

Diagnostic Approach to Patients with Tinnitus

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Tinnitus, a common symptom encountered in family medicine, is defined as the perception of noise in the absence of an acoustic stimulus outside of the body. Because tinnitus is a symptom and not a disease, its underlying cause must be determined to best help patients. Although tinnitus is often idiopathic, sensorineural hearing loss is the most common identified cause. It can also be caused by other otologic, vascular, neoplastic, neurologic, pharmacologic, dental, and psychological factors. More serious causes, such as Meniere disease or vestibular schwannoma, can be excluded during the evaluation. History and physical examination of the head, eyes, ears, nose, throat, neck, and neurologic system guide subsequent evaluation. Almost all patients with tinnitus should undergo audiometry with tympanometry, and some patients require neuroimaging or assessment of vestibular function with electronystagmography. Supportive counseling should begin during the initial evaluation to help patients cope with tinnitus. Counseling may also improve the chances of successful subsequent treatment. (*Am Fam Physician*. 2014;89(2):106-113. Copyright © 2014 American Academy of Family Physicians.)

CME This clinical content conforms to AAFP criteria for continuing medical education (CME). See CME Quiz Questions on page 78.

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► **Patient information:** A handout on this topic, written by the author of this article, is available at <http://www.aafp.org/afp/2014/0115/p106-s1.html>. Access to the handout is free and unrestricted.

Tinnitus is a common symptom encountered in family medicine. It is defined as sound perception in the absence of sound input external to the patient. Subjective tinnitus is the most common type, and is audible only to the patient, without internal or external sound input. Objective tinnitus is rare, accounting for less than 1% of cases.¹ It involves the perception of an internal sound, such as a bruit, as tinnitus. The causes of objective tinnitus are chiefly vascular or muscle dysfunction.²

Tinnitus lasting at least five minutes was reported by 30% of persons 49 years and older in an Australian population-based cohort.³ In a U.S. population-based study, slightly more than 8% of persons 48 years and older were affected by tinnitus that was moderately severe or that impacted sleep.⁴ Although it can be transient, older persons are more likely to have persistent tinnitus.⁵ A simple and efficient approach to the evaluation and diagnosis of tinnitus can safely detect the minority of persons with more serious etiologies, such as Meniere disease or vestibular schwannoma.

Etiology

Because tinnitus is a symptom and not a disease, there is no objective test to confirm its existence or grade its severity.⁶ For this reason, much of the workup for tinnitus is

without evidence. The etiology of tinnitus is often multifactorial, but in many cases, it is a response of the central nervous system to insufficient or abnormal input from the ear, comparable to phantom limb syndrome.⁷ In this syndrome, persons perceive their limb even after it has been amputated. In a similar manner, tinnitus persists even after surgical transection of the auditory nerve.⁷

Tinnitus occurs in most persons with normal hearing who are exposed to silence.⁸ It can be affected by input outside of the auditory system. For example, in patients with somatosensory tinnitus, movements of the head, neck, or limbs or palpation of myofascial trigger points can modulate or reproduce tinnitus.⁹ The belief that tinnitus is entirely psychosomatic is outmoded. Appreciation of a person's subjective response to tinnitus can help establish a therapeutic alliance that will support further education and therapy.

Table 1 lists the common etiologies of tinnitus.^{1,2,10-15} The condition most often associated with tinnitus is sensorineural hearing loss caused by presbycusis or occupational noise exposure.^{3,16} Conductive hearing loss from cerumen impaction, middle ear effusion, or otosclerosis is also associated with tinnitus.⁴ Although certain metabolic conditions were previously thought to cause tinnitus, there is little evidence to support this.¹⁵ A Medline search found no studies linking

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	Evidence rating	References
Patients with chronic tinnitus should undergo pure tone audiometry.	C	34, 49
Patients with unilateral tinnitus or asymmetric sensorineural hearing loss should undergo neuroimaging.	C	33
Non-contrast-enhanced T2- and T2*-weighted MRI may be equivalent to contrast-enhanced MRI for the evaluation of vestibular schwannoma.	C	33
Patients with pulsatile tinnitus should be evaluated with neurovascular imaging. The choice of imaging technique depends on whether arterial or venous tinnitus is suspected.	C	51, 55

MRI = magnetic resonance imaging.

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to <http://www.aafp.org/afpsort>.

Table 1. Selected Causes of Tinnitus

Subjective

Otologic: hearing loss, cholesteatoma, Meniere disease, vestibular schwannoma

Toxicologic: medication or substance use

Somatic: temporomandibular joint dysfunction, head or neck injury

Traumatic: cerumen removal¹⁰

Neurologic: multiple sclerosis, spontaneous intracranial hypotension,¹¹ type I Chiari malformation,¹² idiopathic intracranial hypertension,¹³ vestibular migraine¹⁴

Infectious: viral, bacterial, fungal

Metabolic (weak evidence): hyperlipidemia, diabetes mellitus, vitamin B₁₂ deficiency

Objective

Patulous eustachian tube

Vascular: arterial bruit, venous hum, arteriovenous malformation, vascular tumors, carotid atherosclerosis, dissection, or tortuosity; Paget disease

Neurologic: palatal myoclonus, idiopathic stapedial or tensor tympani muscle spasm

NOTE: Causes listed in approximate order from most to least common.

Information from references 1, 2, and 10 through 15.

tinnitus with thyroid disease. The search found a single case report and a weakly suggestive observational study linking tinnitus with vitamin B₁₂ deficiency,^{17,18} as well as several case series and small observational studies inconsistently suggesting a link between tinnitus and hyperlipidemia or diabetes mellitus.¹⁹⁻²⁴

Diagnosis depends on the presence of typical symptoms, with or without audiometrically documented hearing loss.³¹ When hearing loss is documented in patients with Meniere disease, it is usually noted in lower pitches.³⁰

Vestibular schwannoma, formerly known as acoustic neuroma, is a benign tumor of the acoustic nerve

More than 130 medications have been reported to cause tinnitus or hearing loss.²⁵ To decrease the risk of iatrogenic tinnitus, patients should avoid using more than one potentially ototoxic agent concurrently, and should limit the dose and duration, especially if other risk factors for tinnitus are present. *Table 2* lists common medications consistently associated with tinnitus.^{25,26}

Some studies suggest that a somatosensory component may be present in some patients with tinnitus, although this is not fully understood. Tinnitus may be associated with temporomandibular joint dysfunction²⁷ and whiplash,³ and may be modulated by body movements.²⁸ Several neurologic conditions can produce tinnitus. For example, spontaneous intracranial hypotension is an uncommon but increasingly recognized cause of daily or thunderclap headache; it results from a spontaneous cerebrospinal fluid leak often following a forceful Valsalva maneuver.¹¹ Spontaneous intracranial hypotension is characterized by a positional headache similar to a post-dural puncture headache, and patients may have associated tinnitus. Idiopathic intracranial hypertension is another neurologic cause of tinnitus that occurs primarily in women who are overweight.¹³ Vestibular migraine is characterized by vertiginous symptoms coincident at least twice with migraine symptoms in persons with a history of recurrent vestibular symptoms and migraine.¹⁴

Meniere disease is characterized by intermittent episodes of vertigo, fluctuating hearing loss, tinnitus, and ear pressure secondary to malabsorption of endolymphatic fluid in the inner ear.²⁹ Patients may present with any one of these symptoms or a combination of symptoms, making early diagnosis a challenge.³⁰ Meniere disease has an annual incidence of 4.3 per 100,000 persons, a prevalence of 17 to 46 per 100,000 persons, and a peak age of onset of 40 to 60 years.²⁹

Table 2. Medications Associated with Tinnitus

<i>Drug class</i>	<i>Specific agents</i>	<i>Comments</i>
Anti-inflammatory agents	Aspirin	Especially doses > 2.7 g per day
	Nonsteroidal anti-inflammatory drugs	All agents implicated
	Sulfasalazine (Azulfidine)	—
Antimalarial agents	Quinine and chloroquine (Aralen)	—
Antimicrobial agents	Aminoglycosides	Tinnitus usually is an initial sign of ototoxicity, often with rapid progression to hearing loss; hearing loss or tinnitus that persists two to three weeks after discontinuing the drug is likely permanent
	Macrolides	Erythromycin: rare at dosages < 2 g per day
	Tetracyclines: doxycycline, minocycline (Minocin)	Higher risk in women; symptom onset one to three days after initiation of therapy
	Vancomycin	Rare if not used with other ototoxic agents
	Other anti-infectives: imipenem/cilastatin (Primaxin), linezolid (Zyvox), sulfonamides, fluoroquinolones, voriconazole (Vfend), amphotericin B, ganciclovir (Cytovene), ribavirin (Rebetol)	—
Antineoplastic agents	Vinca alkaloids, etoposide, protein kinase inhibitors, platinum derivatives	—
Loop diuretics	All agents implicated	Highest risk with high doses and/or rapid infusions
Miscellaneous agents	Antiarrhythmics, anticonvulsants, antihypertensives, antiulcer drugs, hormones, psychotropic drugs, atorvastatin (Lipitor), bupropion (Wellbutrin), risedronate (Actonel), varenicline (Chantix)	—
Regional anesthetics	Lidocaine (Xylocaine), bupivacaine (Marcaine)	—
Topical agents	Topical otic preparations containing ototoxic drugs	—

Information from references 25 and 26.

presenting most commonly as progressive unilateral or asymmetric hearing loss, with or without tinnitus, vertigo, or both.³²⁻³⁴ It is rare for tinnitus to be the only symptom.³⁵ Only 1% to 2% of patients presenting with the classic symptoms of unilateral hearing loss with or without unilateral tinnitus have a schwannoma.³⁶ The incidence is about one per 100,000 persons in the United States,³⁷ although it has been increasing over the past several decades, in part because of better detection.³⁸ Although some investigators have speculated that cellular telephone use has contributed to this apparent increase,³⁹ studies do not show a consistent association.

Tinnitus is associated with increased prevalence of depression and anxiety; however, the precise timing and sequence of the relationship is unclear.^{40,41} There is no evidence that affective disorders cause tinnitus, but anxiety or depression may alter a patient's toleration of tinnitus or be exacerbated by tinnitus.^{40,41} Individual personality factors, such as being more socially

withdrawn, less tolerant of stress, and more likely to feel victimized by life's circumstances, were associated with longer duration of and greater annoyance from tinnitus in a New Zealand cohort.⁴²

Clinical Diagnosis

HISTORY AND PHYSICAL EXAMINATION

History and physical examination are the primary diagnostic tools for tinnitus. *Table 3* lists historical items that are helpful in the evaluation of a patient with tinnitus.^{2-4,6,11,13,16,25,27,30-32,43-49} Key elements of the physical examination include the head, eyes, ears, nose, throat, neck, and neurologic system. *Table 4* provides physical examination findings that are helpful in the evaluation of a patient with tinnitus.^{2,3,10,11,32,35,45,50,51}

ANCILLARY STUDIES, IMAGING, AND DIAGNOSTIC TESTS

Figure 1 is a suggested evaluation algorithm based on tinnitus chronicity, accompanying features, and

Table 3. Patient History Findings for Evaluating Tinnitus

General category	Finding	Comments
Associated events	Hearing change, previous chronic noise exposure, acoustic trauma, otitis media, head or neck trauma, dental treatment	Hearing loss and noise exposure are the most consistent risk factors associated with tinnitus ^{3,16} Head injury, ⁴³ whiplash, ³ dizziness, ³ and otosclerosis ⁴ are risk factors for incident tinnitus Dental treatment may produce somatosensory tinnitus via temporomandibular joint disorders or neck stress
	Use of a medication known to cause tinnitus	More than 130 agents are reported to cause tinnitus or hearing loss (Table 2) ²⁵
Associated symptoms	Headaches	Spontaneous intracranial hypotension can cause tinnitus with orthostatic headache, ¹¹ whereas obesity, headache, and tinnitus suggest idiopathic intracranial hypertension ^{2,13} ; both are indications for neuroimaging, lumbar puncture, or myelography ^{2,11}
	Hearing loss	Most common risk factor for tinnitus, occurring in at least one-third of patients ³
	Noise annoyance, intolerance, or pain	Hyperacusis is present in up to 40% of patients with tinnitus ⁴⁴
	Temporomandibular joint or neck pain	May be associated with tinnitus ²⁷ ; therapy for pain may improve tinnitus
	Vertigo	Suggests Meniere disease especially if unilateral, episodic, and associated with hearing loss, ^{30,31} although vestibular schwannoma can also produce vertigo ⁴⁵
Description of tinnitus	Fluctuation, pitch, quality, loudness	Ringling, buzzing, or cicada-like sound is most common ⁴⁶ ; low frequency most typical of Meniere disease ³⁰ ; persons with unilateral pulsatile tinnitus are 80 times more likely to have a vascular loop adjacent to cranial nerve VIII than those without that symptom ⁴⁷
Family history of tinnitus, hearing loss, or neurofibromatosis	—	Meniere disease and otosclerosis are heritable, but overall, tinnitus has a very small genetic component ⁴⁸
Impact of tinnitus	Percent of time the patient is aware of or annoyed by tinnitus (e.g., interference with daily activities, sleep, work, or leisure; auditory perceptual difficulties; effects on general health ⁶)	Helps assess severity and subjective impact of tinnitus
Location	Unilateral or bilateral	Two-thirds of patients have bilateral tinnitus ⁴⁶ ; unilateral is more likely somatosensory, vestibular schwannoma, or Meniere disease
Onset	Gradual or abrupt	Abrupt onset is more likely with somatosensory etiology and less likely to be vestibular schwannoma ³²
Timing	Intermittent or continuous	—

Information from references 2 through 4, 6, 11, 13, 16, 25, 27, 30 through 32, and 43 through 49.

examination findings. Almost all patients presenting with persistent tinnitus should undergo pure tone audiometry with assessment of air and bone conduction, speech discrimination testing, and tympanometry.^{34,49} Table 5 provides sensitivity and specificity data for several ancillary and imaging studies in the evaluation of patients with tinnitus.^{29,33,36,52,53} The optimal definition for asymmetric hearing loss has not been studied by randomized

controlled trials linked to long-term patient outcomes. A recent cross-sectional study of all published definitions of asymmetric hearing loss found an average difference of 10 dB or greater over 1 to 8 kHz had the best sensitivity for vestibular schwannoma. An average difference of 15 dB or greater over 0.5 to 3 kHz maximized specificity.³⁶ Previously, the auditory brainstem response (ABR) test has been used to screen for vestibular schwannoma

Table 4. Physical Examination Findings for Evaluating Tinnitus

General category	Finding	Comments
Ear	Cerumen impaction	Removing cerumen aids examination, and may have a therapeutic benefit in tinnitus ⁵⁰ Cerumen removal is infrequently associated with development of tinnitus ¹⁰
	Effusion, cholesteatoma, or retrotympenic lesion	Physical cause for hearing loss is the most common risk factor for tinnitus ³
Eye	Papilledema or visual field changes	Increased intracranial pressure may suggest mass or idiopathic intracranial hypertension
Musculoskeletal	Change in perception of tinnitus during teeth grinding, side-to-side or resisted head twisting	Diminished or enhanced tinnitus perception with these maneuvers suggests a somatosensory component
	Neck tenderness or limited range of motion	Tenderness in these areas may suggest a somatosensory component and need for dental or otolaryngology referral
	Temporomandibular joint tenderness, pain, or crepitus with motion	
	Tenderness of mastication muscles	
Neurologic	Abnormal cranial nerve testing Abnormal equilibrium, finger-to-nose test, or dysdiadochokinesia	Increased likelihood for vestibular schwannoma ^{32,35,45} ; can be associated with intracranial hypertension ² or hypotension ¹¹
Vascular	Bruits or murmurs over the ear canal, periauricular area, orbits, neck, and chest	May suggest etiology in patients with pulsatile tinnitus ⁵¹
	Change in tinnitus with light pressure to the ipsilateral internal jugular vein	In patients with pulsatile tinnitus, resolution of the tinnitus with this maneuver suggests venous pulsatile tinnitus ²

Information from references 2, 3, 10, 11, 32, 35, 45, 50, and 51.

in patients with asymmetric sensorineural hearing loss, unilateral tinnitus or hearing difficulties, symptoms of vestibular dysfunction, or abnormal neurologic examination.³⁵ The ABR test uses a series of clicks to stimulate the ear and then monitors for afferent nerve propagation of the resulting signal.³³ However, a systematic review of magnetic resonance imaging (MRI) for the evaluation of suspected vestibular schwannoma concluded that MRI superseded ABR testing.³³ Most U.S. otolaryngologists order a contrast-enhanced MRI of the brain and internal auditory canals in the setting of asymmetric hearing loss to rule out vestibular schwannoma. Conversely, a systematic review from the United Kingdom concluded that non-contrast-enhanced T2- and T2*-weighted MRI was the most cost-effective initial examination second to abnormal audiometry because its sensitivity was expected to be comparable with locally available contrast-enhanced MRI studies.³³ Similar reviews have not been performed in the United States. In patients unable to undergo MRI, ABR testing or computed tomography is an acceptable alternative.³³

For patients with suspected Meniere disease, vestibular testing with electronystagmography, in addition to audiometry and neuroimaging, can help exclude other vestibular disorders. Electronystagmography is a

battery of four tests designed to record eye movements in response to visual or vestibular stimuli, thus assessing the peripheral vestibular system. The most common electronystagmography finding in patients with Meniere disease is unilateral vestibular hypofunction.²⁹ No single test confirms a diagnosis of Meniere disease. Rather, the diagnosis is based on a history of fluctuating episodes of tinnitus, hearing loss, aural pressure, vertigo, or a combination of these symptoms.

In patients with pulsatile tinnitus, the choice of imaging test depends on whether arterial or venous tinnitus is suspected. In addition to audiometry, patients with suspected arterial pulsatile tinnitus, retrotympenic lesions, or arterial bruits in the head should be assessed by head and neck computed tomography angiography.⁵¹ An arterial bruit confined to the neck can be assessed using carotid Doppler ultrasonography, neck computed tomography angiography, or magnetic resonance angiography.⁵¹ Suspected venous pulsatile tinnitus requires MRI, magnetic resonance venography, and, in patients with suspected idiopathic intracranial hypertension, a lumbar puncture and measurement of cerebrospinal fluid pressure.⁵¹ In a case series of 145 patients with pulsatile tinnitus, the most common diagnoses were idiopathic intracranial hypertension, atherosclerotic carotid

artery disease, and glomus tumors; these diagnoses accounted for two-thirds of all patients in the study.⁵⁴

Laboratory tests seldom reveal a treatable cause of tinnitus, and little existing evidence supports their use. Laboratory testing should be guided by clinical suspicion rather than the presence of tinnitus because the diagnostic yield is likely to be low. For patients with asymmetric sensorineural hearing loss and tinnitus, syphilis and Lyme serologies are cost-effective ways to identify treatable causes.⁵⁵

Information for Patients

A population-based study of older patients found that tinnitus persisted in more than 80% of individuals during a follow-up of five years and increased in severity in nearly 50%.⁵ However, most of the relatively small number of persons with “very annoying” tinnitus at baseline expressed less annoyance at five years, suggesting improved coping over time.⁵ Because many patients with chronic tinnitus do not have a specific etiology beyond presbycusis,⁴⁴ supportive counseling can help

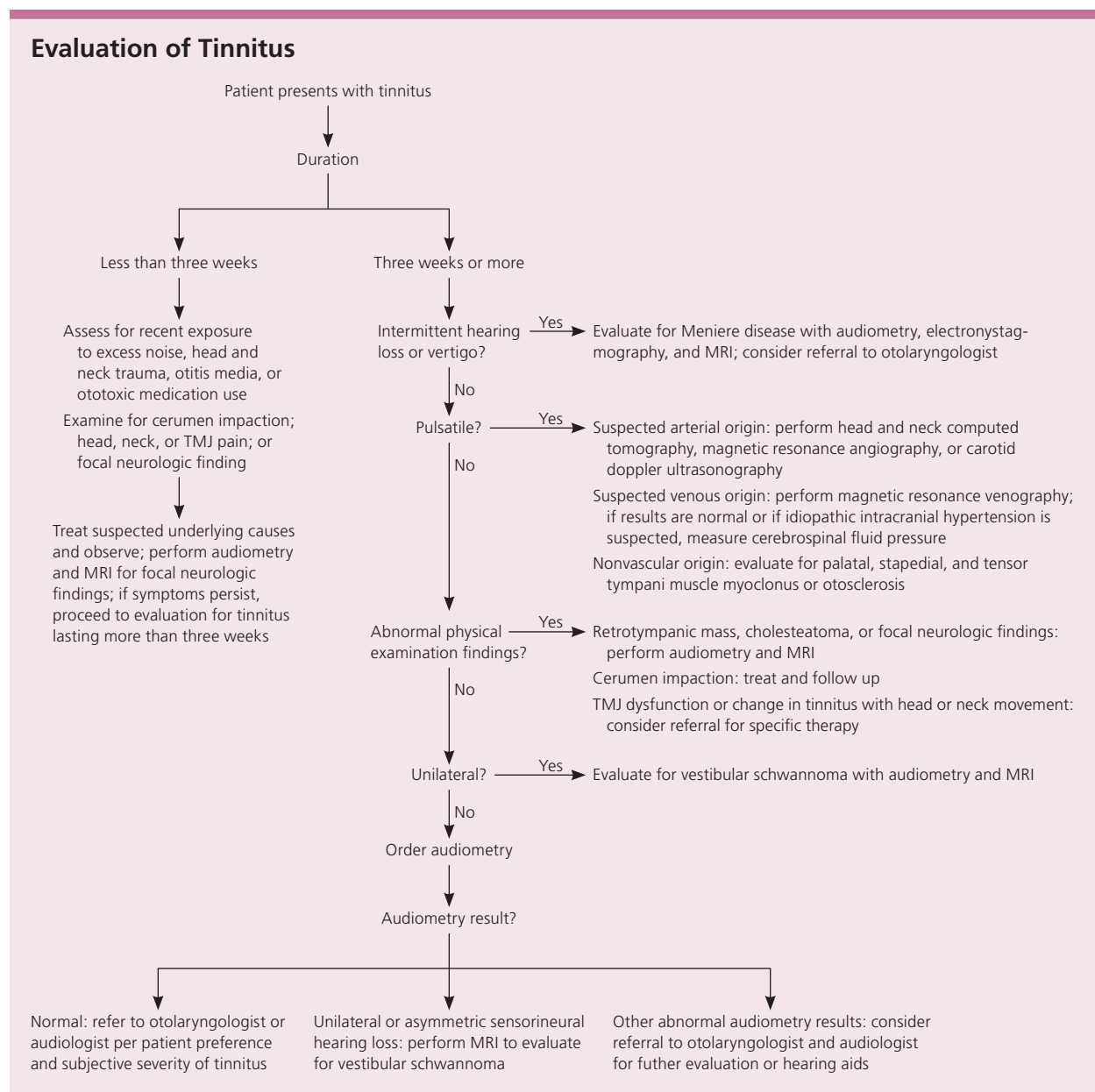


Figure 1. Algorithm for the evaluation of tinnitus. (MRI = magnetic resonance imaging; TMJ = temporomandibular joint.)

Table 5. Ancillary and Specialized Tests for Evaluating Tinnitus

Test	Description	Sensitivity (95% confidence interval)	Specificity (95% confidence interval)
Auditory brainstem response	Measures propagation of sound stimuli along acoustic nerve	For vestibular schwannoma: 85% (82% to 87%) ³³	For vestibular schwannoma: 77% (73% to 81%) ³³
Contrast-enhanced MRI	—	For vestibular schwannoma: 100% ³³	For vestibular schwannoma: near 100% ⁵²
MRI	—	For vestibular schwannoma: T2-weighted MRI: 98% (94% to 99%) ³³ T2*-weighted MRI: 96% (86% to 99%) ³³	For vestibular schwannoma: T2-weighted MRI: 90% to 100% ³³ T2*-weighted MRI: 86% to 99% ³³
Pure tone audiometry	Usually measures air and bone conduction between 250 and 8,000 Hz In Meniere disease, hearing loss is typically "upsloping," affecting low frequencies (below 2,000 Hz) ²⁹	For vestibular schwannoma: 92% ³⁶	For vestibular schwannoma: 45% ³⁶
Speech discrimination	Scored recognition of phonetically balanced words	For vestibular schwannoma: 45% ⁵³	—
Vestibular testing	Electronystagmography	For Meniere disease: 50% ²⁹	—

MRI = magnetic resonance imaging.

Information from references 29, 33, 36, 52, and 53.

patients understand the generally benign nature of tinnitus and learn to cope with it. Counseling can also support subsequent therapeutic efforts. In those with intractable and life-altering tinnitus, several otolaryngology and audiology programs offer clinics for learning coping techniques.

Data Sources: A PubMed search was completed in Clinical Queries with the term tinnitus. The search included meta-analyses, randomized controlled trials, clinical trials, and reviews. Essential Evidence was also searched for the terms Meniere disease, tinnitus, and acoustic neuroma. Using the key word tinnitus, a search was performed in Evidence Based Medicine, Bandolier, Cochrane Database of Systematic Reviews, ACP Journal Club, and Database of Abstracts of Reviews of Effectiveness (DARE), Effective Health Care, Institute for Clinical Systems Improvement guidelines, and the National Guideline Clearinghouse. Also searched in PubMed Clinical Queries Diagnosis were the terms acoustic neuroma and Meniere disease. Additionally, a full PubMed search was performed for tinnitus crossed with benign intracranial hypertension, diabetes, thyroid disease, hyperlipidemia, and vitamin B₁₂ deficiency. Search dates: June and July 2010, and July 2013.

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REFERENCES

- Folmer RL, Martin WH, Shi Y. Tinnitus: questions to reveal the cause, answers to provide relief. *J Fam Pract.* 2004;53(7):532-540.
- Sismanis A. Pulsatile tinnitus. *Otolaryngol Clin North Am.* 2003;36(2):389-402, viii.
- Gopinath B, McMahon CM, Rochtchina E, Karpa MJ, Mitchell P. Risk factors and impacts of incident tinnitus in older adults. *Ann Epidemiol.* 2010;20(2):129-135.
- Nondahl DM, Cruickshanks KJ, Wiley TL, Klein R, Klein BE, Tweed TS. Prevalence and 5-year incidence of tinnitus among older adults: the epidemiology of hearing loss study. *J Am Acad Audiol.* 2002;13(6):323-331.
- Gopinath B, McMahon CM, Rochtchina E, Karpa MJ, Mitchell P. Incidence, persistence, and progression of tinnitus symptoms in older adults: the Blue Mountains Hearing Study. *Ear Hear.* 2010;31(3):407-412.
- McCombe A, Baguley D, Coles R, McKenna L, McKinney C, Windle-Taylor P. Guidelines for the grading of tinnitus severity: the results of a working group commissioned by the British Association of Otolaryngologists, Head and Neck Surgeons, 1999. *Clin Otolaryngol Allied Sci.* 2001;26(5):388-393.
- Møller AR. Tinnitus: presence and future. *Prog Brain Res.* 2007;166:3-16.
- Tucker DA, Phillips SL, Ruth RA, Clayton WA, Royster E, Todd AD. The effect of silence on tinnitus perception. *Otolaryngol Head Neck Surg.* 2005;132(1):20-24.

9. Rocha CA, Sanchez TG. Myofascial trigger points: another way of modulating tinnitus. *Prog Brain Res*. 2007;166:209-214.
10. Folmer RL, Shi BY. Chronic tinnitus resulting from cerumen removal procedures. *Int Tinnitus J*. 2004;10(1):42-46.
11. Schievink WI. Spontaneous spinal cerebrospinal fluid leaks and intracranial hypotension. *JAMA*. 2006;295(19):2286-2296.
12. Albers FW, Ingels KJ. Otoneurological manifestations in Chiari-I malformation. *J Laryngol Otol*. 1993;107(5):441-443.
13. Jindal M, Hiam L, Raman A, Rejali D. Idiopathic intracranial hypertension in otolaryngology. *Eur Arch Otorhinolaryngol*. 2009;266(6):803-806.
14. Neff BA, Staab JP, Eggers SD, et al. Auditory and vestibular symptoms and chronic subjective dizziness in patients with Meniere's disease, vestibular migraine, and Meniere's disease with concomitant vestibular migraine. *Otol Neurotol*. 2012;33(7):1235-1244.
15. Crummer RW, Hassan GA. Diagnostic approach to tinnitus. *Am Fam Physician*. 2004;69(1):120-126.
16. Sindhusake D, Golding M, Newall P, Rubin G, Jakobsen K, Mitchell P. Risk factors for tinnitus in a population of older adults: the blue mountains hearing study. *Ear Hear*. 2003;24(6):501-507.
17. Shemesh Z, Attias J, Ornan 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.
18. Cochran JH Jr, Kosmicki PW. Tinnitus as a presenting symptom in pernicious anemia. *Ann Otol Rhinol Laryngol*. 1979;88(2 pt 1):297.
19. Gosselin EJ, Yanick P Jr. Audiologic and metabolic findings in 90 patients with fluctuant hearing loss. *J Am Audiol Soc*. 1976;2(1):15-18.
20. Kazmierczak H, Doroszevska G. Metabolic disorders in vertigo, tinnitus, and hearing loss. *Int Tinnitus J*. 2001;7(1):54-58.
21. Klagenberg KF, Zeigelboim BS, Jurkiewicz AL, Martins-Bassetto J. Vestibulocochlear manifestations in patients with type I diabetes mellitus. *Braz J Otorhinolaryngol*. 2007;73(3):353-358.
22. Pessin AB, Martins RH, Pimenta Wde P, Simões AC, Marsiglia A, Amaral AV. Auditory evaluation in patients with type 1 diabetes. *Ann Otol Rhinol Laryngol*. 2008;117(5):366-370.
23. Pulec JL, Pulec MB, Mendoza I. Progressive sensorineural hearing loss, subjective tinnitus and vertigo caused by elevated blood lipids [published correction appears in *Ear Nose Throat J*. 1998;77(2):145]. *Ear Nose Throat J*. 1997;76(10):716-720, 725-726, 728 passim.
24. Sutbas A, Yetiser S, Satar B, Akcam T, Karahatay S, Saglam K. Low-cholesterol diet and antilipid therapy in managing tinnitus and hearing loss in patients with noise-induced hearing loss and hyperlipidemia. *Int Tinnitus J*. 2007;13(2):143-149.
25. Seligmann H, Podoshin L, Ben-David J, Fradis M, Goldsher M. Drug-induced tinnitus and other hearing disorders. *Drug Saf*. 1996;14(3):198-212.
26. Cianfrone G, Pentangelo D, Cianfrone E, et al. Pharmacological drugs inducing ototoxicity, vestibular symptoms and tinnitus: a reasoned and updated guide. *Eur Rev Med Pharmacol Sci*. 2011;15(6):601-636.
27. Bernhardt O, Gesch D, Schwahn C, et al. Signs of temporomandibular disorders in tinnitus patients and in a population-based group of volunteers: results of the Study of Health in Pomerania. *J Oral Rehabil*. 2004;31(4):311-319.
28. Pinchoff RJ, Burkard RF, Salvi RJ, Coad ML, Lockwood AH. Modulation of tinnitus by voluntary jaw movements. *Am J Otol*. 1998;19(6):785-789.
29. Sajjadi H, Paparella MM. Meniere's disease. *Lancet*. 2008;372(9636):406-414.
30. Havia M, Kentala E, Pyykkö I. Hearing loss and tinnitus in Meniere's disease. *Auris Nasus Larynx*. 2002;29(2):115-119.
31. Committee on Hearing and Equilibrium guidelines for the diagnosis and evaluation of therapy in Meniere's disease. American Academy of Otolaryngology-Head and Neck Foundation, Inc. *Otolaryngol Head Neck Surg*. 1995;113(3):181-185.
32. Baguley DM, Humphriss RL, Axon PR, Moffat DA. The clinical characteristics of tinnitus in patients with vestibular schwannoma. *Skull Base*. 2006;16(2):49-58.
33. Fortnum H, O'Neill C, Taylor R, et al. The role of magnetic resonance imaging in the identification of suspected acoustic neuroma: a systematic review of clinical and cost effectiveness and natural history. *Health Technol Assess*. 2009;13(18):iii-iv, ix-xi, 1-154.
34. Saliba I, Martineau G, Chagnon M. Asymmetric hearing loss: rule 3,000 for screening vestibular schwannoma. *Otol Neurotol*. 2009;30(4):515-521.
35. Lustig LR, Rifkin S, Jackler RK, Pitts LH. Acoustic neuromas presenting with normal or symmetrical hearing: factors associated with diagnosis and outcome. *Am J Otol*. 1998;19(2):212-218.
36. Cheng TC, Wareing MJ. Three-year ear, nose, and throat cross-sectional analysis of audiometric protocols for magnetic resonance imaging screening of acoustic tumors. *Otolaryngol Head Neck Surg*. 2012;146(3):438-447.
37. Propp JM, McCarthy BJ, Davis FG, Preston-Martin S. Descriptive epidemiology of vestibular schwannomas. *Neuro Oncol*. 2006;8(1):1-11.
38. Tos M, Stangerup SE, Cayé-Thomasen P, Tos T, Thomsen J. What is the real incidence of vestibular schwannoma? *Arch Otolaryngol Head Neck Surg*. 2004;130(2):216-220.
39. Hardell L, Carlberg M, Söderqvist F, Mild KH. Pooled analysis of case-control studies on acoustic neuroma diagnosed 1997-2003 and 2007-2009 and use of mobile and cordless phones. *Int J Oncol*. 2013;43(4):1036-1044.
40. Guitton MJ. Tinnitus and anxiety: more than meets the ear. *Curr Psychiatry Rev*. 2006;2(3):333-338.
41. Holmes S, Padgham ND. Review paper: more than ringing in the ears: a review of tinnitus and its psychosocial impact. *J Clin Nurs*. 2009;18(21):2927-2937.
42. Welch D, Dawes PJ. Personality and perception of tinnitus. *Ear Hear*. 2008;29(5):684-692.
43. Nondahl DM, Cruickshanks KJ, Wiley TL, et al. The ten-year incidence of tinnitus among older adults. *Int J Audiol*. 2010;49(8):580-585.
44. Lloyd SK, Baguley DM. A patient with tinnitus. *Clin Otolaryngol*. 2008;33(1):25-28.
45. Kentala E, Pyykkö I. Clinical picture of vestibular schwannoma. *Auris Nasus Larynx*. 2001;28(1):15-22.
46. Sindhusake D, Mitchell P, Newall P, Golding M, Rochtchina E, Rubin G. Prevalence and characteristics of tinnitus in older adults: the Blue Mountains Hearing Study. *Int J Audiol*. 2003;42(5):289-294.
47. Chadha NK, Weiner GM. Vascular loops causing otological symptoms: a systematic review and meta-analysis. *Clin Otolaryngol*. 2008;33(1):5-11.
48. Kvestad E, Czajkowski N, Engdahl B, Hoffman HJ, Tambs K. Low heritability of tinnitus: results from the second Nord-Trøndelag health study. *Arch Otolaryngol Head Neck Surg*. 2010;136(2):178-182.
49. Langguth B, Goodey R, Azevedo A, et al. Consensus for tinnitus patient assessment and treatment outcome measurement: Tinnitus Research Initiative meeting, Regensburg, July 2006. *Prog Brain Res*. 2007;166:525-536.
50. Goodey R. Tinnitus treatment: state of the art. *Prog Brain Res*. 2007;166:237-246.
51. Sismanis A, Girevendoulis A. Pulsatile tinnitus associated with internal carotid artery morphologic abnormalities. *Otol Neurotol*. 2008;29(7):1032-1036.
52. House JW, Bassim MK, Schwartz M. False-positive magnetic resonance imaging in the diagnosis of vestibular schwannoma. *Otol Neurotol*. 2008;29(8):1176-1178.
53. Ferguson MA, Smith PA, Lutman ME, Mason SM, Coles RR, Gibbin KP. Efficiency of tests used to screen for cerebello-pontine angle tumours: a prospective study. *Br J Audiol*. 1996;30(3):159-176.
54. Sismanis A. Pulsatile tinnitus. A 15-year experience. *Am J Otol*. 1998;19(4):472-477.
55. Wilson YL, Gandolfi MM, Ahn IE, Yu G, Huang TC, Kim AH. Cost analysis of asymmetric sensorineural hearing loss investigations. *Laryngoscope*. 2010;120(9):1832-1836.