
THEORETICAL/REVIEW ARTICLE

Tinnitus is an increasing health concern across all strata of the general population. Although an abundant amount of literature has addressed the many facets of tinnitus, wide-ranging differences in professional beliefs and attitudes persist concerning its clinical management. These differences are detrimental to tinnitus patients because the management they receive is based primarily on individual opinion (which can be biased) rather than on medical consensus. It is thus vitally important for the tinnitus professional community to work together to achieve consensus. To that end, this article provides a broad-based review of what is presently known about tinnitus, including prevalence, associated factors, theories of pathophysiology, psychological effects, effects on disability and handicap, workers’ compensation issues, clinical assessment, and various forms of treatment. This summary of fundamental information has relevance to both clinical and research arenas.

KEY WORDS: tinnitus, treatment, hearing disorders, mechanisms, review

Tinnitus is the perception of sound for which there is no acoustic source external to the head. It poses a significant clinical problem for millions of people in the United States and is proportionally problematic in countries where epidemiological data have been reported. Although an abundance of literature concerning tinnitus has been published in the last 25 years, there is lack of uniformity with respect to all aspects of its clinical management. Furthermore, understanding of pathological mechanisms of tinnitus generation remains in the stage of hypothesis and conjecture. This article provides an overview of what is presently known about tinnitus.

Definitions

Many people may experience transient “ear noises,” which are often described as a whistling sound, in association with sudden temporary hearing loss (Kiang, Moxon, & Levine, 1970). These transient auditory symptoms occur periodically and generally resolve within a few minutes. Consensual criteria that differentiate such normal ear noises from pathologic tinnitus have not been developed. Some authors have specified that tinnitus must exceed a 5-min duration (Coles, 1984; Davis, 1995; Hazell, 1995). Dauman and Tyler (1992) proposed that pathologic tinnitus is head noise lasting at least 5 min that occurs more than once per week. Both of these definitions would constitute...
low-fence criteria to define an internal sound that is present most or all of the time for the typical tinnitus patient (Meikle, Creedon, & Griest, 2004).

A distinction is often made between subjective and objective tinnitus (A. R. Møller, 2003). Subjective tinnitus refers to an internal sound that is perceived only by the patient, whereas objective tinnitus is considered real noise that can be heard by the patient and the examiner (Ciocon, Amede, Lechtenberg, & Astor, 1995; Lockwood, Salvi, & Burkard, 2002; Perry & Gantz, 2000). Hazell (1995) objected to this distinction on the basis that tinnitus is, by definition, always subjective. Hazell distinguished between tinnitus that is generated either neurophysiologically or somatically. Somatic tinnitus (somatosounds) would usually have a vascular, muscular, respiratory, or temporomandibular joint (TMJ) origin. The presence of somatosounds normally indicates an underlying medical condition that warrants medical evaluation. Some of the causes of somatosounds include a variety of vascular lesions, benign intracranial hypertension, high cardiac output, middle-ear disease, patulous Eustachian tube, and palatal myoclonus (Ciocon et al., 1995; Hazell, 1995; Perry & Gantz, 2000). Regardless of the cause of tinnitus, the signal is eventually processed by the central auditory nervous system and consciously perceived in the auditory cortex.

**Prevalence**

The prevalence of tinnitus has been estimated on the basis of data obtained from epidemiologic studies conducted in different countries (Brown, 1990; Davis, 1995; Hinchcliffe, 1961; Leske, 1981; Office of Population Census and Surveys, 1983; Sindhusake et al., 2003). These studies contained at least one question about tinnitus, either written or conducted by interview, that was administered to random population samples (Davis & Refaie, 2000). Unfortunately, the questions used were not standardized, and they did not always differentiate normal ear noises from pathologic tinnitus. In spite of their limitations, these studies indicated that the prevalence of tinnitus in adults falls in the range of 10% to 15%. Hoffman and Reed (2004) compared six studies that obtained age-specific tinnitus prevalence data in adults. Each of these studies showed a trend of increasingly greater prevalence at higher age decades. The studies also showed a plateau in tinnitus prevalence in either the 60–69 years or the 70–79 years age ranges, with a subsequent decline in prevalence for higher age groups.

**Severity**

The term severity in a clinical context refers generally to the impact of a health condition on quality of life. With respect to tinnitus, its degree of severity would reflect the “nature and extent of patients’ tinnitus-related problems” (Meikle, 2003, p. 59). The prevalence of tinnitus is much higher than the number of patients who seek treatment (Brown, 1990; Davis, 1995; Hinchcliffe, 1961; Leske, 1981; Office of Population Census and Surveys, 1983), thus indicating that many individuals who experience tinnitus do not find it to be a significant or debilitating problem. Tinnitus becomes a problem when it is perceived as a threat, appears continually intrusive, or when patients have difficulty coping with tinnitus as a stress factor (Hazell, 1998b). In many patients, the emergence of tinnitus as a problem occurs long after the underlying medical condition (most commonly hearing loss). The trigger for the adverse or intrusive effects of tinnitus is sometimes unrelated to the associated condition. Emotional stress, psychological factors, bereavement, unemployment, or various physical or mental illnesses can trigger intrusive tinnitus. In such cases, the patient’s focus and preoccupation with tinnitus can produce a repeating cycle of annoyance, mood changes, fear, anxiety, and depression—all of which are associated with tinnitus severity.

**Use of Questionnaires to Assess Severity**

Proper methodology for measuring tinnitus severity has been debated for years (Dobie, 2002; Meikle & Griest, 2002; Tyler, 1993). There are at least a dozen published outcome instruments that are used to obtain tinnitus severity ratings, and there is no consensus regarding their use across tinnitus treatment centers (Newman & Sandridge, 2004).

**Index scores.** Most tinnitus questionnaires provide an index score to quantify the impact of tinnitus on the patient’s everyday life. For example, individual index scores for the Tinnitus Severity Index (TSI; Meikle, Griest, Stewart, & Press, 1995) can range from 0 to 48—from lesser to greater severity. Some tinnitus instruments specify different ranges of their index scores to categorize levels of tinnitus severity. A patient’s score on the Tinnitus Handicap Inventory (Newman, Sandridge, & Jacobson, 1998), for example, places the patient into one of four “handicap severity” categories (none, mild, moderate, and severe).

Although an index score is generally helpful in establishing the need for treatment, it can over- or underestimate tinnitus severity. This point is illustrated by a controlled clinical trial that we recently completed (J. A. Henry, 2004b; J. A. Henry, Schechter, Regelein, & Dennis, 2004). For the trial, it was important to ensure that study participants had enough of a problem with their tinnitus to justify 18 months of individualized management. We carefully screened...
about 800 individuals to assess their tinnitus severity, of which 123 were enrolled (and 111 completed treatment). The mean TSI index score (with a possible range of 0–48) for the 123 participants was 28.3 (SD = 8.7). The rigorous screening process ensured that all of these participants had tinnitus of such severity as to qualify for long-term treatment. Their TSI scores, however, ranged from 9 to 48—spanning 75% of the possible range. Participants in our study also completed the Tinnitus Handicap Questionnaire (Kuk, Tyler, Russell, & Jordan, 1990; Tyler, 1993) and the Tinnitus Handicap Inventory (Newman, Jacobson, & Spitzer, 1996; Newman et al., 1998). The baseline index scores for these instruments showed intersubject variability comparable with that for the TSI. Clearly, an examiner cannot rely solely on an index score to make a clinical severity judgment.

**Impediments to the effectiveness of tinnitus questionnaires.** The objective for any tinnitus questionnaire is to accurately identify and quantify each patient’s tinnitus-associated problems. There are two primary reasons why accomplishing this objective has been so difficult. First, effects of tinnitus on an individual are often multidimensional. Tyler and Baker (1983) obtained lists of difficulties caused by tinnitus from members of a tinnitus self-help group. These authors commented, “Perhaps the most striking aspect of these findings is the diversity and gravity of the difficulties that were reported” (p. 152). Tyler (1993) commented, “There are thousands of possible questions one could ask about the distress caused by tinnitus” (p. 378).

The second primary factor hindering the development of a universally effective tinnitus questionnaire is the need to properly weight the different problematic aspects of tinnitus. A question may be irrelevant to the patient’s circumstances, yet the question may receive equal weight when calculating the index score (Newman & Sandridge, 2004). If a questionnaire addresses mostly irrelevant items for a particular patient, the index score might be spuriously low. For example, a patient’s only reported problem may be difficulty falling asleep. The questionnaire used might focus more on emotional consequences of tinnitus, in which case this patient’s index score would be low, even though an individual question about sleep disturbance might indicate a severe problem. Another patient might report being generally bothered by tinnitus most of the day. This patient’s problem would affect most life activities, which would result in a correspondingly high index score. These two hypothetical patients each have a severe problem with tinnitus. The latter patient would provide an index score reflecting severe tinnitus, whereas the former patient would not.

**Individual outcome questions.** Another option for assessing overall tinnitus impact is to use a single outcome question that is global in nature. In our clinical trial, we asked global-severity questions such as, “How much of a problem is your tinnitus?” and “How annoying is your tinnitus?” Responses were based on 0–10 analog scales. The mean responses for the 123 enrolled participants were 6.5 (SD = 2.0; range = 2–10) and 6.4 (SD = 2.2; range = 0–10) for the two questions, respectively. These mean responses are what might be expected for a group of patients such as these who require intensive rehabilitative management. The ranges of individual responses, however, indicate that a single overall response is often not an accurate gauge of severity.

**Intake interviews.** The use of written, self-administered questionnaires is important for any tinnitus assessment, but they should be used with an awareness of the above-mentioned caveats. Determination of the severity of tinnitus is a very complex issue. It requires an astute examiner to identify specifically how tinnitus is a problem for a patient, and to what degree. Written questionnaires, which have been documented for response validity (such as the TSI), are generally helpful in this process. In addition, an intake interview is helpful and should be conducted. We have published two in-depth interviews that can be used for this purpose, one specific to management with tinnitus masking (Schechter & Henry, 2002) and one for use with tinnitus retraining therapy (TRT; J. A. Henry, Jastreboff, Jastreboff, Schechter, & Fausti, 2003). A properly conducted interview is informative for the examiner in revealing each patient’s concerns, which will need to be addressed in managing the problem. The interview also can serve to provide the clinician opportunities to answer a patient’s questions about the tinnitus condition.

**Quality of Life**

It is largely unknown why about 80% of the people with chronic tinnitus suffer little or no treatment-seeking effects from their tinnitus, whereas about 20% manifest a clinically significant condition (Davis & Refaie, 2000; P. J. Jastreboff & Hazell, 1998). Assessing the effects of tinnitus on an individual’s quality of life is a complex issue than is due to a variety of factors: (a) Tinnitus, like pain, is an entirely subjective experience that can only be described by patient report (Jacobson, 2000; Meikle, 2003); (b) results of psychoacoustic testing of tinnitus perception have been shown repeatedly to have little if any correlation with the degree of tinnitus impact (Coles, 2000; J. A. Henry & Meikle, 2000; Sullivan et al., 1988); (c) as with chronic pain syndromes, psychological factors may play a critical role in determining an individual’s reactions.
to tinnitus (e.g., some people are accustomed to solving their own problems whereas others need a great deal of support; Erlandsson, 2000; Sullivan et al., 1988); (d) women are more likely to report emotional reactions to their tinnitus than men (Dineen, Doyle, & Bench, 1997; Hallberg & Erlandsson, 1993; Meikle & Griest, 1989); (e) the rate of personality disturbances is greater for male than for female tinnitus patients (Erlandsson, 2000); and (f) there are individual differences in daily lifestyle, as well as individually distinct acoustic environments, that may make some patients more prone to intrusive tinnitus.

The effects of tinnitus on quality of life are highly individualized, and personality characteristics may predispose a person to experience tinnitus as a “distressing” symptom (J. L. Henry & Wilson, 2001). Subjectively loud tinnitus can, however, be an intrusive and challenging condition for anyone so afflicted. Quality of life is most certainly reduced to some degree for anyone with chronic tinnitus, as its mere presence precludes the serenity that many associate with a quiet environment. Accordingly, the notion of peace and quiet is no longer an option for many tinnitus patients. It is not surprising, therefore, that sleep disturbance is reported by about one half of those individuals who complain of tinnitus (Erlandsson, 2000; Jakes, Hallam, Chambers, & Hinchcliffe, 1985; Meikle & Walsh, 1984; Tyler & Baker, 1983).

Sleep deprivation is one of the primary effects of tinnitus, along with the effects on cognition, emotional status, and hearing (Dobie, 2004b; Tyler, Noble, Preece, Dunn, & Witt, 2004), and can manifest as a single independent problem. These primary effects of tinnitus, however, are not mutually exclusive and there is overlap for most patients. For example, chronic sleep deprivation caused by tinnitus may result in trouble focusing attention (effects on cognition) and give rise to associated frustration and anger (effects on emotional status). These kinds of effects can be self-perpetuating, with the potential to affect every aspect of life. A comment is in order concerning patients’ common complaint that tinnitus affects their hearing. Although this is certainly possible, tinnitus is generally not a significant factor contributing to an audiometric deficit (Coles, 1995; Dobie, 2004b; Zaugg, Schechter, Fausti, & Henry, 2002).

One’s reaction to tinnitus can affect further the ability (or desire) to interact normally with other people and can result in a state of chronic stress that can impact such basic activities as eating, driving, and performing any kind of chore. Sahley and Nodar (2001) have proposed a model to explain how stress can exacerbate tinnitus. Their idea is that glutamate activity at the synaptic bases of inner hair cells is enhanced in response to opioid dynorphins that are released into the synapses during stressful situations. Clinical depression and anxiety are additional potential sequelae for patients who are most affected by tinnitus (Dobie, 2003; Halford & Anderson, 1991; Kirsch, Blanchard, & Parnes, 1989).

**Conceptualization of Tinnitus Impact**

The need to provide uniform and reliable clinical measures for defining the negative impact of tinnitus has been recognized for many years. One approach that has been suggested for meeting this need is to use the concepts of impairment, disability, and handicap, as defined by the World Health Organization (WHO; 1980; see also, Newman et al., 1996; Shulman, 1991; Tyler, 1993). Meikle (2003) described a uniform conceptual framework for assessing tinnitus severity on the basis of the WHO method for categorizing the negative effects caused by chronic conditions. Meikle argued that such standardization would provide a rational basis for stratifying tinnitus patients into different groups and would further enable meta-analyses and other types of comparisons among treatment centers.

WHO (2001) now uses a new International Classification of Functioning, Disability and Health (ICF). The new model conceives of a person’s disability as a dynamic interaction among health conditions and personal factors and is referred to as the ICF interactive model. Using this model, disability is an umbrella term covering three levels of reduced function: impairment of body structure or function (body level), limitation in activities (person level), or restriction in participation (society level; Australian Institute of Health and Welfare, 2002). Each of these levels should be addressed when treating any form of disability.

A health condition can cause three hierarchical levels of life impact, which are shown in Figure 1. At the lowest level, the condition would cause a “problem in body function such as significant deviation or loss” (Australian Institute of Health and Welfare, 2002, p. 3). A problem in body function can lead to activity limitation, which refers to difficulties an individual may have in executing activities. Activity limitation can further lead to participation restriction, which refers to problems an individual may experience being involved in life situations. There also are environmental and personal factors that can be either barriers to, or facilitators of, the person’s ability to function.

Tinnitus impact on function can be conceptualized for a typical patient using the ICF interactive model shown in Figure 2. The “health condition” is tinnitus, and the “problem in body function” for the patient is the perception of tinnitus as a loud ringing sound.

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The patient’s main tinnitus-related problem is that the tinnitus makes it difficult to concentrate (i.e., activity limitation), and, in turn, the concentration difficulty hinders work performance (i.e., participation restriction). The main environmental factor influencing the patient’s tinnitus-related problem is that the family is unsympathetic toward the tinnitus condition and does not provide emotional support. The main contributing personal factor is the patient’s predisposition to anxiety. The model clarifies how these different components and factors interact with each other to determine the degree of impact on human function.

On the basis of the ICF interactive model, 100% of individuals with permanent tinnitus have an impaired body function because of the condition. For the 20% of individuals with clinically significant tinnitus (Davis & Refaie, 2000; P. J. Jastreboff & Hazell, 1998), the associated problems impact to some degree at the activity-limitation and participation-restriction levels.

Impact on Society

Workers’ Compensation

Workplace noise can be a significant hearing-health hazard (Gabriels, Monley, & Guzeleva, 1996), not only in causing hearing loss but also in precipitating tinnitus (Axelsson & Barrenas, 1992). Severe tinnitus is frequently more disabling than hearing loss, but tinnitus has not typically been monitored in noise conservation programs (Gabriels et al., 1996). Tinnitus that is associated with noise-induced hearing loss can severely affect or even end a person’s occupation (Coles, 2000). In spite of these realities, workers’ compensation boards are inconsistent in how they address tinnitus claims (Tyler, 2003). In some cases, compensation boards will not accept a claim for tinnitus by itself; rather, they assign impairment secondary to hearing loss. Tinnitus is, however, becoming increasingly the primary or only complaint, and awards for tinnitus can greatly exceed those for hearing loss (Coles, 2000).

Workers’ compensation programs in 29 of the 50 United States compensate workers for tinnitus (Dobie, 2001). In 13 of these states, tinnitus is compensated only if hearing impairment is also present. In most states, statutes of limitations (which define the period within which legal action can be taken) range from 1 to 5 years. In some states, the statute of limitations is only 30–200 days.

Vernon (1996) and Tyler (2003) have noted that tinnitus litigation involves establishing the presence, etiology, and severity of tinnitus. Vernon also noted the necessity of establishing the permanency of tinnitus, and Dobie (2001) stressed the importance of historic documentation. Because tinnitus is by nature subjective, there is no objective measure to prove its existence or to verify the reported severity. Tinnitus assessment, like pain assessment, depends on subjective scaling, self-report, and medical history. Thus, any litigation involving tinnitus must rely on the reliability of tinnitus psychoacoustic measures, consistency of verbal responses
Economic Impact

The U.S. Department of Veterans Affairs (VA) regards tinnitus as a disabling condition (J. A. Henry, Schechter, et al., 2004). U.S. military veterans can submit claims for tinnitus that they believe was caused by exposure to noise during their military service. Approved claims usually result in a monetary benefit as compensation for the disability. As of September 2004, 289,159 veterans had received a disability award for their tinnitus, and most of these individuals were entitled to receive monthly compensation benefits for their service-connected disability. The total yearly compensation amount was estimated at $345,495,552. Every veteran who is service-connected for tinnitus (whether they receive compensation benefits) is eligible for clinical services at VA medical centers.

There are no known studies that have attempted to estimate the economic impact of tinnitus on the general population. Reich (2002) addressed this issue and concluded, “It isn’t possible to assign a specific cost figure to tinnitus” (p. 18). To conduct such a study would require obtaining estimates of (a) loss of job productivity, (b) medical costs associated with tinnitus, (c) costs of litigation associated with tinnitus, and (d) compensation awards and payments for tinnitus. These costs must be substantial, as evidenced by the cost to the Veterans Health Administration. Moreover, the overall costs are expected to increase as the public becomes better educated about tinnitus and as noise exposure increases as a problem in our society.

Factors Associated With Tinnitus

Many factors are known to be associated with tinnitus. These factors are often considered “causes” of tinnitus when a connection between the two is established. An event, such as impulse noise, may indeed cause the onset of tinnitus. The mechanism responsible for sustained tinnitus, however, remains unknown even when the causal event is unequivocal. The present understanding of cellular events and other pathologic mechanisms that underlie tinnitus onset is insufficient to enable their identification even when a tinnitus-causing event seems obvious. We therefore use the terms cause and etiology in the context of events leading to the onset of tinnitus.

It is often reported that the most common cause of tinnitus is noise exposure (Axelsson & Barrenas, 1992; Penner & Bilger, 1995). Medications are frequently associated with permanent or temporary tinnitus. More than 300 prescription and over-the-counter medications list tinnitus as a side effect (DiSorga, 2001). Less known are the complex interactions between multiple medications that may contribute to the etiology of tinnitus. Other factors associated with tinnitus include aging, head and neck injuries, vascular and cerebrovascular diseases, systemic disorders, infectious disease, autoimmune disorders, ear conditions, and TMJ disorders (Perry & Gantz, 2000; Vernon & Møller, 1995).

In many cases, the cause of tinnitus is identified as idiopathic (Meikle & Griest, 1989). Patients from the Oregon Tinnitus Clinic (OTC) complete extensive questionnaires, including a description of what they believe has caused their tinnitus. In a sample of 2,369 of these patients, 40% reported no known events associated with their tinnitus onset. Patients who could identify a precipitating event revealed four general categories of tinnitus etiology: (a) noise-related, (b) head and neck trauma, (c) head and neck illness, and (d) other medical conditions. The percentages by category of the associated condition (and subcategories of each condition) for the OTC patient sample are shown in Table 1.

Hearing Loss and Aging

There is a clear relation between hearing loss and tinnitus (Axelsson & Barrenas, 1992; Davis & Refaie, 2000; Meikle, 1991), and the majority of tinnitus patients have some degree of hearing loss (Axelsson & Ringdahl, 1989; Davis & Refaie, 2000; J. L. Henry & Wilson, 2001; Vernon, 1998). Vernon and Meikle (2000) reported that 70% to 80% of tinnitus patients have “significant hearing difficulties” (p. 327).

Approximately 34 million Americans are hearing impaired (Blackwell, Collins, & Coles, 2002). Because hearing loss and tinnitus are so closely related, populations with more prevalent hearing loss have a correspondingly greater prevalence of tinnitus (Hoffman & Reed, 2004). The prevalence of hearing loss increases with age, especially after age 65 (Axelsson & Barrenas, 1992; Beck et al., 2002). Thirty percent of all individuals between the ages of 65 and 74 years and 50% of those persons 75 years and older have hearing loss (Blackwell et al., 2002).

Although tinnitus is commonly associated with hearing loss in older patients, other medical factors become increasingly prevalent and must be considered as potential causes of tinnitus. These factors include conditions such as vascular disease, middle-ear disease, diabetes, hypertension, autoimmune disorders, and degenerative neural disorders, with or without concomitant hearing loss (Perry & Gantz, 2000). Furthermore, these medical conditions are accompanied by increasing use of medications, which may also cause tinnitus emergence or
Two percent of a sample of 1,630 OTC patients reported medications as the cause of their tinnitus (Meikle et al., 2004). There are also changes in life that occur with aging. These changes, such as illness, retirement, loss of function, loss of spouse or friends, or reduced social activity may bring on changes in mood, depression, and anxiety. These stressful life events have the potential to increase the perceived loudness of tinnitus or to exacerbate tinnitus reactions (J. L. Henry & Wilson, 2001). Some patients report stress as the precipitating factor for their tinnitus (Meikle & Griest, 1989).

The increase in tinnitus prevalence in older patients does not necessarily mean that tinnitus as a separate and distinct symptom will increase with age (Hoffman & Reed, 2004). The International Work Group on Hearing Problems and the Elderly (Salamon, 1986) concluded that the incidence of tinnitus was not greater than that expected for older patients with hearing loss and other age-related diseases; in addition, the pathophysiology of tinnitus in older patients was the same as that in younger patients. Others have argued that age-related tinnitus exists as a distinct pathology and is related to degeneration at all levels of the auditory system (McFadden, 1982). For the aged population, tinnitus may go unreported or may be given less importance in the context of other significant medical problems.

### Psychological Aspects of Tinnitus

Pain and tinnitus may cause emotional and psychological distress that is out of proportion to the magnitude of the injury. Because both conditions are subjective phenomena, which are most reliably measured by self-report, standardized scales can be helpful in quantifying the associated psychological reactions (Farrar, Portenoy, Berlin, Kinman, & Strom, 2000; Newman & Sandridge, 2004). Moreover, both pain and tinnitus are associated with dysfunctional, inappropriate coping strategies or ideations (Sweetow, 2000). In this regard, tinnitus is similar to other phantom sensations such as phantom pain and phantom limb (P. J. Jastreboff, 1990, 1995).

Not all patients experience tinnitus in the same way, and intrinsic and extrinsic factors such as personality, psychosocial factors, and environment contribute to the patient’s tinnitus reaction (P. House, 1981). Some patients barely notice tinnitus, whereas others are severely affected by difficulty with concentration, sleep disturbances, anxiety, depression, or despair (Tyler & Baker, 1983). Hallam, Rachman, and Hinchcliffe (1984) noted that the “majority” of people with tinnitus generally develop a tolerance to the symptom and that the great majority of patients learn to accept tinnitus as “part of their environment” within a year (Hazell, 1979, p. 88). Furthermore, the number of problems people associate with their tinnitus is greatest when tinnitus has been present for only a short period of time (Tyler & Baker, 1983). Hallam et al. surmised that an adaptation effect seems to take place that causes tinnitus to become less of a problem over time. On the basis of these, and a number of other observations, the authors proposed “an habituation model of tolerance for tinnitus” (Hallam et al., 1984, p. 44) that was designed to explain this apparent adaptation effect. It is important that they postulated that habituation to tinnitus is the “normal” state, whereas the inability to habituate occurs far less frequently and is due primarily to certain psychological factors.

Sound carries meaning. Sound can evoke strong emotional reactions because of its importance to survival and because it is the primary medium for spoken language (Hallam et al., 1984; Hazell, 1999; Iversen, Kupfermann, & Kandel, 2000; Sweetow, 1995). Our ears are always searching the world around us for meaningful or threatening sounds. Most sounds that have little meaning or are not interpreted as a threat are quickly habituated (not reacted to) by the central

#### Table 1. Conditions associated with tinnitus, as reported from a group of 2,369 Oregon tinnitus clinic patients.

<table>
<thead>
<tr>
<th>Condition</th>
<th>% (as single cause)</th>
<th>As single cause</th>
<th>As one of multiple causes</th>
</tr>
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<tbody>
<tr>
<td>Noise related</td>
<td>18%</td>
<td>22%</td>
<td></td>
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<tr>
<td>Long-duration noise</td>
<td>10%</td>
<td></td>
<td></td>
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<tr>
<td>Explosion</td>
<td>5%</td>
<td></td>
<td></td>
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<tr>
<td>Brief intense noise</td>
<td>3%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head and neck trauma</td>
<td>8%</td>
<td>17%</td>
<td></td>
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<tr>
<td>Head injury</td>
<td>4%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whiplash/cervical trauma</td>
<td>3%</td>
<td></td>
<td></td>
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<tr>
<td>Concussion</td>
<td>&lt;1%</td>
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<td></td>
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<tr>
<td>Skull fracture</td>
<td>&lt;1%</td>
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<tr>
<td>Head and neck illness</td>
<td>8%</td>
<td>10%</td>
<td></td>
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<tr>
<td>Ear infection, inflammation</td>
<td>3%</td>
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<td></td>
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<tr>
<td>Cold/sinus infection</td>
<td>3%</td>
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<td></td>
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<tr>
<td>Other ear problems</td>
<td>2%</td>
<td></td>
<td></td>
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<tr>
<td>Sudden hearing loss</td>
<td>1%</td>
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<tr>
<td>Allergies/hay fever</td>
<td>1%</td>
<td></td>
<td></td>
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<tr>
<td>Other medical conditions</td>
<td>7%</td>
<td>13%</td>
<td></td>
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<tr>
<td>Other illnesses</td>
<td>2%</td>
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<tr>
<td>Drugs, medications</td>
<td>2%</td>
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<tr>
<td>Stress</td>
<td>1%</td>
<td></td>
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<tr>
<td>Surgery</td>
<td>1%</td>
<td></td>
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</tr>
<tr>
<td>Possible TMJ syndrome</td>
<td>1%</td>
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<tr>
<td>Barotrauma</td>
<td>1%</td>
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</table>

**Note.** TMJ = temporomandibular joint.
nervous system (Domjan & Burkhard, 1986). A good example is the sound of an electric fan. Unless an individual attends to the sound, the person is not consciously aware that the sound is present. The link between emotion and sound may be the basis for psychological distress in some tinnitus patients (Hallam et al., 1984; P. J. Jastreboff, Gray, & Gold, 1996). Tinnitus is not habituated in patients who typically seek treatment but rather may become intrusive, annoying, or disturbing for them and may persist as a problem even though the underlying condition has been present for months or even years. Once tinnitus emerges and the patient begins to attend to it, the tinnitus becomes audible. If the patient continues to focus on the tinnitus and attaches negative beliefs to it (e.g., “the tinnitus will get worse,” “the tinnitus will make me go deaf,” or “the tinnitus means I have a terrible ear disease”), then he/she may suffer increasing worry, anxiety, fear, distress, despair, and depression. In other words, just as changes in psychological state can trigger tinnitus, psychological reactions and negative associations can exacerbate the condition and, in effect, amplify the perception of tinnitus. Effective control of these maladaptive emotional reactions and beliefs is an important component of tinnitus management (Hallam et al., 1984; Hazell, 1998a; Lindberg, Scott, Melin, & Lyttkens, 1987; Scott, Lindberg, Lyttkens, & Melin, 1985; Sweetow, 1986, 2000).

Attributes of Tinnitus in a Clinical Population

The Oregon Tinnitus Data Archive (Meikle et al., 2004) contains a valuable description of tinnitus attributes from 1,630 OTC patients. Patient data were included in this database if the patient (a) was at least 18 years of age, (b) completed a tinnitus evaluation with at least 75% of questionnaire items having “acceptable reliability,” (c) was interviewed to confirm questionnaire responses, and (d) gave informed consent. These data, which may be generalized to only treatment-seeking patients, suggest more than twice as many men as women are in the archive and that 80% of all patients are over age 40. The onset of tinnitus is reported to be “gradual” slightly more often than “sudden.” Tinnitus is perceived more often in the left ear or left side of the head than in the right. More than half of patients report that their tinnitus consists of a single sound, whereas most of the rest can identify two or more sounds. The great majority of patients report that their tinnitus sounds like “ringing” or a “clear tone,” whereas only 3% report their tinnitus to be perceived as a “hum,” “clicking,” “roaring,” or “pulse.” These latter sounds are likely to be related to vascular or ear disease. Additional findings from analyses of the archive data included the trend for perceived tinnitus loudness to appear higher than suggested by the patients’ matched level (dB SL) to the tinnitus. Furthermore, about 85% of patients reported the perceived loudness of their tinnitus as 5 or more on a 0–10 loudness-rating scale (10 = very loud), whereas 70% matched their tinnitus loudness to pure tones presented at 6 dB SL or less. In contrast, only 20% of patients matched their tinnitus at 6 dB SL or less when the match was performed to a 1000-Hz tone (i.e., a frequency outside the range of hearing loss in most patients). These matching results support the well-known phenomenon of loudness recruitment, which is often thought to contaminate tinnitus-loudness measures (J. A. Henry, 1996; Tyler & Conrad-Arms, 1983a).

Lockwood, Salvi, and Burkard (2003) reported findings in a survey of more than 500 patients. The mean length of time patients waited to seek attention was 5 years. By the time they did seek medical attention, 60% thought they had a serious medical problem and 55% thought they would go deaf—underscoring the recognition of the patient’s misconception of the often-benign tinnitus symptom. Twenty-two percent reported tinnitus as being equal in both ears, 34% reported unilateral tinnitus, and most reported some degree of lateralization. As in the Oregon database, patients reported a wide variety of tinnitus qualities, including “ringing” (38%), “buzzing” (11%), “crickets” (9%), and “humming” (5%). Thirty-four percent of patients rated the severity as 8 on a 1–10 subjective loudness scale (10 = very loud). The tinnitus frequency was usually related to the pattern of the hearing loss and was usually above 3000 Hz.

Sleep problems are a particular concern for many tinnitus patients. From the sample of 1,630 Oregon patients, more than 70% reported that their tinnitus caused sleep disturbances (Meikle et al., 2004). Other clinical data have revealed that the most frequently encountered problem experienced by tinnitus patients is sleep disturbance (Axelsson & Ringdahl, 1989; Jakes et al., 1985; Tyler & Baker, 1983). In addition, numerous studies have reported that patients with tinnitus-related sleep problems tend to report the most severe tinnitus (Axelsson & Ringdahl, 1989; Erlandsson, Hallberg, & Axelsson, 1992; Folmer & Griest, 2000; Meikle, Vernon, & Johnson, 1984; Scott, Lindberg, Melin, & Lyttkens, 1990). These data emphasize the importance of addressing the issue of sleep with all tinnitus patients.

Perception of Tinnitus

The inner ear transforms mechanical energy into electro–chemical energy. Auditory information is transmitted in the form of neural activity that travels from the auditory nerve into the brainstem and to higher processing centers in the thalamus and cortex.
How does the central nervous system distinguish inputs generated by external events from aberrant self-generated events? The fact that tinnitus is qualitatively similar to externally generated sounds suggests that the neural pathways that mediate tinnitus are the same as those that process normal sound perception. If the central nervous system processes aberrant neuronal firing, the result might be a perception of sound that has no external cause (Eggermont & Sininger, 1995). Regardless of the causal or underlying mechanism, the end product is the same: perception of sound in the absence of external stimulation.

Masking of Tinnitus Versus Masking of External Sounds

Many studies have revealed that masking of tinnitus does not follow the psychoacoustic rules that were established for the masking of one sound by another (Feldmann, 1971; Formby & Gjerdingen, 1980; Hazell, 1981; Mitchell, 1983; Mitchell, Vernon, & Creedon, 1993; Penner, 1987; Penner & Klafter, 1992; Shailer, Tyler, & Coles, 1981; Tyler & Conrad-Armes, 1984). All investigators have agreed that the usual marked frequency dependence seen in conventional tone-on-tone masking is not characteristic of most attempts to mask tinnitus. That is, unlike sounds that originate externally, the frequency of the masker is not a reliable predictor of tinnitus masking (Feldmann, 1971). Furthermore, in a normal psychoacoustic masking experiment, low frequencies are more effective maskers than high frequencies. Tinnitus masking usually shows no such dependence.

Normally, it is not possible to mask a broadband or thermal noise with a tone. In tinnitus masking, however, a tonal masker (at any frequency) can be effective in providing relief in some patients (Feldmann, 1971). Tinnitus masking usually shows no such dependence. Regardless of the causal or underlying mechanism, the end product is the same: perception of sound in the absence of external stimulation.

Coherent” Versus “Incoherent” Tinnitus

There are fundamental phenomena concerning directional hearing and localization of sounds that relate to the perception of tinnitus (Feldmann, 1995). Acoustic signals presented binaurally will form a single, centered, auditory image if the signals are diotic (i.e., when the acoustic characteristics of the two sounds are essentially equivalent, especially their time pattern). The signals must be sufficiently different for them to be perceived as two distinct stimuli.

Most tinnitus patients can lateralize their tinnitus to the right or the left ear, to both ears simultaneously, or to some location in the head. Tinnitus that can be heard in each ear lacks coherence and would presumably originate from two sources distal to the medial superior olive (Feldmann, 1995), which is the lowest level of the brainstem at which binaural convergence of auditory stimuli takes place (Gelfand, 1998). On the basis of the premise that tinnitus is not normally associated with mechanical action of the cochlear partition, it follows that tinnitus that can be heard in each ear must originate somewhere between the synapse of the inner hair cells and the superior olivary complex. Because cochlear damage resulting from exposure to noise is the most common cause of tinnitus, and because it is generally believed that tinnitus does not originate from the cochlear partition, it would seem reasonable to postulate that “incoherent” tinnitus originates at the base of the inner hair cells (“peripheral” origin). In contrast, tinnitus that is perceived as a fused (coherent) image could be conjectured to originate at some level above the superior complex (“central” origin). It may also be true, however, that tinnitus that is perceived as fused could simply reflect two cochleae that are very similar in pathological state, resulting in very similar aberrant signals.
Theorized Mechanisms of Pathophysiology

Numerous structures within the auditory system and neighboring anatomical regions could contribute to the generation of tinnitus, but at present no test can identify these regions accurately (Brix, 1995; Hazell, 1995). The study of these structures in relation to tinnitus is problematic, and, not surprisingly, a causal relation between measurable neurophysiologic functions and tinnitus generation has not yet been demonstrated scientifically.

The study of tinnitus mechanisms is vitally important to develop effective treatments for tinnitus in all its forms and manifestations. If pathological mechanisms of tinnitus could be defined for different classifications of tinnitus origin, treatment could be directed toward addressing the cause of the disorder rather than just the consequences. Zenner and Pfister (1999) have proposed three broad classes of tinnitus, on the basis of anatomical and functional divisions of the auditory system, that include conductive, sensorineural, and central tinnitus generation sites. Conductive tinnitus would be caused by some type of vibration in the middle ear. Sensorineural tinnitus would have numerous subclasses, including tinnitus generated from (a) outer hair cells (“motor” tinnitus), (b) inner hair cells (“transduction” tinnitus), (c) the auditory nerve (“transformation” tinnitus), and (d) extrasensory structures (vascular, muscular or other somatic sources of “objective” tinnitus). Central tinnitus would involve tinnitus originating anywhere in the central auditory pathways. Every conceivable site of tinnitus generation is covered in this classification framework. What remains to be learned, however, is the precise mechanisms of action that result in generation of the tinnitus neural signal. These mechanisms are likely to be multiple, even in the same individuals (Baguley, 2002; A. R. Möller, 2003).

Many theories and models have been proposed to explain the pathophysiological basis of tinnitus (Baguley, 2002; Eggermont, 2000; Kaltenbach, 2000; A. R. Möller, 2003; Vernon & Möller, 1995). The most prevalent theories involve hair cells, the auditory nerve, and the central auditory nervous system. Theories involving hair cells include discordant hair cell function (P. J. Jastreboff, 1990), calcium imbalance (Eggermont, 2000), loss of outer hair cell function as a trigger of tinnitus (Kaltenbach et al., 2002), activation of cochlear NMDA (N-methyl-D-aspartate) receptors (Guitton et al., 2003), excitatory drift in hair cells (LePage, 1987), and enhanced glutamate activity from the inner hair cells in response to stress (Sahley & Nodar, 2001). Theories involving the auditory nerve include synchronization of spontaneous activity in auditory nerve fibers that is due to cross-talk (Eggermont, 1990; Möller, 1984, 1995), deafferentation hyperexcitability (Kiang et al., 1970), abnormal temporal pattern in auditory nerve-fiber spontaneous activity (Eggermont, 1984), and differential activity in tonotopically adjacent fibers (Kiang et al., 1970). Theories involving the central auditory nervous system include effects of the efferent auditory system (Hazell, 1987), increased spontaneous activity in the dorsal cochlear nucleus (Brozoski, Bauer, & Caspary, 2002; Kaltenbach & Afman, 2000; Kaltenbach et al., 2002; Zacharek, Kaltenbach, Mathog, & Zhang, 2002), generation of tinnitus by broad multimodal networks of neurons (Cacace, 2003), and cortical plasticity (Lockwood et al., 1998). There are, of course, many other theories.

Peripheral Versus Central Site of Tinnitus Generation

The most fundamental question regarding the mechanism of tinnitus is its neural site of generation. Zenner and Pfister’s (1999) classification model described above emphasizes that tinnitus can originate anywhere between the peripheral ear and the central auditory pathways. Tinnitus generated in the middle ear is relatively rare, but it does occur. Sensorineural tinnitus is thought to be the most common form of tinnitus, mainly because noise exposure is so commonly associated with tinnitus onset, and the damage caused by noise is cochlear. In addition, there are many other causes of cochlear pathology. Coles (1995) referred to the “clinical dictum” (p. 13) that whatever factors are responsible for causing hearing loss are also likely to have caused an associated tinnitus.

The earliest speculations about the site of tinnitus generation suggested a cochlear origin. This site of origin was posited because of the strong association observed between tinnitus and hearing loss caused by cochlear damage (Kiang et al., 1970). Theories that tinnitus originated in the inner ear were further supported by observations that patients often perceived tinnitus in the ears (P. J. Jastreboff, 1990). Because surgical sectioning of the auditory nerve did not always eliminate intractable tinnitus (Fisch, 1970; J. W. House & Brackmann, 1981; Pulec, 1984), the cochlear origin theory was not universally accepted (Douek, 1987; Feldmann, 1995). Over time, the prevailing view suggested that tinnitus was generated in the central nervous system and was triggered by cochlear damage (Penner & Bilger, 1995). More recently, studies have presented arguments to support both a cochlear (Penner & Bilger, 1995; Zenner & Ernst, 1995) and a central (Eggermont & Siningher, 1995; Lockwood et al., 1998) locus of tinnitus origin. A. R. Möller (2003)
took the view that pathology in the ear and auditory nerve results in abnormal input that causes changes in central structures. More specifically, abnormal auditory signals activate neural plasticity within central auditory structures, which can be expressed as tinnitus.

**Tinnitus Associated With Hair Cell Damage**

There are many theories that describe the underlying cause of tinnitus as being associated with the loss of cochlear hair cells, some of which were listed above. The discovery of spontaneous otoacoustic emissions (SOAEs) generated by outer hair cells led to the logical speculation that they might prove to be an objective correlate of some forms of tinnitus (Kemp, 1979). A number of studies that attempted to link SOAEs with tinnitus found that they were generally independent events (Prieve & Fitzgerald, 2002). It does appear, however, that some patients have tinnitus caused by SOAEs. Penner (1990), for example, observed that the prevalence of tinnitus caused by SOAEs was 4%. Norton, Schmidt, and Stover (1990) reported that several studies found an association between SOAEs and tinnitus in 6% to 12% of patients.

With the loss of hair cells or hair cell function, afferent neurons appear to trigger aberrant auditory sensations at frequencies at or near the focus of the lesion (P. J. Jastreboff, 1990). This “edge effect” theory could explain why tinnitus is frequently associated with hearing loss, why the frequency of tinnitus is often related to the involved frequencies of the hearing loss, and why tinnitus persists beyond the time expected for normal recovery from noise exposure (Eggermont & Sininger, 1995).

**Noise exposure.** The histopathology associated with noise exposure, as it is for many causes of cochlear tinnitus, is related to hair cell damage (Coles, 1995). However, the mechanism of tinnitus and its association with noise exposure is unknown, but this cochlear damage somehow appears involved in the onset of tinnitus. We know that high noise levels cause hair cell damage that begins with the stereocilia of the outer hair cells and progresses to loss of the hair cells themselves (Harrison & Mount, 2001). Outer hair cells are damaged first, followed by damage to inner hair cells. Progressive noise damage can eventually result in complete destruction of both inner and outer hair cells in certain regions of the basilar membrane. There can also be degeneration of associated neural elements at each stage of damage.

The structural changes that are observable with hair cell damage are preceded by molecular damage (Wenthold, Schneider, Kim, & Dechesne, 1992). Whereas molecular changes associated with noise damage are potentially reversible, the subsequent structural changes are not. Wenthold et al. have suggested a hypothetical sequence of molecular changes that correlate with different stages of morphological changes—from initial responses to hair cell death. Initial biochemical changes involve damage to the structural proteins that reside in the stereocilia and on the cuticular plate. Any of these changes could play a role in the onset of tinnitus. For example, there is a group of proteins, referred to as heat shock proteins (or stress proteins), that are upregulated in response to any condition that stresses a cell. It is thought that stress proteins bind to damaged proteins to facilitate cellular repair and protection from further cell damage. It is thus possible that stress proteins can protect the cochlea from damage caused by noise. A person who has a deficient stress protein response system may be at increased risk of incurring hearing loss when exposed to noise. It would follow that such a person could also be at greater risk for incurring tinnitus.

It is also important to mention that calcium balance is essential for many aspects of normal cochlear function (Wenthold et al., 1992). Any disruption to this calcium balance could result in tinnitus (Eggermont, 2000). Normal functioning of hair cells requires that calcium concentrations be precisely maintained on both sides of the hair cell membranes. It has been shown that intense sound can increase the concentration of cytoplasmic calcium in isolated outer hair cells, suggesting that such changes in calcium concentration may be involved in the long-term pathogenesis of noise damage to hair cells (Fridberger & Ulfendahl, 1996). Eggermont (2000) suggested that increased levels of intracellular calcium, causing an increase in neurotransmitter release from the cells and a subsequent increase in the spontaneous firing rates of associated afferent fibers, may be a causal factor in tinnitus.

**Ménière’s disease.** Tinnitus is also a symptom of Ménière’s disease (endolymphatic hydrops), for which the underlying pathology again appears to be hair cell damage—possibly related to potassium toxicity (“leaky” potassium channels; Zenner, 1986; Zenner & Ernst, 1995) or changes in osmolarity (Dulon, Aran, & Schacht, 1987; Feldmann, 1995).

**Perplexing observations.** Not all persons with hearing loss have tinnitus (Hazell, 1998a; J. L. Henry & Wilson, 2001). Persons with profound hearing loss (deafness) often do not complain of tinnitus, nor report significant levels of tinnitus (Coles, 1995; Hazell, 1998a). In other words, cochlear damage does not always result in tinnitus. It may also be the case that cochlear damage may be slight and not significant enough to cause substantive change in auditory sensitivity. The variety of theories and the variability in the
Theories of Central Tinnitus Generation

In addition to the observation of tinnitus in patients who had undergone transection of the auditory nerve (Fisch, 1970; J. W. House & Brackmann, 1981; Pulec, 1984), other support for a central origin for tinnitus has come from a study that showed tinnitus patients with reduced amplitudes of auditory event-related potentials (Attias, Urbach, Gold, & Shemesh, 1993). Lockwood et al. (1998) used positron emission tomography (PET) to map brain regions that showed activity in response to changes in tinnitus loudness. Participants in this study had the unusual ability to alter the loudness of their tinnitus by performing oral facial movements. The interesting result was that self-induced changes in tinnitus loudness resulted in unilateral changes in cerebral flow, which suggested that the spontaneous neural activity responsible for their tinnitus was generated centrally. Later imaging studies produced the same general findings (Lockwood et al., 2001; Reyes et al., 2002).

Additional support for tinnitus of central origin has come from a series of animal studies that examined noise-induced hyperactivity in the dorsal cochlear nucleus (DCN). Kaltenbach and Afman (2000) observed that the DCN exhibits a spontaneous activity pattern following noise exposure that is very similar to the activity induced by a low-level tone. DCN hyperactivity also has been induced by the chemotherapeutic drug cisplatin (Rachel, Kaltenbach, & Janisse, 2002). These two findings suggest that DCN hyperactivity could be a neurophysiologic correlate of noise- and cisplatin-induced tinnitus, although both noise and the cisplatin have their damaging effects primarily on the outer hair cells. In fact, outer hair cell damage caused by cisplatin has been documented in association with DCN hyperactivity (Kaltenbach et al., 2002). Zacharek et al. (2002) addressed the question of whether DCN hyperactivity originates centrally or peripherally following exposure to intense noise. They ablated noise-damaged cochleae with the result that DCN hyperactivity persisted. Thus, the central hyperactivity was not dependent on cochlear input, which suggested that the hyperactivity originated centrally.

There is growing evidence that tinnitus may originate from an anatomical location that is central to the site of initial pathology (A. R. Møller, 2003). Cacace (2003) reviewed the evidence and proposed a view that networks of neurons may be involved in generating and sustaining tinnitus. This view would explain why tinnitus might persist following transection of the auditory nerve or ablation of a damaged cochlea. Moreover, Cacace suggested that these neural networks might be so extensive as to involve brain regions subserving emotions, directed attention, and conscious perception.

Similarities With Pain

Tinnitus has been viewed as a phantom auditory perception, not unlike pain (Meikle, 1995; A. R. Møller, 2003). Pain and tinnitus have similar features in terms of the physiology, assessment, and management (A. R. Møller, 1987, 2000). Tinnitus and chronic pain appear to be mediated by neuropathic mechanisms and seem to involve central generation in more severe cases. Pain theories have postulated that pain is caused by rearrangement of cortical circuits that is due to deafferentation, which can result in phantom limb sensation and associated pain (Kandel, 2000; Tonndorf, 1987). Similarly, it has been theorized that tinnitus can be caused by deafferentation of the cochlear nerve, with subsequent reorganization of neural connections at higher level auditory pathways (Feldmann, 1988).

Additional Comments

Whether tinnitus is generated centrally or peripherally has been the subject of considerable debate (Feldmann, 1971; Lockwood et al., 1998). Because tinnitus is perceived consciously as sound, however, it must by definition involve retrocochlear and cognitive processes. Peripheral and subcortical auditory centers are responsible for transduction, transmission, and processing of neural information, but perception occurs at the level of the cortex. The locus of tinnitus must ultimately be physiologically verified, and a number of studies are under way with this objective in mind. In addition, psychoacoustic data concerning perceptual attributes of tinnitus offer a rich source of information in providing evidence supporting the site(s) of tinnitus generation (Meikle, 1995; Penner & Bilger, 1995).

Assessment of the Tinnitus Patient

Medical Evaluation

Although it may not be feasible in many situations, all tinnitus patients should be referred to an otolaryngologist or otologist for an otologic physical examination (Perry & Gantz, 2000; Wackym & Friedland, 2004). The exam enables the identification of underlying physical pathologies that could potentially be treated, or even cured, medically or surgically. The otologic exam is especially important for patients...
with pulsatile tinnitus, which often has an identifiable physical pathology (Sismanis, 1998; Wackym & Friedland, 2004). The examination results in radiologic and/or laboratory testing only if it is determined that there is a reasonable chance that there is a correctable cause for the tinnitus (Perry & Gantz, 2000; Wackym & Friedland, 2004).

**Audiological Evaluation**

Because most tinnitus patients also have some hearing impairment, routine audiological testing is recommended for all patients who seek treatment for their tinnitus (Henry, 2004a). Audiometric evaluation should include pure-tone thresholds, speech-recognition thresholds, word-recognition scores, and immittance measures. When conducting audiometric testing, it is critical for the clinician to be aware that many tinnitus patients are also hypersensitive to sound. Thus, extreme care must be taken when testing acoustic reflexes, which generally is not advised (J. A. Henry, Jastreboff, Jastreboff, Schechter, & Fausti, 2002). Because loudness intolerance can impact testing procedures, loudness discomfort levels (LDLs) should be determined at audiometric frequencies to assess the upper limit of the auditory dynamic range for each patient. This information should be used to ensure that patients are not exposed to any sound during testing that exceeds their LDLs.

**Reduced Loudness Tolerance**

When LDLs are evaluated at different audiometric frequencies, the average of these measurements will generally be below 100 dB HL when there is a loudness tolerance problem (P. J. Jastreboff & Hazell, 1993). The LDLs provide a “reasonable estimate of the problem” (M. M. Jastreboff & Jastreboff, 2002, p. 75), although there is no consistent relation between patient’s complaints of loudness intolerance and their LDLs (Henry, 2004a; M. M. Jastreboff & Jastreboff, 2002).

Patients with a clinically significant problem of reduced loudness tolerance are generally said to have hyperacusis. Unfortunately, there is at present no consensus definition of hyperacusis (Vernon, 2002). Vernon and Press (1998) have defined hyperacusis as “the collapse of loudness tolerance so that almost all sounds produce loudness discomfort” (p. 223). This definition would not be compatible with M. M. Jastreboff and Jastreboff’s (2002) report that 30% of tinnitus patients require “specific treatment for hyperacusis” (p. 75). A number of other clinics have reported that up to 45% of their patients have decreased loudness tolerance (Coles, 1996; Gold, Frederick, & Formby, 1999; Hazell, 1999; P. J. Jastreboff, 2000).

M. M. Jastreboff and Jastreboff (2002) have distinguished hyperacusis from the condition of misophonia. They described hyperacusis as a condition of hypersensitivity to sound that is restricted primarily to the auditory pathways. Thus, when sound that is normally well tolerated causes physical discomfort, hyperacusis is indicated. Misophonia refers to dislike of sound and involves primarily an emotional reaction that is mediated by the limbic and autonomic nervous systems. The reactions are context dependent, and thus the same sound might evoke different responses in different circumstances. If one of the emotional responses is fear, the condition of phonophobia is indicated. Phonophobia is thus a specific case of misophonia when fear is involved. Jastreboff and Jastreboff emphasized that none of these conditions has any relation to loudness recruitment, which refers to abnormally rapid growth in the perception of loudness (Vernon, 1976).

Because reduced loudness tolerance is so often associated with tinnitus, it is important for the clinician to be skilled in making a proper diagnosis and providing appropriate intervention. As with tinnitus, however, management of this condition is not standardized. M. M. Jastreboff and Jastreboff (2002) have provided a comprehensive schema for identifying different components of the condition and treating each accordingly. They distinguished primarily between a physiological (hyperacusis) versus a psychological (misophonia) cause. These constitute very different conditions and treatment should differ accordingly. Pure hyperacusis is like a pain response with no psychological overlay. Its treatment involves desensitization of the auditory pathways through the use of sound. With misophonia, however, the patient has learned to react emotionally to certain sounds. This is a much more complex condition to treat, especially when there is a fear component (phonophobia). Treatment of misophonia in all its manifestations primarily involves counseling, and some patients may require the services of a psychologist.

It is often the case that hyperacusis leads to some form of misophonia. That is, the experience of auditory discomfort can lead to a psychological reactive state involving the inappropriate use of ear protection and/or avoidance of sound. These behaviors would constitute overprotection of hearing, which could exacerbate the condition because sound deprivation can further reduce loudness discomfort levels (Formby & Gold, 2002; Formby, Sherlock, & Gold, 2002). The different components of reduced sound tolerance are clearly not mutually exclusive; thus, the challenge for the diagnosing clinician is to identify the relative contribution of each. The assessment can be facilitated by measuring LDLs, and by administering the sound tolerance section of the TRT Initial Interview (J. A. Henry, Schechter, et al., 2002, 2003; M. M. Jastreboff & Jastreboff, 1999).
Tinnitus Psychoacoustic Assessment

A tinnitus psychoacoustic assessment should also be part of the intake evaluation. The clinical relevance of these measures depends on the form of treatment used because the measures have limited diagnostic value. With masking treatment, it is always crucial to measure or document the effects of masking stimuli on the perception of tinnitus (J. A. Henry & Meikle, 2000; Schechter & Henry, 2002). Therefore, minimum masking levels and trial use of sound-generating devices must be a part of the tinnitus assessment to perform masking. Tinnitus measures also generally are important for individualized counseling purposes, especially in treatments such as TRT (P. J. Jastreboff, 1995). Psychoacoustic measures also are valuable in evaluating and verifying the patient’s subjective reports of his/her tinnitus condition when the patient is involved in legal action related to the tinnitus (Henry, 2004a).

Loudness and pitch. Tinnitus researchers have attempted to unify methodology for the psychoacoustic evaluation of tinnitus (Axelson, Coles, Erlandsson, Meikle, & Vernon, 1993). This need was recognized as early as 1903 (Spaulding, 1903). The advent of electroacoustic equipment made it technically feasible to use pure tones to match tinnitus pitch and loudness (Jones & Knudsen, 1928; Josephson, 1931; Wegel, 1931). Edmund Fowler made important contributions to tinnitus measurement in the 1930s and 1940s. He described a method for measuring loudness recruitment by balancing the loudness of sounds between ears, which he termed the alternating binaural loudness balance (ABLB; Fowler, 1936, 1937). Fowler later applied the ABLB technique as a test with patients to balance the loudness of tinnitus in one ear with the loudness of a tone in the contralateral ear (Fowler, 1938). The level of the comparison tone, expressed in dB Sensation Level (SL), provided an indication of the tinnitus loudness as experienced by the patient.

Fowler (1940) stated that it was important to duplicate, or match, the loudness and pitch of tinnitus to contralateral tones. For pitch matching, he stressed the importance of presenting stimuli at levels equal to the tinnitus intensity. Fowler (1942, 1943) noted that although patients described their tinnitus as very loud, they usually matched the loudness to tones presented at only 5–10 dB SL. Many subsequent studies used Fowler’s same basic technique or variations of it (Graham & Newby, 1962; Hazell et al., 1985; Kodama & Kitahara, 1990; Penner, 1983b, 1986, 1988; Reed, 1960; Roesser & Price, 1980; Tyler, 1992; Tyler & Conrad-Armes, 1983a, 1983b, 1984). Tinnitus loudness matching has been observed to be a reliable measure for most patients (J. A. Henry, Flick, Gilbert, Ellingson, & Fausti, 1999), but pitch matching yields highly variable responses (J. A. Henry, Flick, Gilbert, Ellingson, & Fausti, 2004; Penner & Bilger, 1995; Tyler & Conrad-Armes, 1983b).

A protocol for loudness and pitch matching was described in detail by Vernon and Meikle (1981). Their protocol involves three separate procedures that are alternated systematically among threshold testing, loudness matching, and pitch matching. The objectives are to achieve a pitch match and a loudness match at the pitch-match frequency. In general, the tinnitus frequency is approached gradually by presenting successive pairs of tones from which the patient selects the tone that is “closest in pitch” to the tinnitus. Patients often confuse pitch and loudness during testing; thus, all tones used for pitch matching are presented only at the levels previously matched to their tinnitus loudness.

Loudness and pitch matching can be performed using a clinical audiometer as follows (J. A. Henry, 2004a). After a pure-tone audiogram has been obtained, the examiner focuses on obtaining a pitch match and a loudness match at the pitch-match frequency. Initial loudness-matching tones should be presented at 10–20 dB SL at frequencies where hearing sensitivity is essentially normal and at 5–10 dB SL at frequencies where there is hearing loss. The testing should be started at 1000 Hz. The patient is asked, “Is the pitch of your tinnitus higher or lower than the pitch of the tone?” Most patients will indicate “higher,” and the next tone is presented at 2000 Hz. The testing progresses in this manner to bracket the tinnitus pitch to within an octave. Interoctave frequencies are then tested the same way to determine a pitch match to the closest half octave. Patients often confuse octaves when pitch matching (Graham & Newby, 1962; Vernon, Johnson, Schleuning, & Mitchell, 1980), so the examiner should alternate presentation of the pitch-matched tone with tones an octave higher and an octave lower to identify the final pitch match. At the pitch-matched frequency, a hearing threshold and tinnitus loudness match are then obtained in 1-dB steps.

Matching to noise. The clinical data reported earlier in the Attributes of Tinnitus in a Clinical Population section reveal that the majority of tinnitus clinic patients report that their tinnitus sounds “tonal” (Lockwood et al., 2003; Meikle et al., 2004). Although most patients can match their tinnitus to a pure tone, a closer match may be achieved when bands of noise are varied systematically to obtain a noise match. Limited noise matching can be done with an audiometer, and more precise noise-band matching can be done with special equipment.

The noise-matching protocol performed using an audiometer follows the pitch match. All stimuli used
with noise matching should be presented at levels approximating the patient’s tinnitus loudness. The first objective is to determine whether the tinnitus sounds more like a pure tone or more like noise. Narrowband noise, centered at the pitch-matched frequency, is presented in alternation with the pitch-matched tone, and the patient is asked which sounds more like the tinnitus. If the tone is selected, then no further noise matching is necessary because the tone is considered the best match. If the noise is chosen, then it is necessary to determine whether narrowband or broadband noise sounds more like the tinnitus. Narrowband noise is presented in alternation with white noise and the patient again chooses the best match. A hearing threshold and tinnitus loudness match are obtained in 1-dB steps for the noise stimulus that is selected.

A more precise noise match is possible if properties of the noise bands can be adjusted systematically to widen or narrow the width of the band that provides the best match. In addition, sweeping a band of noise up and down the frequency range can add further precision.

Tinnitus maskability. The minimum masking level (MML) is the minimum level to which broadband noise must be raised to render an individual’s tinnitus inaudible. The MML is a common measurement made in many tinnitus clinics and has been reported to be a measure that correlates with treatment efficacy (P. J. Jastreboff, Hazell, & Graham, 1994). That is, as patients reported improvement with their tinnitus problem, the MMLs were observed to decrease. Otherwise, MMLs are not generally thought to contribute any predictive information concerning treatment efficacy, other than when tinnitus masking is used for treatment. It has been reported for tinnitus masking that the decibel difference between the MML and the tinnitus loudness match is generally predictive of the effectiveness of treatment (Vernon, Griest, & Press, 1990; Vernon & Meikle, 2000). If the MML is less than the loudness match, benefit is considered likely. If the MML is greater than the loudness match, success is less likely.

Clinical MML testing can be done using monaural or binaural stimuli (J. A. Henry, 2004a). It is often difficult for patients when only monaural stimuli are presented because of difficulties determining when the tinnitus becomes inaudible for only one side of the head. Therefore, we recommend presenting binaural stimuli for routine clinical testing. Broadband noise from the audiometer is used and monaural hearing thresholds are first obtained for the noise stimulus. The majority of patients will be masked by noise within 10 dB SL (Meikle et al., 2004), so it is appropriate to use 1-dB step sizes to obtain thresholds and MMLs using broadband noise. After obtaining thresholds, the noise is set for binaural presentation at the threshold levels. The noise is raised binaurally in 1-dB steps until the patient reports that the tinnitus is inaudible. If during testing the patient reports that the tinnitus has become inaudible for one side, the noise is raised in only the contralateral ear until the tinnitus is completely inaudible.

Residual inhibition. Residual inhibition is the temporary suppression or elimination of tinnitus that is often observed following auditory stimulation (Vernon, 1982; Vernon & Meikle, 1988). Residual inhibition was first reported by Spaulding (1903) and later by Josephson (1931). The phenomenon was formally described by Feldmann (1971) and named “residual inhibition” by Vernon and Schleuning (1978) in recognition of Feldmann’s work. Residual inhibition, which occurs with most patients who receive the appropriate type of auditory stimulation (Meikle & Walsh, 1984; Tyler, Babin, & Neibuhr, 1983; Vernon, 1981, 1988), has received little investigation in terms of the specific stimulus parameters that are responsible for activation, or extension, of the effects for longer time periods.

Residual inhibition testing is performed in our evaluation protocol immediately after MML testing with the same broadband noise. The patient is instructed to listen to the noise for 1 min and then to report if there is any kind of change in the tinnitus. The noise levels established for MML are raised 10 dB and presentation of the noise lasts 60 s. If the patient reports a reduction in tinnitus intensity, it is helpful to ask if there is a 10%, 25%, 50%, 75%, or 90% reduction of the usual loudness (J. A. Henry, 2004a). The patient continues to report one of these percentages until the tinnitus has recovered to its usual loudness (100%).

Attempts to standardize tinnitus psychoacoustic assessment. The many attempts to quantify tinnitus using psychoacoustic techniques have had sporadic success, and these techniques currently have limited clinical and research application. Formal efforts over 20 years ago promoted the establishment of standardized tinnitus evaluation procedures (Evered & Lawrenson, 1981; McFadden, 1982; Vernon & Meikle, 1981). A tinnitus assessment battery was recommended that included pitch and loudness matching, tinnitus maskability, and residual inhibition. Standardized procedures for these tests have still not been universally adopted, partially because of the need for specialized testing equipment. Such equipment used to be available commercially (from Danavox, Starkey, and Norwest companies), but these instruments have been out of production for many years. Audio bench equipment (tone generators, attenuators, amplifiers, filters, etc.) can be adapted to perform tinnitus matching.
but the use of bench equipment is not realistic for the typical clinic. Most audiologists who conduct tinnitus evaluations use their audiometers in some manner to obtain some or all of these measurements.

Work at the VA National Center for Rehabilitative Auditory Research (NCRAR) has addressed the need for standardization of tinnitus psychoacoustic assessment (J. A. Henry et al., 1999, 2000, 2004; J. A. Henry, Flick, Gilbert, Ellingson, & Fausti, 2001; J. A. Henry & Meikle, 1999). The techniques described to date use laboratory equipment. Efforts are presently under way to develop testing instrumentation for routine clinical application.

### Methods of Treatment

In the Theorized Mechanisms of Pathophysiology section, we considered a number of potential mechanisms that may underlie the tinnitus condition. The present lack of knowledge concerning these mechanisms, however, precludes the establishment of differential treatment techniques to address the cause rather than just the symptom. Virtually all tinnitus treatment techniques seek ultimately to reduce the impact of tinnitus on a person’s life. In some cases, the tinnitus sound itself can be reduced, but there is no systematic method for accomplishing this in patients. Accordingly, practitioners either attempt a trial-and-error approach to find a treatment that works for the patient or they use a generic treatment method that is intended to work for all forms of tinnitus.

Many treatment protocols have been proposed for tinnitus management (reviewed in Vernon, 1998). Most patients cannot be helped by medical or surgical treatment, and, therefore, seek other forms of professional intervention when the tinnitus becomes intrusive. Primary methods of tinnitus treatment are described below.

### Hearing Aids

The use of hearing aids to treat tinnitus has long been a mainstay of tinnitus treatment provided by audiologists (Melin, Scott, Lindberg, & Lyttkens, 1987; Saltzman & Ersner, 1947; Surr, Montgomery, & Mueller, 1985). Even for patients who are marginal hearing aid candidates, high-frequency amplification (i.e., primary gain at 3 and 4 kHz) may be readily accepted and beneficial. The majority of audiologists do not possess specialized tinnitus expertise, but they are aware that hearing aids may have a beneficial effect for some patients with tinnitus.

Surr et al. (1985) reported that 62% of 200 new hearing aid users experienced tinnitus. Use of hearing aids resulted in “partial or total” tinnitus relief for half of those with tinnitus. In a follow-up study, Surr, Kolb, Cord, and Garrus (1999) administered the Tinnitus Handicap Inventory (THI; Newman et al., 1996) to new hearing aid users before and 6 weeks after the hearing aid fitting. A statistically significant reduction in the mean TSI scores was seen at 6 weeks. These studies are important in showing that hearing aids can, in some cases, provide tinnitus relief as an independent outcome, even when treatment is for a different purpose.

Hearing aids are an important component of tinnitus treatment with tinnitus masking and TRT (P. J. Jastreboff & Hazell, 1998; Vernon, 1988). For these methods, hearing aids are usually incorporated into a combination instrument (amplification and noise generator combined), although regular hearing aids are also used. The primary purpose of either the combination instruments or the hearing aids is to treat the tinnitus. Improvement in hearing is considered of secondary benefit of treatment with these devices. Hearing aids, therefore, can be fitted either for the primary purpose of providing tinnitus relief or to offer tinnitus relief as a secondary benefit.

It has been estimated that up to 90% of tinnitus patients may benefit from amplification (Johnson, 1998; Schechter, Henry, Zaugg, & Fausti, 2002). The benefit may be due to reduced stress associated with hearing loss, which often accompanies tinnitus, and/or result from amplification of ambient sounds that tend to mask the tinnitus or make it less noticeable. There are different philosophies concerning the use of amplification for tinnitus patients; thus, there is no clear agreement as to when a patient would benefit from amplification. Some clinics have reported that only 20% to 30% of their patients are fitted with amplification (Gold, Gray, Hu, & Jastreboff, 1996; P. J. Jastreboff et al., 1996). Wedel, Wedel, and Walger (1998) reported that their clinic dispensed amplification to 60% of their patients.

### Counseling

Counseling is an important component of every form of tinnitus treatment (Hall & Ruth, 1999; Sweetow, 1986, 2000). Some methods are based entirely on specific psychological techniques, whereas others use counseling to augment another modality of treatment. There are a number of common counseling points that are appropriate for all tinnitus patients, such as (a) avoiding high-level noise exposure, (b) making lifestyle changes that would be conducive to minimizing tinnitus intensity (e.g., reducing stress, getting adequate sleep, limiting intake of alcohol, caffeine, tobacco, etc.), (c) maintaining a constant background of sound
(avoiding silence) to reduce the prominence of tinnitus, and (d) staying busy with meaningful activities to distract attention away from the tinnitus.

**Psychological Treatments**

Various psychological treatments have been used for tinnitus. Hypnosis has been observed to provide some limited positive effects, but studies attempting to document these effects were methodologically flawed (J. L. Henry & Wilson, 2001). Biofeedback was advocated for tinnitus treatment in the 1970s (Grosson, 1976; J. W. House, Miller, & House, 1977), and some studies have shown its effectiveness, but methodological problems were again an issue (J. L. Henry & Wilson, 2001). Progressive muscular relaxation training has been used to help patients cope with the tension and anxiety that result from tinnitus or to relieve stress that can exacerbate tinnitus. The literature concerning relaxation training has produced mixed results (J. L. Henry & Wilson, 2001).

In the 1980s, psychological researchers made a paradigm shift in their approach to tinnitus treatment, including a decreased emphasis on relaxation training and biofeedback and an increased focus on cognitive aspects of tinnitus (J. L. Henry & Wilson, 2001). This shift was due to a number of factors: (a) Relaxation and biofeedback were ineffective for many people, (b) greater benefit was observed using cognitive–behavioral therapy (Jakes, Hallam, Rachman, & Hinchcliffe, 1986), and (c) numerous studies had demonstrated efficacy of cognitive–behavioral therapy for psychological problems (especially depression and anxiety; J. L. Henry & Wilson, 2001). Another influencing factor was the observation that tinnitus had many similarities with pain (A. R. Møller, 1987), and numerous studies had demonstrated the efficacy of cognitive–behavioral therapy for management of chronic pain. For all of these reasons, cognitive–behavioral therapy as applied to pain management was modified for use with tinnitus patients (J. L. Henry & Wilson, 2001).

Cognitive–behavioral therapy is a type of counseling that identifies negative behaviors, beliefs, and reactions and assists the patient in substituting appropriate and positive reactions (Sweetow, 2000). Cognitive–behavioral therapy is performed most commonly by psychologists. The method can, however, be administered by hearing specialists, including audiologists, because of its many similarities with aural rehabilitation (J. L. Henry & Wilson, 2001; Sweetow, 2000). Professionals who wish to learn this method require general training in cognitive–behavioral techniques, which they must adapt to the treatment of tinnitus. A book is available that describes in detail the methodology to administer cognitive–behavioral therapy to tinnitus patients (J. L. Henry & Wilson, 2001).

Hallam et al. (1984) were instrumental in establishing many parameters that are accepted today as key issues in the psychological management of tinnitus. At the time their seminal paper was written, no controlled trials had been reported that involved counseling or psychological or psychotherapeutic techniques. They systematically reviewed evidence that supported psychological variables as factors responsible for tinnitus becoming clinically problematic. In addition, Hallam et al. proposed a model to describe how habituation to tinnitus occurs as normal response. They suggested that certain conditions can delay habituation. For example, chronic stress and anxiety (“tonic arousal”) can “lower the threshold of emotional responsiveness in general” (p. 45), which could exacerbate the annoyance caused by tinnitus.

J. L. Henry and Wilson (2001) reviewed the studies that have been conducted to evaluate the efficacy of cognitive–behavioral therapy for tinnitus distress. They concluded that these studies offer “considerable support” (J. L. Henry & Wilson, 2001, p. 247) for using this form of treatment. They noted that best results are achieved with at least eight sessions. They further noted that pure cognitive therapy does not maximize therapeutic potential and that it should be combined with attention control, imagery training, and relaxation methods.

Andersson and Lyttkens (1999) conducted a meta-analysis of studies that have used psychological approaches to treat tinnitus. The various forms of treatment included cognitive–behavioral (n = 11), relaxation training (n = 4), education (n = 2), hypnosis (n = 2), biofeedback (n = 2), and stress management (n = 3). These 24 groups had been included in 18 published studies. Criteria for studies to be included in this meta-analysis included the following: written in English, published in a scientific journal, and reported information, including description of procedures, number of participants, self-reported outcome measures, and interpretable statistics. Outcome measures were classified into four groups: loudness, annoyance, negative experiences (including depression and anxiety), and sleep difficulties. Of these four, effects of treatment were greatest on tinnitus annoyance. Effects on negative experiences were weaker than for annoyance. Effects on sleep were weak. There was an immediate effect on tinnitus loudness that was generally not sustained at follow-up evaluations.

Dobie (1999), in his review of randomized clinical trials, offered a contrasting opinion regarding the various treatments that he evaluated, including cognitive–behavioral therapy. He concluded that, “No treatment can yet be considered well established in terms of
patients.

particular tinnitus expertise. Thus, psychological in-
address these issues, even if they do not have any
tinnitus. Psychologists are clearly well qualified to
logical problems that can cause exaggerated reactions to
hand, some tinnitus patients have preexisting psycho-
cause of numerous psychological problems. On the other

Measurement of outcomes at 12 months posttreatment
showed improvements on some outcome scales that
cognitive–behavioral group, after 6 weekly sessions,
behavioral therapy (J. L. Henry & Wilson, 1996). The
domized to receive intervention that involved either
and Lyttkens (1999), most of the studies did not prop-
except on the part of the patient that expert treatment
is being provided (Isenberg, 1998). Roberts, Kewman,
Hovell (1993) concluded that, when both
patient and clinician believe strongly that a treatment
is efficacious, improvement occurs up to two thirds of
the time regardless of the treatment’s efficacy.

Because the nonspecific effects of therapy can be
powerful, studies of psychological forms of tinnitus
treatment must be interpreted with this consideration
in mind. In the meta-analysis conducted by Andersson
and Lyttkens (1999), most of the studies did not proper
control for patient expectation effects. One possible
exception was a study in which patients were ran-
domized to receive intervention that involved either
education alone or education combined with cognitive–
behavioral therapy (J. L. Henry & Wilson, 1996). The
cognitive–behavioral group, after 6 weekly sessions,
showed improvements on some outcome scales that
were statistically greater than for the education group.
Measurement of outcomes at 12 months posttreatment
indicated, however, that the benefits of therapy had
dissipated.

Tinnitus is an auditory condition that can be the
cause of numerous psychological problems. On the other
hand, some tinnitus patients have preexisting psycho-
logical problems that can cause exaggerated reactions to
tinnitus. Psychologists are clearly well qualified to
address these issues, even if they do not have any
particular tinnitus expertise. Thus, psychological in-
tervention in general can be helpful to many tinnitus
patients.

Pharmacological Approaches

Many medications have been proposed to relieve
tinnitus. Antianxiety drugs, antipsychotics, sedatives,
antidepressants, antihistamines, anticonvulsants, and
even anesthetics have been reported to relieve tinnitus
for some people (Perry & Gantz, 2000). Some of the
medications that have been associated with tinnitus
treatment are discussed below.

Lidocaine. Lidocaine is a local anesthetic used fre-
quently by dentists. Intravenous (IV) administration of
lidocaine has been shown to abolish tinnitus in about 50%
of cases and to reduce it in many others (den Hartigh
et al., 1993; Hulshof & Vermeij, 1984; Israel, Connelly,
McTigue, Brummett, & Brown, 1982; Melding, Goodey,
& Thorne, 1978). IV administration of lidocaine is not
a practical treatment for tinnitus (because of its short
half-life and serious side effects), but its effectiveness
proved that tinnitus can be abolished temporarily in
some patients by a drug (Coles, 1998).

The effectiveness of lidocaine led to a search for an
oral analogue that provides the same effect on tinnitus
but without the side effects (Coles, 1998). The two
main groups of drugs that have been studied are the
antiarrhythmic (e.g., tocainide) and anticonvulsant (e.g.,
carbamazapine) drugs. The antiarrhythmic oral drugs
are closely related to lidocaine, but seven randomized
clinical trials failed to show replicable benefit against
tinnitus (Dobie, 1999). Carbamazapine (Tegehotel;
Novartis Pharmaceuticals Corp.) failed to show benefit
in four randomized clinical trials.

Benzodiazepines. The benzodiazepines are a class of
drugs having anxiolytic, hypnotic, and anticonvulsant
properties (Dobie, 1999). Benzodiazepines that have been
studied for tinnitus relief include diazepam (Valium;
Hoffman-La Roche Inc.) and alprazolam (Xanax; Pfizer
Inc.). Most controlled studies have shown no conclusive
benefit of any of these drugs (Lockwood et al., 2003).
Johnson, Brummett, and Schleuning (1993) demonstrated
that Xanax reduced tinnitus loudness in a controlled
study, but changes in life impact were not measured.
There are risks of dependence on the benzodiazepines
and more controlled studies are needed (Dobie, 1999).

Antidepressant drugs. Tricyclic antidepressants
and serotonin-specific reuptake inhibitors (SSRIs) are
the primary drugs that are used for treating major
depression (Dobie, 1999; Julien, 1995). These drugs,
such as the tricyclic amitriptyline (Elavil; Astra Zeneca)
and the SSRI fluoxetine (Prozac; Eli Lilly), also have
been used to treat tinnitus (Brummett, 1989; Dobie,
Sakai, Sullivan, Katon, & Russo, 1993; J. W. House,
1989). Placebo-controlled studies have shown that the
antidepressants are effective at reducing depression,
but it is not clear whether they actually reduce the

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perception of tinnitus (Dobie & Sullivan, 1998). Tinnitus patients with sleep problems and/or major depression should be considered for antidepressant therapy (Dobie, 1999).

Additional medications. A great many drugs have been evaluated for their potential effectiveness in providing tinnitus relief. In addition to the drugs and classes of drugs mentioned above, additional drugs that have been studied include Cylandelate (vasodilator), Amylobarbitone (barbiturate), Baclofen (gamma-aminobutyric acid [GABA] agonist and muscle relaxant), Misoprostol (prostaglandin analog), Betahistine (histamine-like drug), and Cinnarizine (antihistamine). Dobie (1999) reviewed the randomized clinical trials that were conducted to evaluate these different drugs. He determined that none of them offered any definitive advantage over placebo.

Psychiatric treatment. In the Psychological Treatments section, we pointed out that psychological intervention in general can be helpful to many tinnitus patients, even if the psychologist is not a tinnitus specialist. Similarly, many psychological and mood disorders that are associated with tinnitus are treatable through the use of medications by a psychiatrist.

Tinnitus Retraining Therapy (TRT)

TRT combines low-level, steady background sound with a structured program of directive counseling (J. A. Henry, Schechter, et al., 2002; P. J. Jastreboff, et al., 1996). A patient's emotional reactions to tinnitus is thought to be caused by beliefs, threats, and fears that are inappropriately ascribed to the tinnitus. The counseling addresses these inappropriate reactions and emotions as the necessary first step to achieve tinnitus habituation (i.e., the gradual reduction or elimination of the annoyance and the conscious perception of tinnitus that is the primary goal of treatment with TRT). The other component of TRT for achieving habituation is sound therapy, which is the use of continuous background sound throughout the day. A primary purpose of the sound is to reduce the contrast between the low-level acoustic background and the tinnitus. Sound therapy is accomplished best using ear-level sound generators for patients who do not require amplification (P. J. Jastreboff & Jastreboff, 2001). Hearing aids or combination devices (amplification in a combination unit with a sound generator) are used with TRT when hearing loss is a problem. Open-ear fittings are recommended to facilitate maximal entry of low-frequency environmental sound into the ear canal (P. J. Jastreboff & Jastreboff, 2000). If an open-ear configuration is not available, then the largest possible vent should be used. Treatment with TRT usually requires 1 to 2 years to achieve the desired result.

A number of clinics have reported results of treatment with TRT, both prospectively and retrospectively (reviewed in J. A. Henry, Schechter, et al., 2002). These studies report success rates between 70% and 85%. Although these results may appear impressive, there are as yet no properly controlled clinical studies of TRT efficacy reported in the peer-reviewed literature. A prospective, controlled clinical study was recently completed at the Portland VA Medical Center. The study...
compared the relative efficacies of TRT and the tinnitus-masking approach. Results of that study have thus far been only briefly summarized (Henry, 2004b; Henry, Schechter, et al., 2004). However, the results of this controlled trial were comparable with previous clinical reports of TRT efficacy.

Although TRT has become a relatively well-known method of treating tinnitus, it has not been immune to censure. Of note, the Development and Evaluation Service of the Wessex Institute (Wessex Institute for Health Research and Development, 1998) concluded that there is no evidence substantiating the effectiveness of TRT. Critics of TRT have reported a number of methodological shortcomings that include lack of standardized assessment measures (J. L. Henry & Wilson, 2001; Lockwood et al., 2003). Wilson, Henry, Andersson, Hallam, and Lindberg (1998) were critical of the directive counseling protocol, stating that an education approach to counseling patients is “nothing new” (p. 276) and that this type of approach was recommended many times prior to 1990. An additional criticism is that the only way to receive formal TRT training is to attend expensive instructional courses conducted by a few specialists who have pioneered and promoted the successes of the treatment.

Electrical Stimulation

Electrical stimulation for suppression of tinnitus has been researched for many years (Dauman, 2000). Hatton, Erulkar, and Rosenberg (1960) reported unexpected tinnitus suppression in patients when performing routine galvanic stimulation tests for vestibular function. They tested this finding in a group of 33 tinnitus patients by applying direct current (DC) to various locations on the head. In 45% of these patients, the tinnitus intensity was decreased in response to the DC treatment. Chouard, Meyer, and Maridat (1981) used transcutaneous electrical neural stimulation (TENS), similar to the TENS portable unit used for treating intractable pain. In a study of 53 participants, 47% reported some relief. None of these patients reported complete suppression, and tinnitus returned after a period of days or weeks.

Commercial electrical tinnitus-suppression devices were developed but are no longer available. Morgan, Dubreuil, Disant, and Chanal (1984) used the Tinitop (Audiologie-Recherche-Applications; which produces a pulsed electrical sine wave) along with a 144-Hz acoustic stimulus to treat 50 patients who complained of “intense, continuous tinnitus.” Treatments were administered during six 45-min sessions repeated every 10 days. Twenty-seven (54%) of these patients reported complete or partial, although temporary, relief. A number of studies were conducted with another tinnitus-suppression device, the Audimax Theraband (Audimax Corp.). This wearable device uses a headband to hold electrodes, which deliver low-level alternating current (AC), against each mastoid. The best controlled of the Theraband studies showed some limited therapeutic benefit (Dobie, Hoberg, & Rees, 1986). Other studies also indicated some limited effectiveness, although placebo effects could have influenced the results (Dauman, 2000).

The many studies that have used electricity to stimulate the auditory system for tinnitus relief indicate that this is a promising area of investigation (Dauman, 2000; Rubinstein & Tyler, 2004). However, the underlying mechanisms and most effective parameters of the stimuli have yet to be determined. The general conclusion is that DC is the most effective type of current, but DC causes tissue damage (Aran, 1983; Dobie et al., 1986; Hazell, Meerton, & Ryan, 1989; Staller, 1998). AC does not cause tissue damage, but is only effective for a limited number of patients. Electrical stimulation, therefore, is still considered an experimental treatment and has not been demonstrated as useful for common clinical practice.

Cochlear implants. Cochlear implants were reported early on to produce tinnitus suppression in nearly 80% of implanted patients (W. F. House, 1976). This benefit may arise from masking by ambient sounds that are newly perceived (Vernon, 2000) or from electrical stimulation of the auditory nerve (Dauman, 2000). Other studies have demonstrated that cochlear implants can provide tinnitus relief in 53% to 83% of patients (Berliner, Cunningham, House, & House, 1987; Hazell et al., 1989; McKerrow, Schreiner, Snyder, Merzenich, & Toner, 1991). Cochlear implants are a viable tinnitus treatment option for individuals who are profoundly deaf with severe intractable tinnitus.

TMJ Treatment

Tinnitus can be a symptom of a TMJ dysfunction. In such cases, dental treatment or bite realignment can help relieve TMJ pain and associated tinnitus (Morgan, 1996). This management strategy would be expected to be successful only in a very small percentage of appropriately selected tinnitus patients.

Complementary and Alternative Treatments

A variety of nutritional supplements have been advocated to treat tinnitus, including minerals such as magnesium or zinc, herbal preparations such as ginkgo biloba, homeopathic remedies, and B vitamins (Seidman & Babu, 2003). Other complementary and alternative
treatments include acupuncture (Park, White, & Ernst, 2000), ear-canal magnets (Coles, Bradley, Donaldson, & Dingle, 1991), hyperbaric oxygen therapy (Kau, Sendtner-Gress, Ganzer, & Arnold, 1997), ultrasound (Rendell, Carrick, Fielder, Callaghan, & Thomas, 1987), and low-power laser (Nakashima et al., 2002). Some of these therapies have been studied to verify anecdotal claims, but there is no convincing scientific evidence that these remedies are effective (Dobie, 2004a). Reviews of complementary and alternative methods of tinnitus treatment can be found in Dobie (1999, 2004a) and Seidman and Babu (2003). We briefly discuss here some of the more popular methods that continue to receive attention.

Ginkgo biloba. Much has been written about ginkgo biloba as a potential remedy for tinnitus. It is important to be aware of the literature about this herb because numerous commercial “tinnitus products” contain ginkgo in combination with other herbs and minerals. Patients often ask about these commercial formulas, or about the use of ginkgo alone. Ernst and Stevinson (1999) reviewed five randomized controlled trials that assessed the efficacy of ginkgo for treating tinnitus. They concluded that all of these trials were methodologically flawed, although the three best controlled studies reported beneficial effects with ginkgo. Holstein (2001) reviewed 19 clinical trials that had used the standardized ginkgo extract EGb 761 (Tebonin). Eight of these trials were controlled (five of them randomized and double blinded). Holstein concluded that these studies indicated that ginkgo is generally effective in the treatment of tinnitus regardless of its origin or duration. Drew and Davies (2001) conducted a double-blind, randomized, placebo-controlled study with 1,121 participants. The entire study was conducted by mail and by telephone, and effects of tinnitus were assessed through the use of questionnaires. The authors concluded that ginkgo provided no more benefit than placebo. Most recently, Hilton and Stuart (2004) conducted a multiple-database search of randomized, controlled trials of ginkgo used for adults complaining of tinnitus. They evaluated rigorously the methodological quality of more than 100 studies and determined that most trials were poorly controlled. They concluded that there is no evidence that ginkgo is effective for the primary complaint of tinnitus.

In reviewing the numerous studies of ginkgo and tinnitus, few of these studies were properly controlled. Moreover, methodological variances between those studies that were well controlled prevent any definitive conclusions. However, because there are a number of reported positive outcomes, there may be potential for ginkgo to be an effective treatment for some patients. Patient factors that might be predictive of benefit need to be identified and verified through research. It thus seems worthwhile to conduct properly controlled trials of ginkgo using treatment regimens that would optimize the possibility of achieving successful outcomes.

Zinc. The trace element zinc, which is mentioned often as a tinnitus treatment, is used commonly in commercial tinnitus formulas. Paaske, Pedersen, Kjems, and Sam (1991) performed a placebo-controlled, randomized, double-blind investigation of zinc in 48 patients. Only 1 of these patients had a reduced level of serum zinc. Their study did not result in any beneficial effect from zinc on tinnitus. They suggested a possible reason for this negative finding was that their patients had serum zinc levels in the normal range at the start of treatment. Ochi et al. (1997) reported reduced serum levels of zinc in tinnitus patients relative to controls. The tinnitus patients were reported to have a significant reduction on a subjective tinnitus scale when treated with a regimen of zinc. Yetiser et al. (2002) did not find reduced zinc levels in a group of 40 tinnitus patients and reported that zinc therapy was effective only for some patients. Arda, Tuncel, Akdogan, and Ozluoglu (2003) conducted a randomized, placebo-controlled study in 41 tinnitus patients. Those patients who received zinc showed a significant reduction in subjective tinnitus severity, whereas those receiving placebo did not. The authors reported that 31% of their patients had low zinc levels but that tinnitus improvement was not limited to these patients. These studies indicate that zinc is effective for some patients, but it cannot be stated that zinc is generally helpful for most patients.

Acupuncture. Acupuncture is a method that can be effective in controlling pain. Tinnitus and pain are often theorized to share a common mechanism. Accordingly, acupuncture has been studied for its effects on tinnitus. Park et al. (2000) reviewed six randomized, controlled trials, of which four were blinded. Methodology and outcome measurements varied between studies. Although the two unblinded studies showed a positive result, the four blinded studies showed no significant improvement with acupuncture.

Efficacy of Treatments for Tinnitus

Dobie’s (1999) review of 69 randomized, clinical trials included many drug studies as well as nondrug treatments that included psychotherapy (cognitive-behavioral therapy), electrical stimulation, magnetic stimulation, ultrasound, biofeedback, acupuncture, and hypnosis. Dobie updated his review to include randomized, clinical trials that had been conducted between 1998 and 2003 (Dobie, 2002, 2004a). Although he noted some promising treatments, he emphasized the difficulty in performing these studies to control for nonspecific (placebo) effects.
Nonspecific effects that are due to patient expectations could be the reason that most tinnitus treatment efficacy studies have shown good success rates (Axelsson, 1998; Dobie, 2004a; Tyler, Haskell, Preece, & Bergan, 2001). To conduct such studies properly, there should be rigorous design standards concerning the following: qualifications for study inclusion, the use of validated outcome measures, and randomization into treatment groups, including a no-treatment (i.e., waiting list) group (Dobie, 2004a). It is critical to include a nonspecific control group that addresses the concern that a patient's tinnitus condition might improve simply as a function of the amount of professional attention received (Isenberg, 1998; Roberts et al., 1993). This attention effect could be controlled for by providing equal clinician attention but in a nonspecific fashion (Streiner & Norman, 1998). A well-controlled study, mentioned above, involved tinnitus intervention either with education alone or with education combined with cognitive–behavioral therapy (J. L. Henry & Wilson, 1996). Although patients in both groups received the same duration of intervention, the cognitive–behavioral group received both the education and the cognitive–behavioral training. The authors noted, “The material was provided at a slower pace in the education-only program” (p. 11). Thus, the cognitive–behavioral group received greater intensity of clinician interaction that could have confounded the outcome measurements. This greater intensity involved instruction regarding various cognitive and behavioral modifications designed to improve coping skills. The education-only group received purely didactic information devoid of instruction concerning potential strategies for making their tinnitus less bothersome.

A cursory review of the tinnitus literature would suggest that many diverse methodologies are effective in the treatment of tinnitus (Axelsson, 1998). A closer look at these studies reveals that very few of them were well controlled, thus “The state of our therapeutic knowledge is still quite primitive” (Dobie, 2004a, p. 270). Future studies should be randomized, controlled trials that adhere to rigorous experimental methods. Research that follows the recommendations outlined by Dobie (2004a) and Isenberg (1998) would produce outcome data that are valid, comparable, and clinically useful.

**Discussion**

This article reviews what amounts to an extensive literature relating to tinnitus. Two dominant points define the field: (a) There is increasing interest in the phenomenon of tinnitus, both in terms of understanding its underlying mechanisms and in the development of management techniques; and (b) many inconsistencies exist regarding definitions and terminology, outcome measures, methods of assessment, and approaches to treatment. These two points emphasize the need for professionals to work together to bring uniformity to this field.

There have been numerous attempts to unify the tinnitus profession to standardize techniques for evaluation and treatment (Evered & Lawrenson, 1981; McFadden, 1982; Meikle & Griest, 2002; Vernon & Fenwick, 1984). Clearly, however, clinicians and scientists have not formed any common union to achieve consensus regarding needed standardization. There are several different schools of thought for tinnitus treatment, each advocating their own brand of therapy, which ultimately leaves the tinnitus patient unable to make well-informed decisions regarding the best course of treatment.

There are four areas that must be addressed to achieve standardization within the field: (a) establishing clinical methodology (procedures for screening, assessment, outcome measures, and intervention), (b) obtaining research evidence to support clinical methodology, (c) developing training for professionals (graduate programs, seminars, online courses, textbooks, etc.), and (d) developing patient education (various media to provide educational resources). Some suggestions are mentioned briefly below to address these four areas.

**Clinical Methodology**

It bears repeating that tinnitus is a clinically significant condition for only about 20% of individuals who experience permanent tinnitus (Davis & Refaie, 2000; P. J. Jastreboff & Hazell, 1998). It is therefore important to be able to establish whether clinical intervention is necessary and, if so, how much intervention is required. This would require, first, an efficient and effective method of screening to determine if the patient is in need of clinical services. We have developed the Tinnitus-Impact Screening Interview (TISI) for this purpose (Henry, Schechter, et al., 2004). The TISI consists of six interview questions and has worked well to rapidly assess the potential need for services. In the process of administering the TISI, many of the individual’s questions are answered, which often provides sufficient information for the individual to be satisfied that nothing further is required.

Delivering clinical services in the most expeditious manner would require a progressive intervention approach (Henry, Schechter, et al., 2004). That is, patients should receive only as much treatment as necessary to meet their need. The lowest tier of intervention would be written information, or possibly a tinnitus educational
video. The next tier might be some kind of structured group educational session(s). If additional help was still needed, a clinical assessment would be performed along with some basic counseling. The assessment and minimal counseling would either suffice for meeting the patient’s need or would suggest the need for comprehensive, long-term intervention (the highest level of service).

**Research Evidence**

The delivery of health care services should be based on research evidence (Darby, 1998; Feussner, 1998; Meikle & Griest, 2002). This is not generally the case for tinnitus for which there are many treatments but little supportive research. Properly controlled clinical trials need to be conducted. These trials should use a common set of guidelines to assess outcomes of treatment (Dobie, 2004a; Isenberg, 1998). There is the important need to conduct well-designed, large-scale epidemiology studies to define the problem of tinnitus and to identify factors that cause tinnitus to become problematic for some people. Mechanisms of tinnitus are being studied, and these efforts are essential to ultimately develop cures for tinnitus.

**Professional Training**

Because hearing-health specialists do not normally receive training in the management of tinnitus, methods of training need to be developed that will work for busy professionals. All professionals who are at the point-of-contact for tinnitus patients should be aware that tinnitus is a preventable and treatable problem. They need to know that telling the patient that “nothing can be done—learn to live with it” dispenses erroneous information that can exacerbate the problem. A rudimentary level of professional training would thus involve only a brief informational sheet that would contain information about tinnitus: Why it occurs, how it affects patients, and methods of treatment. Professionals should be provided with brochures that can be given to their tinnitus patients. These brochures should contain basic information about tinnitus and should list resources to obtain further information. Brochures of this kind can now be obtained from the American Tinnitus Association.

Professional training of clinicians to manage tinnitus patients is generally lacking. Various levels and formats of training should be developed to appeal to different levels of interest among clinicians. These formats may range from a brief training booklet or a short website training course to continuing education and seminars that provide step-by-step training instructions. Most important, audiologists and otolaryngologists should have training as part of their formal educational program, which would obviate these types of postcurricular programs for these professions.

**Patient Education**

Any individual who experiences tinnitus should have readily available accurate, up-to-date, comprehensive information about tinnitus. Most important, the individual needs to know that help is available. The American Tinnitus Association hosts a comprehensive website (www.ata.org) containing information about tinnitus and a list of clinical resources. Tinnitus sufferers can greatly benefit from the information provided in this website. What they cannot learn, however, is where they can receive “standard treatment” for tinnitus. Standard practice for tinnitus is not yet a reality, and its development will require a professional organization of tinnitus researchers and clinicians, with a leadership body that works together to establish standards and guidelines for the field.

**Summary**

This article provides an overview of the state of knowledge in the field of tinnitus. Tinnitus may be caused by a somatic disorder, or it may have a neurophysiological (sensorineural) origin. Whereas tinnitus of somatic etiology is often correctable through surgery, sensorineural tinnitus can generally only be managed. Fortunately, of the approximately 10% to 15% of the population who experience tinnitus, about 80% do not require clinical intervention. All individuals with tinnitus, however, want their tinnitus sound “silenced.” Many research studies are under way in the attempt to achieve this goal. Ultimately, mechanisms-based research offers the greatest hope for completely and safely silencing tinnitus. In the meantime, numerous methods exist for the treatment of tinnitus. Although research evidence is scant, some of these methods can be effective for the majority of patients when the protocols are performed properly. Future efforts will undoubtedly unify the field of tinnitus professionals and lead to a consensual and evidence-based standard of practice for the clinical management of tinnitus.

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