

Role of Angiography in the Evaluation of Patients With Pulsatile Tinnitus

Edward J. Shin, MD; Anil K. Lalwani, MD; Christopher F. Dowd, MD

Objectives/Hypothesis: Pulsatile tinnitus in the face of normal findings on otoscopy is a common otological diagnostic dilemma and can be due to serious vascular malformations such as transverse or sigmoid sinus dural arteriovenous fistula (transverse or sigmoid sinus [TS] DAVF). Left untreated, TS DAVF may result in significant morbidity and mortality. TS DAVF can be suspected or diagnosed with computed tomography (CT), magnetic resonance imaging (MRI), and magnetic resonance angiography (MRA), with the gold standard being angiography. Our objective was to assess the utility of these various diagnostic modalities in the diagnosis of dural arteriovenous fistula. **Study Design:** Retrospective clinical review. **Methods:** Between 1986 and 1996, 54 patients were evaluated and treated for TS DAVF. Between 1996 and 1999, an additional 33 patients underwent MRI combined with MRA for the evaluation of pulsatile tinnitus. A retrospective review of the medical records for both groups, with special attention to clinical presentation, diagnostic evaluation, therapy, and outcome, was performed. **Results:** All patients had pulsatile tinnitus with normal findings on otoscopy. CT scan was relatively insensitive in the detection of TS DAVF. MRI and MRI/MRA were significantly more sensitive than CT. In the evaluation of patients with subjective pulsatile tinnitus, MRI/MRA defined anatomical abnormalities that may contribute to pulsatile tinnitus in 63% of patients. **Conclusions:** In the absence of objective pulsatile tinnitus, MRI/MRA is an appropriate initial diagnostic step. When a patient has an objective bruit, the clinician may choose to proceed directly to angiography to make certain that a TS DAVF is not missed. **Key Words:** Pulsatile tinnitus, transverse sinus or sigmoid sinus dural arteriovenous fistula, angiography, magnetic resonance angiography.

Laryngoscope, 110:1916–1920, 2000

From the Division of Otolaryngology, Neurotology and Skull Base Surgery (E.J.S., A.K.L.), Department of Otolaryngology—Head and Neck Surgery, and the Division of Neurointerventional Radiology (C.F.D.), Department of Radiology, University of California, San Francisco, San Francisco, California.

Editor's Note: This Manuscript was accepted for publication July 18, 2000.

Send Correspondence to Anil K. Lalwani, MD, Department of Otolaryngology—Head and Neck Surgery, 400 Parnassus Avenue, Room A 730, San Francisco, CA 94143-0342, U.S.A.

INTRODUCTION

Pulsatile tinnitus in the face of normal findings on otoscopy is a common otological diagnostic dilemma. The differential diagnosis is diverse and includes hydrocephalus, intracranial hypertension, atherosclerosis, and valvular heart disease, and the condition can be due to serious vascular malformations such as aneurysms or arteriovenous fistulas (Table I).

A thorough clinical history and physical examination direct further evaluation and management in patients with pulsatile tinnitus and normal findings on otoscopy. For young obese women with blurred vision, headaches, and papilledema a diagnosis of benign intracranial hypertension (BIH) may be established by lumbar puncture after magnetic resonance imaging (MRI).¹¹ Patients with a history of coronary artery disease, hypertension, and neck bruit are likely to have atherosclerotic carotid artery disease and are diagnosed by carotid artery duplex ultrasound.⁷ However, for patients without symptoms suggestive of coronary artery disease or BIH, but with a complaint of subjective or objective pulsatile tinnitus, initial diagnostic evaluation may begin with any number of radiographic examinations: computed tomography (CT), MRI, magnetic resonance imaging combined with magnetic resonance angiography (MRI/MRA), and angiography. These symptoms may be due to serious vascular malformations such as transverse or sigmoid sinus dural arteriovenous fistula (transverse or sigmoid sinus [TS] DAVF). Left untreated, TS DAVF may result in significant morbidity and mortality. As the disease progresses, the tinnitus may soften or may disappear as obstruction of venous outflow occurs. This may be a harbinger of more severe symptomatology of TS DAVF, including visual disturbances and intracranial hemorrhage, which may result in permanent neurological disability and potentially death. Previously we have demonstrated that the severity of the lesion at the time of detection has tremendous impact both on preoperative and postoperative intervention morbidity and on potential adverse consequences of therapeutic attempts.¹⁸ Early diagnosis and intervention may save patients from these potentially devastating consequences. Our objective was to assess the utility of these various modalities in establishing a diagnosis in this subset of patients suspected of having a vascular malformation.

TABLE I.
Causes of Pulsatile Tinnitus Reported in the Literature.

Type of lesion	Etiology
Arterial lesion	AVM ¹
	DAVF ²
	Carotid cavernous fistula ³
	Aneurysm of the ICA ⁴
	Fibromuscular dysplasia of the ICA ⁵
	Dissection of the ICA ⁶
	Atherosclerosis ⁷
	Vascular anomalies of ear ⁸
	Vascular compression of the eighth nerve ⁹
Venous lesion	Jugular bulb anomalies ¹⁰
	Benign intracranial hypertension ¹¹
Skull base, temporal lesions	Abnormal condylar/mastoid emissary veins ¹²
	Histiocytosis X ¹³
	Paget's disease ¹⁴
	Cavernous hemangioma ¹⁵
Miscellaneous	Glomus tumor ¹⁶
	Anemia, high cardiac output ¹⁷

AVM = arteriovenous malformation; DAVF = dural arteriovenous fistula; ICA = internal carotid artery.

PATIENTS AND METHODS

Between 1986 and 1996, 54 patients in the neurovascular service were evaluated and treated for possible TS DAVF. A retrospective review of the medical records with special attention to clinical presentation and diagnostic evaluation was performed. All patients had undergone a meticulous clinical evaluation including a thorough history, otoscopy, and auscultation of the mastoid, head, neck, and chest with a stethoscope. Tinnitus was defined as pulsatile when the patient described sounds synchronous with the heart rate and as objective when heard by the examining physician. Further diagnostic evaluation depended on decisions made by the treating physician. While there were differences among clinicians about the type of radiological studies ordered, most patients underwent some imaging study before angiography to identify possible vascular malformations. Sixteen patients were investigated by CT, 16 by MRI, and 3 by MRI/MRA. All patients ultimately underwent evaluation by angiography.

RESULTS

Clinical Features

The mean age of the 54 patients was 52.8 years (age range, 34–77 y); there were 32 women and 22 men. Of the 54 patients in our series, pulsatile tinnitus was the presenting symptom in 44 patients. The remaining 10 patients presented with neurological symptoms (i.e., headache, changes in mental status). Objective bruit on auscultation of the cranium (mastoid and ear canal) and neck was present in 72% (39 of 54) of the patients. Only one of 10 patients presenting with neurological symptoms had an objective bruit on examination. The remaining nine patients had no bruit on examination.

Ten patients presenting with pulsatile tinnitus had a history of trauma ranging in duration from 3 months to several years. Types of injury ranged from motor vehicle accident (MVA) or assault to fall. Eighty percent of pa-

tients with a history of trauma had an audible bruit, and all had identifiable origins or causes of their tinnitus.

Etiology

In 46 of 54 patients (85%) the underlying pathological factor was found to be TS DAVF. Remaining diagnoses included vertebral fistula (three), narrowing of transverse sinus (two), fibromuscular dysplasia (one), and carotid cavernous fistula (one). In one patient, the origin of pulsatile tinnitus remained undefined.

Computed Tomography

Ten of 16 CT scans were performed when presenting symptoms involved focal neurological changes revealing subarachnoid or parenchymal hemorrhage ultimately resulting in angiography and diagnosis of TSDAVF. Of the 10 patients presenting with bleeding, only 1 had a history of objective pulsatile tinnitus, and a second patient experienced objective tinnitus after the event. Of the remaining six patients, only one of six CT scans correctly diagnosed the underlying vascular abnormality. Misdiagnoses included scalp DAVF, TS DAVF, vertebral fistula, and narrowing of TS.

Magnetic Resonance Imaging and Magnetic Resonance Angiography

Of 19 MRI scans performed for pulsatile tinnitus, 12 were diagnostic of TS DAVF and 1 of vertebral arteriovenous fistula (AVF). The six nondiagnostic studies were read as normal. Of three MRA studies performed, all three were diagnostic of the underlying lesions. Table II summarizes the efficacy of the various diagnostic methods used to evaluate patients with suspected DAVF.

TABLE II.
Diagnostic Techniques Employed in 54 Patients With Possible TS DAVF (Correctly Diagnosed/Number Performed).

Diagnosis	CT	MRI	MRI/MRA	SA
TS DAVF	1/3	9/13	3/3	46/46
Scalp DAVF	0/1	0/1		1/1
Narrow transverse sinus	0/1			2/2
Fibromuscular dysplasia		1/1		1/1
Carotid cavernous fistula				1/1
Vertebral fistula	0/1	1/3		3/3
SAH	10/10			
Unknown		0/1		0/1

CT = computed tomography; MRI = magnetic resonance imaging; MRA = magnetic resonance angiography; SAH = subarachnoid hemorrhage; TS DAVF = transverse/sigmoid sinus dural arteriovenous fistula; SA = selective angiography.

Review of Patients Presenting With Pulsatile Tinnitus Evaluated by Magnetic Resonance Imaging and Magnetic Resonance Angiography

Because of the small number of MRA/MRI studies performed in the original group, a second review was undertaken to further evaluate the utility of this combined diagnostic modality in the setting of pulsatile tinnitus. Between 1996 to 1999, a search of the University of California, San Francisco (UCSF) database of MRI/MRA studies performed for patients with the diagnosis of pulsatile tinnitus revealed an additional 33 patients. A retrospective review was performed of these medical records as well. Further evaluation depended on the results of the study and the treating physician. There were 20 female and 13 male patients, with a mean age of 51 years (age range, 22–87 y). Tinnitus was unilateral in 28 patients and bilateral in 5. There was right ear involvement in 11 patients and left ear involvement in 17. All patients had normal findings on otoscopy, except four patients who presented with pulsatile tinnitus thought to be secondary to serous otitis media. Pulsatile tinnitus failed to resolve in two of four patients who had ventilation tubes placed; their radiographs confirmed persistent mastoid disease with no vascular abnormalities. Only one of the 33 patients had objective pulsatile tinnitus; his MRI/MRA finding was normal and no further workup was performed.

TABLE III.
Causes of Pulsatile Tinnitus Found in 33 Patients Evaluated by MRI/MRA.

Causes of pulsatile tinnitus	Patients (n)
High riding jugular bulb	9
Dominant transverse sinus	5
Attenuated transverse sinus	3
Fibromuscular dysplasia	1
Carotid dissection-healing	1
Serous otitis media	4
TS DAVF	1
Unknown	9

TS DAVF and fibromuscular dysplasia were diagnosed within the same patient.

The remaining 32 patients had subjective pulsatile tinnitus.

Of 33 patients evaluated by MRA/MRI, 5 subsequently underwent angiography. One MRI/MRA result was suspicious for carotid dissection which angiography confirmed. Two results were suggestive of TS DAVF. Angiography was performed on both patients, with the presence of TS DAVF and fibromuscular dysplasia confirmed in one, and no lesion defined in the other. A fourth patient had an MRI/MRA result consistent with a high-riding jugular bulb; persistent symptoms led to evaluation by angiography, which confirmed the prior results with no new findings. A fifth patient had an abnormal linear enhancement of lateral wall of cavernous sinus; subsequent angiography was normal.

Of the remaining patients, nine patients had no radiographic abnormalities on MRI/MRA to explain their pulsatile tinnitus. Three patients had an attenuated transverse sinus. Five patients had a dominant transverse sinus, and nine had a high-riding jugular bulb on the side of their tinnitus. As mentioned above, four patients had serous otitis media, one patient had carotid dissection, and one patient had TS DAVF (Table III).

DISCUSSION

Pulsatile tinnitus of vascular origin results from non-laminar blood flow generated by increased blood flow or lumen stenosis. Previous studies, as well as this study, have indicated that history and physical examination are critical in evaluating patients with pulsatile tinnitus. Conditions such as serous otitis media, high-riding jugular bulb, and glomus tympanicum may be diagnosed by otoscopy. Systemic disorders of high output (i.e., anemia, thyrotoxicosis, valve heart disease), arteriovenous malformations, DAVFs, arterial wall diseases (i.e., dissection, atherosclerosis, aneurysm), skull base tumors, and intracranial hypertension may all cause pulsatile tinnitus with normal findings on otoscopy.^{1,4,7,11,15–17}

In patients with normal findings on otoscopy and pulsatile tinnitus, experience from this study suggests that CT scan is less sensitive in diagnosis of vascular abnormalities. Only 16% (1 of 6) of the CT studies correctly diagnosed the lesion (excluding the 10 patients

who presented with subarachnoid or parenchymal hemorrhage that was correctly diagnosed by head CT, who later were diagnosed with TS DAVF on angiography). Each of these patients later underwent angiography for further evaluation of pulsatile tinnitus. Their diagnosis included scalp DAVF, TS DAVF, vertebral fistula, and narrowing of the transverse sigmoid sinus.

In addition, MRI alone detected only 68% (13 of 19) of abnormalities. Further evaluation by angiography revealed DAVF, vertebral fistula, or no distinct cause or origin of their tinnitus. MRI/MRA, on the other hand, appears to have a much higher sensitivity than MRI alone. In our study, 100% (3 of 3) of cases of TS DAVF were detected by MRI/MRA. However, small TS DAVFs may be missed by MRI/MRA.¹⁹ Because of the small number of MRI/MRAs reviewed, and to better assess the utility of this diagnostic modality, the evaluation of 33 additional patients who underwent MRI/MRA for pulsatile tinnitus (32 of 33 patients had subjective pulsatile tinnitus) was reviewed. Sixty-three percent of patients who underwent MRI/MRA had anatomical abnormalities and vascular malformations, potentially explaining their symptoms. Various nonpathological anatomical abnormalities (i.e., high-riding jugular bulb, dominant or attenuated transverse sinus) have been described as causes of pulsatile tinnitus.^{10,20,21} While the literature has cited these various findings as potential causes for pulsatile tinnitus and the chosen examples in Figures 1–3 are the likely cause for their symptoms, it is unlikely that the observed anatomical finding explains pulsatile tinnitus in every case.

The presence of an objective bruit heard in the mastoid or the cranium mandates further clinical evaluation. Seventy-two percent (38 of 54) of patients suspected of vascular malformation had an objective bruit. Excluding patients who presented with focal neurological change, 84% (37 of 44) patients had objective pulsatile tinnitus. Radiographic evaluation of these patients ranged from CT scan, MRI, and MRI/MRA to angiography. Of the 38 patients with an objective bruit, all but 3 had pathological vascular abnormalities that ultimately were diagnosed and treated by angiography and embolization. Narrowing

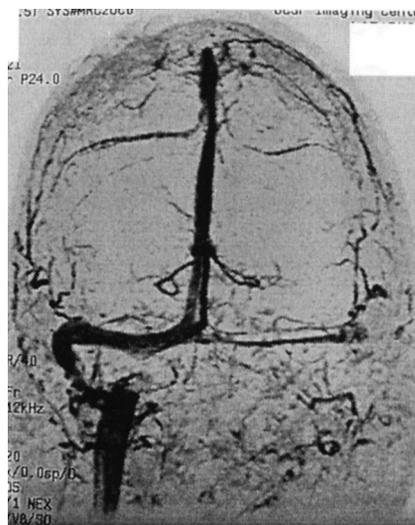


Fig. 2. Magnetic resonance angiography/magnetic resonance imaging of dominant transverse sinus and jugular bulb.

of the transverse sinus (in two cases) and no disease (in one case) explained the remaining three patients' symptoms. Thus objective pulsatile tinnitus is a harbinger of serious underlying vascular abnormality and should prompt evaluation by MRI/MRA or angiography.

In contrast, the underlying cause or origin of subjective pulsatile tinnitus is not always as easily identifiable and frequently is not pursued clinically for lack of physical findings to substantiate further costly and potentially invasive evaluation. With the advent of MRI/MRA, the clinician is armed with a noninvasive diagnostic tool with greater sensitivity than previously available. While it may be reasonable to directly proceed to diagnostic angiography with objective pulsatile tinnitus, this is unwarranted in patients with subjective pulsatile tinnitus.

One limitation of this retrospective review is that only five of the patients underwent subsequent angiography to validate observations made on MRA. To truly evaluate the incidence of disease with subjective pulsatile tinnitus and the sensitivity of MRA in the evaluation, angiographic evaluation in each of these patients would be required. Nonetheless, MRA detected abnormalities in 6% of patients with

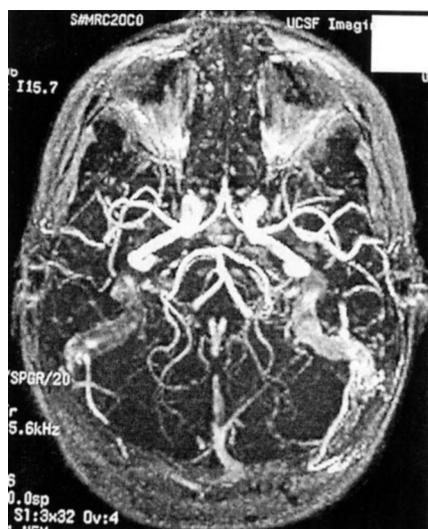


Fig. 1. Magnetic resonance angiography combined with magnetic resonance imaging of transverse or sigmoid sinus dural arteriovenous fistula (TS DAVF).



Fig. 3. Magnetic resonance angiography/magnetic resonance imaging of fibromuscular dysplasia.

CLINICAL EVALUATION

Normal otoscopy

Suspected abnormality	Diagnostic exam	Etiology
Increased ICP	MRI/MRA Neuro-Ophthalmology LP	BIH Hydrocephalus Thrombosis of dural sinus
Carotid/Cardiac Abnormality	Duplex Ultrasound Echocardiogram	ACAD Carotid tortuosity Valve dysfunction
Vascular malformation Subjective pulsatile tinnitus Objective pulsatile tinnitus	MRI/MRA Angiography	See Table 1

Fig. 4. Pulsatile tinnitus: diagnostic algorithm.

subjective pulsatile tinnitus that, if left untreated, could potentially lead to a catastrophic outcome. The appropriate use of MRI/MRA in the setting of subjective pulsatile tinnitus may reduce the necessity of angiography and the morbidity associated with it, and may lead to the detection of potentially life-threatening abnormalities.

Magnetic resonance imaging/magnetic resonance angiography is useful for diagnosing anatomical abnormalities that may be contributing to pulsatile tinnitus. Angiography, however, remains the gold standard, because of its high sensitivity and specificity. In patients with an audible bruit and pulsatile tinnitus and in patients with a history of trauma with new-onset pulsatile tinnitus, angiography is particularly warranted. Figure 4 summarizes our diagnostic algorithm, which is recommended for the evaluation of patients with pulsatile tinnitus with normal findings on otoscopy.

CONCLUSION

In the absence of objective pulsatile tinnitus, MRI/MRA is an appropriate initial diagnostic step. Radiographic screening ensures that potentially life-threatening disorders or diseases are not missed. When a patient has an objective bruit, the clinician may choose to proceed directly to angiography to make certain that pathological vascular abnormalities are not missed.

BIBLIOGRAPHY

- Arenberg IK, McCreary HS. Objective tinnitus aurium and dural arteriovenous malformations of the posterior fossa. *Ann Otol Rhinol Laryngol* 1972;80:111-120.
- Waldvogel D, Mattle HP, Sturzenegger M, Schroth G. Pulsatile tinnitus—a review of 84 patients. *J Neurol* 1998;245:137-142.
- Sismanis A. Pulsatile tinnitus: a 15-year experience. *Am J Otol* 1998;19:472-477.
- Levine SB, Snow JB, Jr. Pulsatile tinnitus. *Laryngoscope* 1987;97:401-406.
- Dufour JJ, Lavigne F, Plante R, et al. Pulsatile tinnitus and

- fibromuscular dysplasia of the internal carotid. *J Otolaryngol* 1985;14:293-295.
- Saeed SR, Hinton AE, Ramsden RT, et al. Spontaneous dissection of the intrapetrous internal carotid artery. *J Laryngol Otol* 1990;104:491-493.
- Sismanis A, Stamm MA, Sobel M. Objective tinnitus in patients with atherosclerotic disease. *Am J Otol* 1994 15:404-407.
- Steffan TN. Vascular anomalies of the middle ear. *Laryngoscope* 1968;78:171-197.
- Lesinski SG, Chambers AA, Komray R, et al. Why not the eighth nerve neurovascular compression: probable cause for pulsatile tinnitus. *Otolaryngol Head Neck Surg* 1979;87:89-94.
- Adler JR, Ropper AH: Self-audible venous bruits and high jugular bulb. *Arch Neurol* 1986;43:257-259.
- Sismanis A. Otolologic manifestations of benign intracranial hypertension syndrome: diagnosis and management. *Laryngoscope* 1987;97(Suppl 42):1-17.
- Lamberg P, Cantrell R. Objective tinnitus in association with abnormal posterior condylar emissary vein. *Am J Otolaryngol* 1986;7:204-207.
- Bonafe A, Joomye H, Jaeger P, Fraysse B, Manelfe C. Histiocytosis X of the petrous bone in the adult: MRI. *Neuroradiology* 1994;36:330-333.
- Gibson R. Tinnitus in Paget's disease with external carotid ligation. *J Laryngol Otol* 1973;87:299-301.
- Taber JR. Cavernous hemangioma of the middle ear and mastoid. *Laryngoscope* 1965;75:673-677.
- Spector GJ, Ciralsky RH, Ogura JH. Glomus tumors in the head and neck, III: analysis of clinical manifestations. *Ann Otol Rhinol Laryngol* 1975;84:73-79.
- Cochran JH, Kosmicki PW. Tinnitus as a presenting symptom of pernicious anemia. *Ann Otol Rhinol Laryngol* 1979;88:297.
- Shah SB, Lalwani AK, Dowd CF. Transverse/sigmoid sinus dural arteriovenous fistulas presenting as pulsatile tinnitus. *Laryngoscope* 1999;109:54-58.
- Koenigsberg RA. Spontaneous pulsatile tinnitus secondary to a dural malformation not visualized by magnetic resonance angiography. *Clin Imaging* 1996;20:95-98.
- Russell EJ, De Michaelis BJ, Wiet R, Meyer J. Objective pulse-synchronous 'essential' tinnitus due to narrowing of the transverse dural venous sinus. *Int Tinnitus J* 1995;1:127-137.
- Buckwalter JA, Sasaki CT, Virapongse C, et al. Pulsatile tinnitus arising from jugular megabulb deformity: a treatment rationale. *Laryngoscope* 1983;93:1534-1539.