Pulsatile tinnitus

Pulsatile tinnitus requires hearing, as there is usually a genuine physical source of sound.

Pulsatile tinnitus is therefore included under the umbrella terms “physical tinnitus” and “somatosounds”.

Less than 10% of tinnitus patients suffer from pulsatile tinnitus

**Etiology**

Most cases are due to vascular lesions.

1. pulse synchronous:
   
a) carotid cavernous fistula
   
b) AVM:
      ● cerebral (pial) AVM
      ● dural AVM
   
c) glomus jugulare tumor
   
d) cerebral aneurysm: (rare) possibly with turbulent flow in giant aneurysm
   
e) hypertension
   
f) hyperthyroidism
   
g) idiopathic intracranial hypertension (pseudotumor cerebri)
   
h) transmitted bruit: from heart (e.g. aortic stenosis), carotid artery stenosis (especially external carotid)
   
i) dehiscent jugular bulb or high riding jugular bulb: normal venous variant
   
j) rarely with posterior fossa tumors: CP-angle tumors e.g. vestibular schwannoma or meningioma, vascular intraparenchymal tumors e.g. hemangioblastoma (especially in CPA)
   
k) lesions that can present with a red tympanic membrane
      ● aberrant carotid artery in middle ear
      ● persistent stapedial artery: rare. Arises from aberrant ICA or from junction of horizontal and vertical petrous ICA. Foramen spinosum is absent on the affected side. Enlargement of anterior tympanic segment of seventh nerve canal
      ● glomus tympanicum tumor
      l) sigmoid sinus diverticulum
2. non pulse-synchronous: asymmetrical enlargement of sigmoid sinus and jugular vein may produce a low grade hum

There are two plausible causes of pulsatile tinnitus:

Bloodflow accelerates, or changes in bloodflow disrupt laminar flow, and the resulting local turbulence is audible. Normal flow sounds within the body are perceived more intensely, either as a result of alterations in the inner ear with increased bone conduction or as a result of disturbance of sound conduction leading to loss of the masking effect of external sounds.

Pulsatile tinnitus is usually unilateral, unless the underlying vascular pathology is bilateral. Recently, a disorder known as “somatosensory pulsatile tinnitus” has been discussed. This is bilateral tinnitus with no vascular cause.

It is often possible to identify the cause of pulsatile tinnitus. In addition to the patient’s medical history and targeted clinical examination, imaging procedures also play an important role in diagnosis. However, despite careful examination, no cause is found in up to 30% of patients.

Pulsatile tinnitus of vascular origin may arise in arterial or venous structures. Many authors have reported the association of pulsatile tinnitus with anomalies of dural venous sinuses and the jugular bulb.

see Venous sinus diverticulum.

Obstructive hydrocephalus secondary to enlarged Virchow Robin space (VRS) is a rare entity, with only a handful of cases reported in the literature. Presenting symptoms vary widely from headaches, through to dizziness.

Donaldson et al. report a 31 year old male who presents with pulsatile tinnitus and an MRI showing obstructive hydrocephalus secondary to tumefactive VRS. After a CSF diversion procedure in the form of an ETV he had almost complete resolution of his symptoms.

This is the first case of obstructive hydrocephalus secondary to enlarged Virchow-Robin spaces, presenting with pulsatile tinnitus.

Patients with idiopathic intracranial hypertension most frequently present with headaches, transient visual obscurations, papilledema, and/or pulsatile tinnitus, but may also be asymptomatic.

Workup

1. MRI without and with enhancement: to look for tumors, e.g. glomus jugulare
2. angiogram: include internal and external carotid injections
3. tests that are usually not helpful and should not be ordered routinely
a) carotid ultrasound: nonspecific, not sensitive

b) MRI/MRV: may miss small dural fistulas and do not give details needed for treatment for large ones
