

# Tinnitus: Questions to reveal the cause, answers to provide relief

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## Practice recommendations

- If no treatable cause of tinnitus is found, assess the severity of tinnitus, secondary problems (such as depression, anxiety, and insomnia), and implement tinnitus management strategies (SOR: **B**).
- Acoustic therapy is effective for tinnitus management (SOR: **B**).
- All patients should wear hearing protection when they are exposed to loud sounds such as a gas lawnmower, leaf blower, power tools, or gunfire (SOR: **A**).
- Successful management of insomnia, anxiety or depression will decrease the severity of tinnitus for most patients (SOR: **B**).

**T**innitus—the perception of sound that does not have an external source—can be constant or intermittent and perceived as ringing, buzzing, hissing, sizzling, roaring, chirping, or other sounds.

Acute tinnitus, which can last days or weeks, may be caused by ear infection, medications, head or neck injury, excessive sound exposure, earwax, and changes in blood pressure or metabolism. With appropriate evaluation, such underlying conditions usually can be identified and treated, often with resultant resolution of tinnitus.

Chronic tinnitus (persistence for 6 months or more) can also result from these conditions and is more likely to occur in people who have hearing loss.<sup>1</sup> (See **Prevalence of tinnitus**.) Even though a true “cure” for most cases of chronic tinnitus is not available, patients can obtain relief from the symptom with assistance from clinicians who are familiar with tinnitus management strategies.

## ■ DAMAGE DEPENDS ON INTENSITY, LENGTH OF EXPOSURE

Tinnitus is most commonly caused by exposure to excessively loud sounds such as gunfire, power tools, machinery, or music. Ringing in the ears occurs because of damage to stereocilia, microscopic appendages attached to the apical ends of hair cells in the cochlea.

Moderate sounds (80 decibels sound pressure level [dB SPL] or lower) normally cause stereocilia to make tiny movements, triggering the release of neurotransmitter molecules from the basal

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ends of hair cells that activate auditory neurons in the eighth cranial nerve.

Excessive sound exposure (85 dB SPL or louder) causes stereocilia to bend more than they should. People then perceive high-pitched ringing tinnitus because hair cells that respond to higher-frequency sounds are located at the base of the cochlea and are the first to be damaged by loud noise.

If the damage is modest and infrequent, stereocilia can recover, returning to their normal function in a few minutes or hours. The patient's hearing will be restored and the tinnitus will stop. However, repeated exposure to hazardous sounds eventually causes irreparable damage to stereocilia and hair cells, resulting in permanent sensorineural hearing loss and possibly chronic tinnitus.

In addition to noise exposure, any condition that causes hearing loss or damages the auditory system can contribute to the generation of tinnitus (**Table**). Imaging studies using functional magnetic resonance imaging<sup>7</sup> or positron-emission tomography<sup>8-9</sup> demonstrated that the perception of chronic tinnitus usually occurs as a result of hyperactivity within central auditory areas of the human brain, especially the auditory cortex. As portions of the auditory system degenerate during the aging process or acquire damage from noise exposure, disease, and accidents, the natural balance of central auditory excitation vs inhibition is disrupted. In patients who hear tinnitus, excitatory pathways within the auditory system are active when they shouldn't be: in quiet environments. This gives patients the perception of tinnitus sounds.

### Objective tinnitus

Objective tinnitus—which can be heard also by people in proximity to the patient's ear—can be caused by vascular abnormalities (congenital arteriovenous fistula, acquired arteriovenous shunt, glomus jugulare, high-riding carotid artery, carotid stenosis, persistent stapedial artery, dehiscent jugular bulb or a vascular loop

## Prevalence of tinnitus

Seidman and Jacobson<sup>2</sup> estimated that 40 million people in the United States experience chronic tinnitus. The prevalence of tinnitus increases with age: 27% of males and 15% of females aged 45 years or older experience the symptom.<sup>3</sup>

Tinnitus is rare in children who have normal hearing.<sup>4</sup> However, the prevalence of tinnitus in children with severe or profound hearing loss has been reported as 33%<sup>5</sup> or 64%.<sup>6</sup> More males than females experience tinnitus because men traditionally have had a greater amount of noise exposure in military service, in the workplace, and during recreational activities. Consequently, hearing loss and tinnitus are both more prevalent among men aged 45 years or older compared with women in the same age group.<sup>3</sup>

such as anterior inferior communicating artery [AICA] or posterior inferior communicating artery [PICA] compressing the auditory nerve) or mechanical disorders (abnormally patent Eustachian tube, palatal myoclonus, temporomandibular joint disorder, or stapedial muscle spasticity).<sup>10</sup> However, objective tinnitus is rare, accounting for <1% of all cases. The vast majority of tinnitus cases are subjective—sounds are perceived only by the patient.

## ■ PATIENT EVALUATION

**Figure 1** is an algorithm that outlines steps in the evaluation and management of patients who experience tinnitus. The first step is to collect as much information as possible about the patient and his condition.

### Tinnitus history

Determine the duration of tinnitus and whether circumstances such as upper respiratory infection, otalgia, noise exposure, head trauma,

TABLE

**Causes of subjective tinnitus**

Presbycusis: hearing loss due to aging
Prolonged noise exposure: Noise-induced hearing loss
Acoustic trauma: one-time exposure to high intensity sound
Otosclerosis: abnormal accumulation of calcium on middle ear ossicles or cochlea
Infections: bacterial, viral, fungal
Autoimmune hearing loss
Meniere's disease or endolymphatic hydrops: abnormally high inner ear pressure
Neoplasms: for example, acoustic neuroma or cholesteatoma
Genetic predisposition
Ototoxicity <ul style="list-style-type: none"> <li>– Medications: aminoglycoside antibiotics (such as gentamicin); valproate; quinine; cisplatin; loop diuretics (such as furosemide)</li> <li>– Heavy metals such as lead</li> </ul>
Vascular <ul style="list-style-type: none"> <li>– Hypertension; arteriosclerosis; cerebral aneurysm; cerebrovascular accident</li> </ul>
Metabolic <ul style="list-style-type: none"> <li>– Anemia; hypothyroidism; hyperthyroidism; diabetes mellitus</li> </ul>
Head or neck injury

sudden hearing loss, or vertigo occurred at the time of tinnitus onset. Ask the patient to describe the tinnitus: Is it intermittent or constant? High- or low-pitched? Unilateral or bilateral? Pulsatile or steady?

Unilateral tinnitus and hearing loss provide preliminary evidence for acoustic neuroma or cerebrovascular accident. High-pitched tinnitus is usually associated with high-frequency hearing loss caused by presbycusis (hearing impairment in the aged) or excessive noise exposure. Low-pitched roaring tinnitus is sometimes associated

with low-frequency hearing loss exhibited by patients with Meniere's disease. Pulsatile tinnitus, especially if synchronous with the patient's pulse, can indicate vascular abnormalities.

Ask the patient if fatigue, stress, noise exposure, or any medications exacerbate the tinnitus. Also ask if masking sounds (such as water running in the shower), medications, or any other factors provide relief from tinnitus. This information can be used to formulate a tinnitus management program.

Assess the severity of the patient's tinnitus using an instrument such as the Tinnitus Severity Index (**Figure 2**).<sup>11</sup> A score of 36 or higher indicates bothersome tinnitus (level of evidence [LOE]: 2).<sup>12</sup> Higher scores indicate that patients perceive their tinnitus to be a significant, even debilitating problem.

**Hearing history**

If possible, determine the presence and type of hearing loss (congenital, sudden, sensorineural, conductive, or mixed). Note the patient's history of ear infections, surgeries, noise exposure (occupational or recreational), otalgia, otorrhea, and vertigo or other balance problems. Ask whether immediate family members have experienced hearing loss or tinnitus.

**Health history**

Look particularly for conditions that can contribute to hearing loss and tinnitus, such as hypertension, hypothyroidism, diabetes mellitus, arteriosclerosis, and autoimmune disorders (eg, lupus or rheumatoid arthritis). Also consider ototoxic medications, such as aminoglycoside antibiotics, cisplatin, furosemide, valproic acid, and high doses of quinine-containing compounds. When possible, patients with hearing loss or tinnitus should be given alternative medications free from ototoxicity.

Excessive use of alcohol, caffeine, and aspirin or other nonsteroidal anti-inflammatory drugs can exacerbate tinnitus for some patients. However, moderate use of these products is often possible.

**FIGURE 1**

**Evaluation and management of tinnitus**

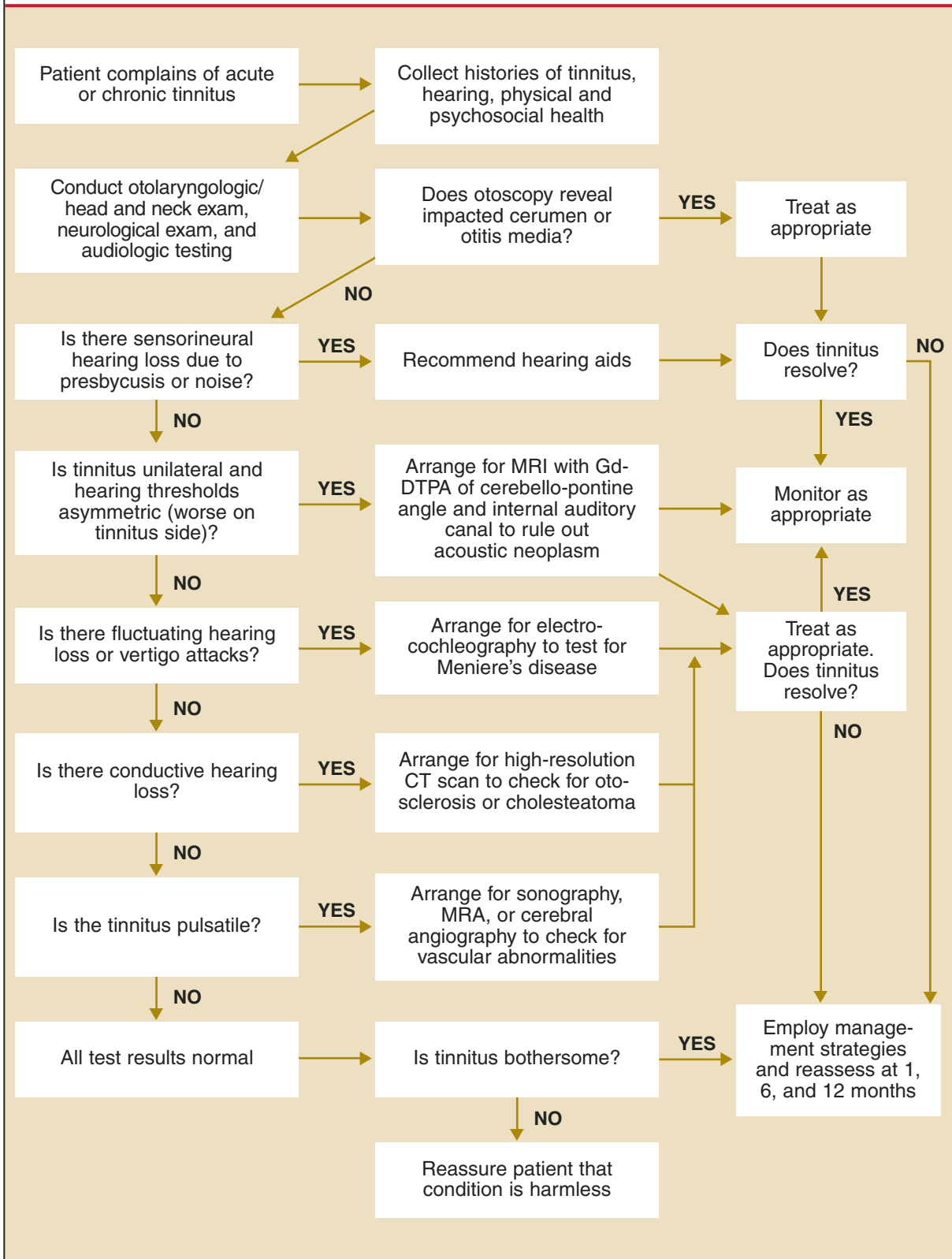


FIGURE 2

## Tinnitus Severity Index

DIRECTIONS: For the questions below, please CIRCLE the number that best describes you

Does your tinnitus	Never	Rarely	Sometimes	Usually	Always
1. Make you feel irritable or nervous?	1	2	3	4	5
2. Make you feel tired or stressed?	1	2	3	4	5
3. Make it difficult for you to relax?	1	2	3	4	5
4. Make it uncomfortable to be in a quiet room?	1	2	3	4	5
5. Make it difficult to concentrate?	1	2	3	4	5
6. Make it harder to interact pleasantly with others?	1	2	3	4	5
7. Interfere with your <i>required</i> activities? (Work, home, care, or other responsibilities)	1	2	3	4	5
8. Interfere with your social activities or other things you do in your leisure time?	1	2	3	4	5
9. Interfere with your overall enjoyment of life?	1	2	3	4	5
10. Interfere with your ability to sleep?	1	2	3	4	5
11. How often do you have difficulty ignoring your tinnitus?	1	2	3	4	5
12. How often do you experience discomfort from tinnitus?	1	2	3	4	5

**Psychosocial history**

Inquire about the patient's marital and occupational status. Unemployed patients living alone often perceive tinnitus to be more severe than do employed patients who have supportive social networks. Also ask about any history of insomnia, anxiety, depression, obsessive-compulsive disorder, or psychosis. A questionnaire such as the abbreviated Beck Depression Inventory<sup>13</sup> can be used to assess the presence and severity of depression.

**Physical exam and testing**

Patient evaluation should include the following physical examinations and tests.

**Otolaryngologic/head and neck exam.**

Otoscopic examination can detect infections such as otitis media, which will usually be accompa-

nied by complaints of ear pain or fullness, and possibly hearing loss in combination with tinnitus. Otoscopy can also detect impacted earwax (cerumen), which can occlude the ear canal or cause immobilization of the tympanic membrane, resulting in conductive hearing loss, tinnitus, and a feeling of fullness in the ear. Symptoms usually resolve when the earwax is removed.

If the tinnitus is synchronous with the patient's pulse, it suggests a vascular contribution for the symptom. Auscultation of blood vessels in the neck can reveal venous hums or other types of bruits audible to the patient. Venous hum can be diagnosed by temporarily blocking blood flow through the jugular vein on the side where tinnitus is perceived.

**Neurologic exam.** A complete neurologic exam should include the Romberg test, Dix-Hallpike

maneuver (if the patient experiences vertigo), gait testing, and cranial nerve function tests.

**Audiologic testing.** Audiologic tests should include pure tone air and bone conduction thresholds, speech discrimination testing, tympanometry, and most comfortable loudness (MCL) and uncomfortable loudness level (UCL) tests. Tympanometry is used to assess middle-ear function. Abnormal tympanograms and significant differences between air and bone conduction thresholds can indicate otitis media, otosclerosis, or cholesteatoma.

MCL and UCL tests are used to assess the dynamic range of patients' hearing. Patients with UCLs that are only 5 to 20 dBs above their MCLs have a reduced dynamic range of hearing that can be caused by recruitment or hyperacusis. The audiometer can also be used to match the tinnitus for pitch and loudness and to test the effects of masking sounds on the patient's tinnitus.

**Additional evaluations.** Results of patient examinations and history collection might warrant additional evaluations. For example, asymmetrical hearing loss (15 dB or greater asymmetry at 2 or more consecutive test frequencies) and unilateral tinnitus can indicate a retrocochlear lesion such as acoustic neuroma (also known as vestibular schwannoma).

One test for retrocochlear pathology is the auditory brainstem response (ABR). In this test, clicks are presented through earphones while scalp electrodes record brain responses to the sounds. Abnormal ABR waveforms can indicate retrocochlear lesion (such as acoustic neuroma) as a possible cause of ipsilateral hearing loss and tinnitus. If positive ABR results are obtained, MRI evaluation of the cerebellopontine angle with contrast material (such as gadolinium) should be performed.

Low-pitched roaring, ringing, or hissing tinnitus; hearing loss, which may be temporary or permanent; vertigo; and a feeling of pressure or fullness in the ear can indicate endolymphatic hydrops or Meniere's disease. Symptoms usually occur in the form of "attacks" that increase in fre-

quency during the first few years of the disease, then decrease in frequency as hearing thresholds stabilize. Electrocochleography testing is one way to diagnose endolymphatic hydrops. Patients who exhibit vestibular disorders should undergo electronystagmography testing to assess the severity and characteristics of their symptoms.

Pulsatile tinnitus associated with abnormalities of blood vessels in the neck can be evaluated with sonography, conventional angiography, or magnetic resonance angiography. Conditions such as a dehiscent jugular bulb or stenosis of carotid arteries can sometimes be treated surgically. However, many forms of pulsatile tinnitus are not caused by these conditions. Pulsatile tinnitus is often a consequence of hearing loss, arteriosclerosis, or weight loss or weight gain. These physiologic changes can cause patients to hear blood pulsing or "swishing" in vessels—sounds they did not perceive previously. Surgery is not recommended for most cases of pulsatile tinnitus.

Sudden hearing loss, especially if bilateral, might indicate autoimmune inner ear disease. Diagnostic tests include the Western blot immunoassay.

## ■ TREATMENT OF ACTIVE DISEASE PROCESSES

Many contributors to tinnitus can be treated surgically or with medication.

**Otitis media.** Successful treatment of the infection with oral antibiotics usually resolves all auditory symptoms.

**Allergies, sinus congestion, or infection.** When inflammation subsides, tinnitus associated with these conditions usually resolves.

**Otosclerosis.** Abnormal accumulations of calcium on middle-ear ossicles (especially the stapes) or the cochlea can result in slowly progressing conductive or sensorineural hearing loss, tinnitus, and vestibular disturbances. Stapedectomy surgery—including implantation of ossicular prostheses—is often successful for advanced cases associated with significant hearing loss. Hearing aids also benefit some patients.



**Meniere's disease or other forms of endolymphatic hydrops.** Meniere's disease, characterized by abnormally high fluid pressure within the cochlea, has an estimated prevalence of 1% in the US.<sup>14</sup> Management includes meclizine, antiemetics and diuretics, and a low-sodium diet.<sup>15</sup> If patients do not respond to meclizine, diazepam can be prescribed to reduce the severity of vertigo attacks. Surgical intervention—including installation of an endolymphatic shunt, labyrinthectomy, or vestibular neurectomy<sup>16</sup>—or trans-tympanic injections of gentamicin<sup>17</sup> are options in severe cases.

**Autoimmune inner ear disease.** This disease has an estimated prevalence of 0.1% in the US.<sup>18</sup> Symptoms include sudden hearing loss in one ear that usually progresses to the second ear. Patients may also feel fullness in the ear and experience vertigo as well as ringing, hissing, or roaring tinnitus. Most patients with autoimmune inner ear disease respond to initial treatment with oral prednisone.

**Auditory neoplasms.** Growths such as acoustic neuroma or cholesteatoma can cause tinnitus. Acoustic neuroma (or vestibular schwannoma) is a benign neoplasm that arises from the vestibular division of the eighth cranial nerve. Symptoms include unilateral hearing loss, tinnitus, and vestibular disturbances. Surgical resection or radiation treatment of the tumor can resolve these symptoms, especially if the neoplasm is detected while it is small.

Cholesteatoma is a benign epithelial cell mass that grows in the middle-ear cavity. Over time, cholesteatomas can enlarge and destroy middle-ear ossicles. Hearing loss, tinnitus, dizziness, and facial muscle paralysis can result from continued cholesteatoma growth. Early detection and surgical resection of auditory neoplasms can reduce the likelihood of residual symptoms.

**Hyper- or hypotension.** Of these two disorders, hypertension is more likely to contribute to tinnitus. Maintenance of blood pressure within the optimum range can decrease or resolve tinnitus for some patients.

**Metabolic disorders.** Disorders such as diabetes mellitus, hyperthyroidism, or hypothyroidism can contribute to tinnitus. Successful management of these conditions can reduce or resolve the patient's tinnitus.

## ■ MANAGING PERSISTENT TINNITUS

Successful treatment of the disorders discussed can resolve or reduce tinnitus. However, if tinnitus continues to bother the patient after other diseases have been treated, shift the clinical focus from treatment to management of the symptom. At this point, the clinician should do 1 of 2 things: 1) spend the time necessary to help the patient manage tinnitus using strategies described in the following sections of this article; or 2) refer the patient to a comprehensive tinnitus management program with experienced personnel who are willing and able to spend a substantial amount of time with each patient.

Like other neurologic symptoms, tinnitus can be considered chronic if it persists for 6 months or more. Approximately 90% of cases of chronic tinnitus are associated with some degree of sensorineural hearing loss.<sup>19</sup> Because sensorineural hearing loss is irreversible, most cases of chronic tinnitus cannot be "cured." Duckro et al<sup>20</sup> wrote: "As with chronic pain, the treatment of chronic tinnitus is more accurately described in terms of management rather than cure."

The goal of management is not *necessarily* to mask or remove the patient's perception of tinnitus. In many cases, this is not possible. Successful management enables patients to pay less attention to their tinnitus. An effective management program helps patients to understand and gain control over their tinnitus, rather than allowing it to control them. The ultimate goal is to reduce the *severity* of tinnitus. Clinicians should strive to help patients progress to where tinnitus is no longer a negative factor in their lives.

## Establishing tinnitus severity

Only 25% of people who experience chronic tinnitus consider the symptom to be a significant

problem.<sup>2</sup> These are the patients most likely to seek treatment. If a patient is not bothered by tinnitus and no active disease processes are detected, no treatments are necessary. The clinician should reassure such patients that tinnitus is a harmless perception of sound and does not usually portend more serious medical conditions.

What differentiates the majority of people not bothered by tinnitus from the minority who perceive it as a significant, even debilitating problem? Is it the matched loudness, pitch, or other qualities of the sound(s) they hear? Several studies have concluded that tinnitus severity is not correlated with any of these psychoacoustic parameters.<sup>21–23</sup>

Tinnitus severity can be defined and quantified several ways: by how much or how often a patient is bothered by tinnitus; by how much or how often tinnitus detracts from the patient's enjoyment of life; or by how disabling patients perceive their tinnitus to be. Instruments such as the Tinnitus Severity Index<sup>11</sup> can be used to assess tinnitus severity (Figure 2).

## ■ TINNITUS MANAGEMENT STRATEGIES

Once underlying conditions have been treated or ruled out, reassure and counsel patients regarding factors that could exacerbate or improve their condition. If patients understand their tinnitus is nothing more than a perception of sound, they will be better able to pay less attention to it. This process of patient education and counseling helps to “demystify” the symptom of tinnitus and encourages patients to view their tinnitus with a more realistic perspective.

The severity of tinnitus is often associated with problems such as insomnia,<sup>24</sup> anxiety,<sup>25</sup> and depression.<sup>26</sup> Such issues can form a vicious circle, with each one exacerbating the others.<sup>23</sup> Tinnitus is not always the starting point of this cycle—many patients experience depression, insomnia, or anxiety before tinnitus. Medication or psychotherapy will often reduce the severity of these symptoms and associated tinnitus (LOE: 2).<sup>27–28</sup>

**Because sensorineural hearing loss is irreversible, most cases of chronic tinnitus cannot be “cured”**

Because each patient has a unique medical, psychological, and social history, management programs should be individualized. In fact, the most successful tinnitus management programs employ multimodal strategies designed to address the specific needs of each patient (LOE: 2).<sup>27,29</sup>

Recommendations should be formulated and explained to the patient: appropriate acoustic therapy; use of hearing protection (all patients should wear earplugs or ear muffs when they are exposed to excessively loud sounds [LOE: 1]);<sup>30</sup> and strategies for management of insomnia, anxiety, or depression. As appropriate, provide patients with referral and contact information for physical or psychiatric evaluations, psychological counseling, and other recommended services or products.

### Acoustic therapy

Patients should add pleasant sounds (music, relaxation CDs, or a tabletop sound machine) to any environment that is too quiet, and listen to them through speakers or headphones. Patients who experience chronic insomnia because of tinnitus may find relief in using a tabletop sound machine in combination with a pillow embedded with speakers (such as the Sound Pillow, distributed by Phoenix Productions, San Antonio, TX).

Patients with normal or nearly normal hearing might benefit from in-the-ear sound generators (such as those manufactured by General Hearing Instruments, Harahan, LA) that produce a broadband sound to muffle or mask the tinnitus.<sup>31</sup> Significant, aidable hearing loss can often be lessened with hearing aids or combination instruments (hearing aid and sound generator in one unit). Hearing aids not only improve communication ability, the devices can also reduce the perception of tinnitus.<sup>32</sup>



## Follow-up

Encourage patients to ask questions about recommended tinnitus management procedures and to report their progress. Reassess patients at 1 month. If necessary, recommendations can be modified to facilitate patient improvement. Follow-up questionnaires can be mailed to patients 6 and 12 months after their initial appointment to assess the effectiveness of the tinnitus management program.

A customized combination of recommendations is effective for many patients with chronic and bothersome tinnitus (LOE: 2),<sup>27</sup> but the process can be very time consuming. For a certain number of patients with severe tinnitus, only a comprehensive management program can help them to improve their condition.

## REFERENCES

- Schleuning A. Medical aspects of tinnitus. In: Vernon JA, ed. *Tinnitus Treatment and Relief*. Boston, Mass: Allyn and Bacon; 1998:20–27.
- Seidman MD, Jacobson GP. Update on tinnitus. *Otolaryngol Clin N Amer* 1996; 29:455–465.
- Adams PF, Hendershot GE, Marano MA. Current estimates from the National Health Interview Survey, 1996. Hyattsville, Md: National Center for Health Statistics, 1999.
- Stouffer JL, Tyler RS, Booth JC, Buckrell B. Tinnitus in normal-hearing and hearing-impaired children. In: Aran J-M, Dauman R, eds. *Tinnitus 91: Proceedings of the Fourth International Tinnitus Seminar*. Amsterdam: Kugler Publications; 1992:255–258.
- Druker GS. The prevalence and characteristics of tinnitus with profound sensori-neural hearing impairment. *Amer Ann Deaf* 1989; 134:260–264.
- Graham J. Paediatric tinnitus. *J Laryngol Otol* 1981; 95(Suppl):117–120.
- Levine RA, Benson RR, Talavage TM, Melcher JR, Rosen BR. Functional magnetic resonance imaging and tinnitus: preliminary results. *Abstr Assoc Res Otolaryngol* 1997; 20:65.
- Arnold W, Bartenstein P, Oestreicher E, Romer W, Schwaiger M. Focal metabolic activation in the predominant left auditory cortex in patients suffering from tinnitus: a PET study with [18F]deoxyglucose. *ORL J Otorhinolaryngol Relat Spec* 1996; 58:195–199.
- Lockwood AH, Salvi RJ, Coad ML, Towsley ML, Wach DS, Murphy BW. The functional neuroanatomy of tinnitus: evidence for limbic system links and neuroplasticity. *Neurology* 1998; 50:114–120.
- Ciocon JO, Amede F, Lechtenberg C, Astor F. Tinnitus: a stepwise workup to quiet the noise within. *Geriatrics* 1995; 50:18–25.
- Meikle MB, Griest SE, Stewart BJ, Press LS. Measuring the negative impact of tinnitus: a brief severity index. *Abstr Assoc Res Otolaryngol* 1995;167.
- Folmer RL, Stevenson EA, Tran A. Factors associated with long-term improvements in tinnitus severity. In: Patuzzi R, ed: *Proceedings of the Seventh International Tinnitus Seminar*. Crawley: University of Western Australia; 2002:115–123.
- Beck AT, Beck RW. Screening depressed patients in family practice: a rapid technic. *Postgrad Medicine* 1972; 52:81–85.
- daCosta SS, deSousa LC, Piza MR. Meniere's Disease: overview, epidemiology, and natural history. *Otolaryngol Clin N Amer* 2002; 35:455–495.
- Thai-Van H, Bounaix MJ, Fraysse B. Meniere's disease: pathophysiology and treatment. *Drugs* 2001; 61:1089–1102.
- Hillman TA, Chen DA, Arriaga MA. Vestibular nerve section versus intratympanic gentamicin for Meniere's disease. *Laryngoscope* 2004; 114:216–222.
- Diamond C, Hornig JD, Liu R, O'Connell DA. Systematic review of intratympanic gentamicin in Meniere's disease. *J Otolaryngol* 2003; 32:351–361.
- Hain TC. Autoimmune inner ear disease. American Hearing Research Foundation Web site. March 23, 2002. Available at [www.american-hearing.org/name/autoimmune.html](http://www.american-hearing.org/name/autoimmune.html). Accessed on May 15, 2004.
- Meikle MB. Electronic access to tinnitus data: The Oregon Tinnitus Data Archive. *Otolaryngol Head Neck Surg* 1997; 117:698–700.
- Duckro PN, Pollard CA, Bray HD, Scheiter L. Comprehensive behavioral management of complex tinnitus: a case illustration. *Biofeedback and Self-Regulation* 1984; 9:459–469.
- Meikle MB, Vernon J, Johnson RM. The perceived severity of tinnitus. *Otolaryngol Head Neck Surg* 1984; 92:689–696.
- van Veen ED, Jacobs JB, Bensing JM. Assessment of distress associated with tinnitus. *J Laryngol Otol* 1998; 112:258–263.
- Folmer RL, Griest SE, Martin WH. Chronic tinnitus as phantom auditory pain. *Otolaryngol Head Neck Surg* 2001; 124:394–400.
- Folmer RL, Griest SE. Tinnitus and insomnia. *Am J Otolaryngol* 2000; 21:287–293.
- Attias J, Shemesh Z, Bleich A, Solomon Z, Bar-Or G, Alster J, Sohmer H. Psychological profile of help-seeking and non-help-seeking tinnitus patients. *Scand Audiol* 1995; 24:13–18.
- Folmer RL, Griest SE, Meikle MB, Martin WH. Tinnitus severity, loudness and depression. *Otolaryngol Head Neck Surg* 1999; 121:48–51.
- Folmer R. Long-term reductions in tinnitus severity. *BMC Ear Nose and Throat Disorders* 2002; 2:3. Available at: [www.biomedcentral.com/1472-6815/2/3](http://www.biomedcentral.com/1472-6815/2/3).
- Folmer RL, Shi YB. SSRI use by tinnitus patients: interactions between depression and tinnitus severity *ENT J* 2004; 83:107–117.
- Sullivan M, Katon W, Russo J, Dobie R, Sakai C. Coping and marital support as correlates of tinnitus disability. *Gen Hosp Psychiatry* 1994; 16:259–266.
- Lusk SL. Preventing noise-induced hearing loss. *Nurs Clin North Amer* 2002; 37:257–262.
- Henry JA, Schechter MA, Nagler SM, Fausti SA. Comparison of tinnitus masking and tinnitus retraining therapy. *J Amer Acad Audiol* 2002; 13:559–581.
- Surr RK, Montgomery AA, Mueller HG. Effect of amplification on tinnitus among new hearing aid users. *Ear and Hearing* 1985; 6:71–75.