

# 1

## Definitions and Background



Tinnitus is a surprisingly complex subject. Numerous books would be required to adequately cover the current body of knowledge. The present handbook focuses on describing procedures for providing clinical services for tinnitus using the methodology of progressive tinnitus management (PTM).

In this opening chapter we establish common ground with respect to terminology and contextual information. Relevant definitions are provided, many of which are operational due to lack of consensus in the field. Additional background information includes brief descriptions of epidemiologic data, patient data, and conditions related to reduced tolerance (hypersensitivity) to sound.

### Basic Concepts and Terminology

Tinnitus is the experience of perceiving sound that is not produced by a source outside of the body. The “phantom” auditory perception is generated somewhere in the auditory pathways or in the head or neck. Tinnitus often is referred to as “ringing in the

ears.” And, in fact, the word “tinnitus” is derived from the Latin word *tinniere*, which means “to ring.” Patients report many different sounds—not just ringing—when describing the sound of their tinnitus, as we discuss later in this chapter.

### Transient Ear Noise

It seems that almost everyone experiences “transient ear noise,” which typically is described as a sudden whistling sound accompanied by the perception of hearing loss (Kiang, Moxon, & Levine, 1970). No systematic studies have been published to date describing the prevalence and properties of transient ear noise; thus, anything known about this phenomenon is anecdotal.

The transient auditory event is unilateral and seems to occur completely at random without anything precipitating the sudden onset of symptoms. Often the ear feels blocked during the episode. The symptoms generally dissipate within a period of about a minute. Although transient ear noise has been described as “brief spontaneous tinnitus”

(Dobie, 2004b), any reference to tinnitus in this book does not include this auditory phenomenon.

## Chronic Tinnitus

Patients often confuse transient ear noise with chronic tinnitus. What differentiates the two? It has been suggested that tinnitus is ear noise that lasts at least five minutes (Coles, 1984; A. C. Davis, 1995; Hazell, 1995). Dauman and Tyler (1992) suggested that the noise must last at least five minutes *and* occur at least two times per week. These are reasonable criteria to define tinnitus, but superfluous for the typical patient who experiences tinnitus all or most of the time. Nonetheless, a distinction must be made between transient ear noise and chronic tinnitus, and Dauman and Tyler's criteria are sufficient for this purpose.

## Origin of Tinnitus: Somatic Versus Neurophysiologic

By definition, the *perception* of tinnitus results from activity in the auditory nervous system. The neural activity that is perceived as tinnitus can be referred to as a "tinnitus neural signal." As for most sounds that activate the auditory system, at any point in time the tinnitus neural signal may or may not be part of the conscious experience. Whenever tinnitus is consciously perceived, the tinnitus neural signal is undergoing active processing by the auditory cortex (J. A. Henry, Trune, Robb, & Jastreboff, 2007a). Although the final destination of the tinnitus neural signal always is the auditory cortex, the *origin* of tinnitus can be from either outside or within the auditory nervous system (referred to as *somatic* and *neurophysiologic* tinnitus, respectively) (Hazell, 1998a).

If the tinnitus has a somatic origin, it can be referred to as *somatic tinnitus* or *somatosound(s)*. If the tinnitus has a neurophysiologic origin, it can be referred to as *neurophysiologic tinnitus* or *sensori-neural tinnitus*. The word *tinnitus* generally refers to neurophysiologic tinnitus because this is the condition that is experienced by the great majority of patients.

## Somatic Tinnitus (Somatosound)

Somatic tinnitus (*somatosound*) has an origin that usually is vascular, muscular, skeletal, respiratory, or located in the temporomandibular joint (TMJ) (J. A. Henry, Dennis, & Schechter, 2005). These "body sounds" thus have an internal acoustic source (Dobie, 2004b). Theoretically, the acoustic signal associated with any somatosound could be detected and characterized if the proper sensing equipment were available for this purpose.

### Somatosound—Pulsatile Tinnitus

The most common type of somatosound is *pulsatile* tinnitus (Lockwood, Burkard, & Salvi, 2004). Also referred to as *venous hum* or *vascular noise*, pulsatile tinnitus pulses in synchrony with the heartbeat (Sismanis, 2003). There are many potential sites for pulsatile tinnitus, which often can be identified by an experienced physician (Lockwood et al., 2004; Sismanis, 1998; Wackym & Friedland, 2004). It is essential that these patients receive a medical examination as pulsatile tinnitus might indicate a more serious medical problem (such as intracranial and carotid artery abnormalities) (Hazell, 1990; Sismanis, 2007). The condition is treatable in some patients (Sismanis, 1998).

### Nonpulsatile Somatosounds

Somatosounds also can be *nonpulsatile*, meaning that they have a nonvascular source (typically muscular, respiratory, or TMJ). Examples of nonvascular conditions that can cause somatosounds are muscular flutters or spasms and patulous eustachian tube.

### Symptoms of Somatosounds

When evaluating a patient who complains of tinnitus, it is essential to determine if the symptoms suggest a somatic origin. If so, then the possibility exists that the tinnitus is amenable to medical management. Information about diagnosing somatic tinnitus is available in several publications (Hazell, 1990; Levine, 2004; Perry & Gantz, 2000; Schwaber, 2003; Wackym & Friedland, 2004). (Please see Chapters 4 and 5 for further information about somatosounds.)

### **Somatosounds Require Medical Evaluation**

The presence of somatosounds always indicates the need for medical evaluation. The physician (usually an otolaryngologist or otologist) should have expertise in the diagnosis and treatment of somatic tinnitus. If the physician cannot resolve the problem with medical or surgical intervention, then the audiologist should provide appropriate clinical services, as described in later chapters.

### **Neurophysiologic (Sensorineural) Tinnitus**

By far the majority of patients who complain of tinnitus have *neurophysiologic* (or *sensorineural*) tinnitus, that is, tinnitus that originates somewhere within the auditory nervous system. Although there is no known cure for neurophysiologic tinnitus, patients can learn to manage their reactions to tinnitus, thereby improving quality of life.

#### **Origin of Neurophysiologic Tinnitus**

All we can know for certain about the origin of a patient's sensorineural tinnitus is that it is generated somewhere within the auditory nervous system. The cochlea seems a likely site because damage to the cochlea from noise exposure and other factors often results in tinnitus. However, some desperate patients have undergone surgical severing of cranial nerve VIII, which extends from the cochlea to the brain, although it did not stop their tinnitus (Fisch, 1970; House & Brackmann, 1981; Pulec, 1984). This finding indicated that tinnitus can be generated centrally. Evidence is accumulating that supports the central generation of tinnitus (Møller, 2003). Cacace (2003) proposed that central networks of auditory system neurons may be involved in generating and sustaining tinnitus, which would explain the persistence of tinnitus following auditory nerve transection.

Understanding the pathophysiology of sensorineural tinnitus is a goal that is being pursued by an ever increasing number of researchers. Many theories have been proposed regarding tinnitus etiology, but are beyond the scope of this clinically

oriented book. The interested reader has a choice of myriad publications written on this topic (e.g., Bauer, 2004; Eggermont & Roberts, 2004; P. J. Jastreboff, 1990; Kaltenbach, Zhang, & Finlayson, 2005; Kaltenbach, Zhang, & Zacharek, 2004; Nuttall, Meikle, & Trune, 2004; Vernon & Møller, 1995).

### **Neurophysiologic Tinnitus Is a Phantom Auditory Perception**

Phantom sensations such as phantom pain and phantom limb have characteristics that are analogous to those of tinnitus (P. J. Jastreboff, 1990, 1995; Meikle, 1995; Møller, 2003). Tinnitus thus has been referred to as a *phantom auditory perception* (P. J. Jastreboff, 1990). Pain and tinnitus are similar with respect to their physiology, assessment, and treatment (Møller, 1987, 2000). These analogous conditions also may have similar neuropathic generating mechanisms. Because of these similarities, strategies of pain management can offer valuable clues to the management of tinnitus. In fact, the method of psychotherapy, cognitive-behavioral therapy (CBT), has been shown to be effective in treating chronic pain (P. H. Wilson, J. L. Henry, & Nicholas, 1993). The success of CBT for the treatment of pain led to the development of CBT for tinnitus (J. L. Henry & P. H. Wilson, 2001).

### **Subjective Versus Objective Tinnitus**

#### **Objective Tinnitus**

By definition, *objective tinnitus* is tinnitus that is audible to the examiner (Dobie, 2004b). Objective tinnitus is relatively rare and always is a somatosound with an internal acoustic source (which as already discussed indicates an underlying condition requiring a medical evaluation by an otolaryngologist or otologist). Not all somatosounds are detectable by the examiner, and thus not all somatosounds would meet the definition of objective tinnitus. As a somatosound is an acoustically generated signal, the signal should be detectable with proper measurement techniques.

### **Subjective Tinnitus**

*Subjective tinnitus* is perceived only by the patient. Any description of subjective tinnitus comes only from the patient, as there are no means to directly measure the intensity or other characteristics of the tinnitus. *Tinnitus matching* is performed to *indirectly* measure subjective tinnitus, (J. A. Henry, 2004; J. A. Henry, Zaugg, & Schechter, 2005a). For the remainder of this book, the word *tinnitus* generally refers to subjective tinnitus, that is, to neurophysiologic or sensorineural tinnitus that does not have an internal acoustic source. When patients have somatosounds, behavioral methods can be applied to manage reactions to these sounds if medical management does not resolve the symptoms.

### **Auditory Imagery, Auditory Hallucinations, and Musical Hallucinations**

Auditory imagery, auditory hallucinations, and musical hallucinations are different forms of phantom auditory perceptions. There often is confusion about how these different auditory perceptions differ from tinnitus.

#### **Auditory Imagery**

Auditory imagery is a normal phenomenon that occurs for all people. It generally refers to the *imagination of sound*, such as repeating a phone number in one's head, or recalling a musical passage (Kraemer, Macrae, Green, & Kelley, 2005; Seal, Aleman, & McGuire, 2004). "Auditory imaginations" can be under conscious control, but they also can occur outside of conscious control. When not under conscious control, auditory imaginations can be mildly distressing, as when you have a song "stuck in your head."

#### **Auditory Hallucinations**

Auditory hallucinations have been estimated to occur in 10 to 15% of the general population (Nicolson, Mayberg, Pennell, & Nemeroff, 2006; Sommer et al., 2008). Perhaps surprisingly, of those who experience auditory hallucinations, most do not have a psychotic disorder. In the nonpsychiatric popula-

tion, auditory hallucinations have been described in conjunction with various diseases, injury, trauma, bereavement, sensory deprivation, religious experiences, near-death experiences, and drugs (Nicolson et al., 2006). Auditory hallucinations also may have nothing to do with the ability to hear, as people born profoundly deaf can experience them (du Feu & McKenna, 1999; Schonauer, Achtergarde, Gotthardt, & Folkerts, 1998).

Auditory hallucinations occur in 70 to 80% of patients with schizophrenia (Hugdahl et al., 2008). Auditory hallucinations normally ascribed to psychiatric illness can be perceived as "voices, cries, noises, or rarely, music" (Wengel, Burke, & Holemon, 1989, p. 163). Musical hallucinations, however, are not necessarily associated with psychopathology, and tend to occur in people with advancing age and marked hearing loss (Sacks, 2008). Bauman (2004) has distinguished "two basic types of auditory hallucinations—psychiatric auditory hallucinations and nonpsychiatric auditory hallucinations. People with mental illnesses often experience the former, while people who are hard of hearing often experience the latter" (pp. 17–18). Focusing on the latter, nonpsychiatric, type, Bauman makes the following points:

- Auditory hallucinations can consist of music, sounds, or voices.
- "Unformed" auditory hallucinations sound distorted and indistinct, whereas "formed" auditory hallucinations sound clear and recognizable.
- Auditory hallucinations typically are experienced by hard of hearing, socially isolated, elderly people who also have tinnitus.
- People who experience auditory hallucinations typically don't admit to the experience.
- At least 10% of hard of hearing people experience auditory hallucinations.
- Many people actually find auditory hallucinations to be pleasant. Based on this information, it is reasonable to expect some patients to report auditory hallucinations that are not associated with psychopathology.

If a patient who reports auditory hallucinations remains unconvinced that the sounds do not have an external acoustic source, then the experience may indicate presence of comorbid mental illness. In addition, the psychiatric population tends to experience auditory hallucinations as frequent, intrusive, and distressing, whereas the nonpsychiatric population may experience them as more positive and nonthreatening (Choong, Hunter, & Woodruff, 2007). One study has reported data suggesting that auditory hallucinations that occur in nonpsychiatric individuals suggest a general susceptibility to schizophrenia (Sommer et al., 2008).

Whenever auditory hallucinations are reported, patients should be referred to both audiology and mental health for a thorough history and to evaluate the auditory experiences—to determine if the sounds are tinnitus or auditory hallucinations and to determine if mental illness is involved.

### Permanent Versus Temporary Tinnitus

Tinnitus can be a temporary or a permanent condition.

#### Permanent Tinnitus

It is, of course, impossible to determine if and when a person's tinnitus becomes permanent. In general, the longer a person has experienced tinnitus the more likely it is to be permanent. A general guideline is that tinnitus of at least 12 months duration has a high likelihood of being a permanent condition (Dobie, 2004b). However, it also has been suggested that a person must have experienced tinnitus for at least two years before it should be considered permanent (Vernon, 1996).

#### Temporary Tinnitus

Exposure to loud noise can cause temporary threshold shift as well as temporary tinnitus (Nuttall et al., 2004). Tinnitus induced in this fashion likely will resolve within a few days following the insult. Repeated episodes of noise exposure increase the likelihood that the tinnitus will become permanent.

Tinnitus also can be induced by a number of medications and drug interactions (DiSorga, 2001)

(see Chapter 5). Such tinnitus usually is temporary (typically lasting 1 to 2 weeks postexposure), but can be permanent—especially with the use of aminoglycoside antibiotics or the cancer chemotherapeutic drug cisplatin (Fausti, J. A. Henry, & Frey, 1995; Rachel, Kaltenbach, & Janisse, 2002). Aspirin (containing salicylate) is well known to cause temporary tinnitus, although the dosage generally has to be rather high to induce tinnitus (Eggermont, 2004; Puel & Guitton, 2007). Other medications that can cause temporary tinnitus include NSAIDs, loop diuretics, and quinine. Drugs used to treat mental health and sleep conditions also may trigger or exacerbate tinnitus. Patients have reported exacerbation of tinnitus due to alcohol and caffeine.

### Onset of Tinnitus

The onset of tinnitus is described as *gradual* for some and *sudden* for others (Axelsson & Barrenas, 1992). In a population study of older adults with tinnitus, 55% reported a gradual onset, 24% reported a sudden onset, and 21% did not know (Sindhusake, Golding, et al., 2003). Uncertainty about the onset of tinnitus can make it difficult to identify a precipitating event. Indeed, as discussed further below, many patients are unable to identify anything that was associated with the onset of their tinnitus.

#### Recent-Onset Tinnitus

Some patients report that they have experienced tinnitus for only a short period of time, usually measured in weeks or up to a few months. For these patients with *recent-onset tinnitus*, it first is important to rule out vestibular schwannoma or any other medical condition that might be causing the symptoms (which is routine practice for audiologists). If medical causes can be ruled out, an attempt should be made to determine if psychological factors such as stress, anxiety, depression, or lifestyle changes might have triggered the tinnitus onset. Patients always should be questioned about any exposure to loud noise. Identifying a potentially triggering event helps to focus the counseling most appropriately.

Patients with recent-onset tinnitus are particularly susceptible to acquiring fears or concerns that

the internal sound indicates the presence of a serious medical condition. These patients may be quite anxious about the potential ramifications of the auditory symptoms. It therefore is critical to provide them with only positive and reassuring information to allay any fears. They should be counseled that (a) their tinnitus may be a temporary condition; (b) they should protect their ears in the presence of damaging sound (or to avoid loud sounds entirely) to optimize the potential for spontaneous resolution of the tinnitus; (c) tinnitus often raises concerns when it is new, but most people who have long-term tinnitus are not particularly bothered by it (Dobie, 2004b; Hallam, Rachman, & Hinchcliffe, 1984); and (d) if the tinnitus becomes bothersome to feel welcome to contact or return to the clinic to discuss ways to manage their reactions to it.

Some patients may remain unconvinced that the recent-onset tinnitus does not indicate a more serious condition, even after thorough audiologic and medical evaluations. These patients may need to be referred to a mental health clinician for assessment of comorbid psychological conditions.

### **Delayed-Onset Tinnitus**

*Delayed-onset tinnitus* is thought to occur weeks, months, or even years following some precipitating event (e.g., exposure to loud noise, traumatic brain injury, treatment with ototoxic medications, etc.). It is not uncommon for patients to make a claim of delayed-onset tinnitus for litigation purposes. The possibility of such a claim being valid relates to the complex interaction among the presumed precursor event and more recent events that might have triggered the tinnitus onset. Evaluating a claim of delayed-onset tinnitus requires taking a detailed history that covers all possible circumstances that might have caused damage to the auditory system.

A better understanding of the mechanisms of tinnitus generation is needed before the existence of delayed onset of tinnitus can be positively confirmed or rejected (Humes, Joellenbeck, & Durch, 2006). However, it does seem likely that noise exposure or other experiences that could have caused auditory damage can result in delayed-onset tinnitus, even when the tinnitus onset occurs years after the event.

## **Epidemiology of Tinnitus**

### **Prevalence of Tinnitus**

Prevalence estimates from numerous epidemiologic studies indicate that about 10 to 15% of all adults experience tinnitus (H. J. Hoffman & Reed, 2004). The American Tinnitus Association (ATA) estimates that 40 to 50 million Americans experience tinnitus as a chronic condition and that of these, 10 to 12 million seek some form of medical help, and 2.5 million are “debilitated” by their tinnitus (S. C. Brown, 1990). Men have a higher incidence of tinnitus than women, likely due to occupational and recreational differences (H. J. Hoffman & Reed, 2004; Meikle & Walsh, 1984).

### **“Causes” of Tinnitus**

We often refer to “causes” of tinnitus (i.e., tinnitus *etiology*), but in fact we never know the specific cause of sensorineural tinnitus (J. A. Henry, Dennis, et al., 2005). For example, we may say that noise exposure “caused” a person’s tinnitus. The noise probably caused some cochlear damage, but it did not *cause* the tinnitus. Technically, instead of “causes,” we should use terminology that implies indirect causality, such as *precipitating factors, events associated with tinnitus onset, tinnitus precursor events, and tinnitus triggering events*.

### **Auditory Pathologies Associated with Tinnitus**

Numerous auditory pathologies have been associated with tinnitus. Sweetow (1996) has listed these with respect to conductive and sensorineural auditory pathologies (Table 1–1). It is important to realize that anything that can cause hearing loss also can trigger the onset of tinnitus (Coles, 1995; Dobie, 2004b).

### **Risk Factors**

A number of studies have obtained tinnitus epidemiology data in a systematic fashion. H. J. Hoffman and Reed (2004) reviewed these studies and summarized the various factors that were shown

**Table 1-1.** Auditory Pathologies Associated With Tinnitus

Conductive	Sensorineural
Impacted cerumen	Endolymphatic hydrops
External otitis	Perilymph fistulas
Tympanic membrane perforations	Noise damage
Otitis media	Vestibular schwannoma
External auditory meatus tumors	Presbycusis
Cholesteatoma	Viral diseases
Ossicular chain fixation or discontinuity	Bacterial infections
Atresias	Ototoxicity
Otosclerosis	Meningionoma
Carcinoma	And so forth
And so forth	

Source: Sweetow (1996).

**Table 1-2.** Risk Factors for Tinnitus

“Definite” Risk Factors	“Possible” Risk Factors
Acoustic neuroma	Alcohol
Age	Anxiety
Cardiovascular and cerebrovascular disease	Depression
Drugs or medications	Familial inheritance
Ear infections/inflammation	Geographic region
Head/neck trauma and injury	Health status—fair/poor
Hyper- and hypothyroidism	Heavy weight or high body mass index
Loud noise exposure	Limited education
Ménière’s disease	Low height
Otosclerosis	Low socioeconomic status
Presbycusis	Low weight or low body mass index
Sudden deafness	Rural residence
	Smoking (cigarettes)

Source: Adapted from “Epidemiology of Tinnitus,” by H. J. Hoffman & G. W. Reed, 2004, in *Tinnitus: Theory and Management* by J. B. Snow (Ed.), (pp. 16–41), Lewiston, NY: BC Decker Inc. Copyright 2004 BC Decker, Inc. Adapted with permission.

to be associated with tinnitus. Their summary is divided between “definite” and “possible” risk factors (Table 1-2). Thus, people are “definitely” or “possibly” more likely to have tinnitus if these factors apply. These epidemiology data reveal factors that are *correlated* with the presence of tinnitus and thus are not necessarily causative agents.

The most common risk factor for the onset of sensorineural tinnitus is noise exposure (Axelsson & Barrenas, 1992; Penner & Bilger, 1995). Also, a direct correlation exists between degree of hearing loss and prevalence of tinnitus—the odds of having tinnitus increase as hearing loss increases (Coles, 2000). This is true regardless of the type or the cause of the hearing loss. Dobie (2004b) concluded that tinnitus tends to occur more frequently in men, the elderly, blue-collar workers, and people with certain health problems.

**Pathophysiology of Tinnitus**

Many researchers are attempting to discover the pathophysiologic basis of tinnitus, with the ultimate goal of finding a cure for tinnitus. Numerous theories and models have been proposed;

currently, there is no consensus regarding tinnitus mechanisms. It is beyond the scope of this book to go into detail regarding possible mechanisms of tinnitus generation. The interested reader is advised to review numerous excellent publications on this topic (e.g., Baguley, 2002; Eggermont, 2000; Kaltenbach, 2000; Møller, 2003; Tyler, 2006; Vernon & Møller, 1995). Because everything we hear (including tinnitus) results from neural activity in the auditory nervous system, tinnitus-mechanisms research has focused on understanding abnormal neural activity that is associated with tinnitus. Most theories involve hair cells, the auditory nerve, and the central auditory nervous system (J. A. Henry, Dennis, et al., 2005). A few examples of proposed mechanisms include (there are many others):

- *Hair cells:* discordant function between inner and outer hair cells (Jastreboff, 1990); damaged outer hair cells causing excessive release of neurotransmitter (glutamate) from inner hair cells

producing sustained cochlear activity (Patuzzi, 2002).

- *Auditory nerve*: synchronization of spontaneous activity in auditory nerve fibers due to cross-talk (Eggermont, 1990; Møller, 1984, 1995); cortical reorganization following changes in the auditory periphery resulting in a disproportionately large number of neurons becoming sensitive (tuned) to frequencies at upper and lower borders representing peripheral hearing loss (Salvi, Lockwood, & Burkard, 2000).
- *Central auditory nervous system*: Increased spontaneous activity in the dorsal cochlear nucleus (Brozoski, Bauer, & Caspary, 2002; Kaltenbach & Afman, 2000; Kaltenbach et al., 2002; Zacharek, Kaltenbach, Mathog, & Zhang, 2002).

### Data from Tinnitus Clinic Patients

#### Tinnitus Data Archive

The Oregon Health and Science University (OHSU) Tinnitus Clinic was started in 1975 (Vernon & Schleuning, 1978). Extensive data from thousands of patients have been collected, and these data have been used to develop the Tinnitus Data Archive (Meikle, Creedon, & Griest, 2004) (<http://www.tinnitusarchive.org/>). The summarized data in the Archive were collected from 1,630 patients seeking clinical intervention for their tinnitus and are not generalizable to individuals with tinnitus who do not seek intervention.

#### Main Findings of the Tinnitus Data Archive

Some of the main findings of the Tinnitus Data Archive include (J. A. Henry, Dennis, et al., 2005):

- There are about 2½ times more male than female patients.
- 80% of all patients are at least 40 years of age.

- Tinnitus onset is reported as “gradual” or “sudden” about equally.
- Left-sided tinnitus is reported more often than right-sided tinnitus.
- More than half of the patients describe their tinnitus as a single sound (most of the remainder identify two or more sounds).
- Most patients describe their tinnitus as “ringing” or “clear tone” (3% report “hum,” “clicking,” “roaring,” or “pulse”).
- 85% of patients indicated that their perceived tinnitus loudness was a 5 or more on a 0 to 10 loudness-rating scale (10 = “very loud”).

#### Factors Associated with Tinnitus Onset

When OHSU Tinnitus Clinic patients were asked to describe the circumstances of their tinnitus onset, 43% indicated that no known events were associated. Most of the remainder reported that one factor was associated with their tinnitus onset (8% reported more than one factor).

For those patients describing factors associated with their tinnitus onset, the factors could be placed into one of four broad categories (Meikle et al., 2004): (1) noise-related; (2) head and neck trauma; (3) head and neck illness; and (4) other medical conditions.

#### Years “Aware of” Tinnitus

OHSU Tinnitus Clinic patients are asked how long they have been aware of experiencing tinnitus. From the Tinnitus Data Archive (Meikle et al., 2004), 40% of patients had experienced their tinnitus for 2 years or less; 55% for 5 years or less; 70% for 10 years or less; and 85% for 20 years or less.

#### Types of “Sounds” Patients Hear

Patients are asked to describe what their tinnitus sounds like from a list of sounds commonly reported by patients, or by describing a sound that is not on the list. The most common sound reported by far



is “ringing.” The second most common sound is “hissing.” The third most common sound is “clear tone.” Numerous additional sounds are reported, including “high-tension wire,” “buzzing,” “transformer noise,” “sizzling,” “crickets,” “whistle,” “hum,” and “clicking.”

### Intermittency of Tinnitus

From the Tinnitus Data Archive, 91% of patients reported that their tinnitus is a constant sound. In 5% of patients, tinnitus is intermittent and heard more than 50% of the time. In 1% of patients, tinnitus is intermittent and heard less than 50% of the time.

### Reduced Tolerance (Hypersensitivity) to Sound

“Hyperacusis” often is reported concurrently with tinnitus, and audiologists need to know how to recognize hyperacusis and how to provide appropriate intervention if it is a significant condition. There is no consensual definition of hyperacusis (P. J. Jastreboff & M. M. Jastreboff, 2004; Vernon, 2002). It has been defined as “the collapse of loudness tolerance so that almost all sounds produce loudness discomfort” (Vernon & Press, 1998). At the other extreme, clinics have reported that up to half of their patients experience decreased loudness tolerance (Coles, 1996; Gold, Frederick, & Formby, 1999; Hazell, 1999; P. J. Jastreboff, 2000). When evaluating the patient, the critical factor is to determine if loudness sensitivity is a significant problem in the patient’s life.

Our definitions of conditions pertaining to decreased loudness tolerance are consistent with those published by J. A. Henry, Zaugg, and Schechter (2005a), which were adapted from P. J. Jastreboff and Hazell (2004).

### Hyperacusis

Hyperacusis is a physical condition of discomfort or pain caused by sound. The effect is restricted primarily to the auditory pathways. Thus, a con-

dition of “pure” hyperacusis causes physical discomfort, but no emotional responses are involved. For a given patient with pure hyperacusis, sound would be uncomfortably loud at levels most people find comfortable—*regardless of the type of sound*. This means that a patient who reports that he or she can tolerate some sounds at louder levels than other sounds is probably not experiencing pure hyperacusis.

### Misophonia

M. M. Jastreboff and P. J. Jastreboff (2002) introduced the term misophonia. The term means “dislike of sound,” and implies that there is an emotional reaction to sound. A misophonic reaction is a learned response. This means that a misophonic patient might report that a particular sound is problematic in some situations, but not in others. When questioned in detail, these patients often report reactions that would be inconsistent with pure hyperacusis. For example, they might report that certain “unpleasant” sounds become uncomfortably loud at levels below which “pleasant” sounds are tolerated comfortably.

### Phonophobia

P. J. and M. M. Jastreboff (2000) defined use of the term phonophobia for clinical application. Phonophobia is a fear response caused by sound, and is considered a subcategory of misophonia. Misophonia can cause any kind of negative emotional response, but phonophobia specifically causes a fear reaction. A defining feature of phonophobia is the *anticipation* that sound will be uncomfortably loud. Thus, phonophobia refers to a person’s state of mind with respect to sounds and sound environments.

### Loudness Recruitment

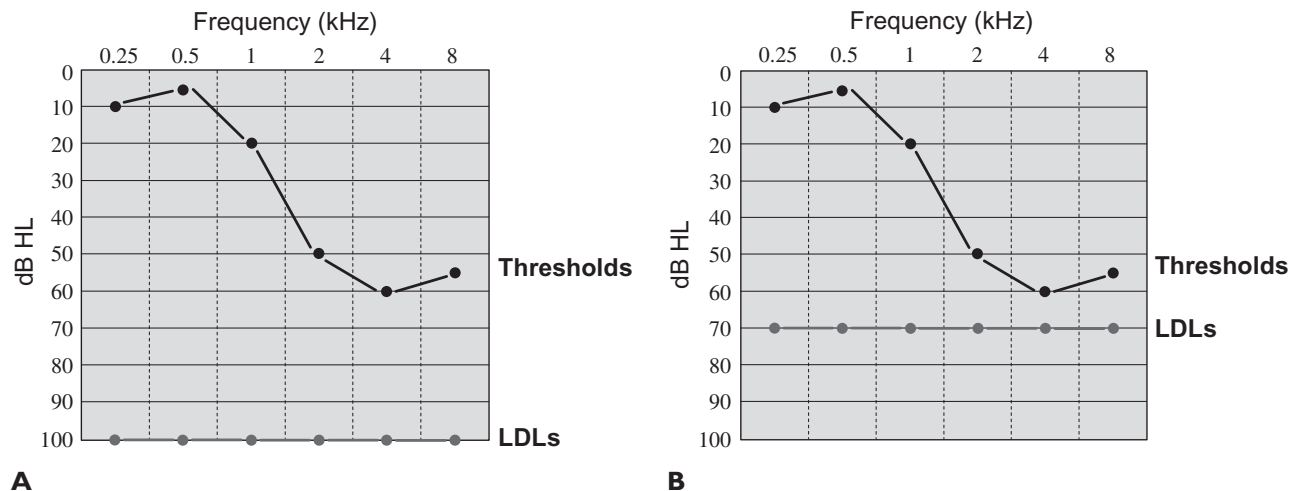
Loudness recruitment often is confused with hyperacusis (Vernon, 2002). Recruitment refers to abnormally rapid growth in the perception of loudness (Vernon, 1976). It usually is a phenomenon of cochlear

or sensorineural hearing loss. Recruitment generally is associated with reduced auditory thresholds and normal loudness discomfort levels (Figure 1–1). Thus, the dynamic range is compressed, but there is normal tolerance to louder sounds.

### Treatment for Conditions of Reduced Sound Tolerance

In Chapter 6, we describe methodology for the treatment of reduced sound tolerance. The overall approach is to first determine if the condition

is a severe problem for the patient. If so, then the patient should receive special treatment that focuses on the condition. If the condition is a mild or moderate problem, then the patient needs to be educated about the *sensitizing* effects of using hearing protection, and the *desensitizing* effects of using therapeutic sound (Formby & Gold, 2002). Normally, the use of sound that is advocated for tinnitus management will indirectly provide adequate treatment for reduced sound tolerance in mild and moderate cases. Consultation with a mental health provider might be useful if a patient fears sound and/or has other intense fears.



**Figure 1–1.** Tolerance to sound can be estimated by obtaining loudness discomfort levels (LDLs), which indicate the threshold level at which sound becomes uncomfortably loud. **A.** LDLs at and above about 100 dB HL are in the normal range. Note that the hearing sensitivity is reduced in the higher frequencies, resulting in a compressed dynamic range at those frequencies—and loudness recruitment. However, the LDLs at 100 dB HL reflect normal tolerance to louder sounds. **B.** LDLs are reduced to 70 dB HL, indicating a condition of reduced loudness tolerance. From *Tinnitus Retraining Therapy: Patient Counseling Guide* (p. 168), by J. A. Henry, D. R. Trune, M. J. A. Robb, & P. J. Jastreboff, 2007, San Diego, CA: Plural Publishing, Inc. Copyright 2007 by Plural Publishing. Reprinted with permission.