The American Journal of Otology
19:472–477 © 1998, The American Journal of Otology, Inc.

Pulsatile Tinnitus

A 15-Year Experience

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Purpose: This study aimed to report the author's 15-year experience with the systematic evaluation and treatment of patients with pulsatile tinnitus.
Methods: Between August 1981 and August 1996, 145 patients with pulsatile tinnitus were evaluated. The diagnostic protocol was individualized according to the clinical findings and included appropriate radiologic testing, ultrasonography studies, and lumbar puncture with cerebrospinal fluid pressure measurement. Treatment was directed toward the etiology of the tinnitus.
Results: Benign intracranial hypertension syndrome (pseudotumor cerebri) was the most common diagnosis (56 patients), followed by atherosclerotic carotid artery disease (24 patients) and glomus tumors (17 patients). In 13 patients, no specific diagnosis could be reached.
Conclusions: Thorough history and physical examination are the two most important factors in evaluating patients with pulsatile tinnitus. Evaluation should be individualized and may include radiologic testing, ultrasonography studies, and lumbar puncture with cerebrospinal fluid pressure measurements. The majority of patients have a treatable underlying etiology.
Key Words: Pulsatile tinnitus—Intracranial hypertension.
Am J Otol **19**:472–477, 1998.

Pulsatile tinnitus (PT), although an infrequent otologic symptom, often presents a diagnostic and management dilemma to the neurotologist. Correct diagnosis is imperative because in the majority of cases, there is a treatable underlying etiology. In addition, failure to make proper diagnosis may be disastrous because in some patients, a life-threatening intracranial disease may be present. The purpose of this article is to present the author's 15-year experience with patients with PT.

MATERIALS AND METHODS

Between August 1981 and August 1996, 145 patients with PT were evaluated at the Medical College of Virginia/Virginia Commonwealth University. Our diagnostic protocol has been described previously (1) and is summarized as follows: All patients undergo a meticulous clinical evaluation including a thorough history, micro-otoscopy, and auscultation of the ear canals, head, neck, and chest with a modified electronic stethoscope (Fig. 1). Pure-tone and speech audiometry is performed on all patients.

During the first 4 years of the study, temporal bone and brain computed tomography as well as carotid angiography were obtained in all patients. After 1985, when intracranial hypertension was identified as a common cause of PT, carotid angiography was performed only on selected patients. Magnetic resonance imaging (MRI) was initiated in 1987 and magnetic resonance angiography (MRA) in 1992.

Duplex carotid ultrasound studies were obtained on patients suspected of having atherosclerotic carotid artery disease (ACAD). Cardiac echocardiography was used for patients with heart murmurs.

Patients suspected of having benign intracranial hypertension (BIH) syndrome were referred for a neuro-ophthalmologic consultation. Funduscopic existence of Papilledema is highly suggestive of BIH syndrome, even though its absence does not exclude this entity (2). After a head MRI, the diagnosis of BIH syndrome was confirmed with lumbar puncture (LP) and measurement of cerebrospinal fluid (CSF) pressure (3). Our diagnostic algorithm is depicted in Figure 2.

Treatment of these patients was individualized according to the underlying etiology. Weight loss, acetazolamide, and furosemide were recommended for patients with BIH syndrome (3). Gastric bypass surgery was considered for failure of conservative management.

RESULTS

Table 1 summarizes the radiologic results. An empty sella was the most common finding followed by small ventricles on intracranial imaging. Carotid angiography

Presented at the American Neurotology Society annual meeting, Washington, DC, September 28, 1996.

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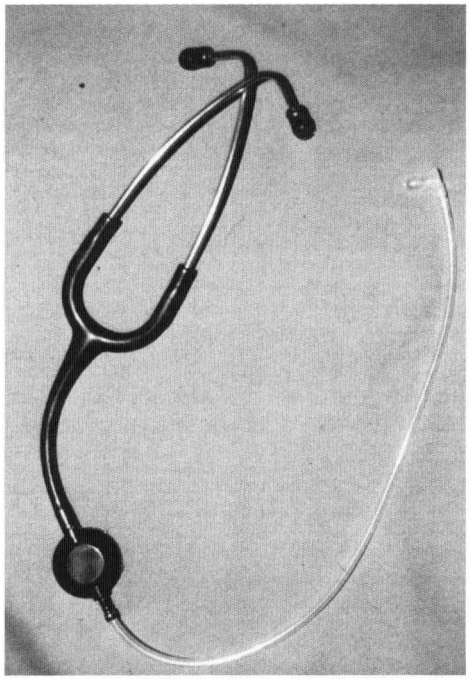

FIG. 1. Modified electronic stethoscope.

showed superior sagittal sinus thrombosis in one patient and significant stenosis in the carotid siphon in another patient. The MRA was suspicious for an arteriovenous fistula in five patients with confirmative carotid angiography in three (one between the internal carotid artery and the cavernous sinus and two between the external carotid vessels and the transverse sinus).

Table 2 depicts the various etiologies of PT in this study. The following paragraphs describe the results of the most common diagnoses among our patient population.

Benign intracranial hypertension (pseudotumor cerebri) syndrome

There were 56 patients in this group, 54 females and 2 males, with a mean age of 34.2 years (range, 20–52 years). Thirty-nine patients were black and 17 were white. Thirty-seven of these patients were overweight and 16 of them were morbidly obese (100 lb above ideal body weight). Tinnitus was unilateral in 45 patients and bilateral in 11 patients. There was right ear involvement in 31 patients and left ear involvement in 14 patients. Tinnitus was objective in 40 patients and subjective in 16 patients. In all patients, PT subsided while light digital pressure was applied over the ipsilateral internal jugular vein (IJV). Blurred vision without any significant loss of vision was present in 17 patients. Bilateral blindness developed in one female patient. Papilledema was identified in 21 patients. Fronto-occipital headaches were present in 44 patients. Lightheadedness–disequilibrium was present in 21 patients, and 1 patient had episodes of true vertigo. One female patient had associated bilateral recurrent facial paralysis and episodic unilateral trigeminal neuralgia.

A mild low-frequency sensorineural hearing loss with excellent discrimination was identified in 24 patients. Hearing loss normalized in all patients after elimination of the PT by applying light digital pressure over the ipsilateral IJV.

Elevated CSF pressure (>200 mm of water) was confirmed with LP in all 56 patients. Four additional patients had typical clinical and radiologic findings for BIH syndrome; however, they declined LP with CSF pressure measurement.

The majority of patients responded well to medical treatment. Ten patients, however, failed to respond to conservative management and were treated surgically. Early on in this study, four patients underwent a lumbar subarachnoid–peritoneal shunt with immediate resolution of their symptoms. Shunt malfunctioned in two of these patients because of obstruction. Both patients declined any further surgical treatment. Six morbidly obese patients un-

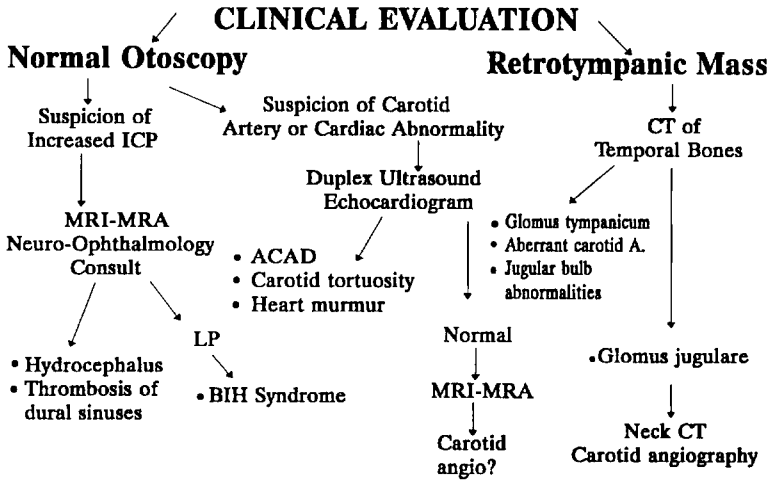

FIG. 2. Pulsatile tinnitus evaluation algorithm.

TABLE 1. *Radiologic results*

Result	No.
Empty sella	16
Small ventricles	4
Arteriovenous fistulae	3
Hydrocephalus stenosis of Sylvius aqueduct	1
Cerebellar atrophy	1
Superior sagittal sinus thrombosis	1
Carotid siphon stenosis	1

derwent gastric bypass surgery with significant reduction of their weight and resolution of their tinnitus.

Atherosclerotic carotid artery disease

Twenty-four patients, 18 females and 6 males, were diagnosed with ACAD. The mean age of these patients was 62.5 years (range, 46–82 years). Objective tinnitus was identified in 12 patients, and a neck bruit was present in 23 patients. In all patients, there was at least one condition and/or risk factor for ACAD present (e.g., transient ischemic attacks, hypertension, angina, diabetes mellitus, previous cerebrovascular accident, smoking). Mild-to-severe carotid artery stenosis was confirmed with Duplex ultrasound studies in 23 patients. In one patient, severe stenosis of the carotid siphon was confirmed with cerebral angiography.

Glomus tumors

Glomus tumors were diagnosed in 17 female patients. The mean age was 43 years (range, 22–77 years). In all patients, a retrotympanic mass was detected otoscopically. In none of these patients, however, could differentiation between glomus tympanicum and glomus jugulare tumor be made on the basis of otoscopic examination alone. Computed tomography of the temporal bone was extremely helpful in making this distinction. Tinnitus was subjective in all patients of this group.

TABLE 2. *Pulsatile tinnitus etiologies*

Etiology	No.
Increase intracranial pressure	
BIH syndrome (pseudotumor cerebri)	56
Possible BIH syndrome*	4
Lupus erythematosus	2
Hydrocephalus Sylvius aqueduct stenosis	1
ACAD	24
Glomus tumors	17
Otosclerosis	4
Hypertension	
On enalapril maleate	3
On verapamil	2
Cardiogenic (murmur)	4
Tortuous vessels	
Internal carotid artery	3
Basilar artery	3
Arteriovenous fistula	3
Aneurysm internal carotid artery	2
Anemia	1
Auditory muscle myoclonus	1
Vascular loop	1
Angioma of cerebellum	1
Idiopathic	13
Total	145

BIH, benign intracranial hypertension; ACAD, atherosclerotic carotid artery disease.

*Not confirmed with lumbar puncture and CSF measurement.

Hypertension

Three patients with hypertension had PT develop while receiving an angiotensin-converting enzyme inhibitor (enalapril maleate) and two while receiving a calcium channel blocker (verapamil hydrochloride). In three of these patients, tinnitus started immediately after initiation of treatment and subsided soon after cessation of therapy.

Tortuous vessels

Diagnosis of a tortuous internal carotid artery was confirmed by Duplex ultrasound studies in three patients. A tortuous basilar artery was diagnosed by MRI/MRA in three patients.

Idiopathic

In 13 patients, 10 females and 3 males, no diagnosis could be established. Tinnitus was subjective in 10 and objective in 3 of these patients. The mean age was 42.5 years (range, 21–65 years).

DISCUSSION

Pulsatile tinnitus of vascular origin results from blood turbulence generated by either increased flow volume or lumen stenosis. According to the vessel of origin, vascular PT can be classified as either **arterial** or **venous**. Applying light digital pressure over the ipsilateral to the tinnitus IJV is helpful in differentiating venous from arterial PT. In patients with venous type, this maneuver results in immediate cessation of PT. Venous PT can originate not only from primary venous diseases but also from conditions producing increased intracranial pressure (ICP) by transmission of arterial pulsations (3). Pulsatile tinnitus originating from other structures or pathologic conditions is classified as **nonvascular**. Tables 3 through 5 summarize the most common etiologies of PT reported in the literature.

The experience gained from this study indicates that history and physical examination are the two most important factors in evaluating patients with PT. The BIH syndrome, ACAD, and glomus tumors composed two thirds of definable causes. Each one of these entities has very characteristic clinical manifestations that will direct the astute neurotologist toward the correct diagnosis. Careful auscultation of the ear, periauricular region, orbits, neck, and chest with a modified electronic stethoscope is important to document objective PT or bruits or both. Electronic auscultation has been found to be more sensitive than auscultation with the Toynbee tube (43). Pulsatile tinnitus in patients with BIH syndrome is of the venous type, whereas in patients with ACAD, it is of the

TABLE 3. *Arterial etiologies of pulsatile tinnitus*

Intracranial and extracranial arteriovenous malformations[4–6]
Intracranial arteriovenous fistulae and aneurysms[4–8]
Atherosclerotic carotid artery disease and subclavian artery disease[9, 10]
Atherosclerotic occlusion of the contralateral common carotid artery[11]
Fibromuscular dysplasia of the carotid artery[12–14]
Extracranial carotid artery dissection[8]
Intrapetrous internal carotid artery dissection[15]
Brachiocephalic artery stenosis[16]
External carotid artery stenosis[17]
Ectopic intratympanic carotid artery[18, 19]
Persistent stapedial artery[20]
Aberrant artery in the stria vascularis[21]
Vascular compression of the eighth cranial nerve[22]
Increased cardiac output (anemia, thyrotoxicosis, pregnancy)[23, 24]
Aortic murmurs[25]
Paget's disease[26, 27]

arterial type (9). Patients with ACAD almost always have an associated ipsilateral or bilateral neck bruit. The three patients with arterio-venous malformations (AVFs) in this study had a loud bruit either in the retroauricular area or in the orbit region. Funduscopic examination should be performed routinely on patients with PT, especially when BIH syndrome is suspected (obese, young, female patients). Table 6 summarizes the characteristics of PT in various pathologic conditions.

Radiologic evaluation is important and should be individualized according to the clinical findings. For patients with normal otoscopic findings, a head MRI/MRA should be the initial evaluation. For patients with a retrotympanic lesion, a computed tomographic scan of the temporal bones should be ordered to detect for aberrant carotid artery, abnormalities of the jugular bulb, and glomus tumors. In the past, carotid angiography was recommended in all patients with PT. Currently, carotid angiography should be considered for patients with objective PT or head bruits or both who are suspected of having arteriovenous malformations–fistulae or fibromuscular dysplasia and for patients with glomus tumors who are surgical candidates. Currently, MRA cannot be considered a substitute for conventional angiography.

The BIH syndrome (pseudotumor cerebri or idiopathic intracranial hypertension) was the most common diagnosis made in our patients and deserves special attention. This condition is characterized by increased ICP without focal neurologic signs, except for an occasional sixth or seventh nerve palsy (44). The pathophysiologic mechanism responsible for PT in these patients probably is secondary to the transmission of systolic pulsations of the CSF to the exposed medial aspect of the dural venous sinuses and the resulting periodic compression of their walls, which convert the normally laminar blood flow to turbulent (3). Diagnosis of this syndrome is made by LP and measurement of CSF pressure after a head MRI to rule out other etiologies of intracranial hypertension. Elimination of the masking effect of PT by the application of light digital pressure over the ipsilateral IJV explains the normalization of the pure tones in patients with BIH with hearing loss. Treatment of this syndrome consists of weight reduction and administration of acetazolamide and furosemide (3). The majority of patients in this study responded well to this type of management. For morbidly obese patients who have failed conservative treatment, gastric bypass surgery should be considered (45). This procedure was performed in six patients with resolution of their PT. Lumbar subarachnoid–peritoneal shunting often becomes obstructed in these patients because of their obesity. This occurred in two of the four patients who had the procedure.

TABLE 4. *Venous etiologies of pulsatile tinnitus*

Benign intracranial hypertension[3,28,29]
Jugular bulb abnormalities[30]
Abnormal condylar and mastoid emissary veins[31,32]
Narrowing of the transverse dural venous sinus[33]
Idiopathic tinnitus (venous hum, essential tinnitus)[34–36]

TABLE 5. *Nonvascular etiologies of pulsatile tinnitus*

Vascular neoplasms of the skull base and temporal bone[37–40]
Palatal, tensor tympani, and stapedial muscle myoclonus[4,35,41]
Patulous eustachian tube[42]
Cholesterol granuloma of the middle ear[25]

Treatment of patients with glomus tumors is mainly surgical and should be individualized according to the location and extent of the lesion. Carotid endarterectomy for patients with ACAD should be considered when obstruction is more than 60% (46).

In the past decade, selective embolization techniques for arteriovenous malformations–fistulae have become very popular, and excellent results with minimal complications have been reported (47).

Pulsatile tinnitus in patients with otosclerosis probably is secondary to neovascularization and formation of arteriovenous fistulae within the temporal bone. The same mechanism of tinnitus also has been reported in Paget's disease, which has very similar histopathologic findings to those of otosclerosis (27). In our experience, stapedectomy usually is effective for relief of the PT in these patients.

Pulsatile tinnitus in patients with hypertension while receiving treatment with angiotensin-converting enzyme inhibitors or calcium channel blockers probably is secondary to hyperdynamic circulation resulting from decreased peripheral vascular resistance (1).

Pulsatile tinnitus should be called idiopathic only after an extensive workup has been completed and specific diagnosis has not been reached. Essential tinnitus and venous hum are other terms used in the literature to describe idiopathic PT (4,35,36). It is speculated that in some of the previously reported cases of idiopathic PT, the underlying etiology could have been undiagnosed increased ICP. Another possible cause of idiopathic PT is turbulent blood flow produced by anatomic abnormalities of the sigmoid–transverse sinuses or at the bend of the IJV as it

TABLE 6. *Characteristics of pulsatile tinnitus*

	BIH syndrome	ACAD	Glomus tumors	AVM/AVF
Age (years)	<40	>50	40 (average)	40 (average)
Sex	Females mainly	More common in females	More common in females	NR
Weight	Obese	NR	NR	NR
Retrotympanic mass	–	–	+	–
Objective PT	+	+	–	+
Arterial type PT	–	+	+	+
Venous type PT	+	–	–	–
Head bruit	–	–	–	+
Neck bruit	–	+	–	–
Papilledema	Common	–	–	–

BIH, benign intracranial hypertension; ACAD, atherosclerotic carotid artery disease; AVF, arteriovenous fistula; AVM, arteriovenous malformation; NR, not relevant; PT, pulsatile tinnitus.

curves around the lateral process of the atlas (30,33,34). Ligation of the IJV has been recommended for patients with idiopathic PT (34). It is strongly recommend that this procedure be considered only after careful elimination of BIH syndrome or other causes of increased ICP. Obliteration of the hypotympanic space with fat from the auricular lobule has been found helpful in these cases (Kamerer DB, personal communication, 1996).

CONCLUSIONS

The results of this study support the following conclusions regarding PT:

1. History and physical examination are the two most important factors in evaluating these patients.
2. Diagnostic testing should be individualized and may include radiologic evaluation, ultrasonography studies, and LP with CSF pressure measurement.
3. Benign intracranial hypertension syndrome (or pseudotumor cerebri) was the most common diagnosis, followed by ACAD and glomus tumors.
4. The majority of these patients have a treatable underlying etiology.

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