Chapter 173: Tinnitus

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Tinnitus is perception of a sound produced involuntarily within the body. It may be the first or the most prominent symptom of various disease processes that threaten the patient's physical health and well-being. Awareness of these diverse disease processes is necessary for prompt diagnosis, effective treatment, and appropriate referral. Even when threatening disease is not present, the quality of life can be impaired severely by the psychologic effects of tinnitus on the patient and indirectly on the involved family. Tinnitus may even precipitate suicide in some patients. The physician must be patient, concerned, and knowledgeable to provide the most effective symptomatic treatment for the patient.

Tinnitus can be divided into two major categories: (1) tinnitus generated by paraauditory structures, usually from vascular or myoclonic sources; and (2) tinnitus generated by the sensorineural auditory system. This division is useful because it parallels our anatomic and physiologic distinctions, and generally, diagnostic evaluation and treatment are quite different. Further understanding may allow delineation of cochlear tinnitus, auditory nerve tinnitus, central tinnitus, etc. Separation of tinnitus into subjective tinnitus and objective tinnitus, which could be heard by the examiner as well, has limited usefulness. The same disease processes might produce subjective tinnitus in some patients and objective tinnitus in others. In addition, studies of cochlear emissions have revealed that a sound that examiner can hear in the ear canal may not be heard by the patient (Glanville et al, 1971) and often originates in the inner ear (Kemp, 1981; Norton et al, 1990; Tyler, 1982; Zurek, 1981).

Tinnitus Generated by Paraauditory Structures

The body continually produces noise that can be transmitted to the base of the skull. This noise is usually inaudible because of its intensity and spectral characteristics relative to certain mechanical characteristics of the ossicular chain (von Bekesy, 1960) and the threshold sensitivity of the ear at the frequencies in question. A patient becomes aware of such noise for several reasons. Wth venous hums or occasionally with acute middle ear inflammation, normal body sounds may increase in intensity. With conductive hearing loss of diverse etiology, externally produced background noise is attenuated, making normal skull sounds more audible. Finally, abnormal arteriovenous malformations (AVMs) or palatal myoclonus may be present.

Evaluating rhythmicity of the tinnitus in relation to the peripheral pulses and examining the head and neck thoroughly are the keys to diagnosing the following entities that produce tinnitus.

Vascular neoplasm

Most cases of paraauditory tinnitus arise because the cochlea detects blood flow. In terms of morbidity and mortality, vascular tumors potentially have the most serious prognosis. The intensity of the tinnitus varies with the complexity of the vascular bed, the pressure and flow through the tumor, and the neoplasm's proximity to the ear.

Diagnosis

Determining that the tinnitus is pulsatile and that it varies directly with cardiac rate is essential to diagnosing a vascular tumor. These conditions may be confirmed by the history or detected by light exercise. The physician then searches thoroughly for an audible bruit, which includes auscultating the external canal with a Toynbee tube and the orbit, mastoid process, skull, and neck using a stethoscope with both bell and diaphragm. The examiner also can place a small, sensitive microphone (Villchur and Killion, 1975) in the closed ear canal and use amplification to listen to even the softest sounds. When a bruit is found, a palpable thrill is sought. The bruit of a vascular tumor usually is not altered appreciably by light pressure on the neck, head position, posture, or Valsalva maneuver, important points of differential diagnosis. When manipulating the patient's vasculature, the physician must consider the possible presence of significant atherosclerotic vascular compromise.

Tympanometry also can be used in the examination (Leveque et al, 1979). Regular pertubations on the tympanogram or a trace of compilance as a function of time (at the maximal compliance) can be used to calculate the regularity of the pulsations (Fig. 173-1). This can be compared directly with the patient's pulse rate.

Further examination includes visualization of the tympanic membrane. A bluish or reddish mass is highly suggestive of a glomus tumor; obvious pulsations of the mass and paling when positive pressure is applied by a pneumatic otoscope support this diagnosis. Other entities to be considered include hemotympanum, dehiscent jugular bulb, and carotid artery abnormality. Radiographic studies (described later) should precede any surgical procedure such as myringotomy or exploratory tympanotomy.

The physician should check for masses during the rest of the head and neck examination even in the absence of a bruit. Areas such as the scalp, paranasal sinuses, pharynx, oral cavity, floor of the mouth, and neck should not be overlooked. Dentition should be inspected for loosening or bleeding. Because many of these lesions either originate intracranially or have extended there, evaluation of cranial nerve function and cerebellar or long tract compromise is important.

Radiography plays an important role in the evaluation and management of vascular tumors. A simple Panorex may suffice for a mandibular or maxillary tumor. Arteriography is performed on most patients to delineate feeder vessels and the tumor's extent. The tumor and its effect on adjacent structures are well demonstrated by contrast-enhanced computed tomography (CT). Other studies that occasionally may provide useful adjunctive data include jugular venography and digital subtraction angiography, which are discussed in Chapter 186. Magnetic resonance imaging is expected to play an ever-increasing role in the evaluation of these lesions.

Management

Management of vascular tumors is generally by surgical excision. Glomus tympanicum and glomus jugulare tumors are the prototypes for neoplasms occurring with pulsatile tinnitus; their diagnosis and management are detailed in Chapter 192. As with glomus neoplasms, other vascular tumors are more often benign than malignant. Excision provides the histologic diagnosis and prevents potentially life-threatening complications that follow their continued growth or infection. At times, convincing a tumor patient with no other symptoms that the tinnitus is the prelude to neurologic disaster is difficult. (Otolaryngologists usually have the opposite problem of convincing their patients with sensorineural tinnitus that they do not have a brain tumor.) Surgical strategies usually include ligation of feeding arteries and excision of the tumor with as little normal tissue as possible. Neoplasms that are obviously malignant require wider excision and perhaps radiation or chemotherapy, depending on cell type.

Arteriovenous malformations

Diagnosis

AVMs may be difficult to differentiate from neoplasms, but the distinction is important. AVMs are developmental abnormalities and often are considerably more extensive than their symptoms would suggest. Potential microscopic channels may canalize rapidly and result in rapid enlargement with increasing tinnitus. Remaining channels also are responsible for a "recurrence" of an AVM after surgical ablation. These lesions impinge on adjacent structures, causing deformation by a mass effect, and may enlarge rapidly during pregnancy. Less often they may result in enough low-resistance arteriovenous shunting to cause high-output cardiac failure.

The most common AVMs are those of the posterior fossa between branches of the occipital artery and the transverse sinus (Arenberg and McCreary, 1972). AVMs of the mandible are uncommon, but notorious for causing tinnitus (Babin et al, 1983). Communications between the carotid artery and cavernous sinus, most often resulting from trauma, are infrequent but have serios consequences.

Pulsatile tinnitus is often the initial complaint of patients with AVMs of the head and neck. Other symptoms include distortion of the face or neck and discoloration of skin or mucosa. Intracranial AVMs often are associated with headache and nonspecific signs of increased intracranial pressure such as papilledema (Arenberg and McCreary, 1972).

As with vascular neoplasms, AVMs usually have a bruit. The vascular mass, when palpable, exhibits a thrill and generally is more compressible than a tumor. If the degree of arteriovenous shunting is sufficient, the cardiac rate may decrease during such compression. AVMs occurring in the mandible and maxilla appear with loosening of teeth, periodontal bleeding, and mucosal discoloration. The most common cause of death in patients with these lesions is exsanguination during a tooth extraction for biopsy (Lambert et al, 1979).

Management

As with vascular tumors, the treatment of AVMs is usually surgical and is facilitated greatly by selective carotid angiography. When a single branch of the external carotid system is the arterial supply for an AVM of the posterior fossa, face, or neck, vessel ligation and lesion excision should be effective because they eliminate the abnormal flow pattern. As progressively more arterial contributions feed the lesion, the likelihood of complete eradication of both the malformation and the tinnitus diminishes substantially. In this situation more potentially neocanalizing tracts also probably exist, increasing the chance of recurrence. As a general rule, during excision the malformation is larger than it appears radiographically. Arterial embolization may be used as an adjunct to control bleeding during surgery (Latchaw et al, 1980) or as definitive therapy in a lesion not amenable to resection (Castaneda-Zuniga et al, 1979; Harris et al, 1979), such as a traumatic carotid artery - cavernous sinus fistula. When adjunctive embolization is used, maximal benefit will be achieved if subsequent surgery is performed within 72 hours (Babin, 1984). In single-vessel, well-defined fistulas, such as traumatic fistulas in the neck, occlusion may be accomplished by the radiographic placement of a detachable balloon catheter.

Venous hum

Venous hum is another vascular sound that emphasizes the necessity for differentiating high-pressure-flow from low-pressure-flow lesions. The hum results partly from eddy currents in the jugular vein and normally is heard in the neck of many children and some adults, most, notably, young women. These hums have been attributed to the transverse process of the second cervical vertebra impinging on the jugular vein (Cutforth et al, 1970) and less commonly to increased venous return secondary to an AVM (Hardison, 1968) and increased intracranial pressure (Meador, 1982). Venous hums increase in loudness in states of increased cardiac output, such as anemia, thyrotoxicosis, and pregnancy. Once diagnosed, they are significant only for the tinnitus they produce. These and other vascular sounds may become audible when a conductive hearing loss occurs in which external background noise is attenuated, similar to a lateralized Weber test.

Diagnosis

Venous hums may have symptoms that are nearly diagnostic. The tinnitus may be eliminated by gentle pressure on the anterior neck that does not occlude the carotid artery. Turning the head toward the uninvolved side decreases the sound. Turning the head toward the involved side makes it louder, as do deep breathing and Valsalva maneuver. In the past, a negative arteriogram was considered essential for the diagnosis of venous hum (Ward et al, 1975). Without classic symptoms or with the suggestion of associated vascular anomalies (Hardison, 1968), a diagnostic arteriogram still is mandated.

Management

Management of venous hum may consist simply of reassuring the patient. Sound not tolerated by the patient or spouse may respond to a high ligation of the jugular vein. Preoperative angiography is necessary in this instance to verify a patent contralateral venous system. The level at which an effective ligation may be achieved can be determined by inflation of a Fogarty catheter balloon at different levels during a retrograde venogram (Ward et al, 1975). This approach may reveal some patients who still require ablation of their transverse sinus to eliminate the sound. Cary (1961) described the use of a prosthetic device that put gentle, constant pressure on the neck.

Muscle contraction tinnitus

A nonvascular cause of middle ear tinnitus is the synchronous contraction of many or most of the fibers of one or more middle ear or palatal muscles. This condition may occur as discrete single contractions, either voluntary (Collard et al, 1982) or involuntary (Williams, 1980), or as part of the syndrome of palatal myoclonus.

Palatal myoclonus consists of rapid, repetitive contractions (60 to 200 per minute) of the tensor and levator veli palatini, tensor tympani, salpingopharyngeal, or superior constrictor muscles. Patients are typically young and often have other associated neurologic disorders such as brainstem infarction or multiple sclerosis (Herrman et al, 1957). Cerebellar dysfunction has been implicated in many persons with this type of myoclonus (Toland et al, 1984). Unlike many involuntary movements, palatal myoclonus is not inhibited by sleep, barbiturate anesthesia, coma or hemiplegia. Although it continues during phonation and caloric tympanic membrane stimulation, it may be inhibited by those nonacoustic stimuli that trigger the acoustic reflex. The audible click heard by some of these patients is synchronous with the relaxation phase of the myoclonus and tubal closure (Pulec and Simonton, 1961).

Diagnosis

The first task in the diagnosis of muscle contraction tinnitus is to attempt to hear it. The physician should auscultate the mastoid process, neck, skull, and orbit and then determine if the sound is synchronous (vascular) or asynchronous (nonvascular) with the pulse. Because palatal myoclonus may be within the range of a normal pulse rate, the observation that the tinnitus rate does not change when the patient exercises implicates palatal myoclonus. Observation of the palate is not necessarily diagnostic, as opening the mouth may suppress myoclonus. If rhythmic muscle contraction is suggested, tympanometry can be diagnostic. A plot of compliance as a function of time should be obtained at the middle ear pressure equal to maximal compliance; increasing the sensitivity of the instrument sometimes aids in this calculation. This can demonstrate a periodic decrease in compliance synchronous with the tinnitus (Toland et al, 1984). The compliance tracing obtained in cases of muscle-contraction tinnitus has a sawtoothlike waveform and is less periodic than the tracing obtained from vascular tinnitus.

Management

Management of palatal or tensor tympani myoclonus has taken two forms: (1) attempts to decrease the hyperactive motor discharge to the muscles and (2) procedures that render the muscle contractions less symptomatic. Diphenylhydantoin (phenytoin) and carbamazepine have been reported to successfully suppress myoclonus, as has valproic acid (Toland et al, 1984). Opening the mouth, touching the palate, and filling the mouth with water all temporarily suppress myoclonus, and blowing cold air on the cornea reportedly suppresses it for longer periods. In one case of learned tensor tympani cotraction, biofeedback resulted in its suppression (Collard et al, 1982). Surgical approaches have included sectioning of the tensory tympani muscle, dislocation of the tensor veli palatini tendon from the hamulus, and hamulus fracture (Williams, 1980). Even a myringotomy with ventilating tube placement (Kwee and Sturben, 1972) has been attempted.

Sensorineural Tinnitus

Etiology

Unfortunately, little is known about the underlying physiologic mechanisms that cause sensorineural tinnitus. Because the presentation of sound results in an increase of neural discharge rates over spontaneous activity, it is reasonable to suppose that tinnitus may also be accompanied by an increase in the rate of neural discharges. None of the many speculations about mechanisms that produce sensorineural tinnitus have been clearly substantiated. Generally these theories involve hyperactive hair cells or nerve fibers activated by a chemical imbalance across cell membranes or decoupling hair cell stereocilia (Tonndorf, 1980). An abnormally low spontaneous hair cell discharge rate could result in the patient perceiving a sound, similar to an amputee patient perceiving a phantom limb. Kiang et al (1970) also proposed a transition region between areas of normal and abnormal hair cells. A lack of mutual lateral suppression (von Bekesy, 1967) from the abnormal region toward the normal region would result in increased spontaneous activity by the normal cells. Moller (1984) recently suggested that adjacent auditory nerve fibers could become damaged in such a way that artificial synapses between them might occur. This reaction could possibly result in ephaptic transmissions between fibers, and correlated spontaneous activity between fibers could be perceived as tinnitus.

Eggermont (1984) suggested that hypersensitivity, perhaps due to a loss of inhibition, muight be one cause of sensorineural tinnitus. He also suggested that tinnitus might be caused by an abnormal displacement of the basilar membrane toward the scala tympani, resulting in hyperactivity (Sellick et al, 1982). Spoendlin (1987) suggested that it results from the presence of damaged outer hair cells and normal inner hair cells. Tonndorf (1987) also suggested that chronic tinnitus may be related to deafferentation of the nerve fibers, analogous to some theories of pain. Eggermont (1990) continued with the notion that tinnitus results from spontaneous correlated neural activity, possibly resulting from ephaptic excitation of one nerve fiber by adjacent fibers, or from synchronous hair cell discharges (possibly related to spontaneous sodium or calcium influx). Jastreboff (1990b) also emphasized the transfer of calcium in a detailed biologic explanation of tinnitus.

Animal studies

Animal models are an important way to examine these theories about sensorineural tinnitus; however, this approach is difficult because determining whether the animal actually hears any sound is impossible, even if abnormal spontaneous activity occurs. The usual approach is to treat the animal with an agent known to produce tinnitus in human beings and to look for electrophysiologic abnormalities.

Studies of animals who have been deafened artificially by noxious agents, such as noise and kanamycin, have largely found a decrease in the spontaneous discharge rate of auditory neurons (for example, Kiang et al, 1970; Schmiedt et al, 1980). A few studies, however, have reported some evidence of activity that could be related tinnitus.

Evans and Borerwe (1982) found an increase in the mean discharge rate of nerve fibers with normally high spontaneous activity in cats treated with salicylates. Jastreboff and Sasaki (1986) also reported an increase in the spontaneous activity in the inferior colliculus in albino guinea pigs after administration of sodium salicylate. Slightly elevated spontaneous activity in nerve fibers in animals exposed to damaging noise also has been observed (Liberman and Kiang, 1978; Salvi and Ahroon, 1983; Schmiedt et al, 1980). Jastreboff et al (1988) reported abnormally high spontaneous activity in the inferior colliculus of guinea pigs that had been taking salicylates.

Initial work on a behavioral animal model of tinnitus has been proposed by Jastreboff et al (1988a and 1988b). First they deprived rats of water until they were 80% of their initial body weight. They were then trained to lick a water dispenser during the presence of a continuous background noise. In the next acclimation phase, the rats were acclimatized to the 30-second offsets (the conditioned stimulus) of the background noise. This was followed by condition suppression training, where the noise offsets were coterminus with electrical shocks (unconditioned stimulus). Subsequent sessions were extinction trials, where the noise offset trials continued but without further shocks. Control rats lick happily during the continuous background noise and then during the acclimation phase with noise offset intervals. However, with the shocks they quickly reduce their licking activity during the quiet intervals. When the shocks are withheld, they gradually learn that it is appropriate to lick during this phase, and their licking behavior increases again. In one group of rats, salicylates were introduced, presumably producing tinnitus. Some rats received the salicylates before suppression training. The noise probably masked the tinnitus, but the aversive stimulus became the presence of the tinnitus and not the quiet. These rats showed a more gradual extinction period, perhaps because they heard a tinnitus during the extinction trials.

Although these studies show some promise for an animal model for tinnitus, further investigation is needed. Most patients have a prominent pitch associated with their tinnitus, even if their description suggests a tinnitus analogous to a broad-band tinnitus (Tyler and Conrad-Ames, 1983b). Thus an increase in spontaneous activity in nerve fibers should show either a place-dependent increase or some temporal periodicity.

Relationship to different lesions

At present no evidence shows that different types of tinnitus are associated with different lesions, such as noise-induced hearing loss, drug-induced hearing loss, Ménière's disease, or acoustic neoplasms. That is, no measurements of sensorineural tinnitus or perceptual descriptions are pathognomonic of a single lesion. Douek and Reid (1968) noted that patients with Ménière's disease generally had a low-pitch tinnitus (below 500 Hz), but so did patients with other lesions. Stouffer et al (1991) found that patients with Ménière's disease generally have more problems, which they associate with the tinnitus. It is louder, more annoying, and more unstable (it changes more often). Different types of lesions probably produce different types of sensorineural tinnitus. It is hoped that some measures of tinnitus will emerge that selectively differentiate among different etiologies.

Tinnitus and aging

The prevalence and severity of tinnitus increases with age. Axelsson and Rinhdahl (1987) noted that 337 people with tinnitus were identified in a population of 2378 Swedes (14.2%). Fig. 173-2, A, shows that the percentage of the population with tinnitus increases with age. This age effect is consistent with reports from Coles (1987), who indicates that the increase in tinnitus in the aged is independent of noise exposure. Axelsson and Ringdahl also noted that some of the disturbing qualities of tinnitus are greater in the older population (Fig. 173-2, B). As the number of older patients increases, more tinnitus patients will be identified clinically.

Evaluation

The quantification of sensorineural tinnitus is in its infancy. Although many techniques can be applied, such as pitch and loudness matching, their interpretation and application are uncertain. In addition, the test stimulus itself probably has some influence on the tinnitus (Bilger et al, 1983; Tyler and Conrad-Armes, 1983b), adding to the inherent variability of its measurement. This problem is complicated further because tinnitus fluctuates daily in some patients (Penner, 1983; Stouffer and Tyler, 1990). Nevertheless, reliable observations can be reported to increase our understanding. Tinnitus measurements have been used to study treatment (Tyler et al, 1984) and to monitor patient habits, such as caffeine intake (Malatesta et al, 1980). Several parameters can be examined.

Questionnaires can also be useful to better understand patients' problems and to quantify subjective attributes. A Tinnitus Handicap Scale (Kuk et al, 1991; Tyler, Stouffer, and Schum, 1990) can be used to document systematically the problems associated with tinnitus (see box), to compare tinnitus among patients, and to evaluate patients' handicap over time.

Box: Tinnitus Handicap Questionnaire

Indicate to what degree you agree or disagree with the following statements by writing in a number from **0** to **100.**

0 indicates that you strongly disagree
100 indicates that you strongly agree

Numbers between **0** and **100** should also be used to represent your level of agreement with each statement.

PLEASE ANSWER *ALL* THE QUESTIONS

1. I do not enjoy life because of tinnitus.
2. My tinnitus has gotten worse over the years.
3. Tinnitus interferes with my ability to tell where sounds are coming from.
4. I am unable to follow a conversation during meetings because of tinnitus.
5. Tinnitus causes me to avoid noisy situations.
6. Tinnitus interferes with my speech understanding when talking with someone in a noisy room.
7. I feel uneasy in social situations because of tinnitus.
8. The general public does not know about the devastating nature of tinnitus.
9. I cannot concentrate because of tinnitus.
10. Tinnitus creates family problems.
11. Tinnitus causes me to feel depressed.
12. I find it difficult to explain what tinnitus is to others.
13. Tinnitus causes stress.
14. I am unable to relax because of tinnitus.
15. I complain more because of tinnitus.
16. I have trouble falling asleep at night because of tinnitus.
17. Tinnitus makes me feel tired.
18. Tinnitus makes me feel insane.
19. Tinnitus contributes to a feeling of general ill health.
20. Tinnitus affects the quality of my relationships.
21. Tinnitus has caused a reduction in my speech understanding ability.
22. Tinnitus makes me feel annoyed.
23. Tinnitus interferes with my speech understanding when listening to the television.
24. Tinnitus makes me feel anxious.
25. I think I have a healthy outlook on tinnitus.
26. I have support from my friends regarding my tinnitus.
27. I feel frustrated frequently because of tinnitus.

To obtain total score, subtract 100 from questions 25 and 26, add scores from all questions and divide by 27.

Quality

Patients' descriptions of tinnitus include many sounds, such as buzzing, rushing, ringing, roaring, and whistling. Stouffer and Tyler (1990) reported that 38% of their subjects described their tinnitus as ringing and 11% as buzzing. Because these descriptions depend on the patient's vocabulary and previous listening experience, they usually have limited diagnostic significance. Reports of pulsing or popping sounds should alert the physician to a vascular or muscular tinnitus, but these sounds may also occur with sensorineural tinnitus.

Many patients describe hearing two or more sounds; for example, they may report hearing a low-pitched hum and a high-pitched whistle in the same ear. Interestingly, one of the sounds occasionally fluctuates while the other remains constant, or a noise may mask one of the sounds but have no effect on the other. This suggests that the two sounds have separate generators.

Perceptual location

Patients perceive tinnitus in one ear; in both ears; at the back, middle, side, or front of the head; and occasionally outside the head. For example, Stouffer and Tyler (1990) reported that in their patients, 52% reported bilateral tinnitus, 37% unilateral tinnitus, 10% in the head, and less than 1% outside the head. Meikle and Griest (1987) reported 61% bilateral tinnitus, 21% unilateral, 3% in the head, and multiple or other locations in their remaining patients.

At present, however, this information has limited diagnostic importance and may be misleading. A tinnitus perceived in one ear can often be masked as effectively in the ear contralateral to the tinnitus (Tyler and Conrad-Armes, 1983b). This finding suggests that the masking occurs centrally. In some patients, when a tinnitus perceived in one ear is masked by a noise in the same ear, a "new" tinnitus can appear in the opposite ear, perhaps indicating that tinnitus was present in the contralateral ear all along but at a lower level. Thus a distinction between peripheral and central tinnitus cannot be made based on the patient's report of location.

Interestingly, Meikle et al (1987) reported that 9% of their 519 tinnitus patients described a variable location and 19% said it was in a different location from that previously documented. They noted that a common trend is for the tinnitus to initially be audible in one ear and then later to appear bilaterally.

Pitch

Although tinnitus usually does not resemble a single tone, most patients are able to equate the pitch elicited by a pure tone with the most prominent pitch of their tinnitus. Several techniques are available to estimate tinnitus pitch. The patient may be asked which of two tones has a pitch closer to the tinnitus or whether the tinnitus has a higher or lower pitch than that of a pure tone. We have asked used a method of adjustment in which the patient manipulates the frequency of a tone until its pitch is about equal to the most prominent pitch of the tinnitus. We use a graphic equalizer (between the tone generator and the earphones) to keep the intensity of the pure tone above threshold and at a similar loudness

throughout the frequency region as the frequency is varied.

In most patients, the pitch match frequency is above 2000 Hx. Reed (1960) and Penner (1990) both noted that the most common frequency matched is 4000 Hz. Vernon (1988) claims that about 83% of his patients have a pitch-match frequency above 3000 Hz. Some patients show good test-retest reliability for pitch matching and good consistency over time. Many patients, however, are unable to perform this task reliably. Burns (1984) found that the standard deviation of pitch matching to tinnitus could be 10 or 20 times greater than to pure tones (see also Penner 1983, who used binaural stimuli and Tyler and Conrad-Armes, 1983b).

Some patients may provide that pitch matches that are distributed in two clusters, about one octave in frequency apart. These octave confusions could be identified by asking the patient if a tone one octave higher or one octave lower than the frequency selected is closer to the tinnitus pitch. If octave confusions do occur, they could be of important theoretical interest. However, Penner (1983) obtained over 80 matches per subject in three subjects and did not observe octave confusions. A close examination of their existence needs to be evaluated in more patients.

The choice of ear to receive the test stimuli also may influence the results. A different pitch-match frequency may be obtained depending on which ear receives the tone (Tyler and Conrad-Armes, 1983b). If the tinnitus and tone are in the same ear, then the tone may change the tinnitus. When binaural diplacusis is present and the tinnitus and tone are in different ears, inaccurate matches can result. Binaular presentation of the tone may be confounded by both effects, and the patient may "listen" with different ears on different trials. We recommend monaural testing, with the examiner noting the test ear.

Penner (1980) used a unique technique for determining the spectral locus of the tinnitus. Patients were required to adjust the low-frequency cutoff frequency of a high-pass noise - fixed spectrum sound pressure level (SPL) of 43 dB - until it just masked the noise. Then the high-frequency cutoff frequency of a low-pass noise was similarly adjusted. The frequency region common to both was called the "masking interval" and was related to the spectral region and bandwidth of the tinnitus.

It appears that the pitch matching reliability varies widely among patients. This finding is consistent with reports that in about one in every two patients with tinnitus, the pitch varies from day to day or within a day (Stouffer and Tyler, 1990). Furthermore, they report that in one of every five patients, the tinnitus pitch increases over time. Even when the tinnitus pitch is reported to be stable, some patients will have difficulty providing an accurate pitch match. In these cases, several replications of the pitch match must be obtained, perhaps as many as 15 or 20 depending on the variability of the patient. The pitch match frequency, number of matches made, and standard deviation should be reported. It is important to give patients adequate instruction and practice trials.

Loudness

The loudness of tinnitus has been measured by having the subject adjust the level of a pure tone so that it has about the same loudness as the tinnitus. Several researchers, including Fowler (1940), have commented that the level of this pure tone of equivalent loudness was only 3 or 4 dB above threshold, or sensation level (SL), even when the patients complained that the tinnitus was very loud. Although this finding appears inconsistent at first, SL units are not measurements of loudness; the SL in decibels (dB SL) represents the intensity, not the loudness, of the signal above threshold. Goodwin and Johnson (1980) realized this difficulty and noted that the loudness match, in dB SL, could be much greater at a frequency with normal hearing than at a frequency with a threshold loss.

Tyler and Conrad-Armes (1983b) tried to calculate the loudness of tinnitus, taking into account the threshold loss and loudness recruitment. Fig. 173-3 shows their model, which indicates the loudness of a tone in sones (Stevens, 1955), the conventional psychoacoustic units of loudness. he equation shown in Fig. 173-3 attempts to predict tinnitus loudness given to dB SL loudness macth, and the person's threshold, in decibels of hearing level (HL) or equivalent. In normal listeners, a tone of 10 dB SL has a loudness of about 0.1 sones; in a listener with a 35 dB HL threshold, a 10 dB SL tone has a loudness of nearly 2 sones (20 times louder than normal); in a listener with a 70 dB HL threshold, a 10 dB SL tone has a loudness of nearly 20 sones (200 times louder than normal). Although this model is based on an assumed typical recruitment function and, therefore, may not be precise for each individual, it does illustrate the severe loudness and thus the disturbing effect of tinnitus (see also Penner, 1984, 1986, 1988b; and Hinchliffe and Chambers, 1983). Tyler and Conrad-Armes (1983b) also observed patients who had very low dB SL loudness matches in regions of both normal and abnormal thresholds. Loudness recruitment cannot account for the discrepancy between subjective reports of a loud tinnitus and low dB SL measurements in these patients.

The variability of loudness adjustments also differs among patients. Although it can be greater than the variability of matching to an external tone, the variability is not as great as with tinnitus pitch (Burns, 1984; Penner, 1983).

Stouffer and Tyler (1990) reported that over half of their patients experienced fluctuating loudness in their tinnitus, and half reported that it changed from day to day. In one third of patients, the tinnitus loudness increased over time. Meikle et al (1987) reported that approximately 80% of their 519 patients indicated that their tinnitus loudness fluctuated. Tyler and Stouffer (1989) have recently reviewed different procedures for estimating tinnitus loudness.

Masking

In many patients the presentation of a pure tone or noise can mask tinnitus completely. Vernon (1988) reported that 91% of 491 clinic patients could be masked completely. That tinnitus can be masked suggests that the tinnitus and the response to the acoustic stimulus share the same neural channels somewhere in the nervous system.

Patterns. A tinnitus-masking pattern can be measured by determining the minimal level required to mask the tinnitus at several tone frequencies (Bailey, 1979; Formby and Gjerdingen, 1980; Fowler, 1940; Mitchell, 1983).

Feldmann (1971) tested about 200 subjects and classified the masking patterns into five broad categories:

1. Congruence, in which the tinnitus was masked just above threshold throughout the frequency range.

2. Distance, in which the tinnitus was masked at high levels throughout the frequency range.

3. Persistence, in which the tinnitus could not be masked.

4. Convergence, in which the tinnitus could be masked at high SLs at low frequencies and low SLs at high frequencies in subjects with a precipitous high-frequency hearing loss.

5. Divergence, in which the tinnitus could be masked at low SLs at low frequencies and high SLs at high frequencies in subjects with mild-to-moderate hearing loss.

Patients with mild-to-moderate loss were reported to have a low-pitched "whooshing" tinnitus and may have had tinnitus of middle ear origin. Patients with distant, congruent, and convergent types of masking patterns have been observed by others (Mitchell, 1983; Tyler and Conrad-Armes, 1984). At present these categories have no known diagnostic significance.

Psychoacoustic tuning curve. Such tinnitus masking patterns might be useful to establish the site of tinnitus. For example, in the normal ear a low-level, pure-tone signal excites a small region of the basilar membrane. A second pure tone (a masker) can be introduced that masks the original stimuli. When this masking occurs, it is assumed that the excitation pattern of the masker has overlapped that of the signal. As the masker frequency moves further from the signal frequency, greater masker intensities are required for the excitation patterns to overlap. When the minimal level required to mask the signal is graphed as a function of masker frequency, a plot called a *psychoacoustic tuning curve (PTC)* is obtained. This curve represents, at least to a first approximation, the spatial extent of the displacement pattern of signal along the basilar membrane.

If tinnitus originated from a localized region of the basilar membrane, its masking properties should be similar to a low-level pure tone. The broad tinnitus-masking patterns observed by Feldmann (1971) and others, however, were not similar to the sharply tuned PTCs seen in normal listeners. However, because tinnitus patients usually have a threshold loss and because hearing-impaired patients usually exhibit abnormal PTCs (Florentine et al, 1980; Tyler et al, 1982), comparing PTCs and tinnitus-masking patterns in the same patient is more appropriate. In this case the pure-tone signal in the PTC is used to simulate the tinnitus.

Tyler and Conrad-Armes (1984) (see also Dauman and Cazals, 1989) used tinnitus loudness and pitch matches to determine the signal level and frequency for the PTC. Three representative examples of their data are shown in Fig. 173-4. In the top panel, a high-level masker was required to mask the tinnitus at all masker frequencies, but in the middle panel a low-level masker was required at all frequencies. In neither case was the PTC similar to the tinnitus-masking pattern. Thus for these two individuals, the locus of the tinnitus apparently was not a restricted region along the basilar membrane. In the bottom panel, greater masker levels were required to mask the tone than to mask the tinnitus, although the shapes of the two functions were similar. This finding could suggest a peripheral, localized source of the tinnitus, but the loudness match between the tone and the tinnitus resulted in an overestimation of the actual magnitude of the tinnitus. Even in a patient with normal hearing, the tinnitus masking pattern and the PTC were dissimilar (Tyler, 1985).

Formby and Gjerdingen (1980) performed tonal masking experiments with a single tinnitus patient. The use of binaural and monaural maskers resulted in tinnitus masking curves of similar shape, but the binaural maskers resulted in less variability and required greater masker levels. The fine structure of masking curves was also somewhat different between the two masker conditions, perhaps because a different tinnitus was being masked in two conditions.

Burns (1984) also noted a discrepancy in PTCs and tinnitus masking patterns in five patients. He grouped his patients into two broad categories: those who required a roughly constant SPL masker and those who required a roughly constant SL masker to mask the tinnitus.

These observations suggest that the tinnitus does not originate in a single place on the basilar membrane in most cases. The diversity of responses also affirms the likelihood that tinnitus originates in many places in the auditory system.

Noises of different bandwidths. Another technique used to measure frequency resolution involves masking by noises of different bandwidths. In normal listeners the overall level of noise band required to mask a tone is independent of its width and narrow bandwidths. As the masker bandwidth is widened, however, some critical bandwidth is reached, beyond which the overall level must be increased to mask the tone. These normal results are shown by the dashed line in Fig. 173-5. Shailer et al (1981) measured the minimal levels required to mask tinnitus for different bandwidths of noise. The noise level was increased until the tinnitus was masked. In two patients the results resembled those from normal subjects, suggesting that the tinnitus is processed in a discrete frequency channel (Fig. 173-5, A). In two other subjects, the results are clearly abnormal, suggesting that the tinnitus may have a diffuse or central origin (Fig. 173-5, B). The nonmonotonic function of one subject is peculiar. When fitting a tinnitus masker based on the lowest level to mask the tinnitus, careful monitoring of the masker spectrum may be desirable.

Ipsilateral and contralateral masking. In normal-hearing individuals, a pure tone presented to one ear can be masked most easily by a noise presented to the same ear. If a noise less than 40 to 50 dB SPL is presented to the opposite ear, the signal threshold can be increased by a small amount, usually less than 5 dB. This procedure is called central masking because the interaction is presumed to occur between the excitation produced by the signal

and masker in the central nervous system. If a noise greater than 40 to 50 dB SPL is then presented to the opposite ear, the noise can mask the tone in the contralateral ear by bone conduction. This procedure is called crossover, a common concern in clinical audiology when it is necessary to ensure that only the test ear is receiving the signal.

The minimal masking levels of broadband noise required to mask the tinnitus have been studied for maskers presented to the ipsilateral and contralateral sides of the tinnitus (Tyler, 1984; Tyler and Conrad-Armes, 1983a). Surprisingly, the masker levels required in the contralateral ears were similar to the levels in the ipsilateral ears, indicating that crossover masking was not occurring (Fig. 173-6). Similar findings were reported by Murai et al (1987). In these subjects the masking seemed to occur at levels above the cochlear nucleus, as if the tinnitus contained a major central component, even in those patients who localized their tinnitus to one ear. In some patients, once the unilateral tinnitus was masked, a tinnitus of a similar quality suddenly appeared in the opposite ear, as if the tinnitus were present in both ears all the time but was lateralized to one ear because of its greater loudness.

Adaptation of tinnitus masking. If a continuous noise is presented at levels that initially masked the tinnitus, in some patients the tinnitus will reappear after several seconds or minutes. Penner et al (1981) quantified this effect by requiring the patient to increase the noise level to maintain masking of the tinnitus. Fig. 173-7 illustrates this effect for a patient in whom the masker intensity had to be increased by 45 dB over 30 minutes. The authors suggested that this effect was caused by the adaptation of the masker, whereas the tinnitus, being processed differently, did not adapt. Another possible explanation is that the masker is exacerbating the tinnitus, and the tinnitus magnitude is increasing over time (Tyler and Conrad-Armes, 1983a). Further work by Penner (1988b) and Penner and Bilger (1989) indicated that in some of the patients, the masker was not adapting; therefore, it seems likely that the tinnitus may be increased by the noise. It seems that the tinnitus and the masker do not share the same neural channels at the point where the adaptation is taking place. Examiners should be cautious when using this procedure because the high-level, continuous maskers could have undesirable long-term effects on hearing or on the tinnitus.

Postmasking effects

Another intriguing characteristic of tinnitus occurs when a masking noise is turned off. In many cases the tinnitus remains inaudible.

Feldmann (1971) presented pulsed maskers with long intermasker delays. Between the maskers, patients reported that the tinnitus would at first be completely inaudible. Patients were required to decrease the delay between the maskers until they could no longer hear the tinnitus. In one patient with 0.5 second, 100 dB SPL, one-third octave band noise masking pulses, the tinnitus was still inaudible with a 1.5-second delay between pulses.

Terry et al (1983) measured the postmasking effects of tinnitus in eight patients. After the termination of a single masker, patients were required (1) to adjust a dial to indicate tinnitus loudness or (2) to balance the loudness of the tinnitus with the loudness of a tone in the contralateral ear. In the second case the contralateral tone possibly could have influenced the tinnitus in some way. Both techniques showed reasonable agreement in most cases, indicating that longer duration and high-intensity maskers produced longer relief from tinnitus.

The authors reported an absence of postmasking effects when the masker was presented to the ear opposite the tinnitus. They also observed a temporary threshold shift after masking that appeared to have a similar time course as the reduction in tinnitus.

Tyler et al (1984b) studied short-term postmasking effects in 10 patients with sensorineural tinnitus. They presented a single, continuous masker (which lasted from 1 to 60 seconds, depending on the experimental condition) and required patients to indicate when the tinnitus (1) first returned and (2) returned to its normal premasker loudness. Their findings, summarized in Fig. 173-8, show five general types of the postmasking effects, although other detailed patterns also were observed. After the termination of the masker, the tinnitus either (1) returned to normal loudness immediately, (2) returned immediately but initially was softer, (3) was absent before gradually returning to normal, (4) was absent before abruptly returning to normal, or (5) was louder before gradually returning to normal. Masker frequency had little effect on the postmasking patterns, but this finding requires more extensive investigation (see Kitajima et al, 1987). Increasing masker duration and level typically produced longer effects.

Tyler et al (1987) compared postmasking effects with a 5-minute masker, which was in the ear either ipsilateral or contralateral to the tinnitus. They required patients to touch a bar on a computer monitor to indicate the tinnitus loudness at about 5- to 10-second intervals. The results from one subject, shown in Fig. 173-9, indicate that this procedure is repeatable. Postmasking relief occurred in both the ipsilateral or contralateral ear, but it was greater when the masker was presented to the ipsilateral ear.

Penner (1988b) also assessed postmasking effects with a 5-minute masker. Subjects judged whether a tone presented 20 ms after the masker was louder or softer than their tinnitus, and subjectively rated whether their tinnitus was louder, softer, or the same, compared with its premasker loudness. Three of six subjects reported a change but the level of the tone equal in loudness to the tinnitus was not significantly different from a premasker judgment. Penner concluded that the subjective ratings provided by some subjects may reflect the inherent dynamic changes of the tinnitus independent of the influence of the masker. One subject reported that the tinnitus became louder after the masker was used on some trials and softer after others. Penner also observed that postmasking tinnitus was judged louder when the postmasker tone was presented to the ipsilateral ear as compared with contralateral presentation.

Vernon and Meikle (1988) measured postmasking effects in patients with a 1-minute masker presented at 10 dB above the minimum level required to mask the tinnitus (with a noise band from 3000 to 12000 Hz). He reported that 89% of patients demonstrated complete or partial residual inhibition. More than half the patients recovered from the inhibition in 2 minutes or less.

Spontaneous otoacoustic emissions

Spontaneous otoacoustic emissions (Kemp, 1981) are measured by placing a microphone in the ear canal and recording spontaneous acoustic activity. Because tinnitus represents a spontaneous perception without any stimulus, it was quite reasonable to search for a direct correlate of the tinnitus that was emitted into the ear canal. These spontaneous acoustic emissions most likely indicate an active mechanism within the cochlea. They are

particularly interesting for the study of tinnitus because they can be measured objectively and could help in developing animal models of tinnitus.

Zurek (1981) searched for spontaneous emissions in six patients with tinnitus. He was able to record an emission in three of them, but always in the ear contralateral to the tinnitus. Tyler and Conrad-Armes (1982) were able to detect an emission only in 1 of 25 patients with sensorineural tinnitus, and it did not correspond to the tinnitus pitch-match frequency. These subjects found the tinnitus distressing. However, using pitch matching, emission suppression by another tone, and tinnitus matching, Penner (188a, 1989a, 1990) has documented at least three cases in which the tinnitus was directly linked to the spontaneous emission.

At present the relationship between spontaneous emission and tinnitus is unclear. Studies of patients with hearing loss have suggested that threshold losses greater than 30 to 40 dB HL usually correspond to an absence of spontaneous emissions, as well as to another, perhaps related, type of acoustic emission evoked by the tone (Kemp, 1978; Rutten, 1980). Spontaneous emissions that do exist do not generally relate to the pitch of the tinnitus (Bonfils, 1989). Ruggero et al (1983) have argued that a particularly sharp transition between regions of normal and abnormal hair cells may disrupt the normal damping of adjacent regions and produce a spontaneous emission, possibly without a pure-tone threshold loss. In reviewing the relationship between spontaneous emissions and tinnitus, Norton et al (1990) concluded that spontaneous emissions "are more likely to be a source of tinnitus if the subject has normal hearing or at least islands of normal hearing". However, "... emissions ... are a source of problem tinnitus in a relatively small percentage of cases".

Improved recording techniques and further studies may demonstrate that some types of tinnitus coexist with spontaneous acoustic emissions. In addition, other categories of otoacoustic emissions also may reflect some aspect of outer hair cell viability (Brownell, 1990). For example, with evoked emissions, an acoustic tone burst or click is presented to the ear, and a delayed time-locked emission can then be recorded. Tinnitus patients may have a basilar membrane that is particularly sensitive tobeing set into oscillation. They would then exhibit a prolonged ringing or oscillating evoked emission. Norton et al (1990) observed an oscillating or prolonged evoked emission in five of six tinnitus subjects, but none in two subjects without tinnitus. They suggested that " ... the evoked emission and the tinnitus might be related to the same underlying pathology, but the former is not the cause of the later".

Another category of otoacoustic emissions results from the distortion products of two tones delivered to the ear (Brown and Kemp, 1984; Martin et al, 1987). These distortion product emissions are particularly suitable for examining cochlear function because they are present in all normally hearing individuals (Martin et al, 1990), they can be used to estimate functioning from a range of places along the cochlea, and both threshold and suprathreshold measures can be obtained.

Auditory brainstem response

Ikner and Hassen (1990) found little difference between the auditory brainstem response (ABR) in tinnitus and non-tinnitus patients matched for hearing loss. Preliminary data from Jastreboff (1990a), however, demonstrated a difference in click-evoked ABR when an analysis of variance for partially correlated data was applied to the entire ABR waveform.

Spontaneous activity measured at the eardrum

Dolan et al (1990) recently discussed the round window recordings of unstimulated neural activity in the guinea pig. They believe this activity originates from basal nerve fibers. Coincidentally, Sininger (1990) has observed a low-frequency peak in the spectral analysis of the spontaneous electrical activity recorded at the eardrum, which apparently is unique to (at least some) tinnitus patients. The results are preliminary, and further work is necessary to determine appropriate recording parameters.

Magnetic activity of the auditory system

Hoke et al (1989) reported a distinction between the auditory evoked magnetic field in tinnitus and nontinnitus patients. In response to a 1000 Hz tone burst, the magnetic response at about 100 ms was augmented and the response at about 200 ms was delayed or missing when compared with results in normal-hearing persons. A ratio of the two measures was the best discriminator (Fig. 173-10). The tinnitus patients had hearing within normal limits up to 1000 Hz; this is a very important development. Jacobson (1991) has not been able to demonstrate the clear distinction between tinnitus and nontinnitus groups, but he considers his findings preliminary. Presently the cost of the instrumentation and its limited availability restricts its experimental and clinical use.

Management

Many treatments for tinnitus have been attempted over the years, including electrical stimulation (Dobie et al, 1986; Vernon, 1987), acupuncture (Mann, 1974), masking with noise (Tyler and Bentler, 1987), medications (Goodey, 1981), behavior modification (Sweetow, 1987), biofeedback (House and House, 1987), and counseling (Tyler et al, 1990). We shall review a few of them.

Medication

The logical selection of appropriate drugs to treat tinnitus has been hampered by lack of knowledge about the generation site(s) and mechanism(s) of this symptom. (See Murai et al, 1992 for a recent review of the pharmacologic treatment of tinnitus.) Three general approaches have been adopted:

1. Patients with tinnitus who take drugs such as lidocaine for other reasons occasionally note relief of the tinnitus.

2. Investigators have used therapeutic trials of drugs such as nicotinic acid and carbamazepine because their mechanism of action counteracts a mechanism suspected of producing the tinnitus or associated deafness.

3. Some drugs, such as the antidepressant amitriptyline, have been used to help the patient tolerate tinnitus.

Despite the drug selected, the medical treatment of tinnitus is frequently reported anecdotally. The lack of controlled, blinded studies and the inability to measure tinnitus objectively have obscured the results of such management trials (McFadden, 1982; Murai et al, 1992).

Nicotinic acid (vitamin B6), a peripheral vasodilator, has been used with the rationale that tinnitus, as well as associated sensorineural deafness, or Ménière's disease, is the result of cochlear ischemia (Atkinson, 1947). Carbon dioxide, an effective cerebral vasodilator, also has been used for tinnitus (Powers, 1984) but has not gained wide acceptance.

Carbamazepine is thought to be effective by an entirely different mechanism. The decreased sensory input that accompanies peripheral vestibular dysfunction is believed to result in a more central afferent loop analogous to a sensory epilepsy. If this is true, tinnitus, trigeminal neuralgia, and the phantom limb syndrome may be similar phenomena occurring in different sensory systems. Melding and Goodes (1979) postulated that tinnitus resulting from such "hyperactivity" might be reduced by the anticonvulsant carabmazepine, which is known to be effective in trigeminal neuralgia. They treated only patients with "incurable and intolerable" tinnitus with doses of 600 to 1000 mg/day. Patients described the magnitude of their tinnitus on a visual analog scale, and more than half reported significant improvement while taking the drug. When the drug was withdrawn, the original severity of the tinnitus returned. Shea and Harell (1978) also demonstrated relief from tinnitus in an uncontrolled study. Lower doses of carbamazepine, 100 to 400 mg/day, were chosen because of several dose-related side effects, including severe bone marrow depression. The frequency and severity of side effects have limited carbamazepine's acceptance by tinnitus sufferers. Several other anticonvulsants have been tried with less spectacular results, including phenytoin (Dilantin) and primidone (Mysoline) (McFadden, 1982).

Lidocaine was fist used to treat tinnitus in patients with Ménière's disease by Barany and later by Fowler after the observation that intravenous procaine was sometimes effective for tinnitus (Gejrot, 1963). Melding et al (1978) observed independently that patients in Auckland who received intravenous lignocaine (lidocaine) for chronic pain also reported temporary relef of tinnitus. More studies have investigated the effectiveness of lidocaine than of any other drugs (Melding and Goodey, 1979; Medling et al, 1978; Shea and Harrell, 1978). Several carefully controlled double-blind studies have repeatedly demonstrated its benefit (Duckert and Rees, 1983; Israel et al, 1982; Martin and Coleman, 1980). Martin and Coleman (1980) reported a subjective reduction in tinnitus intensity in about two thirds of patients after an intravenous bolus of 1 to 2 mg/kg body weight. More recently, Duckert and Rees (1983) conducted a double-blind, randomized trial to assess the effectiveness of intravenous lidocaine on tinnitus. They reported that although 40% of patients who received lidocaine reported temporary subjective improvement, 30% reported an increase in tinnitus intensity. The mechanism of action is thought to be related to the inhibition of neuronal hypersensitivity at this dosage level. The high incidence of tinnitus aggravation in the Duckert and Rees group, however, does not support this theory. Lidocaine is also used as an anticonvulsant and may also be effective by reducing the excitability of the ganglion, preventing the reverberant activity of a hyperactive nerve (Møller, 1984). Unfortunately, lidocaine must be given intravenously, has a very short half-life (minutes to hours), and is sometimes accompanied by undesirable side effects (disequilibrium, slurred speech, numbness; depending on the dosage).

The response to lidocaine has not proved a valuable predictor of success with other drugs (McFadden, 1982), perhaps due partly to a high false response rate (40%), which can be attributed to the placebo effect of a sole intravenous injection in patients with tinnitus (Duckert and Rees, 1984). No objective improvement in hearing thresholds is reported with lidocaine, although patients sometimes report improved clarity.

The lidocaine trials demonstrate hope for the treatment of tinnitus through drug therapy. More studies that examine the mechanism of tinnitus reduction could be worthwhile. Lyttkens et al (1979a, 1979b) reported that lidocaine binds to melanin in the inner ear. Melanin, involved in energy transformation in neural transduction, has been shown to relate to a person's susceptibility to hearing loss due to noise exposure (Hood et al, 1976; Karsai et al, 1972). McFadden (1982) suggests that melaning be quantified in future tinnitus drug trials.

Tocainide was developed as an analog of lidocaine to obviate the need for intravenous administration and to produce a longer duration of action. Tocainide is orally effective and has a half-life of 11 hours, compared with 1 hour for lidocaine. In a double-blind study, Emmett and Shea (1980) were unable to demonstrate any significant difference in tinnitus relief between a placebo group and a second group of patients who received 200 mg of tocainide four times a day. In a smaller series of patients, they reported tinnitus relief at higher doses, but the study was uncontrolled. We tested 26 patients in a double-blind trial (Babin and Tyler, unpublished data) with increasing doses of oral tocainide after administration of both intravenous lidocaine (100 mg) and intravenous tocainide (250 mg). A visual analog scale was used, as well as pitch and intensity matching and masking. When pooled data were analyzed, no significant difference was found between the placebo and tocainide. Three patients, however, demonstrated an improvement in their tinnitus, but one of these later demonstrated an exacerbation. Tyler et al (1984a) reported on data from two of the subjects from the study, one whose tinnitus increased and one whose decreased. In both cases, the amount of noise required to mask the tinnitus was consistent with the subjective annoyance rating of the subject. Apparently, oral tocainide is a less effective treatment for tinnitus than is lidocaine (see also Cathcart, 1982), and its use is limited by potentially adverse neural and gastrointestinal side effects.

Harker et al (1987) performed a double-blind crossover trial to determine the efficacy of flecainide acetate. This antiarrhythmic agent has potent local anesthetic activity; therefore, the investigators reasoned that it would be an appropriate follow-up to lidocaine and tocainide. Of the 20 patients tested, however, none showed a significant reduction in tinnitus, either on subjective ratings or on psychophysical tasks. A few controlled studies of other drugs have shown a significant treatment effect (Murai et al, 1991). Donaldson (1978) treated tinnitus with amylobarbitone, a rapidly acting barbiturate sedative that is absorbed by the gastrointestinal tract and metabolized by the liver. Its plasma concentration declines in a biphasic manner with a half-life of about 40 minutes for the first phase and 20 to 25 hours for the second phase. It depresses the reticular portion of the central nervous system. Thus if hyperactivity in this area is related to tinnitus, then a decrease of hyperactivity could reduce tinnitus. Twenty patients were treated with the drug and 20 additional patients were used as controls. Subjective ratings by the patients and loudness matching indicated that benefit was obtained by 16 to 20 (80%) patients who used amylobarbitone and only 2 of 20 (10%) control subjects. No side effects were reported. It is not clear, however, whether the control subjects

received a placebo, and the investigation needs to be repeated with a double-blind cross-over design.

Lechtenberg and Shulman (1984) investigated the efficacy of several benzodiazepines to treat tinnitus. Twelve of 23 (52%) patients receiving oxazepam reported benefit, and 18 of 26 (69%) receiving clonazepam reported benefit. Only 4 of 37 (11%) patients receiving antihistamines as a control reported benefit. Clonazepam is an anticonvulsant that is well absorbed from the gastrointestinal tract and is metabolized in the liver. Because lidocaine is also an anticonvulsant, the physiologic mechanism responsible for tinnitus relief may be similar.

Surgery

Surgery has been successful for many conditions in which tinnitus is an associated symptom, such as otosclerosis, acoustic neuroma, Ménière's disease, and glomus jugulare. In a review of these surgical procedures, House and Brackmann (1981) found that relief from tinnitus occurred in about one half of patients. Such procedures must be differentiated from a primary surgical attack on tinnitus itself. Destructive surgical lesions into the peripheral auditory system were reported as early as 1928 by Jones and Knudsen as not only dangerous but surprisingly ineffective. More recent experience with acoustic or vestibular nerve section validates the earlier experience that somehow tinnitus generators are centralized and are not consistently relieved by uncoupling the end organ (Pedersen and Sorenson, 1970). House and Brackmann (1981), for example, found only a 25% chance of improving tinnitus when a cochlear nerve section was added to a vestibular nerve section for Ménière's disease; tinnitus worsened in 1 of 17 (6%) patients.

Another surgical approach to treating tinnitus is based on the ischemia theory. Stellate ganglion block or section is believed to result in increased blood flow to the cochlea. Warrick (1969), in his experience with 66 patients, had positive responses in 56% of patients with Ménière's disease and in 27% of patients with idiopathic tinnitus. The difficulties inherent in interpretation without adequate controls and the capricious nature of Ménière's disease must be considered in such a report. Other procedures directed toward tinnitus have included tonsillectomy, excition of the tympanic plexus, and section of the vestibulofacial anastomoses (McFadden, 1982).

Hearing aids and tinnitus maskers

Because an external noise can render tinnitus inaudible in many patients, it seems reasonable to introduce such a noise to provide temporary relef. Jones and Knudsen (1928) first constructed a device that produce noise for the treatment of tinnitus. Saltzman and Ersner (1947) observed that a hearing aid amplified background noise that could successfully mask the tinnitus. Hearing-impaired patients with tinnitus probably should try a hearing aid before trying a tinnitus masker. A hearing aid interferes less with speech, does not produce an intense noise that could produce damage, and can improve speech understanding.

Vernon (1977) and associates designed a commercially available tinnitus masker, a device that produces a noise, the level of which is controlled by the user. Hearing aids with a tinnitus masker built into them also are available. These combination units allow tinnitus masking and amplification of speech. Although most maskers are behind-the-ear devices, Coles et al (1987) have developed an ear canal masker.

Tinnitus annoys some patients only in specific quiet situations (Tyler and Baker, 1983), such as working in a closed office, reading, or while trying to sleep. In these instances providing a background noise source that can act as a tinnitus masker, such as a radio tuned between stations, is sometimes helpful.

The noise can either completely mask the tinnitus or partly mask it, so that both noise and tinnitus are heard, but the tinnitus is reduced in loudness. There are no clear guidelines for selecting the spectrum characteristic of the masking noise and no indications that measurements of pitch, loudness, or postmasking characteristics can be used to select an appropriate spectrum for the tinnitus masker. The masking data of Feldmann (1971), Shailer et al (1981), and Tyler and Conrad-Armes (1984) suggest that the noise spectrum of the masker need not be centered on the frequency of the tinnitus. In other words, even if the patient matches the most prominent pitch of the tinnitus to a mid-frequency sound, presenting a high-level, broadband high-frequency emphasis noise that has limited interference with speech may be desirable. Narrowband noises have a tonal character, and those with a prominent tonal component are more annoying than broadband noise (Kryter and Pearsons, 1963). Narrowband masking noises (less than a half-octave bandwidth) are undesirable for many patients. In addition, continuous exposure to intense sounds can produce further hearing loss. A conservative approach would be to urge the patient to use the lowest level masker that provides adequate relief and to inform the patient that the masker need not be worn continuously. The patient must understand that the masker is not a cure and that the tinnitus probably will not go away with its continued use. The patient should be told that the masker simply provides an external noise to the tinnitus, and that the patient can control its intensity and turn it on or off as desired.

No protocol is available for deciding which ear to fit with a masker. Tyler and Conrad-Armes (1983a) showed that in many patients the tinnitus can be masked equally wellin both ears; therefore, the clinician always should try to fit the tinnitus masker on each ear separately. Some audiologists also report that fitting two maskers, one on each ear, sometimes is preferable to fitting one on only one ear, although a detailed investigation of this issue is not available. Coles and Hallam (1987), Hazell and Wood (1981), Shulman and Goldstein (1987), Tyler and Bentler (1987), and Vernon (1988) have provided overviews for fitting tinnitus maskers.

Tyler and Stouffer (1990) evaluated binaural tinnitus masking in several conditions: (1) two different noises presented to the two ears (perceived throughout the head), (2) the same noise presented to each ear (typically lateralized in the center of the head), and (3) the same noise waveforms presented binaurally, but with one phase-delayed (so that the focal location of the masker could be manipulated throughout the head as the phase delay was adjusted). They found that most subjects required lower masker levels if the noise was the same in both ears. When the phase delayed was adjusted, 2 of 12 subjects required even lower masker levels.

There is controversy regarding the percentage of the population who can be helped by tinnitus maskers (Erlandsson et al, 1987; Hazell et al, 1985; McFadden, 1982; Roeser and Price, 1980; Rose, 1980; Stephens and Corcoran, 1985). The type of patients fitted, the quality of the counseling, and the encouragement received all affect outcome. Even if the percentage of patients helped by maskers is less than 15% (or, to emphasize the point, less than 1%), maskers are a viable treatment procedure for some. Because treatment is universally efficacious and because tinnitus causes severe problems, tinnitus maskers must be considered an important clinical treatment.

Electrical suppression

Several clinicians and researchers have noted that electrical stimulation can reduce tinnitus in some patients. These studies can be grouped as using either extracochlear or intracochlear stimulation.

Extracochlear studies. Hatton et al (1960), using direct current, produced a reduction in tinnitus in 15 of 33 (46%) patients. They suggested that electrical current was most effective in suppressing tinnitus in profoundly hearing-impaired individuals. Several investigators have confirmed that direct electrical current can be used to suppress tinnitus. For example, Portmann et al (1983) were able to reduce the magnitude of tinnitus in 66% of 72 patients with tinnitus (no selection criteria reported) by presenting a positive current to the cochlear round window. The tinnitus was reduced throughout the stimulation. After the current was terminated, however, the tinnitus reappeared louder than before in several patients (resembling the pattern of postmasking effects shown in Fig. 173-8, E). Aran and Cazals (1981) also noted that round window stimulation was more effective than promontory stimulation. Unfortunately, direct currents (DC) may produce permanent damage and, therefore, cannot be used clinically. Aran and Cazals (1981) suggest tinnitus reduced by electric current probably has a peripheral origin, whereas tinnitus that cannot be reduced probably has a central origin.

Other work has focused on using alternating current. Graham and Hazell (1977) and Hazell et al (1983), using a transtympanic stimulation of the promontory in totally deaf patients, were able to reduce tinnitus in 2 of 13 (15%) and 7 of 12 (58%) patients, respectively. Chouard et al (1981) placed electrodes on the tragus and behind the ear lobe. They evaluated 64 ears and were able to reduce the tinnitus in 30 (47%). Chouard et al (1981) suggested that there was no correlation between etiology and success, although no supporting data are provided. Curiously, they also report that none of their patients experienced a complete suppression of tinnitus, but only a reduction in loudness. Complete reduction of tinnitus was found by Vernon and Fenwick (1985) and Kuk et al (1989). Chouard et al (1981) also thought that several repeat visits may be necessary in some cases, suggesting that patients who were initially unsuccessfully treated may later experience suppression. However, no data were provided to support their claim.

Vernon and Fenwick (1985) used transcutaneous electrodes in preauricular and postauricular regions of the ear with tinnitus. Five of 23 (22%) subjects noted a reduction in their tinnitus. In some cases the reduction lasted 3 hours. The authors retested five patients who had previously shown suppression. Surprisingly, only three of the five demonstrated the same positive response on retest.

Kuk et al (1989) completed a pilot study on electrical suppression of tinnitus using an eardrum electrode. The most effective frequency for tinnitus suppression was different for each subject. A 10-minute electrical stimulation resulted in a poststimulus reduction that varied among patients from 40 seconds to 4 hours.

One extracochlear wearable device to reduce tinnitus, the Theraband (Shulman, 1985), was marketed in the USA. The device used a 60-kHz carrier frequency, which was amplitude modulated (90%) by a sinusoid. The sinusoid continuously changed its frequency from 200 Hx to 20 kHz. No reports were published to justify the use of these stimulus parameters. Dobie et al (1986) demonstrated that only 1 patient out of 20 clearly received benefit from the device. The product has been removed from the market.

Intracochlear studies. Fewer investigators have explored the use of intracochlear electricity to suppress tinnitus. Most of these reports have been observations that some cochlear-implant patients report a reduction in their tinnitus while listening to speech. House and Brackmann (1981) reported that 23 of 29 (79%) 3M/House cochlear-implant patients with tinnitus reported relief of tinnitus while using their cochlear implant. In one follow-up study of this device, Berliner et al (1987) reported that 53% of 65 patients reported that their tinnitus was improved, and 11% reported that their tinnitus worsened. Hazell et al (1989) reported on 28 patients who had received the single-channel intracochlear implant developed in London. Fifteen (54%) reported some relief from their tinnitus while listening through their speech processor. Tyler and Kelsay (1990) noted that 34 out of 42 (81%) of some of the better cochlear-implant users reported that their tinnitus was reduced while using their implant. One patient (2%) reported that tinnitus worsened. These reports may overestimate the actual success, as responses were obtained only after the treatment and no controls were used.

A few patients have received a cochlear implant explicitly for tinnitus reduction and not to improve their hearing ability. JW House (1984) reported that WF House implanted devices in five severe-to-profoundly-hearing-impaired patients for tinnitus relief. No special stimuli were administered to these patients, and only reported a reduction in tinnitus while listening to speech through the cochlear implant.

Sininger et al (1987) reported on one patient implanted with the 3M/House cochlear implant for tinnitus reduction. This patient had normal low-frequency hearing and a moderate-to-severe high-frequency hearing loss. Sinusoids from 400 to 16.000 Hz were ineffective in reducing tinnitus. However, they noted that the current levels may have been insufficient to reduce tinnitus in most conditions because of loudness intolerance induced by bone conduction or electrophonic hearing. Interestingly, the short (6 mm) intracochlear electrode resulted in little change in the residual hearing of this patient. This is noteworthy because it suggests that intracochlear electrodes may eventually be designed for tinnitus suppression in mild-to-moderately impaired patients.

Hazell et al (1989) reported on six totally deaf patients who had received an intracochlear implant and additional experimental trials with sinusoidal stimuli. They were able to reduce the tinnitus in all six patients using a 100-kHz sinusoid. Other stimuli apparently were tried, but no data were reported. At least two of the patients elected to forego their speech processors and to use their implant to suppress their tinnitus. One reported that

turning on the current turned off his tinnitus "like a light switch". This area clearly requires further investigation. Electric stimulation eventually may have diagnostic significance and result in an implantable device for tinnitus reduction.

Other procedures

Because the treatment of tinnitus and its underlying causes may be difficult, other indirect efforts to help the patient cope sometimes are helpful. These procedures include biofeedback (House, 1981) and hyponotherapy (Marlowe, 1973). Although these procedures should not be expected to change tinnitus, they could beneficially modify a peron's reaction to tinnitus. Because acupuncture's anesthetic effect is well established and tinnitus has been considered analogous to pain, attempting to treat tinnitus with acupuncture seems acceptable. Mann (1974) suggested that about 5% of his tinnitus patients were helped with acupuncture, but our experience with six patients has been largely unsuccessful (McCabe, 1985).

Young (Young and Lowry, 1983) unsuccessfully tried to eliminate his own tinnitus by exposing himself to a 107 dB SPL, 2000 Hz pure tone for 10 minutes. The rationale was to eliminate a narrow region of hair cells that might be producing the tinnitus. Because this trial exacerbated the tinnitus and could produce a permanent threshold shift, this procedure should not be used clinically.

Vernon et al (1980) reported that increasing the pressure in the external meatus of a patient reduced his tinnitus. Changing pressure in the ear canal would change the impedance match between the middle ear and the cochlea and could temporarily change the structural relationships in the cochlear partition.

Psychologic considerations

Tinnitus can have a far greater effect on the patient's life-style than is normally associated with hearing loss. Fig. 173-11 shows data obtained by Scott et al (1990), comparing some disturbing psychologic effects common to hearing loss and tinnitus. Patients with hearing loss and tinnitus suffer more than hearing-impaired patients in insomnia, depression, anxiety, and concentration.

Table 173-1 shows a list of the 15 most common problems associated with tinnitus from 72 patients with tinnitus severe enough to join a self-help group (Tyler and Baker, 1983). Note that about 5 or 6 of every 10 respondents reported that their tinnitus made sleep difficult. About half complained that their tinnitus was always present and escape seemed impossible. Although 4 of every 10 thought that tinnitus interfered with hearing speech, this response is difficult to verify because they probably also had a hearing loss. Some respondents noted that their tinnitus was worse in noisy environments, whereas others reported that it was worse in quiet environments.

Other studies have highlighted similar problems. For example, Coles (1984) found that about 1% of the population had such severe tinnitus that the ability to lead a normal life was affected. Slater et al (1987) noted that about 42% of 1000 tinnitus patients were taking medications to help them sleep. Lindberg et al (1984) observed that 59% of patients visiting a hospital hearing center were troubled by tinnitus. Tinnitus patients studied by Jakes et al

(1985) complained that tinnitus interfered with the ability to enjoy music and watch television. The diversity and severity of these problems dramatically emphasize the importance of counseling. Because many of these difficulties are probably interrelated, all of the patient's difficulties must be considered. Many patients requre reassurance that their tinnitus is not an indication of a life-threatening disease. They also should be informed about the high incidence of tinnitus. Discussing the problems tinnitus patients experience and the difficulty in describing the sounds they hear is often helpful. Discussions with the spouse, companion, or family members are encouraged so that they can begin to realize the validity and severity of the problem.

In evaluating their Tinnitus Handicap Scale (see box, p. 3036), Kuk et al (1991) observed that patients' tinnitus problems could be grouped into three categories: the physical, emotional and social consequences of tinnitus; hearing loss; and patient's view of tinnitus.

In a study of over 500 tinnitus patients, Stouffer and Tyler (1990) noted that tinnitus increased in severity over time for about 25% of the patients. Tinnitus was more severe in patients whose primary complaint was tinnitus and in patients with Ménière's disease.

Tyler et al (1990) describe a management model that includes an initial counseling session, a handicap evaluation, psychoacoustical measurement, and an in-depth counseling session. Many patients require only initial counseling. Other strategies are described by Stephens et al (1986) and Scott et al (1985).

Sweetow (1984a, 1984b) has reviewed a group therapy strategy for tinnitus patients, many points of which are also applicable to individuals. He identifies four patient needs: (1) emotional support, (2) realistic understanding of tinnitus and its treatment, (3) positive and active attitude to pursue helpful activities, and (4) a battery of tactics and coping strategies. Initially the patient is informed of the following (Sweetow, 1984a):

1. Tinnitus is real, not imagined, and has a definite physical basis.
2. Tinnitus may be permanent.
3. The person's reaction to tinnitus, not the sound itself, creates the problem.
4. The person's reaction to such a disorder is manageable and subject to modification.

Summary

Tinnitus is a distressing symptom for many patients. The clinician must make a thorough evaluation to try to determine its cause. Appropriate treatment is frequently available, particularly with tinnitus generated by vascular and myoclonic sources. Sensorineural tinnitus probably has many sources; therefore, finding any single treatment that will work for many patients is unlikely. Many effective treatments are available, however, and the task is to try to determine which will work for a specific patient. Patients usually benefit from a frank and sympathetic discussion about the nature of tinnitus and its high prevalence. Several measurement techniques are evolving that should result in a more selective description of subcategories of tinnitus. This in turn should facilitate matching the treatment with the patient.