



Tinnitus

evaluation
in primary care

Abstract: Tinnitus is a common, yet poorly understood problem. This symptom has many causes, both benign as well as serious. Patients can experience significant changes in quality-of-life related to symptom severity and duration. This article explores causes of tinnitus, evaluation in a primary care setting, and management strategies.

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In the United States, approximately 25% of the population experiences tinnitus, yet the condition is not well understood.¹ Tinnitus is a symptom, not a disease, and is frequently associated with hearing loss—85% to 96% of patients with tinnitus report some degree of hearing loss.² Tinnitus is defined as a phantom sound or the perception of sound within the human ear in the absence of external auditory stimulus or sound in the environment.^{3,4}

High-frequency hearing deficit and previous loud noise exposure have been found to be good predictors of tinnitus.^{1,3} Age is also a factor; while tinnitus may occur in children, it is more common in adults ages 60 to 69 with a 31% prevalence rate.¹ Tinnitus is more common in men than in women and affects non-Hispanic Whites more than Blacks or Hispanics.^{1,2,3}

Of the approximately 50 million people who experience tinnitus, about 16 million seek medical help. Of these, 2 to 3 million report symptoms so debilitating that they are unable to lead a normal life.^{1,5} Tinnitus is a significant clinical issue that can impact a patient's quality of life, cause anxiety, and negatively impact activities of daily living. It affects concentration, emotional stability, sleep habits, work productivity, and social activity.^{1,2,4} Depression, anxiety, and even suicide have been reported in individuals with severe tinnitus.^{6,7}

The degree of discomfort may be mild, moderate, or intense and is often unrelated to the loudness of the tinnitus. Patients with mild tinnitus may perceive the symptom only

in particular situations, and a patient with moderate tinnitus is aware of the symptom but is not bothered by it. Patients with intense tinnitus are disturbed by the ever-present sensation and view it as unbearable. These patients often make significant life modifications due to the intractable and unpleasant nature of the symptom.⁶

The exact physiology of tinnitus is unknown, but a variety of conditions and environmental exposures can lead to the development of the symptom. These underlying conditions range from simple cerumen impactions to more serious ones such as acoustic neuromas or arteriovenous malformations that require immediate attention.

■ **Classification of tinnitus**

Tinnitus may occur in one or both ears. This symptom is frequently perceived as ringing, whistling, roaring, or buzzing, and may be pulsatile (synchronous with the heartbeat). Tinnitus is divided into subjective or objective types.² Subjective is internal sound only perceived by the patient and is considerably more common. Objective tinnitus is rare and considered a real noise that can be heard by the examiner and the patient. The sound is audible to the examiner with the use of a stethoscope or Doppler, and the patient frequently describes it as a clicking or pulsing sound.⁸

■ **Causes**

The pathophysiology of tinnitus is not fully understood, and there are many risk factors for the development of subjective and objective tinnitus (see *Causes of objective*

Key words: high-frequency hearing deficit, previous loud noise exposure, pulsatile tinnitus, tinnitus

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Causes of objective and subjective tinnitus^{12,15,16,17}

| Type | Cause |
|----------------------------|--|
| Subjective | |
| Medication related | Adverse reaction of many drugs: antibiotics, diuretics, chemotherapeutic agents (including mechlorethamine and vincristine), high doses of aspirin and nonsteroidal anti-inflammatory drugs, quinine, antidepressants |
| Infections | Otitis media, otitis externa, meningitis, syphilis, Lyme disease, HIV otitis, herpes zoster |
| Sensorineural hearing loss | Trauma—head injury, whiplash, giant cell arteritis, acoustic neuroma, multiple sclerosis |
| Otologic | Ménière disease, cerumen impaction, loud noise or music induced, presbycusis, otitis media or otitis externa, middle ear effusion, otosclerosis |
| Other | TMJ dysfunction |
| Objective | |
| Pulsatile | Carotid stenosis, arteriovenous fistula or malformations, aneurysms, aberrant carotid artery, intracranial hypertension, high cardiac output (anemia, drug induced), glomus tympanicum, vascular tumors (glomus jugular) |
| Anatomic/muscular | Patulous Eustachian tube, palatal myoclonus, stapedial muscle spasm |

and subjective tinnitus). Internal and external risk factors include long-term exposure to noise (leisure time, occupational, and firearms) and associated hearing loss, cochlear dysfunction, history of middle ear infection, sinus infection, cerumen impactions, dizziness, and age. Tinnitus has also been associated with temporomandibular disease, head and neck muscle spasms, and vascular conditions such as arteriovenous malformations or acoustic tumors.⁹ The literature has found the heritability of tinnitus to be low. In a large Norwegian study, researchers found that only 11% of the variance of tinnitus was caused by genetic effects in the population. No gender differences were found in the heritability estimates.¹⁰

Tinnitus may occur without any external or internal event and is associated with the gradual deterioration of neural function related to aging. As part of the normal aging process, the number of functioning nerve fibers declines as a result of the decrease in reserves of the nervous system. There is also an increase in the variation of conduction velocity within the auditory nerve with aging.¹¹ Although hearing loss can occur with aging, tinnitus is not an inevitable correlate.

Medications are frequently associated with permanent or temporary tinnitus. Tinnitus may resolve once the medication is discontinued. Common ototoxic agents that cause tinnitus include: loop diuretics (ethacrynic acid, furosemide, bumetanide); salicylates (aspirin), non-

steroidal anti-inflammatory agents, quinine; antibiotics (aminoglycosides, erythromycin, vancomycin, polymyxin B, neomycin); chemotherapeutic agents (cisplatin, carboplatin, vinblastine, vincristine); topical agents (solvents, propylene glycol); and antiseptics (ethanol).¹² The medication or topical agent can cause damage to the cochlea, auditory nerve (the eighth cranial nerve, also known as the acoustic nerve), and sometimes the vestibular system. The exact pathophysiologic mechanism is unclear.

Sensorineural tinnitus is the most common form of the condition and is often associated with or occurs after hearing loss or presbycusis.¹³

Hearing loss, which is frequently associated with prolonged loud noise exposure, is caused by an injury to the cochlea with damage to the minute hair cells that vibrate in response to sound waves and outer hair cells. These cells convert neural signals into tension on the vibrating basement membrane.¹⁴

Tinnitus is also a symptom of Ménière disease, and the pathology also appears to be hair cell damage. In Ménière disease, the underlying pathology is possibly related to potassium toxicity.⁹ Tinnitus is usually the initial symptom in patients with acoustic neuroma, an uncommon, benign tumor arising from the Schwann cells covering the eighth cranial nerve. Tinnitus in these patients is most often unilateral and may be present for months to years before hearing loss or vertigo are noted.¹³

■ Symptom evaluation

Since tinnitus is a subjective symptom, history and physical exam are important to identify possible causes. Although diagnostic testing is limited in primary care settings, the nurse practitioner (NP) can use basic screening tools to decide if more extensive testing is needed. In addition, benign etiologies can usually be distinguished from those requiring an immediate referral to a specialist.

■ History

A tinnitus evaluation begins with a thorough, focused history. The NP should take a full symptom history including onset, location, characteristics, patterns, and alleviating/exacerbating factors. Past medical/surgical, family, social, and medication history should also be noted. Risk factors such as hypertension, diabetes, smoking, hyperlipidemia, and cerebrovascular disease raise suspicion of atherosclerotic carotid artery disease, especially in an older patient presenting with pulsatile tinnitus.¹⁸ Anemia or hyperthyroidism can result in a hyperdynamic state elevating cardiac output and cerebral blood flow.¹⁹ Focused questions related to the presenting tinnitus will gain specific information that can provide clues to possible underlying causes (see *Focused history questions to evaluate tinnitus*).¹⁷

Progressive hearing loss may be a sign of presbycusis, particularly in aging patients. Sudden onset is usually associated with either head trauma or extreme noise exposure. Location (unilateral or bilateral) can narrow down underlying etiologies. Unilateral presentation, particularly when accompanied by sensorineural hearing loss, can be a sign of acoustic neuroma. Other causes of unilateral tinnitus include cerumen impaction and otitis externa or media.¹³

Assessment of characteristics and pattern is critical. Information related to pattern includes whether the tinnitus is pulsatile or nonpulsatile, continuous or episodic. Although bilateral, nonpulsatile tinnitus is usually benign; pulsatile tinnitus can be a sign of a more serious vascular etiology. Episodic tinnitus, along with concurrent symptoms such as vertigo, hearing loss, and aural fullness are associated with Ménière disease.¹³ History of exposure to loud noise, either acutely and/or chronically, should be elicited, as this is a common cause of hearing loss in all ages. A full medication history is important to identify possible causative or contributing ototoxic agents. Factors that alleviate or exacerbate the symptom are important to determine. For example, lying down can often alleviate tinnitus due to Eustachian tube dysfunction.^{8,13} Factors such as stress and lack of sleep can sometimes exacerbate the symptom.

Focused history questions to evaluate tinnitus¹⁷

When did the symptom first occur?
 Was the onset sudden or gradual?
 How long has the symptom been there?
 Is the symptom unilateral or bilateral?
 Is the symptom continuous or episodic?
 Is the symptom pulsatile or nonpulsatile (that is, can you feel/hear the pulsing)?
 Is there any hearing loss?
 Is there any history of recent ear infections, head trauma, or surgery?
 Are there any other associated symptoms (vertigo, dizziness, aural fullness, pain, headache, tooth pain, jaw clicking or clenching, teeth grinding, pain with chewing, blurred vision, sleepiness)?
 Have you been exposed to environmental noise (recent and/or chronic)?
 What medications are you taking (prescription, over-the-counter, and herbal)?
 Is there anything that makes the symptom better or worse (such as position, background noise, stress, lack of sleep, alcohol, smoking or caffeine use)?
 How does the symptom affect daily life or activities?
 Do you feel depressed or have you had any thoughts about death or suicide? (Note: a positive response will require more extensive evaluation of depression and/or referral.)

■ Physical exam

Exam includes a thorough head, ear, nose, neck, and throat assessment. The exam begins with inspection of the ears for cerumen impaction, foreign body, infection, inflammation, or tympanic membrane perforation or scarring that can affect hearing and result in tinnitus. Otoscopy might reveal a blue or red mass behind the tympanic membrane in cases of pulsatile tinnitus caused by vascular origin.¹⁸ Cranial nerves are assessed to evaluate hearing loss or brainstem dysfunction. Hearing can be assessed using the whisper test as well as the Weber and Rinne tests to determine any sensorineural or conductive hearing loss (see *Determining the type of hearing loss*).^{18,19} An oral inspection can reveal any dental issues or palatal muscle contractions. Temporomandibular joint (TMJ) dysfunction should be assessed, noting any snapping or clicking during palpation.

In the presence of pulsatile tinnitus, gentle pressure can be applied to the ipsilateral internal jugular vein (IJV). Resolution of the tinnitus can indicate a venous etiology.²⁰ If the symptom persists, the source is more likely arterial. The same response is also verified by having the patient turn the head toward the side of the tinnitus. This maneuver causes compression of the IJV. With a venous cause, the tinnitus will decrease in intensity or cease. Turning the

Determining the type of hearing loss¹⁹

| Technique | Rinne | Weber |
|--|---|--|
| | Place the vibrating tuning fork on the mastoid bone. When the patient can no longer hear the sound, place the tuning fork close to the ear and assess if the sound can still be heard. Normal finding: AC > BC | Place the base of the vibrating tuning fork on top of the head or on the forehead Normal finding: equal sound |
| |  |  |
| | Conductive hearing loss BC = AC or BC > AC | Conductive hearing loss Sound lateralizes to impaired ear |
| | Sensorineural hearing loss AC > BC | Sensorineural hearing loss Sound heard in good ear |
| AC = air conduction BC = bone conduction | | |

head in the other direction will cause an opposite effect. If the cause is arterial, the maneuver will not have any effect on the tinnitus.²⁰

The NP should perform a full cardiovascular assessment, especially noting BP and any murmurs and dysrhythmias. Carotid and/or temporal artery bruits are important to note.

■ Diagnostics

The need for lab testing is guided by the patient's presentation. However, routine lab testing without a pertinent history is not recommended.¹⁶ If indicated, testing may include a complete blood cell count with differential to rule out anemia or infection, a complete metabolic panel to determine any underlying health conditions such as diabetes, an erythrocyte sedimentation rate to screen for an autoimmune process, a lipid profile to determine a possible risk factor for atherosclerosis, and thyroid function tests. Vitamin B₁₂ and folate levels might be considered in some patients as deficiencies have been associated with hearing loss or tinnitus.^{21,22} Tests for infectious causes such as syphilis, HIV, or Lyme disease may be warranted based on history and physical exam data.

Tympanometry can be used to measure tympanic membrane compliance in patients with suspected middle ear

effusions.^{15,23} Tympanic membrane stiffness can also be caused by a patulous Eustachian tube, palatal myoclonus, or stapedial muscle spasm.¹³ Simple office audiology, if available, can be used to screen for hearing loss. A hearing threshold level above 25 decibels on audiogram is considered abnormal.^{17,24} Patients with hearing loss or suspected auditory causes should be referred for more extensive auditory testing. Audiologic testing may include measurement of pure tone (measures air and bone conduction), speech reception threshold, word discrimination score, otoacoustic emissions measurement (test integrity of outer hair cells of organ of Corti), acoustic reflexes, reflex-decay threshold, and decay measurement. The acoustic reflex is the involuntary contraction of the stapedius and tensor tympani muscles that occurs in response to a high-intensity sound. The threshold measures the lowest-intensity sound that elicits the reflex. The reflex decay test measures whether the contraction is maintained or weakened during continuous auditory stimulation.²² Changes in the acoustic reflex can occur with conductive hearing loss, otosclerosis, and pathology, affecting the seventh or eighth cranial nerve.²⁴ Other tests may be added based on results and at the audiologist's discretion.²⁵ Expert interpretation of results is generally done collaboratively by an otolaryngologist and audiologist.

All patients with unilateral tinnitus or pulsatile tinnitus should be referred to an otolaryngologist for further evaluation and any radiologic testing. If the patient has pulsatile tinnitus and has had recent head trauma or surgery, an urgent referral to an otolaryngologist is warranted. If specialty services are not available, the patient should be sent to an emergency department.

■ Management

Tinnitus management is based on the underlying cause of the symptom. The symptom may be mild, and removal of the cause, such as infection or medication, can result in reversal or nonprogression. Evaluation may indicate that no treatment is needed. Any obvious underlying health condition, including dental issues or TMJ dysfunction, should be treated. Management of tinnitus often requires an interdisciplinary approach.

The patient will need education about factors that can impact tinnitus. Exposure to loud noise and excessive use of caffeine, nicotine, and alcohol are areas to address. Further hearing loss must be avoided for patients with noise-induced loss. Patients exposed to loud noise in the work environment must wear ear protection consistently. Patients should limit their use of portable music players with ear buds and other, similar devices as these are associated with hearing losses in adolescents.^{26,27} Overuse of medications such as aspirin, nonsteroidal anti-inflammatory drugs, or other potentially ototoxic agents should be avoided. A healthy diet and regular exercise are important components of the management plan as well. Some patients may benefit from stress management and/or coping counseling.²⁸ In a patient with sleep problems, a full sleep evaluation should be performed, as well as cognitive behavioral therapy to address sleep hygiene.

Strategies to address bothersome or more severe tinnitus can include using a hearing aid, if necessary, and sound maskers such as tapes, white noise generators, or radios. Sound devices create a background of noise that make tinnitus less noticeable to the patient.²⁹ Some patients may benefit from referral for cognitive behavioral therapy. Therapies such as biofeedback, acupuncture, and electrical stimulation are other alternatives, although most are lacking clear scientific evidence of efficacy.

Pharmacologic therapy is limited. Efficacy of antiepileptics, antispasmodics, vasodilators, and diuretics are not supported by evidence.^{3,9,17} In a randomized, prospective, placebo-controlled, double-blind trial of 40 subjects with presbycusis, pramipexole showed a significant improvement in tinnitus annoyance but no change in hearing threshold.³⁰

If depression accompanies tinnitus, an antidepressant can be considered. Thoughts of death or suicide indicate an immediate need for psychiatric referral.

Patients with tinnitus may be desperate to find any type of relief and turn to the Internet for information. The NP needs to steer the patient to reliable information. Beneficial resources for patients and families about tinnitus and its management are the American Tinnitus Association (<http://www.ata.org>) and the American Academy of Otolaryngology-Head and Neck Surgery (<http://www.entnet.org>).

■ Implications

Tinnitus can be a bothersome symptom that affects quality of life. In some cases, this symptom may be a warning sign of a more ominous vascular condition. Although the underlying causes are numerous, taking a focused history and physical is the key to determining possible etiologies. Benign causes may require little testing. Unilateral or pulsatile tinnitus warrants immediate referral to the otolaryngologist or emergency department for evaluation of cause. Management of tinnitus is determined by the severity of the symptom, and may warrant an interdisciplinary approach including the NP, physician,

In some cases, the symptom of tinnitus may be a warning sign of a more ominous vascular condition.



audiologist, therapist, or counselor. Severe cases necessitate referral for evaluation by medical or dental specialists. In nonbenign cases, surgical intervention is often necessary. However, in most patients with tinnitus, education and the use of targeted devices are the only necessary treatments. NP

REFERENCES

- Shargorodsky J, Curhan GC, Farwell WR. Prevalence and characteristics of tinnitus among US adults. *Am J Med.* 2010;123(8):711-718.
- Morales-Garcia C, Quiroz G, Matamala JM, Tapia C. Neuro-otological findings in tinnitus patients with normal hearing. *J Laryngol Otol.* 2010;124(5):474-476.
- McFerran DJ, Phillips JS. Tinnitus. *J Laryngol Otol.* 2007;121(3):201-208.
- Agency for Healthcare Research and Quality [AHRQ]. Evaluation and treatment of tinnitus: a comparative effectiveness review. <http://www.effectivehealthcare.ahrq.gov/index.cfm/search-for-guides-reviews-and-reports/?pageaction=displayproduct&productid=811>.
- American Tinnitus Association [ATA]. Top 10 most frequently asked questions. www.ata.org/for-patients/faqs.
- Teggi R, Caldirola D, Perna G, Bussi M. The "emotional side" of tinnitus. In: Bahmad F, ed. *Up To Date on Tinnitus*. http://cdn.intechopen.com/pdfs/25113/InTech-The_emotional_side_of_subjective_tinnitus.pdf.
- Prestes R, Daniela G. Impact of tinnitus on quality of life, loudness and pitch match, and high-frequency audiometry. *Int Tinnitus J.* 2009;15(2):134-138.
- Welch KJ, Nath AR, Lewin MR. Evaluation of tinnitus in the emergency department. In: Bahmad F, ed. *Up To Date on Tinnitus*. http://cdn.intechopen.com/pdfs/25108/InTech-Evaluation_of_tinnitus_in_the_emergency_department.pdf.

9. Henry JA, Dennis KC, Schechter MA. General review of tinnitus: prevalence, mechanisms, effects and management. *J Speech Lang Hear Res.* 2005;48(5):1204-1235.

10. Kvestad E, Czajkowski N, Engdahl B, Hoffman HJ, Tambs K. Low heritability of tinnitus. *Arch Otolaryngol Head Neck Surg.* 2010;136(2):178-182.

11. Roberts LE, Eggermont JJ, Caspary DM, Shore SE, Melcher JR, Kaltenbach JA. Ringing ears: the neuroscience of tinnitus. *J Neurosci.* 2010;30(45):14972-14979.

12. Silverberg M, Lucchesi M. Common disorders of the external, middle and inner ear. In: Tintinalli JE, Stapczynski JS, Cline DM, Mam OJ, Cydulka RK, Meckler GD, eds. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide.* 7th ed. New York, NY: McGraw-Hill; 2011:1518-1526.

13. Crummer RW, Hassan GA. Diagnostic approach to tinnitus. *Am Fam Physician.* 2004;69(1):120-126.

14. Henry JA, Zaugg TL, Myers PJ, Kendall CJ. Definitions and background. *Progressive Tinnitus Management: Clinical Handbook for Audiologists.* Long Beach, CA: VA Employee Education System; 2010:1-10. http://www.ncrar.research.va.gov/Education/Documents/TinnitusDocuments/01_HenryPTM-HB_1-10.pdf.

15. Lalwani A. Disorders of hearing. In: Longo D, Kasper D, Jameson JL, Fauci A, Hauser S, Loscalzo J, eds. *Harrison's Principles of Internal Medicine.* 18th ed. New York, NY: McGraw-Hill; 2012:248-255.

16. Stachler RJ, Chandrasekhar SS, Archer SM, et al. Clinical practice guidelines—sudden hearing loss. *Otolaryngol Head Neck Surg.* 2012;146(suppl 3):S1-S35.

17. Lockwood AH, Salvi RJ, Burkard RF. Tinnitus. *N Engl J Med.* 2002;347(12):904-910.

18. Conlin AE, Massoud E, Versnick E. Tinnitus: identifying the ominous causes. *CMAJ.* 2011;183(18):2125-2128.

19. Bickley LS, Szilagyi P. *Bates' Guide to Physical Examination and History Taking.* 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:226-227.

20. Liyanage SH, Singh A, Savundra P, Kalan A. Pulsatile tinnitus. *J Laryngol Otol.* 2006;120(2):93-97.

21. Cadoni G, Agostino S, Scipione S, Galli J. Low serum folate levels: a risk factor for sudden sensorineural hearing loss? *Acta Otolaryngol.* 2004;124(5):608-611.

22. Shemesh Z, Attias J, Oman M, Shapira N, Shahar A. Vitamin B12 deficiency in patients with chronic-tinnitus and noise-induced hearing loss. *Am J Otolaryngol.* 1993;14(2):94-99.

23. Bhattacharyya M. Evaluation of hearing loss. In: Gorroll A, Mulley A., eds. *Primary Care Medicine: Office Evaluation and Management of the Adult Patient.* 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009:1377-1383.

24. Sweetow RW, Sabes JH. Audiologic testing. In: Lalwani AK, ed. *CURRENT Diagnosis & Treatment in Otolaryngology—Head & Neck Surgery.* 3rd ed. New York, NY: McGraw-Hill; 2012. <http://www.accessmedicine.com/content.aspx?aID=55770577>.

25. Steiger JR, Hamill TA. A proposed clinical pathway for tinnitus evaluation and management. *Hearing J.* 2004;57:26-28.

26. Vogel I, Verschuure H, van der Ploeg CP, Brug J, Raat H. Adolescents and MP3 players: too many risks, too few precautions. *Pediatrics.* 2009;123(6):e953-e958.

27. Vogel I, Brug J, Hosli EJ, van der Ploeg CP, Raat H. MP3 players and hearing loss: adolescents' perceptions of loud music and hearing conservation. *J Pediatr.* 2008;152(3):400-404.

28. Tyler RS, Haskall GB, Gogel SA, Gehringer AK. Establishing a tinnitus clinic in your practice. *Am J Audiol.* 2008;17(1):25-37.

29. Henry J, Zaugg TL, Schechter MA. Clinical guide for audiologic tinnitus management II: treatment. *Am J Audiol.* 2005;14(1):49-70.

30. Sziklai I, Szilvassy J, Szilvassy Z. Tinnitus control by dopamine agonist pramipexole in presbycusis patients: a randomized, placebo-controlled, double-blind study. *Laryngoscope.* 2011;121(4):888-893.

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