contrasts using different flip angle evolution), or DRIVE (driven equilibrium radio frequency reset pulse) sequences of the MRI head and IAC without IV contrast provides high sensitivity and specificity even for small vestibular schwannomas. However, tumors ≤ 3 mm and intralabyrinthine schwannomas are at risk of being missed by the noncontrast study [89]. Vertigo and tinnitus are not uncommon in patients with temporomandibular joint problems for unknown reasons, and there seems to be an association. Dedicated MRI of the temporomandibular joint may be considered if temporomandibular joint pathology is suspected [90,91].

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

Q. MRV head with IV contrast

There is no relevant literature to support the use of MRV head with IV contrast for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

R. MRV head without and with IV contrast

There is no relevant literature to support the use of MRV head without and with IV contrast for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

S. MRV head without IV contrast

There is no relevant literature to support the use of MRV head without IV contrast for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

T. US duplex Doppler carotid artery

There is no relevant literature to support the use of US duplex Doppler carotid artery for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

U. US duplex Doppler transcranial

There is no relevant literature to support the use of US duplex Doppler transcranial for evaluation of unilateral NPT.

Variant 3: Nonpulsatile tinnitus, unilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

V. US head

There is no relevant literature to support the use of US head for evaluation of unilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

Imaging is not helpful in cases of bilateral NPT related to age-related hearing loss, prior exposure to noise, acoustic trauma, ototoxic medications, and chronic bilateral hearing loss [92]. Most cases of bilateral NPT are associated with sensorineural hearing loss, and imaging is typically unrevealing in such cases [34,93,94]. The AAO-HNS guidelines make a strong recommendation against any imaging studies of the head and neck for the subset of patients in whom tinnitus does not localize to 1 ear, is nonpulsatile, and is not associated with focal neurological abnormalities or an asymmetric hearing loss [6,7].

Tinnitus often coexists with many other symptoms such as hearing loss, vertigo, previous head trauma, and neurological deficits. ACR Appropriateness Criteria[®] topics for "[Hearing Loss and/or Vertigo](#)" [8], "[Head Trauma](#)" [9], and "[Cerebrovascular Diseases-Aneurysm, Vascular Malformation, and Subarachnoid Hemorrhage](#)" [10] should be used to guide imaging in these settings.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

A. Arteriography cervicocerebral

There is no relevant literature to support the use of conventional arteriography for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

B. CT head with IV contrast

There is no relevant literature to support the use of CT head with IV contrast for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

C. CT head without and with IV contrast

There is no relevant literature to support the use of CT head without and with IV contrast for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

D. CT head without IV contrast

There is no relevant literature to support the use of CT head without IV contrast for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

E. CT temporal bone with IV contrast

There is no relevant literature to support the use of CT temporal bone with IV contrast for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

F. CT temporal bone without and with IV contrast

There is no relevant literature to support the use of CT temporal bone without and with IV contrast for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no

trauma. Initial imaging.

G. CT temporal bone without IV contrast

There is no relevant literature to support the use of CT temporal bone without IV contrast for evaluation of bilateral NPT.

Variante 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

H. CTA head and neck with IV contrast

There is no relevant literature to support the use of CTA head and neck with IV contrast for evaluation of bilateral NPT.

Variante 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

I. CTA head with IV contrast

There is no relevant literature to support the use of CTA head with IV contrast for evaluation of bilateral NPT.

Variante 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

J. CTV head with IV contrast

There is no relevant literature to support the use of CTV head with iv contrast for evaluation of bilateral NPT.

Variante 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

K. MRA head with IV contrast

There is no relevant literature to support the use of MRA head with IV contrast for evaluation of bilateral NPT.

Variante 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

L. MRA head without and with IV contrast

There is no relevant literature to support the use of MRA head without and with IV contrast for evaluation of bilateral NPT.

Variante 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

M. MRA head without IV contrast

There is no relevant literature to support the use of MRA head without IV contrast for evaluation of bilateral NPT.

Variante 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

N. MRI head and internal auditory canal with IV contrast

There is no relevant literature to support the use of MRI head and IAC with IV contrast for evaluation of bilateral NPT.

Variante 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

O. MRI head and internal auditory canal without and with IV contrast

There is no relevant literature to support the use of MRI head and IAC without and with IV contrast for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

P. MRI head and internal auditory canal without IV contrast

There is no relevant literature to support the use of MRI head and IAC without IV contrast for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

Q. MRV head with IV contrast

There is no relevant literature to support the use of MRV head with IV contrast for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

R. MRV head without and with IV contrast

There is no relevant literature to support the use of MRV head without and with IV contrast for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

S. MRV head without IV contrast

There is no relevant literature to support the use of MRV head without IV contrast for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

T. US duplex Doppler carotid artery

There is no relevant literature to support the use of US duplex Doppler carotid artery for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

U. US duplex Doppler transcranial

There is no relevant literature to support the use of US duplex Doppler transcranial for evaluation of bilateral NPT.

Variant 4: Nonpulsatile tinnitus, bilateral; no hearing loss, and no neurologic deficit, and no trauma. Initial imaging.

V. US head

There is no relevant literature to support the use of US head for evaluation of bilateral NPT.

Summary of Highlights

- **Variant 1:** In the setting of unilateral or bilateral PT with no retrotympanic lesion on otoscopy, a combination of parenchymal and vascular imaging is usually appropriate. This may include either CTA of the head and neck with IV contrast, CTA of the head with IV

contrast, or a combination of MRI head and internal auditory canal without and with IV contrast, MRA head with IV contrast, and/or MRV head with IV contrast, which are complementary examinations. CTA of the head and neck with IV contrast is more commonly used in this setting.

- **Variation 2:** In the setting of unilateral or bilateral PT with a suspected retrotympenic lesion on otoscopy, CT temporal bone without IV contrast is usually appropriate to evaluate for lesions such as glomus tympanicum, glomus jugulotympanicum, and vascular variants like aberrant ICA, dehiscent jugular foramen, and PSA. Dedicated vascular imaging using CTA head, CTV head, and CTA head and neck may be appropriate because thin-section bony reconstructions from the raw data show bony details of the temporal bone and can characterize most retrotympenic lesions.
- **Variation 3:** In the setting of unilateral, NPT without hearing loss, or neurological deficit, or history of trauma, MRI head and IAC without and with IV contrast is usually appropriate to evaluate for a retrocochlear lesion. MRI head and IAC without IV contrast may be appropriate but is considered slightly less sensitive for small lesions.
- **Variation 4:** In the setting of bilateral, NPT without hearing loss, or neurological deficit, or history of trauma, imaging is not recommended.

Supporting Documents

The evidence table, literature search, and appendix for this topic are available at <https://acsearch.acr.org/list>. The appendix includes the strength of evidence assessment and the final rating round tabulations for each recommendation.

For additional information on the Appropriateness Criteria methodology and other supporting documents, please go to the ACR website at <https://www.acr.org/Clinical-Resources/Clinical-Tools-and-Reference/Appropriateness-Criteria>.

Appropriateness Category Names and Definitions

Appropriateness Category Name	Appropriateness Rating	Appropriateness Category Definition
Usually Appropriate	7, 8, or 9	The imaging procedure or treatment is indicated in the specified clinical scenarios at a favorable risk-benefit ratio for patients.
May Be Appropriate	4, 5, or 6	The imaging procedure or treatment may be indicated in the specified clinical scenarios as an alternative to imaging procedures or treatments with a more favorable risk-benefit ratio, or the risk-benefit ratio for patients is equivocal.
May Be Appropriate (Disagreement)	5	The individual ratings are too dispersed from the panel median. The different label provides transparency regarding the panel's recommendation. "May be appropriate" is the rating category and a rating of 5 is assigned.
Usually Not Appropriate	1, 2, or 3	The imaging procedure or treatment is unlikely to be indicated in the specified clinical scenarios, or the

	risk-benefit ratio for patients is likely to be unfavorable.
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Relative Radiation Level Information

Potential adverse health effects associated with radiation exposure are an important factor to consider when selecting the appropriate imaging procedure. Because there is a wide range of radiation exposures associated with different diagnostic procedures, a relative radiation level (RRL) indication has been included for each imaging examination. The RRLs are based on effective dose, which is a radiation dose quantity that is used to estimate population total radiation risk associated with an imaging procedure. Patients in the pediatric age group are at inherently higher risk from exposure, because of both organ sensitivity and longer life expectancy (relevant to the long latency that appears to accompany radiation exposure). For these reasons, the RRL dose estimate ranges for pediatric examinations are lower as compared with those specified for adults (see Table below). Additional information regarding radiation dose assessment for imaging examinations can be found in the ACR Appropriateness Criteria® [Radiation Dose Assessment Introduction](#) document.

Relative Radiation Level Designations

Relative Radiation Level*	Adult Effective Dose Estimate Range	Pediatric Effective Dose Estimate Range
○	0 mSv	0 mSv
☸	<0.1 mSv	<0.03 mSv
☸ ☸	0.1-1 mSv	0.03-0.3 mSv
☸ ☸ ☸	1-10 mSv	0.3-3 mSv
☸ ☸ ☸ ☸	10-30 mSv	3-10 mSv
☸ ☸ ☸ ☸ ☸	30-100 mSv	10-30 mSv

*RRL assignments for some of the examinations cannot be made, because the actual patient doses in these procedures vary as a function of a number of factors (e.g., region of the body exposed to ionizing radiation, the imaging guidance that is used). The RRLs for these examinations are designated as “Varies.”

References

1. Krishnan A, Mattox DE, Fountain AJ, Hudgins PA. CT arteriography and venography in pulsatile tinnitus: preliminary results. *AJNR Am J Neuroradiol.* 2006;27(8):1635-1638.
2. National Institutes of Health. National Institute on Deafness and Other Communication Disorders (NIDCD). Quick Statistics. Available at: <http://www.nidcd.nih.gov/health/statistics/Pages/quick.aspx>.
3. Coelho CB, Santos R, Campara KF, Tyler R. Classification of Tinnitus: Multiple Causes with the Same Name. [Review]. *Otolaryngol Clin North Am.* 53(4):515-529, 2020 Aug.
4. Abdalkader M, Nguyen TN, Norbash AM, et al. State of the Art: Venous Causes of Pulsatile Tinnitus and Diagnostic Considerations Guiding Endovascular Therapy. [Review]. *Radiology.* 300(1):2-16, 2021 07.
5. Fife TD. Neuro-otology of Systemic Disease. In: Lewis SL, ed. *Neurological Disorders due to Systemic Disease.* 1st ed. Oxford, UK: Wiley-Blackwell Health Sciences; 2013:145-54.
6. Dalrymple SN, Lewis SH, Philman S. Tinnitus: Diagnosis and Management. [Review]. *Am Fam Physician.* 103(11):663-671, 2021 06 01.

7. Tunkel DE, Bauer CA, Sun GH, et al. Clinical practice guideline: tinnitus. *Otolaryngol Head Neck Surg* 2014;151:S1-S40.
8. Sharma A, Kirsch CFE, Aulino JM, et al. ACR Appropriateness Criteria® Hearing Loss and/or Vertigo. *J Am Coll Radiol* 2018;15:S321-S31.
9. Shih RY, Burns J, Ajam AA, et al. ACR Appropriateness Criteria® Head Trauma: 2021 Update. *J Am Coll Radiol* 2021;18:S13-S36.
10. Ledbetter LN, Burns J, Shih RY, et al. ACR Appropriateness Criteria® Cerebrovascular Diseases-Aneurysm, Vascular Malformation, and Subarachnoid Hemorrhage. *J Am Coll Radiol* 2021;18:S283-S304.
11. Kumar R, Rice S, Lingam RK. Detecting causes of pulsatile tinnitus on CT arteriography-venography: A pictorial review. [Review]. *Eur J Radiol*. 139:109722, 2021 Jun.
12. Mattox DE, Hudgins P. Algorithm for evaluation of pulsatile tinnitus. *Acta Otolaryngol*. 2008;128(4):427-431.
13. Hofmeier B, Wolpert S, Aldamer ES, et al. Reduced sound-evoked and resting-state BOLD fMRI connectivity in tinnitus. *Neuroimage (Amst)*. 20:637-649, 2018.
14. Yakunina N, Kim SS, Nam EC. BOLD fMRI effects of transcutaneous vagus nerve stimulation in patients with chronic tinnitus. *PLoS ONE*. 13(11):e0207281, 2018.
15. Amukotuwa SA, Marks MP, Zaharchuk G, Calamante F, Bammer R, Fischbein N. Arterial Spin-Labeling Improves Detection of Intracranial Dural Arteriovenous Fistulas with MRI. *AJNR Am J Neuroradiol*. 39(4):669-677, 2018 Apr.
16. Farb RI, Agid R, Willinsky RA, Johnstone DM, Terbrugge KG. Cranial dural arteriovenous fistula: diagnosis and classification with time-resolved MR angiography at 3T. *AJNR Am J Neuroradiol*. 30(8):1546-51, 2009 Sep.
17. Grossberg JA, Howard BM, Saindane AM. The use of contrast-enhanced, time-resolved magnetic resonance angiography in cerebrovascular pathology. [Review]. *Neurosurgical Focus*. 47(6):E3, 2019 12 01.
18. Nishimura S, Hirai T, Sasao A, et al. Evaluation of dural arteriovenous fistulas with 4D contrast-enhanced MR angiography at 3T. *AJNR Am J Neuroradiol*. 31(1):80-5, 2010 Jan.
19. Schoeff S, Nicholas B, Mukherjee S, Kesser BW. Imaging prevalence of sigmoid sinus dehiscence among patients with and without pulsatile tinnitus. *Otolaryngol Head Neck Surg*. 150(5):841-6, 2014 May.
20. Waldvogel D, Mattle HP, Sturzenegger M, Schroth G. Pulsatile tinnitus--a review of 84 patients. *J Neurol*. 245(3):137-42, 1998 Mar.
21. Dong C, Zhao PF, Yang JG, Liu ZH, Wang ZC. Incidence of vascular anomalies and variants associated with unilateral venous pulsatile tinnitus in 242 patients based on dual-phase contrast-enhanced computed tomography. *Chin Med J*. 128(5):581-5, 2015 Mar 05.
22. Nicholson P, Brinjikji W, Radovanovic I, et al. Venous sinus stenting for idiopathic intracranial hypertension: a systematic review and meta-analysis. *J Neurointerv Surg*. 11(4):380-385, 2019 Apr.
23. Sonmez G, Basekim CC, Ozturk E, Gungor A, Kizilkaya E. Imaging of pulsatile tinnitus: a review of 74 patients. *Clin Imaging*. 2007;31(2):102-108.

24. Zhao P, Wang Z, Xian J, Yan F, Liu Z. Persistent petrosquamosal sinus in adults: qualitative imaging evaluation on high-resolution CT venography. *Acta Radiol.* 2014;55(2):225-230.
25. Grewal AK, Kim HY, Comstock RH 3rd, Berkowitz F, Kim HJ, Jay AK. Clinical presentation and imaging findings in patients with pulsatile tinnitus and sigmoid sinus diverticulum/dehiscence. *Otol Neurotol.* 35(1):16-21, 2014 Jan.
26. Harvey RS, Hertzano R, Kelman SE, Eisenman DJ. Pulse-synchronous tinnitus and sigmoid sinus wall anomalies: descriptive epidemiology and the idiopathic intracranial hypertension patient population. *Otol Neurotol* 2014;35:7-15.
27. Liu Z, Chen C, Wang Z, et al. Sigmoid sinus diverticulum and pulsatile tinnitus: analysis of CT scans from 15 cases. *Acta Radiol.* 54(7):812-6, 2013 Sep.
28. Madani G, Connor SE. Imaging in pulsatile tinnitus. [Review] [33 refs]. *Clin Radiol.* 64(3):319-28, 2009 Mar.
29. Narvid J, Do HM, Blevins NH, Fischbein NJ. CT angiography as a screening tool for dural arteriovenous fistula in patients with pulsatile tinnitus: feasibility and test characteristics. *AJNR Am J Neuroradiol.* 2011;32(3):446-453.
30. Ellenstein A, Yusuf N, Hallett M. Middle ear myoclonus: two informative cases and a systematic discussion of myogenic tinnitus. *Tremor Other Hyperkinet Mov (N Y)* 2013;3.
31. Fox GN, Baer MT. Palatal myoclonus and tinnitus in children. *West J Med* 1991;154:98-102.
32. Park SN, Bae SC, Lee GH, et al. Clinical characteristics and therapeutic response of objective tinnitus due to middle ear myoclonus: a large case series. *Laryngoscope* 2013;123:2516-20.
33. Sinclair CF, Gurey LE, Blitzer A. Palatal myoclonus: algorithm for management with botulinum toxin based on clinical disease characteristics. *Laryngoscope* 2014;124:1164-9.
34. Wu V, Cooke B, Eituti S, Simpson MTW, Beyea JA. Approach to tinnitus management. [Review]. *Can Fam Physician.* 64(7):491-495, 2018 07.
35. In 't Veld M, Fronczek R, de Laat JA, Kunst HPM, Meijer FJA, Willems PWA. The Incidence of Cranial Arteriovenous Shunts in Patients With Pulsatile Tinnitus: A Prospective Observational Study. *Otol Neurotol.* 39(5):648-653, 2018 06.
36. Remley KB, Coit WE, Harnsberger HR, Smoker WR, Jacobs JM, McIlff EB. Pulsatile tinnitus and the vascular tympanic membrane: CT, MR, and angiographic findings. *Radiology* 1990;174:383-9.
37. Pelkonen O, Tikkakoski T, Luotonen J, Sotaniemi K. Pulsatile tinnitus as a symptom of cervicocephalic arterial dissection. *J Laryngol Otol.* 2004;118(3):193-198.
38. von Babo M, De Marchis GM, Sarikaya H, et al. Differences and similarities between spontaneous dissections of the internal carotid artery and the vertebral artery. *Stroke.* 2013;44(6):1537-1542.
39. Sismanis A. Pulsatile tinnitus. *Otolaryngol Clin North Am.* 2003;36(2):389-402, viii.
40. Hillman TA, Kertesz TR, Hadley K, Shelton C. Reversible peripheral vestibulopathy: the treatment of superior canal dehiscence. *Otolaryngol Head Neck Surg.* 2006;134(3):431-436.
41. Jacky Chen CH, Nguyen T, Udawatta M, et al. Clinical Assessment of Patients with Bilateral Superior Semicircular Canal Dehiscence. *World Neurosurg.* 126:e1549-e1552, 2019 Jun.
42. Kline NL, Angster K, Archer E, et al. Association of pulse synchronous tinnitus and sigmoid

sinus wall abnormalities in patients with idiopathic intracranial hypertension. *Am J Otolaryngol.* 41(6):102675, 2020 Nov - Dec.

43. Li X, Qiu X, Ding H, et al. Effects of different morphologic abnormalities on hemodynamics in patients with venous pulsatile tinnitus: A four-dimensional flow magnetic resonance imaging study. *J Magn Reson Imaging.* 53(6):1744-1751, 2021 06.
44. Lao Z, Sha Y, Chen B, Dai CF, Huang WH, Cheng YS. Labyrinthine sequestrum: four case studies. *Otolaryngol Head Neck Surg.* 147(3):535-7, 2012 Sep.
45. Cho IK, Jung JY, Yoo DS, Suh MW. 3-Dimensional reconstruction of the venous system in patients suffering from pulsatile tinnitus. *Acta Otolaryngol.* 2012;132(3):285-289.
46. Cunnane MB. Imaging of Tinnitus. [Review]. *Neuroimaging Clin N Am.* 29(1):49-56, 2019 Feb.
47. Mundada P, Singh A, Lingam RK. CT arteriography and venography in the evaluation of Pulsatile tinnitus with normal otoscopic examination. *Laryngoscope.* 125(4):979-84, 2015 Apr.
48. Christie A, Teasdale E. A comparative review of multidetector CT angiography and MRI in the diagnosis of jugular foramen lesions. *Clin Radiol.* 65(3):213-7, 2010 Mar.
49. Bathla G, Hegde A, Nagpal P, Agarwal A. Imaging in Pulsatile Tinnitus: Case Based Review. *J Clin Imaging Sci.* 10:84, 2020.
50. Hewes D, Morales R, Raghavan P, Eisenman DJ. Pattern and severity of transverse sinus stenosis in patients with pulsatile tinnitus associated with sigmoid sinus wall anomalies. *Laryngoscope.* 130(4):1028-1033, 2020 04.
51. Eisenman DJ, Raghavan P, Hertzano R, Morales R. Evaluation and treatment of pulsatile tinnitus associated with sigmoid sinus wall anomalies. *Laryngoscope.* 128 Suppl 2:S1-S13, 2018 10.
52. ETTYREDDY AR, SHEW MA, DURAKOVIC N, et al. Prevalence, Surgical Management, and Audiologic Impact of Sigmoid Sinus Dehiscence Causing Pulsatile Tinnitus. *Otol Neurotol.* 42(1):82-91, 2021 01.
53. Liu Z, He X, Du R, Wang G, Gong S, Wang Z. Hemodynamic Changes in the Sigmoid Sinus of Patients With Pulsatile Tinnitus Induced by Sigmoid Sinus Wall Anomalies. *Otol Neurotol.* 41(2):e163-e167, 2020 02.
54. Wang AC, Nelson AN, Pino C, Svider PF, Hong RS, Chan E. Management of Sigmoid Sinus Associated Pulsatile Tinnitus: A Systematic Review of the Literature. [Review]. *Otol Neurotol.* 38(10):1390-1396, 2017 12.
55. Wang D, Zhao Y, Tong B. Treatment of pulsatile tinnitus caused by anomalies of the sigmoid sinus wall via combined internal and external sigmoid sinus wall reconstruction with 3D temporal bone CT guidance. *Eur Arch Otorhinolaryngol.* 277(9):2439-2445, 2020 Sep.
56. Zhao P, Ding H, Lv H, et al. CT venography correlate of transverse sinus stenosis and venous transstenotic pressure gradient in unilateral pulsatile tinnitus patients with sigmoid sinus wall anomalies. *Eur Radiol.* 31(5):2896-2902, 2021 May.
57. Lenck S, Labeyrie MA, Vallee F, et al. Stent Placement for Disabling Pulsatile Tinnitus Caused by a Lateral Sinus Stenosis: A Retrospective Study. *Oper Neurosurg (Hagerstown).* 13(5):560-565, 2017 10 01.

58. Deuschl C, Goricke S, Gramsch C, et al. Value of DSA in the diagnostic workup of pulsatile tinnitus. *PLoS One* 2015;10:e0117814.
59. Noguchi K, Melhem ER, Kanazawa T, Kubo M, Kuwayama N, Seto H. Intracranial dural arteriovenous fistulas: evaluation with combined 3D time-of-flight MR angiography and MR digital subtraction angiography. *AJR Am J Roentgenol* 2004;182:183-90.
60. Chadha NK, Weiner GM. Vascular loops causing otological symptoms: a systematic review and meta-analysis. *Clin Otolaryngol* 2008;33:5-11.
61. Guevara N, Deveze A, Buza V, Laffont B, Magnan J. Microvascular decompression of cochlear nerve for tinnitus incapacity: pre-surgical data, surgical analyses and long-term follow-up of 15 patients. *Eur Arch Otorhinolaryngol*. 2008;265(4):397-401.
62. Nowe V, De Ridder D, Van de Heyning PH, et al. Does the location of a vascular loop in the cerebellopontine angle explain pulsatile and non-pulsatile tinnitus? *Eur Radiol*. 2004;14(12):2282-2289.
63. Levine SB, Snow JB Jr. Pulsatile tinnitus. [Review] [38 refs]. *Laryngoscope*. 97(4):401-6, 1987 Apr.
64. Weissman JL, Hirsch BE. Imaging of tinnitus: a review. [Review] [49 refs]. *Radiology*. 216(2):342-9, 2000 Aug.
65. Li Y, Chen H, He L, et al. Hemodynamic assessments of venous pulsatile tinnitus using 4D-flow MRI. *Neurology*. 91(6):e586-e593, 2018 08 07.
66. Farid M, Alawamry A, Zaitoun MMA, Bessar AA, Darwish EAF. Relentless pulsatile tinnitus secondary to dural sinovenous stenosis: is endovascular sinus stenting the answer?. *Clin Radiol*. 76(7):526-531, 2021 07.
67. Sundararajan SH, Ramos AD, Kishore V, et al. Dural Venous Sinus Stenosis: Why Distinguishing Intrinsic-versus-Extrinsic Stenosis Matters. *AJNR Am J Neuroradiol*. 42(2):288-296, 2021 01.
68. Gedikli O, Kemal O, Yildirim U, et al. Is there an association between the parameters of arterial stiffness and tinnitus?. *Acta Otolaryngol (Stockh)*. 140(2):128-132, 2020 Feb.
69. Yeh SJ, Tsai LK, Jeng JS. Clinical and carotid ultrasonographic features of intracranial dural arteriovenous fistulas in patients with and without Pulsatile Tinnitus. *J Neuroimaging*. 2010;20(4):354-358.
70. Bierry G, Riehm S, Marcellin L, Stierle JL, Veillon F. Middle ear adenomatous tumor: a not so rare glomus tympanicum-mimicking lesion. *J Neuroradiol*. 37(2):116-21, 2010 May.
71. Lewis S, Chowdhury E, Stockdale D, Kennedy V, Guideline Committee. Assessment and management of tinnitus: summary of NICE guidance. *BMJ*. 368:m976, 2020 Mar 31.
72. Chari DA, Limb CJ. Tinnitus. [Review]. *Med Clin North Am*. 102(6):1081-1093, 2018 Nov.
73. Lee CF, Lin MC, Lin HT, Lin CL, Wang TC, Kao CH. Increased risk of tinnitus in patients with temporomandibular disorder: a retrospective population-based cohort study. *Eur Arch Otorhinolaryngol* 2016;273:203-8.
74. Funnell JP, Craven CL, Thompson SD, et al. Pulsatile versus non-pulsatile tinnitus in idiopathic intracranial hypertension. *Acta Neurochir (Wien)*. 160(10):2025-2029, 2018 10.
75. Ocak E, Kocaoz D, Acar B, Topcuoglu M. Radiological Evaluation of Inner Ear with Computed

Tomography in Patients with Unilateral Non-Pulsatile Tinnitus. *J. int. adv. otol.* 14(2):273-277, 2018 Aug.

76. Willinsky RA. Tinnitus: imaging algorithms. *Can Assoc Radiol J* 1992;43:93-9.
77. Gimsing S. Vestibular schwannoma: when to look for it? *J Laryngol Otol.* 2010;124(3):258-264.
78. Cao W, Hou Z, Wang F, Jiang Q, Shen W, Yang S. Larger tumor size and female gender suggest better tinnitus prognosis after surgical treatment in vestibular schwannoma patients with tinnitus. *Acta Otolaryngol (Stockh).* 140(5):373-377, 2020 May.
79. Jiang ZY, Kutz JW, Jr., Roland PS, Isaacson B. Intracochlear schwannomas confined to the otic capsule. *Otol Neurotol.* 2011;32(7):1175-1179.
80. Springborg JB, Poulsgaard L, Thomsen J. Nonvestibular schwannoma tumors in the cerebellopontine angle: a structured approach and management guidelines. *Skull Base* 2008;18:217-27.
81. Choi KJ, Sajisevi MB, Kahmke RR, Kaylie DM. Incidence of Retrocochlear Pathology Found on MRI in Patients With Non-Pulsatile Tinnitus. *Otol Neurotol.* 36(10):1730-4, 2015 Dec.
82. Arai M, Takada T, Nozue M. Orthostatic tinnitus: an otological presentation of spontaneous intracranial hypotension. *Auris Nasus Larynx* 2003;30:85-7.
83. Isildak H, Albayram S, Isildak H, Spontaneous intracranial hypotension syndrome accompanied by bilateral hearing loss and venous engorgement in the internal acoustic canal and positional change of audiography. *Journal of Craniofacial Surgery.* 21(1):165-7, 2010 Jan.
84. Loureiro RM, Sumi DV, Lemos MD, et al. The role of magnetic resonance imaging in Meniere disease: the current state of endolymphatic hydrops evaluation. *Einstein.* 17(1):eMD4743, 2019 Feb 25.
85. Paskoniene A, Baltagalviene R, Lengvenis G, et al. The Importance of the Temporal Bone 3T MR Imaging in the Diagnosis of Meniere's Disease. *Otol Neurotol.* 41(2):235-241, 2020 02.
86. Patel VA, Oberman BS, Zacharia TT, Isildak H. Magnetic resonance imaging findings in Meniere's disease. *J Laryngol Otol.* 131(7):602-607, 2017 Jul.
87. Perez-Carpena P, Lopez-Escamez JA. Current Understanding and Clinical Management of Meniere's Disease: A Systematic Review. *Semin Neurol.* 40(1):138-150, 2020 Feb.
88. Pyykko I, Zou J, Poe D, Nakashima T, Naganawa S. Magnetic resonance imaging of the inner ear in Meniere's disease. [Review]. *Otolaryngol Clin North Am.* 43(5):1059-80, 2010 Oct.
89. Liudahl AA, Davis AB, Liudahl DS, Maley J, Policeni B, Hansen MR. Diagnosis of small vestibular schwannomas using constructive interference steady state sequence. *Laryngoscope.* 128(9):2128-2132, 2018 09.
90. Chole RA, Parker WS. Tinnitus and vertigo in patients with temporomandibular disorder. *Arch Otolaryngol Head Neck Surg* 1992;118:817-21.
91. Park RJ, Moon JD. Prevalence and risk factors of tinnitus: the Korean National Health and Nutrition Examination Survey 2010-2011, a cross-sectional study. *Clin Otolaryngol* 2014;39:89-94.
92. Chemali Z, Nehme R, Fricchione G. Sensory neurologic disorders: Tinnitus. [Review]. *Handb.*

clin. neurol.. 165:365-381, 2019.

93. Oosterloo BC, Croll PH, de Jong RJB, Ikram MK, Goedegebure A. Prevalence of Tinnitus in an Aging Population and Its Relation to Age and Hearing Loss. *Otolaryngol Head Neck Surg.* 164(4):859-868, 2021 04.
94. Shapiro SB, Noij KS, Naples JG, Samy RN. Hearing Loss and Tinnitus. [Review]. *Med Clin North Am.* 105(5):799-811, 2021 Sep.
95. American College of Radiology. ACR Appropriateness Criteria® Radiation Dose Assessment Introduction. Available at: <https://edge.sitecorecloud.io/americancoldf5f-acrorgf92a-productioncb02-3650/media/ACR/Files/Clinical/Appropriateness-Criteria/ACR-Appropriateness-Criteria-Radiation-Dose-Assessment-Introduction.pdf>.

Disclaimer

The ACR Committee on Appropriateness Criteria and its expert panels have developed criteria for determining appropriate imaging examinations for diagnosis and treatment of specified medical condition(s). These criteria are intended to guide radiologists, radiation oncologists and referring physicians in making decisions regarding radiologic imaging and treatment. Generally, the complexity and severity of a patient's clinical condition should dictate the selection of appropriate imaging procedures or treatments. Only those examinations generally used for evaluation of the patient's condition are ranked. Other imaging studies necessary to evaluate other co-existent diseases or other medical consequences of this condition are not considered in this document. The availability of equipment or personnel may influence the selection of appropriate imaging procedures or treatments. Imaging techniques classified as investigational by the FDA have not been considered in developing these criteria; however, study of new equipment and applications should be encouraged. The ultimate decision regarding the appropriateness of any specific radiologic examination or treatment must be made by the referring physician and radiologist in light of all the circumstances presented in an individual examination.

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