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Review Article

Pulsatile tinnitus

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Abstract

Pulsatile tinnitus is an uncommon otological symptom. Objective pulsatile tinnitus has numerous causes, including benign intracranial hypertension, glomus tumours and atherosclerotic carotid artery disease. History and physical examination can give important information as to the underlying diagnosis. Radiographic evaluation is essential in all patients with pulsatile tinnitus. Many patients have a treatable underlying aetiology. Early diagnosis and appropriate intervention may save patients from unnecessary morbidity.

Key words: Ear Diseases; Pulsatile Tinnitus

Introduction

The word tinnitus is derived from the Latin word *tinnire*, meaning 'to ring' or 'a ringing'. Tinnitus is defined as a 'sound in one or both ears, such as a buzzing, ringing, clicking or whistling, occurring without an external stimulus'.[1] Tinnitus is a frequent presenting symptom, affecting up to 10 per cent of the general population.[2] It affects men more frequently than women, Caucasians more than Afro-Caribbeans, and its prevalence increases with advancing age.[3] It can occasionally present in childhood.[4] Pulsatile sounds are reported in about 4 per cent of unselected patients with tinnitus.[5] The severity of tinnitus can vary, from scarcely noticeable (by the patient) to an unbearable sound that drives some persons to contemplate suicide.[6,7]

The purpose of this review is to present the classification and causes of pulsatile tinnitus (PT), together with techniques used for its evaluation, diagnosis and treatment.

Classification

Many classification schemes have been developed to facilitate the diagnosis and treatment of tinnitus. One classification system groups the different forms of tinnitus into subjective or objective. Objective tinnitus is a real sound produced outside the inner ear, which is audible to the patient and also to the examining physician. Subjective tinnitus, which is more common, is heard only by the patient.

Objective tinnitus can be divided into pulsatile, mechanical and spontaneous. The latter arises from the spontaneous vibrations of the outer hair cells of the cochlea, known as otoacoustic emissions. Clicking may indicate a mechanical cause for the tinnitus. This is often secondary to the repetitive contractions of muscles in the middle ear or nasopharynx. Muscles implicated include: tensor veli palatini, levator palatini, tensor tympani and stapedius.[8,9] A patulous eustachian tube can also result in pulsatile tinnitus, secondary to inward and outward movement of the tympanic membrane in association with respiratory movements.[10]

Pulsatile tinnitus is an infrequent otological symptom which often presents a diagnostic and management dilemma to the otolaryngologist. Accurate diagnosis is imperative, because in many patients a treatable underlying aetiology can be identified. Furthermore, this symptom always deserves a thorough evaluation if unnecessary morbidity is to be avoided.

Causes

The causes of PT may be divided into two broad groups: vascular and nonvascular. Pulsatile tinnitus originating from vascular aetiologies may be the result of turbulence in the blood flow. This turbulence is generated by increased flow volume or stenosis of the vessel lumen. The sound produced due to the turbulent flow of the blood is transmitted directly to the inner ear and hence causes the PT.

According to the vessel of origin, vascular PT can be either arterial or venous. Pulsatile tinnitus due to arterial lesions can be classified into: arteriovenous shunts (which include arteriovenous malformations

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(AVMs) and arteriovenous fistulae (AVF), and Paget's disease), intracranial aneurysms, atherosclerosis and fibromuscular dysplasia of the carotid artery.[11] Among the venous causes of PT, the most frequently reported are benign intracranial hypertension (BIH), jugular bulb abnormalities and dural sinus stenosis.[12]

Venous PT originates from both primary venous disease and conditions that elevate intracranial pressure. The latter is postulated to result from the transmission of arterial pulsations.[13] Venous PT may be differentiated from arterial PT by applying light digital pressure over the internal jugular vein (IJV) ipsilateral to the tinnitus; in patients with the venous type, this results in cessation of the PT.

A comprehensive list of the pathological causes of PT is illustrated in Table I. A 15-year study of 145 patients with PT revealed that BIH, glomus tumours and atherosclerotic carotid artery disease composed two-thirds of definable causes.[12]

Model for nonpathological pulsatile tinnitus

Embryologically, the heart begins to beat and demonstrate unilateral flow at about the same time as the rudimentary carotid systems evolve.[14] It takes a further 10–12 days before the cochlear and semicircular appendages are apparent.[15] It is possible that pulsatile sounds are present within the embryonic ear well before the sensory receptors of the cochlea form.

Anatomically, the major arterial and venous systems of the head and neck lie in close proximity to the hypotympanum. A plate of bone, thinner over the internal carotid artery, separates these vessels from the basal turn of the cochlea.[16] This intimacy of the pulsating vessels, and the fact that laser Doppler interferometry measurements show that 80 dB SPL sounds cause vibration amplitudes at the stapes tendon of 6 Angstrom and at the stapes head of 19 Angstrom,[17] make it remarkable that we do not all have PT. However, habituation in utero could lead to suppression of this. Patients suffering with PT without any definable cause may have suffered an event which is different from their auditory template. Such an event could include: altered arousal, altered selective attention, otological change (e.g. conductive hearing loss), or alteration in cochlear blood flow or trauma (e.g. whiplash injury or forcible neck manipulation). During this event, the patient is straining to hear (Jastreboff model).[18] Following resolution of the event, the patient becomes aware of their physiological PT.

TABLE I

PATHOLOGICAL CAUSES OF PULSATILE TINNITUS

Type of lesion	Aetiology
Arterial	Atherosclerotic carotid artery disease[20]
	Arteriovenous fistula[28]
	Arteriovenous malformation[29]
	Intracranial aneurysm[28]
	Fibromuscular dysplasia of the carotid artery[30]
	Dissection of the carotid artery[31]
	Vascular anomalies of the ear[32]
	Vascular compression of the VIII nerve[33]
Venous	Benign intracranial hypertension[13,19]
	Jugular bulb abnormalities[34]
	Dural venous sinus stenosis[35]
	Abnormal condylar or mastoid emissary veins[36]
	Idiopathic tinnitus, venous hum, essential tinnitus[7,8]
Skull base or temporal	Glomus tumour[21]
	Paget's disease[37]
	Cholesterol granuloma of middle ear[38]
	Meningioma of middle ear[39]
	Cavernous haemangioma[40]
	Histiocytosis X[41]
Miscellaneous	Increased cardiac output (anaemia, thyrotoxicosis, pregnancy)[42]
	Aortic murmurs[38]
	Ménière's disorder[3]

Evaluation

The neurotologic evaluation of a patient with tinnitus includes a detailed history, a thorough medical examination (including an otological assessment), a comprehensive audiological work-up, blood tests and imaging studies. Benign intracranial hypertension, atherosclerotic carotid artery disease and glomus tumours all have characteristic features which, if detected during the clinical process, will direct the otolaryngologist to the correct diagnosis. This is illustrated below.

History

The following aspects should be checked when taking a history: character (pulsatile, continuous or episodic), onset, location, exacerbating and alleviating factors, and distress caused by the tinnitus. Enquiries should be made regarding associated otological symptoms such as hearing loss, vertigo and aural fullness. A thorough drug and past medical history should also be determined.

The typical patient with BIH is a young, obese female, who often has associated symptoms of hearing loss, aural fullness, dizziness, headaches and visual disturbances.[13,19] Suspicion of atherosclerotic carotid artery disease should be aroused in older individuals with PT and one or more of the following risk factors: hypertension, diabetes, smoking, hypercholesterolaemia and cerebrovascular disease.[20] The common presenting symptoms of glomus tumours are conductive hearing loss and unilateral PT.[21] This tinnitus is characteristically subjective, not objective.[8]

Examination

A comprehensive medical examination consisting of the following aspects should be performed.

Careful otoscopy to exclude middle-ear pathology (e.g. glomus tumour or high jugular bulb) is essential. Cranial, carotid and chest auscultation is important to detect objective PT, bruits and murmurs, respectively. This may be done using a modified electronic stethoscope in a sound-proof booth.[22] In patients with palatal myoclonus, listening to the nose, mouth and ear for the rhythmic contractions of the soft

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palate can be useful.[9] Fundoscopic examination is critical, especially when BIH is suspected. Palpation of the pulse for arrhythmia and measurement of the blood pressure to assess for hypertension is also important. Both may result in the formation of a hyperdynamic circulation and the generation of a murmur.

The response to light digital pressure over the ipsilateral IJV should be ascertained. Tinnitus of venous origin (e.g. BIH or idiopathic tinnitus) can be suppressed by compression over the ipsilateral IJV. Such a manoeuvre in patients with arterial-aetiology tinnitus has no effect. Further confirmation can be obtained by asking the patient to turn their head towards the side of the tinnitus; in venous-aetiology tinnitus, this results in a decrease in its intensity or complete cessation. This is due to IJV compression by the contracting sternocleidomastoid muscle. Turning the head away from the tinnitus has the opposite effect.

Position testing can also be useful when assessing for patulous eustachian tubes. The PT in the latter may disappear when the patient lies down with the head in a dependent position.[2] In addition, the effect of the Valsalva manoeuvre should be checked. Both venous-hum tinnitus and patulous eustachian tube tinnitus can be decreased or completely suppressed by such a manoeuvre.

Audiometric assessment

Diagnostic testing should include pure-tone audiometry and tympanometry. Patients with BIH may have a low-frequency hearing loss which is reversible by applying light digital pressure over the IJV ipsilateral to the tinnitus.[13] Tympanometry can be useful to identify middle-ear effusions and changes in tympanic membrane compliance secondary to a patulous eustachian tube, glomus tumours, or myoclonous of the stapedius muscle or muscles of the nasopharynx. Auditory-evoked brainstem responses are a useful adjunct in patients suspected of having BIH. Prolonged interpeak latencies is the most common abnormality detected.[23] Measurement of tympanic membrane displacement can provide a useful noninvasive method of monitoring intracranial pressure in patients with BIH, providing that the stapedial reflex is intact and that the cochlear aqueduct is patent.[24]

Blood tests

A full blood count, vitamin B12 levels and thyroid function tests should be performed if there is any suspicion of anaemia or hyperthyroidism. Both these conditions can result in an elevated cardiac output and cerebral blood flow. A complete biochemistry profile will aid in the diagnosis of Paget's disease; this condition is associated with elevated alkaline phosphatase levels. A cholesterol level is helpful to check for hyperlipidaemia, a risk factor for atherosclerosis.

Radiology

A thorough clinical evaluation should precede any imaging investigations as the former will direct the radiological work-up.

In patients with a retrotympanic mass on otoscopy, the preferred initial imaging is contrast-enhanced computed tomography (CT) of the temporal bone, brain and scalp[1] to detect glomus tumours, abnormalities of the jugular bulb and vascular anomalies (such as an aberrant carotid artery or persistent stapedial artery).

In the case of glomus tumours, whilst CT delineates the anatomic extent of the tumour clearly, magnetic resonance imaging (MRI) can be a useful adjunctive investigation. Tumour enhancement is much easier to identify with MRI than with CT.[25] If the initial CT or MRI study is normal, further imaging of the neck and superior mediastinum should be obtained and may disclose a cause for the PT.[1]

Patients with normal otoscopic findings and objective PT who are suspected of having AVMs, AVF or fibromuscular dysplasia (FMD) may be examined with MRI and magnetic resonance angiography (MRA).[1] If these initial magnetic resonance studies are normal and there is still the serious possibility of an AVM or AVF, then conventional carotid angiography must be obtained.[1,25]

Duplex carotid ultrasonography is helpful in patients with suspected atherosclerotic carotid artery disease. Small ventricles or an empty sella seen on CT or MRI suggest BIH.[1,26]

Invasive tests

A lumbar puncture and measurement of cerebrospinal fluid pressure should be performed in BIH patients.

Management

The management of PT involves the treatment of identifiable causes (Table II) if this is considered to be in the patient's best interests. In conjunction with this, and as with the management of other types of tinnitus, the alleviation of the perception of the tinnitus and the resulting behavioural, emotional, autonomic and cognitive responses, which may cause tinnitus distress, should be addressed.

Patients may experience PT in the absence of identifiable pathology. The absence of external auditory cues generates tinnitus in healthy volunteers placed in a sound-proof room.[27] The Jastreboff neurophysiological model[18] presents a useful synthesis of the role of the peripheral auditory system and the role of the brain and autonomic nervous system. This model would account for the generation and maintenance of PT in the absence of an identifiable cause.

In some patients, PT reflects the presence of good hearing in the low- and mid-range frequencies and a high-frequency sensorineural hearing loss. In other patients, there is a conductive hearing loss which directs physiological vascular sounds to the affected ear. The management of such cases involves offering

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TABLE II

TREATMENT OPTIONS FOR PULSATILE TINNITUS

Aetiology	Treatment options
Benign intracranial hypertension	Medical: weight reduction, aceatazolamide, furosemide[13] Surgical: [a]gastric bypass surgery,[43] [b]lumbo-peritoneal shunting[12]
Glomus tumours	Surgery[12]
Atherosclerotic carotid artery disease	Carotid endarterectomy[44]
AVM/AVF	Selective embolization[45]
Otosclerosis	Stapedectomy[12]
Paget's disease	[c]Medical: bisphosphonates,[46] calcitonin,[47] combination calcitonin & bisphosphonates[48] [d]Surgical: stapedectomy[12]
Idiopathic, venous hum, essential tinnitus	[e]Ligation of internal jugular vein[8,50]

[a]Should be considered in morbidly obese patients who fail conservative treatment; [b]should be considered for patients with visual disturbance secondary to papilloedema and/or those with severe tinnitus who have failed conservative treatment; [c]treatment of symptomatic Paget's disease is almost exclusively medical (bisphosphonates can relieve both the deafness and tinnitus); [d]results of reconstructive middle-ear surgery in Paget's disease have been unsatisfactory[51] – stapedectomy should only be considered for residual stapes fixation after failed medical treatment; [e]should be considered after benign intracranial hypertension and other causes of raised intracranial pressure have been eliminated as may trigger serious intracranial venous hypertension.[49] AVM = arteriovenous malformation; AVF = arteriovenous fistula

adequate ambient sounds, either with the use of ambient sound generators or with hearing aids carefully programmed to correct the hearing loss. A poor hearing aid prescription is likely to aggravate the tinnitus by increasing the tendency to strain to hear. The prescription of sound generators to patients with a significant hearing loss is inappropriate, as they will hear only those components of the sound that are above their hearing thresholds. This would increase the tendency to strain to hear and, with it, the PT. Patients should be cautioned not to use ear plugs as these will increase the straining to hear and the perception of internally generated PT.

Alleviation of the perception of tinnitus involves helping patients alter their selective attention to the tinnitus. This involves cognitive, emotional and behavioural factors, and intervention may include counselling, giving a rational explanation for the tinnitus (particularly that there is no sinister cause) and advising that the PT itself does not cause damage to the ears nor does it represent progressive damage to the ears. Patients with persisting symptoms may require cognitive behaviour therapy, tinnitus retraining therapy and pharmacological intervention. Patients with significant tinnitus distress or psychological symptoms resulting from the PT should be referred for a psychiatric opinion.

Conclusion

Pulsatile tinnitus may be a very distressing symptom. In certain unfortunate individuals, it can prove to be almost unbearable and provoke significant morbidity. Its causes are numerous. Clinical history and physical examination are of the utmost importance in establishing the correct diagnosis. The prudent use of well adapted imaging studies can aid diagnosis. Many patients with PT have a treatable underlying aetiology. Early diagnosis and appropriate intervention may prevent unnecessary morbidity.

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