

TITLE: Tinnitus

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Introduction

Tinnitus is the perception of sound in the absence of external stimuli. The term comes from the Latin term *tinnere* which means ringing, but is used to describe sounds that are buzzing, roaring, pulsatile, or clicking in nature. The sound may be perceived as either unilateral or bilateral. Tinnitus may be the first or only symptom of a disease process which threatens the patient's health or well being.

It is estimated that 40 million people in the United States suffer from tinnitus with 10 million of those severely affected. The prevalence is highest in 40-70 year-olds and it is more common in men than in women. Tinnitus can have a great impact on a patient's quality of life. Ludwig Von Beethoven described his experience with tinnitus in a letter to a friend in 1801, "only my ears whistle and buzz continuously day and night. I can say I am living a wretched life."

Tinnitus may be classified as either objective tinnitus, which may be heard by an examiner, or subjective tinnitus in which the sound is only perceived by the patient. Subjective tinnitus is much more common than objective tinnitus. Tinnitus may also be classified as pulsatile or nonpulsatile, with pulsatile usually indicating a vascular etiology.

Objective Tinnitus

Objective tinnitus is the patient perceiving sound produced by paraauditory structures which may be heard by an examiner. It may be either pulsatile or nonpulsatile in nature. Take a careful history from the patient including the relationship of the sound to the heartbeat, the quality of the sound, aggravating/relieving factors, associated hearing loss, and whether the sound is unilateral or bilateral. Possible etiologies include vascular abnormalities, Eustachian tube dysfunction, or muscle spasm.

Vascular Abnormalities

Tinnitus which is described by a patient as pulsatile or with a rushing sound may be of vascular origin. The sound is produced by the flow of blood through paraauditory structures. Vascular tinnitus may be either objective or subjective. Possible causes are arteriovenous malformations, vascular tumors, venous hum, atherosclerosis, ectopic carotid artery, persistent stapedial artery, dehiscent jugular bulb, vascular loops, cardiac murmurs, increased cardiac output (pregnancy, anemia, hyperthyroidism), Paget's disease, and benign intracranial hypertension.

Arteriovenous malformations are congenital lesions. Types include communication of the occipital artery and the transverse sinus, the internal carotid artery and the vertebral arteries, the middle meningeal artery and the greater superficial petrosal artery, and those of the mandible, brain parenchyma, or dura. Pulsatile tinnitus may be the initial symptom, but AVMs can also be associated with headache, papilledema, or discoloration of skin or mucosa.

Vascular tumors which can cause tinnitus include glomus tympanicum and glomus jugulare. These are paragangliomas which arise in the middle ear or jugular bulb respectively. A glomus jugulare may extend up into the middle ear. When the mass involves the middle ear it may be seen as a reddish mass behind the tympanic membrane.

A venous hum may be present from increased or turbulent flow through the venous system. Possible causes include a dehiscent jugular bulb, transverse sinus obstruction, benign intracranial hypertension, or increased cardiac output from pregnancy, anemia, or thyrotoxicosis. A dehiscent jugular bulb may be seen on otomicroscopy as a bluish mass in the middle ear. Patients with benign intracranial hypertension are usually female, overweight and have symptoms of hearing loss, aural fullness, dizziness, headaches, and visual disturbances.

Patulous Eustachian Tube

In patients with Eustachian tube dysfunction in which the tube remains open abnormally, it is called a patulous Eustachian tube. Patients may describe the sound as an ocean roar in the ear which changes with respiration. They may have relief with lying down or putting the head in a dependent position. A tympanogram may show motion of the tympanic membrane with respiration.

Muscle Spasm

Palatal myoclonus is a muscular cause of tinnitus. It is caused by contraction of tensor palatini, levator veli palatini, tensor tympani, salpinopharyngeal, or superior constrictor muscles. It is described as a clicking sound which is rapid (60-200 beats per minute), repetitive, and intermittent. It is associated with multiple sclerosis, small vessel disease, tumor, and degenerative neurological disorders. The muscle spasms may be seen either transorally or transnasally. A plot of compliance as a function of time on tympanometry will show the rhythmic change in compliance of the tympanic membrane corresponding to the muscle contraction.

Idiopathic stapedial muscle spasm induced tinnitus is described as a rough, rumbling, or crackling noise which may be exacerbated by outside sounds. The tinnitus tends to follow a sound stimulus, is brief and intermittent.

Subjective Tinnitus

Subjective tinnitus is not able to be heard by an examiner. It is more common than objective tinnitus and is usually nonpulsatile in nature. Subjective tinnitus is associated with presbycusis, noise exposure, Meniere's disease, otosclerosis, head trauma, acoustic neuroma, drugs, middle ear effusion, temporomandibular joint problems depression, hyperlipidemia, meningitis, and syphilis.

Conditions which result in a conductive hearing loss, such as middle ear effusion, otosclerosis, or cerumen impaction may decrease the level of external sound to the point where the patient is able to hear normal skull sounds. In these cases, treatment of the cause of the conductive hearing loss may alleviate the tinnitus.

Tinnitus which is sensorineural in origin does not have a clear physiologic explanation. It may be caused by abnormalities of the cochlea, cochlear nerve, ascending auditory pathway, or auditory cortex. Many theories of the origin of tinnitus have been proposed which generally involve hyperactive hair cells or nerve fibers activated by a chemical imbalance across cell membranes or decoupling of stereocilia. A neurophysiologic model of tinnitus has been proposed by Jastreboff. In this theory, tinnitus emerges as the result of interaction of a number of subsystems in the nervous system, with auditory pathways playing a role in the development and appearance of tinnitus as sound perception, with the limbic system responsible for the development of tinnitus annoyance. The perception of tinnitus provides negative reinforcement which enhances the perception of tinnitus and the perception of time the person is aware of its presence. This model has led to the development of tinnitus retraining therapy to habituate the patient to the tinnitus.

Depression has been shown to be more prevalent in patients with chronic tinnitus than in people who do not complain of tinnitus. Folmer et al have reported their finding that patients with depression and tinnitus rated their tinnitus severity higher than patients with tinnitus who did not have depression.

Drugs

Many drugs have been linked to tinnitus. Although almost any medication can be a possible cause of tinnitus the most frequently implicated drugs are the antiinflammatories, antibiotics, and antidepressants. Both aspirin and quinine are associated with tinnitus. This tinnitus is high frequency, tonal in nature, and accompanied by a temporary threshold shift. The tinnitus is reversible with cessation of the medication. Aminoglycoside antibiotics are also often implicated as the cause of drug-induced tinnitus. Other medications include loop diuretics and chemotherapeutic agents such as cisplatin and vincristine. Any of the heterocycline antidepressants (i.e. amitriptyline, imipramine) can cause tinnitus. This is interesting because antidepressants have also been investigated for the treatment of tinnitus.

Evaluation

The evaluation of a patient with tinnitus should start with a carefully taken history. The patient's description of the tinnitus is very important, it can provide key information during the initial evaluation. The quality of the sound, especially whether it is pulsatile or nonpulsatile, the perceived location, the pitch, the loudness, constant or episodic, onset, alleviating/aggravating factors, history of infection or trauma, noise exposure, medication usage, medical history, associated hearing loss/vertigo, family history of hearing loss and associated pain should all be topics of inquiry. Another very important factor is the impact of the tinnitus on the patient. There are several tinnitus-specific self assessment tools available for evaluating the perceived severity of the tinnitus to the patient. The Tinnitus Handicap Inventory is a 25 item survey that provides a total score and three subscale scores for functional, emotional, and catastrophic impact on the patient.

After a thorough history is taken a complete head and neck exam, as well as a general physical exam should be performed. Otomicroscopy should be performed to look for a middle ear mass or motion of the tympanic membrane with respiration. A glomus tympanicum can be seen as a reddish mass in the middle ear or a dehiscent jugular bulb may be seen as a bluish mass. With a history of pulsatile tinnitus, the physician should search for an audible bruit by auscultating the external canal with a Toynbee tube, and over the orbit, mastoid process, skull, and neck using the bell and diaphragm of a stethoscope. The heart should be auscultated for murmurs. The patient should perform light exercise to see if this increases the pulsatile tinnitus. Tinnitus of arterial origin will often worsen with exercise. Venous induced tinnitus may decrease with light pressure on the neck, turning the head, or with the Valsalva maneuver.

All patients may undergo audiometric testing including pure tone audiometry, speech discrimination, tympanometry, and acoustic reflex measurements. The pitch of the tinnitus may be matched by the patient to an administered pure tone. The loudness of the tinnitus may be estimated by having the patient adjust the level of a pure tone to the loudness of their tinnitus. The minimal masking level is the number of decibels required to mask the tinnitus. In the case of vascular or palatomyoclonus induced tinnitus, a graph of tympanic membrane compliance versus time will show changes in compliance which correspond to the pulse or palatal movement respectively. Patulous Eustachian tube induced tinnitus can be diagnosed by a change in the compliance which corresponds to respiration. Unilateral high-frequency sensorineural hearing loss associated with tinnitus and asymmetric speech discrimination scores suggests an acoustic neuroma. Patients with unilateral symptoms may undergo auditory brainstem evoked response testing. These patients should undergo an acoustic protocol MRI to evaluate for acoustic neuroma, which will be further discussed below.

Laboratory studies may be obtained as indicated by the history and physical exam. Possible studies include hematocrit, fluorescent treponemal antibody absorption tests, blood chemistries, thyroid studies, and a lipid battery.

Weissman and Hirsch recently reviewed the imaging of tinnitus. They recommend contrast-enhanced computed tomography of the temporal bones and skull base as the first line

study for evaluating pulsatile tinnitus. The diagnosis of glomus tympanicum tumors is made on the bone algorithm scans which best shows the extent of the mass. It is usually not possible to see enhancement of a small tumor confined to the middle ear on a CT study. Either T1-weighted MRI with gadolinium enhancement or T2 weighted images will show the tumor enhancement. The earliest detectable abnormality on CT of glomus jugulare tumors is erosion of the lateral and anterior walls of the osseous jugular fossa. These tumors enhance significantly with contrast material, it may not be possible to differentiate the internal jugular vein from tumor. As with glomus tympanicum tumors T1-weighted MRI with gadolinium or T2-weighted images will show tumor enhancement. The classical characteristic MRI pattern is a “salt and pepper” appearance. Extracranial arteriovenous malformations as well as brain parenchymal AVMs are usually readily identified on contrast CT and MR studies. A patient with pulsatile tinnitus and a normal otoscopic exam may have a dural AVM or AVF. These lesions are often invisible on CT and MRI. Conventional angiography may be the only study to show the abnormality. Other abnormalities which can be identified on contrast enhanced CT include an aberrant carotid artery, a dehiscent carotid artery, dehiscent jugular bulb, and a persistent stapedia artery. CT findings of a persistent stapedia artery are the appearance of soft tissue on the promontory, enlargement of the facial nerve canal, and absence of the foramen spinosum.

Unilateral tinnitus or asymmetric sensorineural hearing loss is an indication for MRI to evaluate for an acoustic neuroma. A gadolinium enhanced MRI of the cerebellopontine angle is the study of choice to diagnose these lesions.

Treatment

The multiple etiologies and poorly understood mechanisms of tinnitus have led to the attempt at multiple treatment modalities. These include diet modification, medications, habituation, masking, electrical stimulation, acupuncture, hypnosis, and surgery. Some patients need only reassurance that the tinnitus is not a sign of a serious medical disease. Having a physician acknowledge that their symptoms are real and receiving follow up appointments, possibly with repeat audiogram to allay fears of worsening deafness may be of benefit to some patients. Avoidance of stimulants such as coffee, tea, chocolate, cola, and other caffeine containing medications as well as smoking cessation may help some patients. Patients should be instructed to avoid medications, which are known to cause tinnitus such as aspirin and NSAIDs. The use of white noise from a radio or a home masking machine is also helpful in some cases.

Many medications have been researched for the treatment of tinnitus, including lidocaine, tocainide, carbamazepine, benzodiazepines, tricyclic antidepressants, and ginko biloba. Lidocaine administered intravenously has been shown to improve tinnitus but is impractical to use clinically. Tocainide is an oral compound closely related to lidocaine, it has been shown to be ineffective in the treatment of tinnitus. Several randomized-controlled trials have also shown that carbamazepine is ineffective and may cause bone marrow suppression. Johnson et al performed a double-blind, placebo-controlled study of the effectiveness of the benzodiazepine alprazolam and found 76% of patients had improvement in their tinnitus. The risk of dependency to this medication is a significant risk of its use. Interestingly, given that tricyclic antidepressants are implicated as a possible cause of some tinnitus, is that nortriptyline has been

shown to be more effective than placebo. Ginkgo biloba has also shown some benefit in the reduction of tinnitus in some studies.

Hearing aids, maskers, or combinations of the two may help some patients. If the patient has some hearing loss, amplification of background noise by a hearing aid can decrease tinnitus. A masker produces sound to mask the tinnitus and decrease the annoyance to the patient. There are combination hearing aids/maskers which can be used which are called tinnitus instruments.

Tinnitus retraining therapy is a technique of habituation using a combination of masking with low level broadband noise and counseling to achieve habituation of the reaction to tinnitus and the perception of the tinnitus signal itself. In a study of 32 patients Berry et al found a significant improvement in the Tinnitus Handicap Inventory scores of tinnitus patients following six months of tinnitus retraining therapy.

Electrical stimulation of the cochlea has been studied for the treatment of tinnitus. Transcutaneous, round window, and promontory stimulation of the cochlea have shown some benefit. Direct currents may produce permanent damage and cannot be used clinically. Steenerson and Cronin used transcutaneous stimulation of the auricle and tragus to decrease tinnitus in 53% of 500 patients treated in this manner. Cochlear implants have also shown some promise in the relief of tinnitus. Ito and Sakakihara reported 77% of 26 patients with tinnitus who underwent cochlear implantation had relief of their tinnitus and 8% had aggravation of their tinnitus.

Surgical treatment of tinnitus is used in the treatment of arteriovenous malformations, vascular tumors, otosclerosis, and acoustic neuroma. Some authors have reported success with cochlear nerve section in patients with intractable tinnitus that is recalcitrant to all other treatment modalities, however this is not advocated by most otologists.

Other treatments that have been studied are biofeedback, hypnosis, magnetic stimulation and acupuncture. Studies of these modalities have shown conflicting results as to their benefit.

Conclusion

Tinnitus is a symptom that can be classified as objective or subjective. The majority of patients have subjective tinnitus associated with presbycusis. The impact of tinnitus on the patient's well being and quality of life is an important factor in deciding upon treatment. As we are faced with the aging of the population in the United States, tinnitus will surely become even more prevalent. Continued research into the mechanism and treatment of tinnitus is necessary to make progress in relieving the suffering of our patients.

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