Pulsatile tinnitus: contemporary assessment and management

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Purpose of review

Pulsatile tinnitus is an uncommon otologic symptom, which often presents a diagnostic and management dilemma to the otolaryngologist. The majority of patients with pulsatile tinnitus have a treatable cause. Failure to establish correct diagnosis may have disastrous consequences, because a potentially life-threatening, underlying disorder may be present. The purpose of this review is to familiarize the otolaryngologist with the most common causes, evaluation, and management of pulsatile tinnitus.

Recent findings

The pathophysiology, classification, various causes, evaluation, and management of the most common causes of pulsatile tinnitus are presented in this review.

Summary

Pulsatile tinnitus deserves a thorough evaluation and, in the majority of cases, there is a treatable underlying cause. The possibility of a life-threatening cause needs to be ruled out in every patient with pulsatile tinnitus. The otolaryngologist should be familiar with the evaluation and management of this symptom.

Keywords

arteriovenous fistulae, arteriovenous malformation, bruit, cause, idiopathic intracranial hypertension, management, pulsatile tinnitus

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Introduction

Pulsatile tinnitus is an uncommon otologic symptom, which often presents a diagnostic and management dilemma to the otolaryngologist. The purpose of this review is to familiarize the otolaryngologist with the most common causes, evaluation, and management of pulsatile tinnitus.

Pathophysiology and classification

Pulsating tinnitus originates from vascular structures within the cranial cavity, head and neck region, and thoracic cavity, and it is transmitted to the cochlea by bony or vascular structures. Pulsatile tinnitus arises from either increased blood flow or stenosis of a vascular lumen and can be classified as arterial or venous according to the vessel of origin. Pulsatile tinnitus can be further classified as objective or subjective according to whether it is audible to both patient and examiner or to the patient only.

Rarely, objective tinnitus originates from other structures and is classified as nonvascular. High-pitched tinnitus with a pulsatile component, often bilateral and associated with high-frequency sensorineural hearing loss (SNHL), should not be confused with arterial pulsatile tinnitus. This type of tinnitus is subjective and is considered to be related to the SNHL.

The following are the most common causes of pulsatile tinnitus.

Arterial causes

The most common causes of arterial pulsatile tinnitus are as follows.

Atherosclerotic carotid artery disease

Atherosclerotic carotid artery disease (ACAD) is a common cause of pulsatile tinnitus in patients older than 50 years, and often associated risk factors such as hypertension, angina, hyperlipidemia, diabetes mellitus, and smoking are present. Pulsatile tinnitus may be the first manifestation of ACAD, and, therefore, the otolaryngologist may be the first to be consulted [1]. Pulsatile tinnitus is secondary to bruit(s) produced by turbulent blood flow at stenotic segment(s) of the carotid system. In a series of 12 patients with pulsatile tinnitus secondary to ACAD, ipsilateral carotid bruit was present in all of them. Diagnosis can be confirmed in most patients by duplex ultrasound studies [1]. In patients with ACAD at the skull base or the cavernous sinus, duplex ultrasound studies may not be revealing and MRI should be considered. Recently, MRI has been shown to accurately identify carotid plaque features, including intraplaque hemorrhage, neovasculature, and vascular wall inflammation [2•].

Intracranial vascular abnormalities

Intracranial vascular abnormalities are uncommon causes of pulsatile tinnitus; however, a high index of suspicion and proper evaluation are required to avoid misdiagnosis and catastrophic consequences. The majority of these lesions are dural arteriovenous fistulae (AVFs) and arteriovenous malformations (AVMs), and pulsatile tinnitus is the most common manifestation. The transverse and sigmoid dural sinuses are the most commonly involved, followed by the cavernous sinus [3•,4•]. Dural AVFs comprise approximately 15% of intracranial AVMs, which usually become symptomatic during the fifth or sixth decades of life [5,6]. In contrast to AVMs, AVFs are usually acquired and thought to result from dural sinuses, spontaneous or traumatic thrombosis, obstructing neoplasms, surgery, and infection. As the thrombosed segments recanalize, ingrowths of dural arteries take place and arterial to sinus anastomoses are formed [5]. Pulsatile tinnitus in these patients is of the arterial type and is associated with a loud bruit over the involved dural sinus (usually the retroauricular area) as well.

According to a recent report, the most critical anatomical feature is the presence of cortical venous drainage as a finding, which identifies lesions at a high risk for future hemorrhage or ischemic neurological injury [7••]. Patients who present with pulsatile tinnitus have a less aggressive clinical course with an estimated annual rate of intracerebral hemorrhage of 1.4–1.5% [7••]. The mortality from hemorrhage of dural AVFs has been reported between 10 and 20% [5].

Aneurysms of anterior inferior cerebellar artery and dissecting aneurysms of the internal carotid and vertebral arteries are less often associated with pulsatile tinnitus [8,9]. Other manifestations of dissecting aneurysms include pain, transient ischemic attacks, cranial neuropathies, Horner's syndrome, and subarachnoid hemorrhage [5,9–11]. Sudden head rotation, especially when accompanied by extension (i.e. the tennis 'ace serve'), can be a precipitating event. Fibromuscular dysplasia (FMD), various arteriopathies such as Marfan syndrome, and osteogenesis imperfecta are predisposing factors [12].

Glomus tumors of the jugular foramen and middle ear

Subjective pulsatile tinnitus and conductive hearing loss are the most common manifestations of glomus jugulare and tympanicum tumors [13]. Diagnosis is based upon otoscopic findings and imaging studies of the temporal bone and skull base.

Fibromuscular dysplasia

FMD is a nonatherosclerotic, noninflammatory stenosing vascular disease, which primarily affects women of age 20–60 years. This entity most commonly involves the renal and internal carotid arteries. As carotid artery involvement is frequently associated with pulsatile tinnitus [14•], these patients may first consult the otolaryngologist. Cases with carotid or vertebral arteries involvement may develop dissection and/or aneurysms and present with symptoms of transient ischemic attacks or stroke [15,16]. The typical angiographic finding is that of a 'string of beads' [16].

Key points

- Pulsatile tinnitus is an uncommon otologic symptom, which always deserves thorough investigation.
- Most patients have a treatable underlying cause.
- Detailed history and auscultation of the head and neck are of utmost importance.
- Radiologic evaluation should be individualized according to the type of pulsatile tinnitus (arterial or venous), presence of retrotympanic pathology, and audible bruit.
- Treatment is directed toward the correction of the underlying cause.

Tortuous internal carotid artery

Pulsatile tinnitus secondary to a tortuous internal carotid artery is more common in older individuals and is often accompanied by an audible bruit. Rarely this entity may present as an abnormal sensation in the throat associated with a pulsating pharyngeal or cervical mass [17,18]. It is likely that carotid vessels become tortuous with aging, and turbulent blood flow and pulsatile tinnitus are produced. Atherosclerosis and FMD have been reported in association with tortuous internal carotid artery [19]. In the author's experience, this is a benign condition and pulsatile tinnitus usually subsides spontaneously with time. Symptoms of cerebral ischemia warrant consultation with a vascular surgeon. Diagnosis is made with computed tomography angiography (CTA) or magnetic resonance angiography (MRA) [20].

The arterial causes of pulsatile tinnitus are as follows:

(1) Dural, skull base, and cervical region AVMs/AVFs [3•,4•,21–25].
(2) Atherosclerotic carotid and subclavian artery disease [1,26–29].
(3) Glomus tumors of jugular foramen and middle ear [30,31].
(4) Tortuous internal carotid artery [20].
(5) Dehiscence of the superior semicircular canal [32].
(6) FMD of the carotid artery [15,33,34].
(7) Increased cardiac output (anemia, thrombocythemia, thyrotoxicosis, and pregnancy) [35,36].
(8) Extracranial carotid artery dissection [37,38].
(9) Intrapetrous carotid artery dissection and aneurysm [39,40].
(10) Brachiocephalic artery stenosis [41].

(11) External carotid artery stenosis [42].
(12) Vascular anomalies of the middle ear [43–47].
(13) Aberrant artery in the stria vascularis [48].
(14) Vascular compression of the eighth nerve [49–51].
(15) Aortic murmurs [52].
(16) Paget's disease [53–55].
(17) Otosclerosis [56].
(18) Hypertension – antihypertensive agents [56].

Venous causes

The most common causes of venous pulsatile tinnitus are as follows.

Idiopathic intracranial hypertension syndrome

In the author's experience, idiopathic intracranial hypertension (IIH) syndrome is one of the most common causes of venous pulsatile tinnitus, which may be the first or only manifestation of this syndrome [57,58]. Other associated otologic symptoms may include hearing loss, dizziness, and aural fullness [59]. For this reason, the otolaryngologist may be the first to be consulted. The exact pathophysiology of this entity remains unclear; it affects obese women of childbearing age in more than 90% of cases and is associated with increased intracranial pressure (ICP) without focal signs for neurological dysfunction except for occasional fifth, sixth, and seventh cranial nerve palsies [60,61,62•,63,64]. IIH syndrome may also present mainly with posture-dependent headaches and visual changes (blurred vision, transient visual obscurations, retrobulbar pain, and diplopia) because of papilledema [65], which prompt patients to seek consulation with a neurologist or ophthalmologist. Although papilledema is common in these patients, its absence does not exclude this entity [66–68]. Optical coherence tomography (OCT) has recently been used to differentiate papilledema secondary to increased ICP from optic disc swelling secondary to optic neuropathy [65,69]. Children are affected as well, although less often [70]. Diagnosis is established by lumbar puncture and documentation of elevated cerebrospinal fluid (CSF) pressure (>200 mm of water) with normal CSF constituents [71]. In most patients, IIH syndrome has a benign and self-limiting course; however, in 25% of patients it may become chronic [60].

Although many causes have been alleged for this syndrome, few besides obesity, hypervitaminosis A and related compounds, steroid withdrawal, and female sex have been proven [72]. It has been reported that increased ICP in obese patients is secondary to associated elevated intra-abdominal, pleural, cardiac filling, and cerebral venous pressures [73–75]. This pathophysiologic mechanism is further supported by an animal study demonstrating increased ICP upon acute elevation of intra-abdominal pressure [76]. Increased cerebral blood flow secondary to cerebrovascular resistance changes and CSF hypersecretion induced by elevated estrogen levels, which are produced in excess by fat tissues in obese patients, have been reported as pathophysiologic mechanisms as well [77].

Pulsatile tinnitus in IIH syndrome is believed to result from the systolic pulsations of the CSF, which originate mainly from the arteries of the Circle of Willis. These pulsations, which are increased in magnitude in the presence of intracranial hypertension, are transmitted to the exposed medial aspects of the dural venous sinuses (transverse and sigmoid), compressing their walls synchronously with the arterial pulsations [57,78]. The ensuing periodic narrowing of the dural venous sinuses lumen converts the laminar blood flow to turbulent and produces the pulsatile tinnitus [57]. The low-frequency SNHL seen in many of these patients is believed to result from the masking effect of the pulsatile tinnitus. This is supported by the fact that light digital compression over the ipsilateral internal jugular vein (IJV) results in cessation of the tinnitus and immediate improvement or normalization of hearing [57]. Stretching or compression of the cochlear nerve and brain stem, caused by the intracranial hypertension, can also play a role in the hearing loss and dizziness. This is supported by the abnormal auditory-evoked response (ABR) present in one-third of these patients [79].

MRI and magnetic resonance venography (MRV) are necessary to rule out a neoplastic lesion and dural sinus thrombosis [69]. MRI is normal in most patients, although an empty sella, small ventricles, and flattening of the posterior aspect of the globe are suggestive of this entity [56,80]. Anatomic obstruction of the transverse venous sinuses has been reported in IIH syndrome. Direct retrograde cerebral venography with manometry has been recommended in order to establish diagnosis and treatment with stents [81–83]. In another study, MRV identified bilateral dural venous sinus stenosis in 27 of 29 patients with IIH syndrome and only in four of 59 controls. It was not clear whether stenosis was a cause or effect of the intracranial hypertension, and this has been reported by others [84,85].

Idiopathic or essential pulsatile tinnitus

Idiopathic or essential pulsatile tinnitus and venous hum are terms used interchangeably in the literature to describe patients with pulsatile tinnitus of unclear cause [86,87]. Diagnosis of this condition should be made only after appropriate evaluation and elimination of other disorders. As the majority of studies on idiopathic pulsatile tinnitus were reported prior to the introduction of MRI/MRV/MRA and the overall better understanding of the various causes of pulsatile tinnitus, it is possible that some of these patients may have had IIH syndrome or

other pathologies such as AVFs, AVMs, or abnormalities of the dural venous sinuses.

The venous causes of pulsatile tinnitus are as follows:

(1) IIH syndrome [57].
(2) Jugular bulb abnormalities: high location, dehiscence, and diverticula [88–92,93••,94].
(3) Transverse-sigmoid sinus stenosis and aneurysms [95•,96–98].
(4) Abnormal condylar and mastoid emissary veins [99,100].
(5) Increased ICP associated with Arnold–Chiari syndrome and stenosis of the sylvian aqueduct [101].
(6) Idiopathic or essential tinnitus [24,86,87,102].

Nonvascular causes

Myoclonic contractions of the tensor veli palatini, levator veli palatini, salpingopharyngeus, and superior constrictor muscles are the most common nonvascular causes, which can result in objective pulsating sounds. These contractions can range between 10 and 240/min and may be confused with the arterial pulse.

This disorder is often seen in young patients, usually within the first three decades of life, although it may be seen in older individuals as well [103,104,105•,106]. Associated neurologic disorders such as brain stem infarctions, multiple sclerosis, trauma, and syphilis have also been reported. Involvement of the olivary tracts, posterior longitudinal bundle, dentate nucleus, and reticular formation has been identified in these patients [86,107].

Myoclonic contractions of the stapedial muscle have also been reported as a cause of pulsatile tinnitus [108].

Evaluation

The history, otoscopic examination, and auscultation are of utmost importance in evaluating patients with pulsatile tinnitus. Older patients with previous history of cerebrovascular accident, transient ischemic attacks, hyperlipidemia, hypertension, diabetes mellitus, and smoking are suspicious for ACAD [1]. Obese females associated with hearing loss, headaches, aural fullness, dizziness, and visual disturbances are highly suspicious for IIH syndrome [57,59]. Sudden onset of pulsatile tinnitus in association with cervical or facial pain, headache, and symptoms of cerebral ischemia is compatible with extracranial or intrapetrous carotid artery dissection [37,39,109].

Otoscopy is essential as it may reveal a retrotympanic lesion such as a high or exposed jugular bulb, aberrant carotid artery, glomus tumor, and Schwartze's sign. Rhythmic movements of the tympanic membrane, soft palate, or pharynx can be present in patients with tensor tympani myoclonus. Wide opening of the oral cavity during examination may eliminate the soft palate myoclonic contractions [24]. Transnasal fiberoptic inspection of the soft palate and pharynx is more revealing in these patients.

Auscultation of the ear canal, periauricular region, orbits, cervical region, and chest is the most important aspect of the examination. This should preferably be performed with a modified electronic stethoscope in an audio booth [110,111]. Should objective pulsatile tinnitus be detected, its rate should be compared with the patient's pulse rate and the effect of light digital pressure over the ipsilateral IJV should be checked. Pulsatile tinnitus of venous origin, such as in patients with IIH syndrome, decreases or completely subsides with this maneuver [57,59]. In patients with arterial pulsatile tinnitus, however, this maneuver is ineffective. The effect of head rotation on tinnitus intensity should also be checked. Venous pulsatile tinnitus decreases or completely subsides with head rotation toward the ipsilateral side, probably because of compression of the IJV between the contracting sternocleidomastoid muscle and the transverse process of the atlas [59]. A complete neurologic examination should be performed.

Audiologic and electrophysiologic testing

Pure tone (air and bone conduction) and speech audiometry should be performed in all patients. When hearing loss of 20 dB or more is detected in the low frequencies, a repeat audiogram should be obtained while the patient is applying light digital pressure over the ipsilateral IJV. This maneuver typically results in improvement or normalization of pure tones in patients with venous pulsatile tinnitus, such as in IIH syndrome, because of elimination of the masking effect of the tinnitus [57]. Discrimination is typically excellent in these patients.

Impedance audiometry can be useful in patients suspected of tensor tympani myoclonus.

ABRs may be considered in selected cases only. Abnormalities of ABR, consisting mainly of prolonged interpeak latencies, have been detected in one-third of patients with IIH syndrome, which normalize following successful management [79]. Electronystagmography may be considered in patients with associated dizziness [57].

Metabolic workup

Metabolic workup is rarely revealing in these patients; however, complete blood count and thyroid function tests should be obtained in patients with increased cardiac output syndrome to exclude anemia and

hyperthyroidism. Serum lipid profile and fasting blood sugar should be considered in patients suspicious of ACAD.

Ultrasound studies

Duplex carotid ultrasound (including the subclavian arteries) and echocardiogram studies should be obtained in patients suspected of ACAD and valvular disease. These studies should be performed prior to any radiologic evaluation, as they may be the only tests required to establish diagnosis [1].

Radiologic evaluation

The radiologic evaluation is individualized according to the otoscopic findings and pulsatile tinnitus characteristics (arterial/venous type).

Normal otoscopy

Patients with venous pulsatile tinnitus are scheduled for a brain MRI/MRV as initial evaluation. Abnormalities of the jugular bulb/dural venous sinuses and findings present in IIH syndrome (empty sella, small ventricles, and flattening of posterior aspect of globe) can be detected with these studies. Other rare congenital central nervous system abnormalities such as Chiari I malformation and stenosis of the sylvian aqueduct can be detected with brain MRI as well [101].

Patients with arterial pulsatile tinnitus are considered for a CTA as initial evaluation. Experience with CTA suggests this study to be satisfactory in evaluating intracranial vascular lesions [112–116]. This is a fast imaging technique and, as the upper neck is included, cervical vascular pathology, such as a carotid body tumor, can be detected as well. Tortuous carotid vessels, AVF/AVMs, carotid artery dissections/aneurysms, cervical/intracranial ACAD, and FMD can be diagnosed with this study [20].

Patients with isolated cervical carotid bruits are considered for a carotid duplex ultrasound prior to CTA. If ACAD is confirmed, no other imaging study is necessary [117].

Abnormal otoscopy/retrotympanic pathology

These patients are considered for a CTA of the temporal bones and neck at the initial evaluation. For patients with glomus jugulare/tympanicum tumors, the presence of synchronous carotid body tumor(s) can easily be detected in the same study. Lesions of the temporal bones such as glomus tympanicum, ectopic carotid artery, and jugular bulb/ dural venous sinuses abnormalities are easily detected as well.

Carotid angiography is indicated only in cases with strong suspicion of an AVF/AVM, such as patients with loud retroauricular bruits, and for prospective surgical candidates in order to evaluate the collateral circulation of the brain (arterial and venous) in anticipation of possible vessel ligation and/or preoperative tumor embolization [118].

Figure 1 a and b depict algorithms for the evaluation of patients with pulsatile tinnitus.

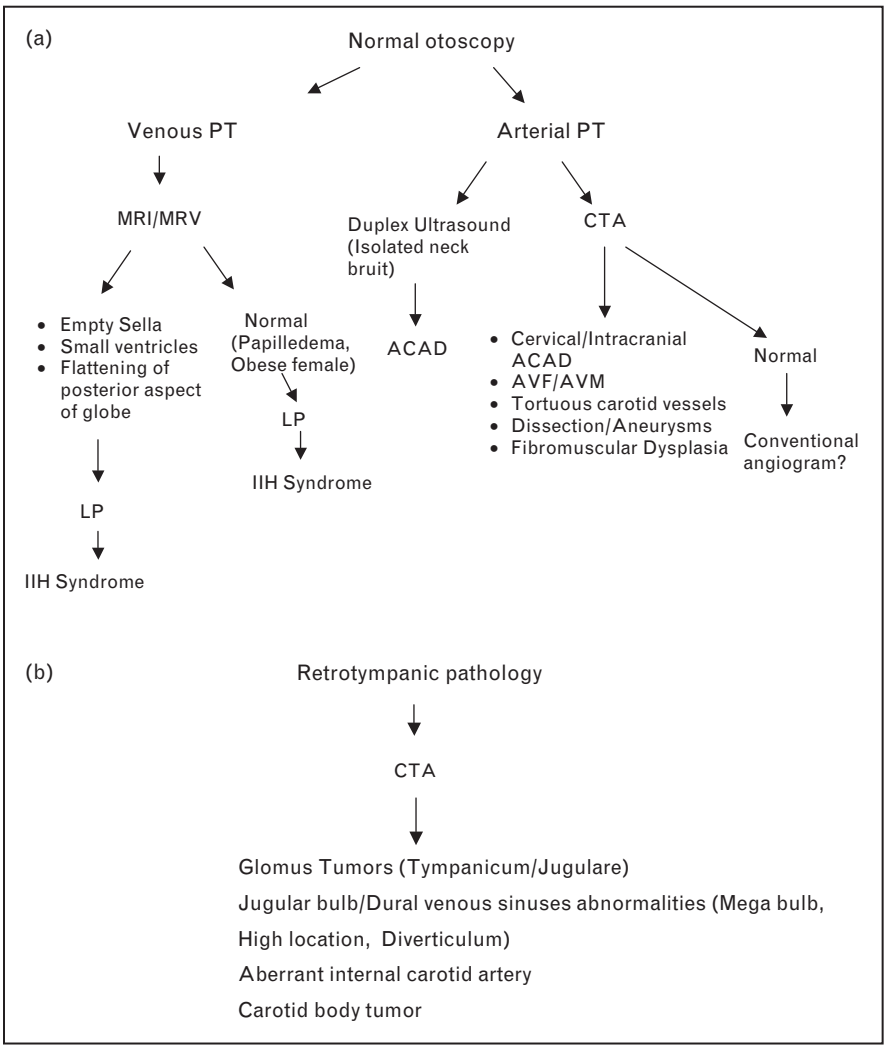
Management

Management should be directed toward treating the underlying cause. The following describes management of the most common pulsatile tinnitus causes.

Patients with IIH syndrome will often present to the otolaryngologist first because of disturbing pulsatile tinnitus. Obese patients should understand the connection between body weight and pulsatile tinnitus. Associated comorbidities such as hypertension, diabetes mellitus, gastroesophageal reflux, and obstructive sleep apnea are common in these patients and appropriate referral to other specialists should be advised. Weight reduction is the most important aspect of management and will reduce or even eliminate pulsatile tinnitus in the majority of patients [62•,119•]. Administration of acetazolamide (Diamox, Lederle Parenterals, Inc.) is thought to reduce CSF production and can be helpful in decreasing tinnitus intensity, although it rarely eliminates this symptom [120]. Lumbar–peritoneal shunt should be considered for patients with progressive deterioration of vision, persistent headaches, and disabling pulsatile tinnitus [56,57,59]. In morbidly obese patients, however, this procedure is often complicated by the occlusion of the shunt secondary to increased intraabdominal pressure [121]. Weight reduction surgery in morbidly obese patients with pulsatile tinnitus is very effective in eliminating this symptom. Thirteen out of 16 patients who underwent this procedure experienced complete resolution of their pulsatile tinnitus [122]. Evaluation and close follow-up by an opthalmologist and neurologist are of outmost importance. Optic nerve sheath fenestration should be considered for patients with progressive visual loss [120,123].

Patients with ACAD and disturbing pulsatile tinnitus should be considered for surgical intervention [124]. Carotid endarterectomy (CEA) and carotid angioplasty with stenting (CAS) are the two procedures for treating ACAD. Randomized controlled trials comparing the efficacy of CEA vs. medical therapy have shown a clear benefit for CEA in patients with symptomatic stenosis of greater than 70% and a lesser benefit in patients with 50–69% stenosis. More recent studies comparing CAS with CEA failed to reach conclusions regarding a clear neurologic outcome advantage of one method over the

Figure 1 Pulsatile tinnitus evaluation algorithm



ACAD, atherosclerotic carotid artery disease; AVF, arteriovenous fistula; AVM, arteriovenous malformation; CTA, computed tomography angiography; IIH, idiopathic intracranial hypertension; LP, lumbar puncture; MRV, magnetic resonance venography; PT, pulsatile tinnitus.

other [125•]. Angioplasty has been reported to relieve pulsatile tinnitus secondary to atherosclerotic obstruction of the subclavian and intracranial carotid arteries [27,126].

Glomus tympanicum tumors are amenable to surgical extirpation with excellent long-term results [127,128]. Treatment of glomus jugulare tumors should be individualized, and, although the traditional management has been surgical removal, stereotactic radiosurgery has recently been revealed to be very effective [129•,130•].

Although there have been no prospective, randomized therapeutic trials for patients with carotid artery dissection, experience shows that anticoagulants such as heparin followed by coumadin are effective in preventing further artery-to-artery emboli [37]. In selected cases of carotid artery dissection with occlusion, stent-assisted angioplasty is effective [131,132].

Repair of symptomatic high/dehisced jugular bulbs and diverticulae has been reported by using bone dust, perichondrium, tragal cartilage, conchal cartilage, and mastoid cortical bone [44,93••,133,134]. Surgical repair of transverse and sigmoid sinus aneurysms is effective in eliminating pulsatile tinnitus [95•,135].

Sectioning of the levator veli palatini muscle has been reported for treating pulsatile tinnitus in cases with palatal myoclonus [24]. However, botulinum toxin injection seems to be a more appropriate treatment [105•,136,137]. Tensor tympani and stapedial myoclonus may respond to sectioning of the respective muscles via

tympanotomy [138,139]. The author's experience with two patients (three ears) has been very satisfactory.

Dural AVFs can be treated very effectively with combined transvenous coil occlusion of the venous sinus segment and transarterial occlusion of supplying arteries [140].

Finally, ligation of the ipsilateral to the tinnitus IJV has been recommended in the literature for patients with idiopathic pulsatile tinnitus. The results of this procedure have been very inconsistent and poor overall. In a series of 13 patients with essential tinnitus, three underwent ligation of the ipsilateral IJV and only 1 benefited permanently. The other two patients experienced return of their pulsatile tinnitus within a few days [102]. This procedure should rarely, if ever, be performed for alleviating pulsatile tinnitus [141].

Conclusion

Pulsatile tinnitus deserves thorough evaluation. Failure to establish correct diagnosis may have disastrous consequences, as potentially life-threatening pathologies such as an AVF may be present. In the majority of cases there is a treatable cause. Detailed history and auscultation of the head and neck are of utmost importance. Radiologic evaluation should be individualized according to the type of pulsatile tinnitus (arterial or venous), presence of retrotympanic pathology, and audible bruit. Treatment is directed toward the correction of the underlying cause. Ligation of the IJV for treatment should rarely, if ever, be performed for alleviating pulsatile tinnitus.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 411).

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Despite the widespread consensus on the importance of these plaque features, testing the vulnerable plaque hypothesis in prospective clinical studies has been hindered by the lack of reliable imaging tools for in-vivo plaque characterization. This article provides a summary of the histological validation of carotid MRI and highlights its application in prospective clinical studies aimed at early identification of the high-risk atherosclerotic carotid plaque.

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Partial treatment did not resolve the clinical symptoms of patients with 'benign' dural AVF in the follow-up and was not clearly superior to conservative management. These results suggest that embolization should be offered only if there is a possibility of a complete cure without major peri-interventional risks.

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Coil embolization of the cavernous sinus led to durable closure of AVF and reliable regression of acute symptoms. However, long-term follow-up showed a 44% rate of persistent cranial nerve deficits with disturbances of oculomotor and visual functions. This may be explained by the underlying fistula size itself and/or the space-occupying effect of the coils. As neuro-ophthalmologic outcome is crucial for control of therapeutic success, patients should be routinely examined by ophthalmologists.

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Fibromuscular dysplasia (FMD) is a nonatherosclerotic, noninflammatory vascular disease that primarily affects women from age 20 to 60, but may also occur in infants and children, men, and the elderly. It most commonly affects the renal and carotid arteries but has been observed in almost every artery in the body. An increasing number of patients are asymptomatic and are only discovered incidentally when imaging is performed for some other reason or by the detection of an asymptomatic bruit. FMD should be considered in the differential diagnosis of a young person with a cervical bruit; a 'swishing' sound in the ear(s); transient ischemic attack, stroke, or dissection of an artery; or in individuals aged 35 years or less with onset of hypertension. Treatment consists of antiplatelet therapy for asymptomatic individuals and percutaneous balloon angioplasty for patients with indications for intervention.

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