Tinnitus: Diagnostic Approach Leading to Treatment

Robert Aaron Levine, MD

1 Department of Otolaryngology, Tel Aviv Sourasky (Ichilov) Medical Center, Tel Aviv, Israel


Abstract

Optimal care for a patient with tinnitus begins with identifying the cause of the tinnitus. Once the cause has been identified then an appropriate treatment plan can be initiated. In this article, the author reviews how to identify the tinnitus etiology and its treatment. The workup begins with the patient’s description of the percept because in some cases, the quality of the tinnitus will make the diagnosis (e.g., clicking, which is readily suppressed pharmacologically); in other cases, it will give direction in the diagnostic evaluation (e.g., pulsatile). With the exception of a small dural arteriovenous malformation, the source of objective pulsatile tinnitus can be determined without conventional cerebral angiography. If the diagnostic workup is unrevealing and the pulsations are not suppressed with somatic testing, then eighth nerve vascular compression becomes the likely etiology, especially if there is some clicking also heard, no matter how minor.

The two major causes of tinnitus are hearing loss and myofascial disorders of the head and neck. Moreover, the two can combine and cause tinnitus even though either condition alone would not have caused tinnitus. Although the tinnitus of hearing loss is not easily treatable, the tinnitus from myofascial disorders is often responsive to an optimized myofascial treatment program. Hyperacusis, a frequent accompaniment of tinnitus, and its treatment are discussed.

Keywords
► tinnitus
► somatic
► eighth nerve
► cranio-cervical
► dorsal cochlear nucleus
► vascular compression
► stress
► hyperacusis

Tinnitus, the perception of sound in the absence of an external sound, is a symptom and like any other symptom can have a diversity of causes. Here an approach to determining the cause of this symptom is described. Once the cause is determined then the appropriate treatment may follow. Like any other symptom, the approach to the patient begins by obtaining the details of the presenting complaint. The presenting complaint can be used as the organizing principle. In the review of systems, a clinician should pay especially close attention to cervical, dental, and head complaints because the two major causes of tinnitus are (1) a disorder of the muscles and tendons of the upper neck and jaw; and (2) a disorder of the auditory system, namely the outer ear, middle ear, inner ear (cochlea), hearing (auditory, eighth) nerve, and the auditory central nervous system (CNS).

Furthermore, tinnitus may be multifactorial. For example, hearing loss can combine with a disorder of the muscles or tendons of the upper neck or jaw to cause tinnitus, whereas either one alone would not cause tinnitus. For these reasons, the physical examination must include close attention to these areas.

The evaluation of tinnitus should include the standard elements of any medical evaluation with close attention to the circumstances surrounding the onset of the tinnitus, including any association with new medications, psychosocial stressors, other auditory, vestibular or neurologic complaints with special emphasis on headaches; neck discomfort, including new neck stress such as from initiating a new or altered exercise program; and jaw pain, as well as bruxism. Exacerbating and remitting factors should be sought including diurnal variations in the tinnitus. The most important features of tinnitus that must be ascertained are its (1) quality, particularly whether or not it is ever pulsatile or ever has a clicking component, even a very minor one; (2) its location, whether it is heard on one side or not; (3) its variability, whether it is intermittent or constant; (4) its pitch,

Issue Theme Neuro-Otology 2013; Guest Editor, Terry D. Fife, MD

Copyright © 2013 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New York, NY 10001, USA. Tel: +1(212) 584-4662.


ISSN 0271-8235.
whether it is predominantly low or high frequency in character; and (5) whether the patient has found anything that can be done to change the characteristics of the tinnitus.

In addition to the standard neuro-otological physical examination, in general the tinnitus exam should include inspection of the teeth for evidence of wear, listening around the ear and neck for sounds similar to the patient’s tinnitus, palpation of the craniofacial musculature for muscle tension and tenderness with special attention to asymmetries, and probing whether the tinnitus percept can be modulated with maximal isometric contraction of the head and neck muscles ("somatic testing") or strong pressure upon these same muscles and the auricle. All patients should have a recent audiogram.

**Tinnitus Quality: Specific**

Some types of tinnitus have such characteristic features that the description alone is the major determinant of the direction of the diagnostic approach. These types of tinnitus can be strictly in one ear or nonlateralized. Patients use a variety of expressions to describe nonlateralized tinnitus, such as “both ears about equally,” “both ears but worse in one ear,” “in the head but not strongly toward either side,” or “in the head toward one side.” Tinnitus with a characteristic quality include sudden brief unilateral tapering tinnitus (SBUTTIs), hallucinations (musical or auditory), exploding head syndrome (auditory sleep starts), and staccato irregular unilateral tinnitus ("typewriter tinnitus"), and "somatosounds"—physical sounds generated by the body and heard by one or both ears.

**Always Unilateral**

Sudden, Brief, Unilateral, Tapering Tinnitus

About 75% of adults report that they have experienced the sudden onset of a tone or noise in one ear that fades away within seconds and seems to have no definite trigger. It can sometimes be associated with a feeling of fullness, pressure, blocking, or hearing loss of the same ear. Once it begins, it remains at a constant loudness for several seconds then wanes until it is no longer perceived. All the while, the quality of the tinnitus remains unchanged. The entire event typically lasts less than a minute with no permanent change in hearing. In a few individuals, the tone or noise can occur more than twice a week; in others, it can occur once a year or even less. In some, it occurs always in the same ear, but for others the ear can vary. It never occurs simultaneously in both ears. In general, it occurs more commonly in the right ear. Sudden, brief, unilateral, tapering tinnitus is benign. It is not associated with the development of hearing loss or chronic tinnitus.¹

Coarse Intermittent Sounds Coincident with Jaw or Head Movements

Coarse intermittent sounds coincident with jaw or head movements are typical of a foreign body such as cerumen, hair, or a liquid (usually water) resting against the tympanic membrane. Inspection of the ear canal will reveal the source.

Fluttering

Stapedius muscle contractions tend to be described as a fluttering. If the fluttering is associated with facial movements, then stapedial contractions are highly likely the cause of the fluttering sound. This is most commonly seen after recovery from Bell’s palsy, unilateral facial paralysis: When the affected side of the face contracts, the ipsilateral stapedial muscle also contracts (synkinesis) due to aberrant facial nerve regeneration. Abnormalities in the pattern of the stapedial reflex or acoustic impedance measurements corresponding to the characteristics of the patient’s tinnitus can occur.² The normal stapedius reflex occurs only for high-intensity sounds (≥ 85 dB HL). Fluttering that is evoked by high-intensity sounds is usually due to stapedius reflex dysfunction and can be treated by releasing the stapedius tendon.

**Always Nonlateralized**

Exploding Head Syndrome

Typically patients report that as they are dropping off to sleep they hear a brief loud sound ("bang," "explosion," "shotgun," "cymbals," etc.) throughout their head. By the time, the subject is wide awake it stops. Most commonly, it occurs with initiation of sleep, but it can also happen during the night or even with napping. One sleep study found it occurring during the transition from nonrapid-eye-movement stage 1 sleep to waking.³ It can occur in any age group including childhood. Often the condition remits for prolonged intervals, even several months.⁴ Reassurance is usually sufficient. If not, benefit from clonazepam, clomipramine, nifedipine, and topiramate has been suggested from isolated case reports.

Special Quality: Sometimes But Not Always Unilateral

Tinnitus that is clicking can occur synchronously in both ears or can be strictly unilateral.

Nonlateralized Clicking

Very few conditions cause synchronous clicking in both ears: Clicks are heard in both ears at the same time. Palatal myoclonus is the only consideration. Often the clicking can be heard by the examiner listening at the patient’s ear or with use of stethoscope or recording with a microphone in the external auditory canal. Inspection of the palate will usually reveal the problem and confirm the diagnosis. Occasionally, nasal endoscopy will be necessary if direct inspection of the palate is not definitive. Medications that have been reported to improve palatal myoclonus include clonazepam, lamotrigine, piracetam, and valproate. Botox injections can provide relief that is temporary; the relief may last about 4 months.

Lateralized Clicking

Unilateral clicking tinnitus on the other hand can have multiple different etiologies. It may be a unilateral form of palatal myoclonus due to contractions of tensor tympani or the nasopharyngeal muscles controlling the patency of the eustachian tube (salpingopharyngeus, dilator tubae, and...
Unilateral Staccato Irregular Intermittent Clicking (Typewriter Tinnitus)

Probably the most common type of unilateral clicking is “typewriter” tinnitus characterized by unilateral staccato irregular intermittent clicking. Besides typewriter, other terms that patients have used to describe what they hear are “popcorn,” “Morse code,” “nail clipper,” “tapping,” “snapping,” “crackling,” and “machine gun.” In some, the clicking can be triggered by a specific class of external sounds (e.g., running water). Most have no associated hearing loss: The audiogram is symmetric. The irregular clicking can occur with no other symptoms, including no other tinnitus and no hearing loss. It typically responds to carbamazepine usually at low dose; it can also respond to other trigeminal neuralgia medications, but less predictably.5,6

Like the other unilateral staccato irregular intermittent cranial nerve syndromes (trigeminal and glossopharyngeal neuralgia, hemifacial spasm, and vestibular parosmia) typewriter tinnitus typically occurs in people above the age of 50, probably because it is most commonly due to vascular compression of the eighth nerve (Case 4); however any condition that distorts the eighth nerve can cause typewriter tinnitus, probably because the eighth nerve becomes hyperexcitable.7

Variants of pure typewriter tinnitus occur. Typewriter tinnitus may occur in the same ear with a second type of tinnitus that is usually associated with a sensorineural hearing loss and is not staccato, but a constant sound such as hissing. The second type of tinnitus can even be pulsatile. In Case 1 as given below, there was definite vascular compression with ipsilateral pulsatile tinnitus that alternated between typewriter (“clicking”) and nontypewriter (“whooshing”). The typewriter tinnitus can begin at the same time, precede, or follow the constant tinnitus by months or years. The typewriter tinnitus component responds to carbamazepine (as well as other medications used for trigeminal neuralgia), while the nonclicking component does not.6

Another variant is bursts of typewriter tinnitus occurring with vertigo (vestibular parosmia). Two cases of paroxysmal unilateral clicking tinnitus and spinning with imbalance occurring at the same time and lasting a few seconds have been reported.8 Both responded to gabapentin, a trigeminal neuralgia medication.

Case 1

In this case, eighth nerve vascular compression was the cause of pulsatile clicking and nonspecific tinnitus.

A 60-year-old woman with a mild symmetric high-frequency sensorineural hearing loss, developed right-sided pulsatile tinnitus that alternated between clicking and whooshing—typewriter tinnitus and nontypewriter tinnitus. Both were cardiac synchronous. When her tinnitus was quieter, it was clicking with no whooshing. When louder, it was whooshing with no clicking. Her MRI showed her right anterior inferior cerebellar artery (AICA) abutting (open curved arrow) the cisternal segment of her right auditory nerve (eighth nerve), causing it to deviate from a direct course between the brainstem and the internal auditory canal (Fig. 1). She declined carbamazepine.

Comment

It is likely that carbamazepine would have abolished the clicking, but not the whooshing as we have observed multiple times previously. From analogy with trigeminal neuralgia and with some supportive imaging data, I have previously suggested that clicking tinnitus is due to eighth nerve vascular compression.5 This case provides further support for this hypothesis as well as the concept that pulsatile tinnitus can be related to eighth nerve vascular compression.

Pulsatile Tinnitus (Cardiac Synchronous)

Whether the tinnitus is pulsatile should be established at the outset of the evaluation. If described as pulsatile, then the next step is to determine whether it is cardiac synchronous. This can be evaluated by comparing the examiner’s silent count of the patient’s cardiac pulse, while the patient is counting the pulsations of his or her tinnitus. The examiner indicates when the counting interval starts and stops and then the two counts are compared. If the counts are virtually identical, then the pulsatile tinnitus is cardiac related and a vascular source must be sought. However, if the two counts are discordant then the tinnitus is not cardiac related and other causes must be considered.
Once it has been established that the pulsatile tinnitus is cardiac synchronous, then the next property that needs to be established is where the percept is perceived.

Nonlateralized Pulsatile Tinnitus

If it is “heard” throughout the head, and not in just one ear, then very few conditions need to be considered that can cause nonlateralized cardiac synchronous pulsatile tinnitus. Two different unilateral sources, one from each side, can be easily excluded because their onset is at different times. A high cardiac output state such as anemia or hyperthyroidism should be ruled out with a complete blood count (CBC) and thyroid profile. A central somatosound is another possibility such as from a carotid-cavernous fistula or aortic stenosis. Once these uncommon possibilities are excluded, then somatosensory pulsatile tinnitus syndrome becomes most likely and is confirmed by somatic testing, which always abolishes the pulsations and often the tinnitus altogether.9

Somatosensory pulsatile tinnitus syndrome was first reported in 2008. Its percept is cardiac synchronous and can be either lateralized or nonlateralized; it is usually high pitched. Its defining feature is that the pulsations can be abolished by somatic testing: a strong muscle contraction of the head or neck or a strong pressure applied to these same muscles (Case 2). In our original report, 70% of the patients could abolish their tinnitus completely with somatic testing; the other 30% converted their cardiac synchronous pulsatile tinnitus to nonpulsatile constant high-pitched tinnitus.9 Usually, the tinnitus is constant, but in some it is intermittent. An exhaustive imaging workup is negative. Continuous auricular electrical stimulation can quiet the tinnitus of many who have this syndrome.10

Case 2

In this case, a patient with somatosensory pulsatile tinnitus syndrome responds to auricular electrical stimulation.

A 58-year-old woman reported 1 year of right ear, constant, high-pitched, cardiac synchronous, pulsatile tinnitus whose loudness increased with stress. In her own words, “Salvation Army Christmas bell ringers were in my ... right ear.” She had noticed that by putting pressure on her sternocleidomastoid tendon insertion at the right mastoid process, her tinnitus quieted by 80%. Her audiogram, MRI, and magnetic resonance angiography (MRA), CBC, and thyroid testing were all normal.

Her right temporalis and sternocleidomastoid muscles were increased in bulk as compared with the left, but there was no associated tenderness. No bruits were heard over the mastoid, jugular regions, globes, or occipital regions at rest or following exercise. Jugular or carotid compression on either side did not affect her tinnitus. Some, but not all somatic maneuvers abolished her pulsations. -Table 1 outlines the maneuvers that changed her tinnitus.

In view of her suppression of the pulsations with somatic testing, she elected to defer further diagnostic testing (Fig. 2) and commence a trial of continuous auricular electrical stimulation.74 After seven weekly applications of the device, her tinnitus had quieted by ≈75%. Her tinnitus continued to quiet despite no further active treatment. Ninety days after starting electrical stimulation “the ringing had been reduced to near zero,” where it has remained for the last 3 years.

Comment

The hallmark of the somatosensory pulsatile tinnitus syndrome is the ability to abolish pulsations with somatic testing, as this subject demonstrates. Note that one of the maneuvers that increased her tinnitus loudness at the same time abolished the pulsations. In an open trial of continuous auricular electrical stimulation, five of seven patients with somatosensory pulsatile tinnitus syndrome had sustained quieting of their tinnitus by more than 50% on their visual analogue (VAS) scale.10

Lateralized Pulsatile Tinnitus

The patient’s history can give clues to the source of the pulsatile tinnitus.11 An association with headaches, blurring of vision, and menstrual irregularities in an obese young woman is suspicious for benign intracranial hypertension, also known as pseudotumor cerebri. Abrupt onset with unilateral neck or head pains suggests a carotid dissection. A change in tinnitus intensity with head turning suggests a venous source for the tinnitus—from a source ipsilateral to the direction that decreases the tinnitus. This can be confirmed with jugular compression. If the patient can obliterate the tinnitus with mild localized pressure in the periauricular region then an emissary vein is probably accounting for the tinnitus.

The physical exam can provide key information about the pulsatile tinnitus, as well. A crescentic purple coloration to

Table 1 Effects of somatic testing upon right pulsatile tinnitus, no left tinnitus

<table>
<thead>
<tr>
<th>Condition</th>
<th>R tinnitus loudness</th>
<th>Pulsatile?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline (0–10 Scale)</td>
<td>5</td>
<td>Yes</td>
</tr>
<tr>
<td>Protrude jaw maximally</td>
<td>9</td>
<td>Yes</td>
</tr>
<tr>
<td>Right lateral neck flexion maximally against resistance</td>
<td>7</td>
<td>No</td>
</tr>
<tr>
<td>Left lateral neck flexion maximally against resistance</td>
<td>3</td>
<td>No</td>
</tr>
<tr>
<td>Intense right splenius pressure</td>
<td>1</td>
<td>No</td>
</tr>
<tr>
<td>Intense right sternocleidomastoid pressure</td>
<td>1</td>
<td>No</td>
</tr>
</tbody>
</table>
the tympanic membrane is diagnostic of a glomus jugulare tumor. Otoscopic observation of a red mass behind the tympanic membrane is evidence for an aberrant carotid artery, dehiscent jugular bulb, or a vascular tumor. A unilateral conductive hearing loss in association with ipsilateral pulsatile tinnitus and an otherwise normal exam suggests otosclerosis, as does Schwartze's sign (a red hue behind the tympanic membrane on otoscopy). Detection of a bruit ipsilateral to the pulsatile tinnitus suggests that the tinnitus is from the same source as the bruit. The source of the bruit then must be sought. If localized to the region of the carotid artery bifurcation, then fibromuscular dysplasia, carotid stenosis, or carotid dissection is suspected; an associated ipsilateral Horner's syndrome would suggest a carotid dissection.

If the bruit is more widely distributed such as throughout the periauricular region or even more widespread, a dural arteriovenous fistula becomes likely. If heard over the globe, a carotid-cavernous sinus fistula is suspected, particularly if there is associated proptosis. Obliteration or reduction in the intensity of the pulsatile tinnitus with ipsilateral jugular compression (light or moderate pressure below the angle of the jaw) implicates a venous source of the tinnitus. If venous pulsations are seen within at least one of the optic fundi, then cerebrospinal fluid pressure is normal and raised intracranial pressure can be ruled out. If somatic testing eliminates the pulsation or cures the tinnitus altogether, then the somatosensory pulsatile tinnitus syndrome becomes the working diagnosis, particularly if all diagnostic testing including imaging is negative (see above section, Nonlateralized Pulsatile Tinnitus and Case 2). If somatic testing does not stop the pulsations, then eighth nerve vascular compression is likely (see below and Case 1).

The diagnostic studies following the initial visit will be guided by the findings of the clinical evaluation and laboratory studies (Fig. 2). Because high cardiac output states such as anemia or hyperthyroidism can cause pulsatile tinnitus (usually bilateral), all patients should have a thyroid profile and a hematocrit count done. If a carotid lesion is suspected, then either a duplex ultrasound study of the carotid, computed tomography angiography (CTA), or MRA should be performed. If a retrotympanic mass is suspected, then a high-resolution contrast-enhanced CT scan of the temporal bones should be obtained. Otherwise, a contrast-enhanced MRI scan of the temporal bone and cranium should be obtained. The MRI scan may not detect anomalous arterial patterns such as a persistent stapedial artery, so a noncontrast high-resolution CT scan of the temporal bone is performed should the MRI scan be normal. If still no etiology is apparent, and neither papilledema nor retinal venous pulsations, were observed then cerebrospinal fluid pressure should be measured via a lumbar puncture. If all the above noninvasive imaging studies have been unremarkable and raised intracranial pressure has been ruled out, then cerebral angiography should be considered because a dural arteriovenous malformation can sometimes go undetected by any other diagnostic study, even though there may or may not be a thrill or bruit on physical examination.

**Eighth Nerve Vascular Compression**

That eighth nerve vascular compression can cause unilateral tinnitus that in some cases is pulsatile has been well

---

**Fig. 2** Pulsatile tinnitus: diagnostic algorithm. Somatic testing and laboratory studies (hematocrit and thyroid profile) should be done before imaging. If somatic testing does not suppress pulsations and diagnostic testing reveals no abnormality then vascular compression of the auditory nerve is very likely. (MRI, magnetic resonance imaging; MRA/MRV, magnetic resonance angiography/venography of cervical and intracranial vasculature; CT, thin section computed tomography of temporal bone).
established by Ryu and colleagues. They made their observations in patients whose tinnitus was not their primary complaint; hemifacial spasm was their primary complaint. From surgically decompressing the facial nerve in the cerebellopontine angle, it was observed that 100% of those with ipsilateral preoperative tinnitus (half of whom were pulsatile) had eighth nerve compression; and in those without tinnitus, only 6% had eighth nerve compression. Furthermore, with decompression 70% of those with tinnitus had their tinnitus completely gone and another 10% was markedly improved. Eighty percent of those with pulsatile tinnitus had their tinnitus resolved. Hence, vascular compression of the auditory nerve can cause unilateral tinnitus including pulsatile tinnitus.

With rare exceptions (Case 1), determining with certainty that an individual’s unilateral tinnitus, whether pulsatile or nonpulsatile, is from eighth nerve vascular compression has unfortunately not yet been possible. A recent report proposed several criteria for establishing the diagnosis of eighth nerve vascular compression; however, the patients had not been evaluated for somatosensory pulsatile tinnitus syndrome. Decompression surgery was performed on 20 patients who met these criteria. From observations at surgery, they all were said to have “proven cochleovestibular compression.” However, only one patient had any major improvement of her tinnitus. These disappointing results suggest that the proposed criteria are not useful and other criteria should be sought such as ruling out somatosensory pulsatile tinnitus syndrome with somatic testing.

From analogy with trigeminal neuralgia and with some supporting imaging data, I have suggested previously that typewriter tinnitus is also due to eighth nerve vascular compression. Case 1, whose MRI (Fig. 1) showed clear-cut right auditory nerve vascular compression, provides more supporting evidence for pulsatile tinnitus and clicking tinnitus related to eighth nerve vascular compression because her tinnitus was always pulsatile: When quieter it was clicking-pulsatile; when louder it was whooshing-pulsatile. She never heard clicking and whooshing together.

**Autophony (Echoing of the Voice) or Blowing Tinnitus**

The characteristics of this type of tinnitus are so unique that the history alone virtually makes the diagnosis, namely a patulous ipsilateral eustachian tube. Patients describe a blowing sound with respiration and an echoing quality to their own voice. Confirmatory features include disappearance of their complaints when their head is in a dependent position and abnormally large changes in the tympanic membrane acoustic impedance with respirations.

**Hallucinations (Nonverbal, Stereotyped Repetitive)**

Unlike the hallucinations associated with psychoses, these patients have no associated thought disorder, and the hallucinations do not have personally relevant content. Rather the hallucinations are either “musical” in which patients report hearing one or a series of familiar tunes incessantly, or “auditory” in which a variety of different sounds are described.

Typically, the strictly musical hallucinations occur in elderly patients (more commonly in women) with a longstanding progressive moderate to severe bilateral hearing loss. The tunes can be vocal and/or instrumental. Although they are usually bilateral, they can be unilateral even with a bilateral hearing loss. Occasionally, they can be precipitated by a new medication, and the hallucinations resolve when the medication is stopped. If the presentation is typical, no brain imaging is necessary.

Auditory hallucinations differ from musical hallucinations in several respects. They are usually abrupt in onset and associated with focal neurologic findings due to a brainstem stroke or space-occupying lesion. There usually is no major preexisting chronic hearing loss and the hallucinations are usually not only musical, but may have a variety of other sounds such as bells or a waterfall. They often are transient.

<table>
<thead>
<tr>
<th>Tinnitus Quality: Nonspecific</th>
</tr>
</thead>
<tbody>
<tr>
<td>Included in this category are a variety of descriptors of tinnitus such as buzzing, tonal, hissing, humming, ringing, roaring, rushing, whistling and whooshing, cicadas, crickets, etc. None of these descriptors point to a specific diagnosis. Roaring, though nonspecific, is often associated with Ménière's syndrome. Because the quality of the tinnitus is nonspecific, aids in making a diagnosis must come from sources other than how the tinnitus sounds. Associated symptoms, circumstances surrounding the onset of the tinnitus, and ameliorating and exacerbating factors are some of the pointers to the diagnostic entity that is accounting for the tinnitus symptom.</td>
</tr>
</tbody>
</table>

In attempting to arrive at a diagnosis for tinnitus in which the characteristics of tinnitus are nonspecific, there are several important considerations that must be kept in mind. The first is that tinnitus is very common in the general population. Many people have tinnitus, but have never complained of tinnitus and have not sought any medical attention for it. The landmark 1953 study of Heller and Bergman found such tinnitus in 94% of people. More recent and better controlled studies of normal hearing subjects have also found large percentages reporting such tinnitus albeit smaller numbers (52–92%).

<table>
<thead>
<tr>
<th>Table 2 Nonspecific tinnitus: Reasons for difficulties in establishing a diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tinnitus is common in the general population.</td>
</tr>
<tr>
<td>For any disease, not all subjects will develop tinnitus.</td>
</tr>
<tr>
<td>The coexistence of tinnitus and a disease does not imply that the two are related (Case 3).</td>
</tr>
<tr>
<td>Tinnitus can be multifactorial (Case 3).</td>
</tr>
</tbody>
</table>
In our study in which normal subjects were placed in a low-noise room, 20% had previously been aware that they had tinnitus in quiet surroundings. For the first time, another 35% noticed that they had tinnitus in quiet surroundings. Seventy-five percent of our subjects reported having experienced SBUTTs, another type of “normal” tinnitus—namely, unilateral tonal/noise tinnitus lasting less than a minute. Another type of transient tinnitus, which is common, follows exposure to loud sound. About 50% of our subjects recalled such tinnitus, lasting from a few minutes to several hours or even days. In fact, patients have occasionally presented to our clinic complaining of one of these types of “normal” tinnitus.

A second important consideration is that with any pathologic process that has been associated with tinnitus, not all subjects will develop tinnitus. In surveys of profoundly deaf subjects, ~80% will have tinnitus, but 20% will not. A similar percentage has been reported for idiopathic sudden sensorineural hearing loss. Hence, the presence of tinnitus and a pathologic process by itself does not imply that the two are related. Because there is no obligatory association between the pathologic process and tinnitus, it remains a possibility that even though a tinnitus patient has a chronic condition known to be associated with tinnitus, his or her tinnitus may not be related to the chronic condition; rather the tinnitus and the pathologic process could coexist, but be unrelated.

Because tinnitus is common in the general population, a third consideration is that the pathologic process only draws the patient’s attention to his or her pre-existing tinnitus. Furthermore, if the disease in and of itself did not cause the tinnitus but hearing loss is part of the disease, the hearing loss could be “unmasking” the pre-existing tinnitus. Just as bringing a normal subject into a low-noise environment can make the subject aware of tinnitus they had not appreciated previously, so could a hearing loss unmask tinnitus. Therefore, the question always remains in any patient with nonspecific tinnitus, whether the tinnitus was pre-existing and unmasked either due to (1) his or her loss of hearing, or (2) his or her attention being drawn to his or her hearing. Casual attention to tinnitus of which people were previously unaware does not appear to be associated with the development of disturbing tinnitus because following the Heller and Bergman type of studies, no subjects developed troublesome tinnitus. Clinical anecdotes suggest that when attention to tinnitus, of which they were previously unaware, is emotionally charged then the tinnitus can become troublesome.

Because nonspecific tinnitus can be physiological, and nonspecific tinnitus is not obligatorily associated with any pathologic process, establishing a diagnosis that is accounting for any tinnitus is more problematic than for most medical symptoms when the condition is chronic and very slowly progressive (e.g., presbycusis). However, when the onset of the condition and new tinnitus are temporally related then the causal relationship is much more certain (such as in sudden sensorineural hearing loss).

In general, there is a level of confidence associated with any diagnosis that might account for a symptom. For tinnitus, some relationships increase this confidence. The first is a temporal association between the tinnitus and the diagnostic consideration. For example, in Ménière’s syndrome, if the tinnitus fluctuates with the hearing loss and vertigo, then this strengthens the confidence of the association between the tinnitus and Ménière’s syndrome. Another way the confidence of the diagnosis is strengthened is if the pitch of the tinnitus corresponds to the audiometric hearing loss pattern. Considering again Ménière’s syndrome, where early in the illness low-frequency hearing loss often predominates, if the tinnitus is described as roaring and/or the pitch match is a low frequency one, then the confidence of the relationship between the Ménière’s syndrome and the tinnitus is strengthened. Likewise, changes in the tinnitus percept that are closely coupled to changes in physical findings strengthen the association between the two.

Another consideration in attempting to establish a diagnosis to account for a patient’s tinnitus is that the cause of the tinnitus may be multifactorial. Tinnitus can be considered a threshold phenomenon, such that, although any one factor such as chronic progressive hearing loss may not be sufficient to elicit a tinnitus complaint, two or more factors may synergistically lead to the tinnitus becoming symptomatic.

Closely related to the threshold idea is the concept of “triggering factors” that can lead to symptomatic chronic tinnitus. Such factors include psychosocial stress (Case 3). Clinical anecdotes suggest that the clinical problem of tinnitus can be precipitated by one (or more) of these triggering factors (Case 3). Although a triggering factor may appear to be responsible for initiating the tinnitus, sometimes the tinnitus will persist despite resolution of the triggering factor.

Case 3

In this case, somatic factors cause intermittent tinnitus ipsilateral to the better hearing ear.

A 48-year-old woman presented with 8 months of fluctuating right ear nonpulsatile tinnitus (like “crickets”) that began a few weeks after receiving new corrective lenses and the onset of frequent frontal headaches. She had no left tinnitus. At times, she could have right tinnitus nearly continuously for as long as a month; at other times, no tinnitus for up to a week. She did not notice any hearing change with her tinnitus or its fluctuations. A subsequent audiogram showed on the right a 40 dB hearing loss at 4 kHz and above, on the left a moderate to severe left-sided hearing loss that was congenital and stable (> 70 dB above 4 kHz). Her speech discrimination score was normal on the right. She denied bruxism. On exam, she had right myofascial trigger points involving her temporalis, masseter, pterygoid and sternocleidomastoid muscles. None were detected in her left-sided muscles. The pitch of her right ear tinnitus changed with...
intense palpation of her right temporalis trigger points. MRI and auditory brainstem response (ABR) were normal.

Comment
Her tinnitus was intermittent and fluctuating and on the side of her myofascial disorder, not the side of her major hearing loss. Because fluctuations are more commonly associated with somatic tinnitus than tinnitus from a high-frequency sensorineural hearing loss, and she had an ipsilateral myofascial disorder, it was concluded that her tinnitus and headaches were primarily upon a myofascial basis with possibly a contribution to her tinnitus from the mild sensorineural hearing loss of her tinnitus ear (Fig. 3). A treatment program involving needling of her trigger points was begun.

Case 4
In this case, a trigger factor in chronic progressive symmetric hearing loss was found.

Following the birth of her second child, a 31-year-old mother with a family history of hearing loss was not hearing the beep of the thermometer. She was otherwise not aware of any hearing loss or tinnitus. She was advised to have an audiogram, which was not obtained until 15 months later. It showed normal thresholds below 1 kHz but a 60 dB loss at 2 kHz and above with absent distortion product otoacoustic emissions. Speech discrimination scores were normal. She reported feeling very distressed from learning of her hearing loss. Because her audiogram associated with the onset of the tinnitus. What is striking is that her hearing loss could easily be accounting for her tinnitus and yet she reported no tinnitus until her first audiogram. The stress of learning about her hearing loss and its prognosis appears to have been the “trigger factor” that precipitated her tinnitus. This raises the general issues discussed above regarding what accounts for the onset of nonlateralized tinnitus in people with a slowly progressive symmetric hearing loss. In this particular individual, the possibility remains that her tinnitus pre-existed her first audiogram and was unmasked when her attention was drawn to her hearing by the abnormal audiogram.

Always Nonlateralized

Chronic Progressive Symmetric Hearing Loss (Presbycusis, Chronic Acoustic Trauma, Hereditary Hearing Loss)
Presbycusis, chronic acoustic trauma, and hereditary hearing loss can be considered together because they affect only hearing, generally have a symmetric hearing loss, and are slowly progressive, albeit at different rates. They are the most common type of tinnitus encountered in the clinic. When their associated tinnitus begins, it is nonlateralized. Presently, it cannot be predicted when, during the course of patients’ progressive hearing loss, their nonlateralized tinnitus will begin, if ever, because 20% of such people will never develop tinnitus despite a profound hearing loss. Should (1) the audiogram be asymmetric (2) the tinnitus begin first in one ear and only later in the other ear, or (3) the tinnitus be lateralized, then one or more other factors must be sought to account for the asymmetry.

The establishment of a causal relationship between chronic progressive hearing loss and tinnitus is problematic because there is no perceptible change in the hearing or audiogram associated with the onset of the tinnitus. What has been well established is that the prevalence and reported loudness of tinnitus increase with increasing hearing loss. However, for any patient with chronic progressive symmetric hearing loss and recent onset of nonlateralized tinnitus a triggering factor or other cause for the tinnitus must be sought (Case 4), even though the yield has been low in my experience. The association between tinnitus and chronic progressive hearing loss must be considered tenuous.
Autoimmune Inner Ear Disease
This condition is like chronic progressive hearing loss except the progression of the bilateral hearing loss is measured in weeks or months rather than years. It can fluctuate and be asymmetric. Due to its more rapid time course, the association of tinnitus with the disease is more compelling. Positive blood tests for general autoimmune disease or inner ear antibodies support the diagnosis.

Central Nervous System Disorder: Rostral to Trapezoid Body
The evidence available indicates that for central nervous system lesions rostral to the trapezoid body (such as involving the inferior colliculus) tinnitus is usually transient and bilateral.27 A lesion located at the junction of the head and body of the caudate nucleus ( locus of caudate neurons or area LC) can quiet tinnitus.28,29

Medication-Related Tinnitus (Including Withdrawal Syndromes)
The temporal association of the onset of the tinnitus with exposure to a toxin establishes the diagnosis particularly if the tinnitus resolves when the toxin is withdrawn. An association is less clear when tinnitus begins just after a new medication was begun, but does not remit when discontinued. Such instances raise the possibility that the new medication acted as a trigger factor for the tinnitus. Cisplatin, aminoglycoside antibiotics, and loop diuretics can cause permanent hearing loss and tinnitus. High-dose aspirin and quinine can cause a reversible hearing impairment and tinnitus. Transient or rarely long-lasting tinnitus can be a part of a sedative withdrawal syndrome,10 which in the case of benzodiazepines can be precipitated by fluoroquinolones because these antibiotics compete directly with benzodiazepines for the benzodiazepine receptor site displacing benzodiazepines and thereby precipitating benzodiazepine withdrawal.31

Always Unilateral—Never with Vestibular Symptoms
Conductive Hearing Loss
Any type of unilateral conductive hearing loss, such as cerumen impaction, ossicular discontinuity, or otosclerosis, can be associated with tinnitus of that ear. The tinnitus may be related at least in part to an unmasking of a normal underlying tinnitus, as discussed above. Otosclerosis may sometimes be associated with inner ear involvement that could be contributing to the tinnitus as well.

Otoacoustic Emissions
Although spontaneous otoacoustic emissions are common (75% of female and 45% of male with normal or near normal ears), tinnitus due to spontaneous otoacoustic emissions is uncommon. It is said to be accounting for the tinnitus of 1 to 2% of the patients of one British tinnitus clinic, but in my experience it has been much less frequent.32 The diagnosis is made by measuring an emission and showing that its suppression abolishes the tinnitus. The emission can be suppressed in either of two ways: (1) presentation of a low-level tone near the emission frequency, or (2) the use of aspirin.33

Eighth Nerve Compression (Usually Vascular)
As discussed earlier, the tinnitus of eighth nerve compression is unilateral, rarely associated with vestibular symptoms, and can take three forms: (1) nonspecific (hissing, buzzing etc.), (2) pulsatile, and (3) staccato irregular intermittent clicking (typewriter). Anyone with unilateral nonspecific tinnitus should be questioned closely about whether there ever is a clicking or pulsating component to their nonspecific tinnitus, even if very rarely. If so, then the possibility of eighth nerve compression becomes more likely.

Always Unilateral—Possibly with Vestibular Symptoms
Ménière’s Syndrome
As a syndrome, this condition has tinnitus as one of its defining features. The full-blown picture consists of episodic attacks of intense vertigo persisting hours, fluctuating unilateral hearing loss typically involving the lower frequencies (in the early stages), ear fullness, and a roaring low-frequency tinnitus. Early in its course, there may be no persistent symptomatology; however, with recurrent episodes any or all of the symptoms can persist and cumulatively progress with each recurrence. Although the tinnitus is often described as roaring early in the illness, with more advanced stages of the syndrome, the tinnitus tends to become more variable in its description. There are no definitive tests to establish the diagnosis; however, electrocochleography can be supportive of the diagnosis, if there is a large ratio of summing potential to action potential amplitudes. Documentation of the fluctuation in hearing with serial audiograms is supportive of the diagnosis. The FTA-abs (fluorescent treponemal antibody-absorption) test can sometimes be positive in this syndrome. Forms of this syndrome may occur. In particular, episodic low-frequency fluctuating hearing loss with a contemporaneous roaring tinnitus and aural fullness may occur without vertigo.

Perilymphatic Fistula
Like Ménière’s syndrome, hearing loss, vertigo, and tinnitus may occur together. However, the tinnitus and hearing loss tend to be high frequency (hissing, crickets, etc.) with no recovery. The fistula consists of a communication between the perilymph of the inner ear fluids and the middle ear through a round or oval window defect or sometimes a defect of the bony labyrinth. The defect can be caused by barotrauma (e.g., airplane descent or ascent from SCUBA diving), head trauma, Valsalva, or erosion of the bony labyrinth due to an inflammatory or neoplastic process, or following middle ear surgery such as stapedectomy. The diagnosis can be suggested by the fistula test—the induction of nystagmus by positive or negative pressure applied to the external auditory canal. If symptoms persist and the findings are suggestive, then the middle ear can be explored for a fistula with patching of the round and oval windows. Generally, the hearing loss
and tinnitus are not improved by patching the oval and round windows whether or not a leak is found at surgery.

**Superior Semicircular Canal Dehiscence**

Superior semicircular canal dehiscence is closely related to perilymphatic fistula. Whereas the perilymphatic fistula is a defect of the otic capsule into the middle ear, superior semicircular canal dehiscence is a defect of the otic capsule into the floor of the middle cranial fossa. Although vestibular symptoms from loud sounds or straining are more commonly the presenting superior semicircular canal dehiscence symptom, in one series 35% of patients reported unilateral high-pitched tinnitus as one feature of their presentation. Other auditory symptoms include monaural heightened bodily sounds (eye movements, heartbeat, footsteps, or voice). The diagnosis is suspected from an air—bone gap with bone thresholds exceptionally good, intact acoustic reflexes, and ipsilateral low thresholds of their vestibular evoked myogenic potential. A CT scan can demonstrate the dehiscence. Much rarer are dehiscences involving the posterior or lateral semicircular canals. A variety of surgically induced dehiscences can also occur.

**Herpes Zoster Oticus**

Intense ear pain followed by ipsilateral tinnitus, hearing loss, vertigo and facial paralysis, known as Ramsay Hunt syndrome, will be recognized as due to herpes zoster once vesicles appear on the pinna, external auditory canal, or tympanic membrane.

**Cerebellopontine Angle Tumors**

The most common presentation of such tumors is a gradual unilateral sensorineural hearing loss with minimal if any vestibular complaints. Dizziness and facial weakness, in general, are either nonexistent or very minor accompaniments of acoustic neuromas at any time. The audiometric pattern is variable. They are more likely to have poor speech discrimination, acoustic reflex decay, and pure tone decay. Other presentations do occur including (1) unilateral tinnitus only, or (2) sudden hearing loss with or without subsequent recovery.

Any unilateral tinnitus with or without unilateral sensorineural hearing loss must be considered suspect for a cerebellopontine angle tumor, even though only ~3% of all patients with acoustic neuromas first present complaining of unilateral tinnitus only. Therefore, should every such patient have a contrast-enhanced or high-resolution T2-weighted MRI scan to exclude the diagnosis because they have a sensitivity “approaching, if not reaching 100%” in 25 years of evaluating all patients with unilateral tinnitus for a cerebellopontine angle lesion, I have detected just two lesions of the cerebellopontine angle. Dawes and Basiony have reported a similar experience; one acoustic neuroma was detected in 174 patients with unilateral tinnitus. Alternatives to obtaining the gold standard contrast-enhanced MRI scan immediately are (1) using the short-latency brainstem auditory evoked potentials (especially stacked ABR), a good but imperfect test, to decide whether or not to proceed to the MRI scan; or (2) following the patient with revisits and repeat audiograms at 6-month intervals, looking for development of an ipsilateral hearing loss before going to the MRI scan. The issue ultimately becomes one of cost effectiveness and availability of MRI scanning because there is virtually no morbidity associated with the MRI scan.

**Central Nervous System: Caudal to Trapezoid Body**

The hallmark of tinnitus due to a disorder of the central nervous system is other neurologic system involvement. If the central auditory nervous system involvement occurs between the ear and the trapezoid body (where the auditory inputs from the two sides intermix and cross) then there may be an associated ipsilateral hearing loss with unilateral tinnitus. The type of associated neurologic involvement will depend crucially upon the precise location of the lesion. With unilateral tinnitus, it can include dizziness, diplopia, limb ataxia, ipsilateral facial weakness, ipsilateral facial paresthesias, or contralateral limb paresthesias. The tinnitus is often transient. Stroke, intrinsic or extrinsic neoplasms, demyelinating disease, inflammatory diseases, and meningiitides can all lead to unilateral tinnitus and hearing loss. The diagnosis will be established by the pattern of neurologic system involvement, the temporal profile of the illness, and the results of ancillary diagnostic studies such as MRI scanning, cerebrospinal fluid examination, or arteriography.

**Sudden Idiopathic Hearing Loss**

Patients can present with an abrupt unilateral tinnitus as their only complaint and upon evaluation are found to have a corresponding unilateral hearing loss of which they may have been unaware. More typically, they complain of both the hearing loss and tinnitus or hearing loss alone. The hearing loss and tinnitus are abrupt in onset and usually unilateral. The quality of the tinnitus usually is closely related to the pattern of the pure tone audiogram. Vestibular symptoms, should they occur, are usually not prominent. Once other rare but identifiable causes have been considered, such as Ménière’s syndrome, cerebellopontine angle tumor, cochlear ischemia, syphilis, or herpes zoster, then the diagnosis of sudden idiopathic hearing loss is secure.

**Maybe Unilateral or Nonlateralized—Always with Hearing Loss**

**Acute Acoustic Trauma**

There is no difficulty establishing the diagnosis of tinnitus due to acoustic trauma when the history is one of immediate development of hearing loss and tinnitus following an intense sound exposure with partial or complete recovery of hearing (temporary threshold shift) over a few days. On the other hand, if the audiogram is normal or the tinnitus does not immediately follow a unilateral intense sound exposure, then other causes for the tinnitus must be sought.

**Possibly No Hearing Loss**

**Somatic (Head or Upper Cervical)**

Observations abound supporting the notion that head and neck somatic events can be associated with tinnitus. Prior to...
their first visit, ~20% of patients in our clinic have noticed that they can modulate their tinnitus somatically, such as by clenching the teeth or pushing on various places on the head. For 2 years, we systematically examined our patients with a battery of isometric head and neck contractions (Case 2). More than 75% of patients could modulate their tinnitus. A variety of changes can occur. Most frequently, the tinnitus gets louder (72%); less often, the tinnitus becomes quieter (25%) as well. In some, the tinnitus became quieter for some maneuvers and louder for others (18%). Less frequently patients describe pitch or location changes.

Tinnitus is generally included among the features associated with pain in the temporal or preauricular region that goes by various names such as Costen’s syndrome, cranio-mandibular disorder, and temporomandibular joint syndrome. Well-designed studies have shown a higher incidence of tinnitus in normal hearing subjects with temporomandibular joint syndrome than in controls. The same is true regarding whiplash. From multiple other observations and case reports, the concept of tinnitus associated with whiplash and temporomandibular joint syndrome can be generalized to include tinnitus associated with any disorder of the upper cervical region and head, including dental pain.

Our observations and those of others have indicated that the tinnitus temporally associated with unilateral somatic disorders is localized to the ipsilateral ear (Case 3). Hence, unilateral tinnitus with no associated auditory or vestibular symptoms such as hearing loss must be suspect for an ipsilateral head or neck somatic disorder. The physical examination should include (1) inspection of the teeth for wear as evidence of bruxism, (2) palpation of the head and neck musculature for tender muscles with increased tension (trigger points), (3) assessment of the effects upon the patient’s tinnitus from somatic testing, including forceful systematic isometric contraction of muscle groups of the head and neck, and deep palpation of these same muscles and the auricle.

At least four factors have been associated with changes in tinnitus attributes. The first is somatic modulation. As described above, it is clear that most, if not all, subjects can somatically modulate their tinnitus. The second is stress. Patients consistently describe that they are more bothered by their tinnitus when stressed. Whether this is due to the patient focusing his or her attention upon the tinnitus or actual changes in the tinnitus loudness usually cannot be distinguished by the patient. In fact, it could be that stress acts through somatic modulation to increase tinnitus loudness because contractions of craniofacial musculature such as clenching the teeth, furrowing the brow, or grimacing often accompany stress. Hence, one way by which stress may lead to increased tinnitus loudness is through increasing head and neck muscle contractions, which in turn lead to louder tinnitus by the somatic mechanism. Third, some patients clearly associate an increase in their tinnitus loudness with exposure to loud sound; in some, the louder tinnitus can persist for hours after the exposure has discontinued. Lastly, benzodiazepine use can result in tinnitus fluctuations because benzodiazepines quiet tinnitus in the majority of people and are commonly used for controlling the insomnia and anxiety associated with tinnitus. Thus, if a patient reports that his tinnitus is intermittent or has wide fluctuations in loudness or other qualities, and there is neither use of benzodiazepines, exposure to intense sound, nor evidence for stress, then the somatic modulation mechanism must be suspected.

A history of variations in tinnitus loudness then raises the suspicion for a somatic factor modulating the percept’s loudness (Table 3). At an extreme are patients who describe that they have periods when their tinnitus cannot be heard, even in the quiet while focusing on their hearing (Case 3). Others report wide variations in the loudness of their tinnitus. For still others, their tinnitus is unilateral when it is relatively quiet, but becomes nonlateralized when the tinnitus is louder. Such phenomena suggest that there are ongoing somatically mediated factors modulating the tinnitus percept.

Diurnal fluctuations in the tinnitus percept also suggest that somatic modulation is operative. Patients who describe their tinnitus as louder upon awakening raise the possibility that somatic factors (such as bruxism—grinding of the teeth) are active during sleep and are causing an increase in tinnitus loudness. Others describe that their tinnitus has usually vanished by the time they awaken and then returns a few hours into the day; this scenario suggests that during the day they are reactivating their tinnitus through somatic mechanisms, such as the tonic muscle contractions required to support the head in an upright position or clenching related to the stress of daily activities. Finally, others describe that their tinnitus is louder after awakening from a nap in a chair; this may relate to somatic factors such as stretching of the neck muscles when their head passively falls forward while dozing in a sitting position.

Although a somatic factor on its own can cause tinnitus, much more frequently somatic factors combine with other factors (such as chronic hearing loss) to act as trigger factors or modulators (Case 3; Fig. 3).

### Head Trauma

The contemporaneous association between head trauma, hearing loss, and tinnitus makes the diagnosis straightforward. The association with trauma becomes less certain when some of these elements are missing, such as a delay between the trauma and the onset of the tinnitus. The longer the delay the less is the confidence of any association between the two. Tinnitus, but no hearing loss following trauma, makes the association more tenuous and raises the possibility that

<table>
<thead>
<tr>
<th>Table 3 Tinnitus properties suggesting a somatic component</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermittency</td>
</tr>
<tr>
<td>Large fluctuations in loudness</td>
</tr>
<tr>
<td>Variability of location</td>
</tr>
<tr>
<td>Diurnal pattern</td>
</tr>
<tr>
<td>No hearing loss, but head or neck disorder</td>
</tr>
</tbody>
</table>

Seminars in Neurology Vol. 33 No. 3/2013
trauma is causing tinnitus through a brainstem mechanism or indirectly such as through a somatic mechanism. Abnormalities of otoacoustic emissions including those involving the brainstem (the contralateral suppression of otoacoustic emissions) have been found in patients with tinnitus and head injury, including some with normal audiograms, raising the likelihood that a brainstem mechanism is accounting for the tinnitus associated with head trauma.49,50

**Postinfectious Tinnitus**

Occasionally, patients report the onset of tinnitus following an upper respiratory infection. Whether this is cause and effect has never been established. Because upper respiratory infections are common and idiopathic tinnitus not uncommon, chance association of the two is to be expected.

**Idiopathic Tinnitus**

Sometimes, despite an exhaustive evaluation of nonspecific tinnitus, no specific diagnosis can be made with any high degree of confidence. Hence, the diagnosis of idiopathic is made.

**Treatment of Tinnitus**

Once the etiology of the tinnitus is tentatively identified, then treating the underlying cause of the tinnitus can lead to its resolution. In a preliminary study of needling of head and neck myofascial trigger points, 25% of patients had total and complete cessation of tinnitus and another 25% experienced decided improvement.45

If the pathological process for the tinnitus cannot be reversed, then suppression of the tinnitus acoustically, pharmacologically, or electrically can be attempted. For people with hearing loss, hearing aids can often act as maskers of their tinnitus. Additionally, when hearing is improved, patients no longer perceive the tinnitus as interfering with their hearing. Consequently, they pay less attention to their tinnitus once their ability to hear external sounds is improved.

With programmable digital hearing aids, it has been reported that there was a more than 50% tinnitus improvement in 65% of subjects, who received one hearing aid, and in 80% of subjects, who received bilateral hearing aids.51 With hearing aids that incorporate linear octave frequency transposition, a very recent study reported a large number of subjects who completely suppressed their tinnitus; these same subjects were said to have had no benefit from hearing aids using classical amplification or nonlinear frequency compression.52

Anecdotal tinnitus suppression has been reported for a large number of medications (fluoxetine, sertraline, paroxetine, risperidone, ginkgo, scopolamine patch, and gabapentin). Only benzodiazepines have been shown to quiet tinnitus in multiple well-designed studies. All three benzodiazepine studies report that in ~75% of subjects the benzodiazepine quiets tinnitus to some degree.46–48 In our clonazepam study using a strict protocol, 7% had their tinnitus completely suppressed. It is likely that in clinical practice the patients who suppress their tinnitus completely will be much higher, because the dose can be individualized and other benzodiazepines can be tried.

Acamprosate was shown to quiet tinnitus in two well-designed studies,53,54 but not in a third.55 One of the studies found that at 90 days the tinnitus was not heard in more than 10% of subjects.54

The medications for trigeminal neuralgia, especially carbamazepine, suppress completely staccato irregular unilateral tinnitus (typewriter tinnitus).

Auditory nerve electrical stimulation has been repeatedly shown to suppress tinnitus in many patients, but no promontory stimulation device is currently available.56,57

If the tinnitus cannot be quieted satisfactorily, then behavioral methods or antidepressants can improve the patient’s tolerance of his or her tinnitus by shifting the patient’s attention away from the tinnitus and/or improving the associated depression.58 Although a recent review of randomized controlled trials concluded that “only studies examining cognitive–behavioral therapy were numerous and similar enough to perform meta-analysis, from which the efficacy of cognitive–behavioral therapy (moderate effect size) appears to be reasonably established,” in the hands of a competent practitioner the behavioral methods may all be about equally effective.59 The other behavioral techniques include tinnitus retraining therapy, neuromonics, mindfulness, binaural beats, and self-hypnosis.60–65

**Hyperacusis**

Hyperacusis refers to reduced sound tolerance. People with hyperacusis report that they find sound intensities considered comfortable by most people to be unbearably loud.66 There is a close relationship between hyperacusis and tinnitus: ~40% of patients with tinnitus report some degree of hyperacusis. Furthermore, though less than 10% of people in the general population have hyperacusis, more than 80% of those with hyperacusis report coexisting tinnitus. In a small number of individuals, hyperacusis is their prime concern; tinnitus is of secondary importance.

There is evidence supporting the notion that sound avoidance as a reaction to new-onset tinnitus creates a negative feedback situation leading to hyperacusis. It has been shown in short-term experiments with people wearing ear plugs that sound avoidance leads to decreased sound tolerance.67–69 The negative feedback then is as follows: Sound avoidance because of tinnitus leads to more sound intolerance, which in turn leads to more sound avoidance. Further support for this concept has come from the success in treating hyperacusis with desensitization programs.70–73 These desensitization programs all have in common the gradual introduction of sounds in a supportive environment that had been previously poorly tolerated, thereby breaking the vicious cycle of negative feedback.

**Conclusion**

In some surveys, ~10% of adults report having chronic tinnitus and one out of 20 of them feel their tinnitus interferes
with their ability to lead a normal life. Yet, in dead silence, between 50 and 90% of adults report an auditory perception. About 75% of adults have experienced sudden unilateral taping tinnitus, typically lasting about a minute or less. About 50% of adults have experienced transient tinnitus following loud noise exposure.

A wide range of disorders can cause tinnitus. Many involve a disturbance of the peripheral auditory system, including various types of hearing loss. However, one of the major, but underappreciated, causes of tinnitus has no involvement with the ear, but causes tinnitus through its connections to the auditory system within the central nervous system. Myofascial disorders of the head and neck cause tinnitus through their activation of the somatosensory system, which then projects to the central auditory system at multiple levels including the dorsal cochlear nucleus and inferior colliculus.

Although myofascial disorders or hearing loss alone can cause tinnitus, they also can combine to cause tinnitus. Therefore, all tinnitus patients should have an audiogram and be examined for a myofascial disorder of the head and neck. Specifically, the history must cover the topics of headache, bruxism, dental disorders, neck and facial pain, trauma, and changes in physical activities such as a new or altered exercise program. The physical exam must include an examination of the head and neck for myofascial disorders and for evidence of tinnitus modulation from activation of the head and neck muscles.

If there is evidence that the tinnitus is related to a myofascial disorder of the head and neck, then a well-designed treatment program directed toward the myofascial disorder can result in a major improvement of the tinnitus in about half of such individuals.

References

25. Fowler EP. Control of head noises: their illusions of loudness and timbre. Arch Otolaryngol 1943;37:391–398