Paparella: Volume II: Otology and Neuro-Otology

Section 3: Diseases of the Ear

Part 1: General Problems

Chapter 17: Tinnitus

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Tinnitus is one of the most common presenting complaints in the practice of otolaryngology. The word tinnitus is a derivative of the Latin word tinnire meaning "to jingle". According to Dorland's Medical Dictionary, tinnitus aurium is a tinkling sound in the ears. Tinnitus cranii, on the other hand, is that sound which is nonlocalized or sensed within the head in general. The term tinnitus as it is now used is more inclusive and is generally accepted as meaning an auditory perception of internal origin (noise), usually unwanted, usually localized as originating from within, and rarely heard by others. Tinnitus is usually perceived as a fairly simple sound. In its simplest form it may be described as a pure tone (in approximately 25 per cent of patients), but it may also include the more diversified sounds of an ocean rumble and the chirping of a cricket. Approximately 70 per cent of all sufferers describe their tinnitus as steam escaping, ringing, or buzzing. Tinnitus must be differentiated from auditory hallucinations, which are more complex sounds, such as voices or music, and which suggest drug intoxication or psychiatric disturbance. It must also be differentiated from autophony, which is the hearing of one's own voice and respiratory sounds due to a conductive hearing loss or patulous eustachian tube.

Although patients may complain bitterly of tinnitus more than 80 per cent of these individuals match the loudness of their tinnitus to sounds zero to 20 dB above their thresholds, and less than 5 per cent of patients will have tinnitus of a magnitude of 40 dB or greater. The aggravation of tinnitus is independent of perceived pitch, duration, and type of sound heard, and in many patients the level of disability is more closely related to interrupted sleep and to the apparent level of anxiety and fatigue rather than to the magnitude of the tinnitus itself. It should be noted, however, that estimates of tinnitus loudness are based on sensation level. Recalling that recruitment produces an abnormally rapid growth of loudness, sounds that ordinarily should be comfortable may be perceived as unpleasant. Evidence to this effect can be obtained by studying frequency-specific loudness discomfort levels in tinnitus patients, as many will demonstrate markedly reduced levels.

The problem and almost ubiquitous nature of tinnitus is well known. Total absence of tinnitus is rare, and when a normal population is placed in an anechoic chamber, about 95 per cent will experience some form of auditory perception. Speculations regarding the source of this phenomenon include the resting discharge of cochlear hair cells, molecular motion of air within the middle ear or anechoic chamber, and circulating blood in or near the organ of Corti. Most people, however, are not aware of their tinnitus, due to the masking effect of ambient noise.

Over 37 million Americans actually complain of tinnitus, and the degree of suffering ranges from occasional awareness to contemplation of suicide. About 20 per cent of these

Americans have tinnitus in what is described as a severe of extreme form, which significantly interferes with the quality and productivity of life.

Of all aural symptoms, tinnitus can be the most devastating, and it often is of such annoyance as to cause the patient constant torture. This distressing situation produces an inability to concentrate and prevents the carrying out of routine daily duties. Family problems are often aggravated, and frequently there is intentional avoidance of friends. Patients experience despair, frustration, irritation, worry, insecurity, and an inability to relax. In many persons, tinnitus finally leads to mental depression and other mental disorders.

Although tinnitus may occur at any time during the life cycle, the majority of sufferers are between the ages of 40 and 80 years. Tinnitus does occur in children, however, and may be a more common pediatric affliction than previously thought. Males and females are equally affected. Approximately 50 per cent of patients with tinnitus localize the noise to one ear, while the other 50 per cent identify it as coming from both ears or the head in general. Involved frequencies range from near 0 Hz to over 10,000 Hz, with a fairly even distribution, except for a peak incidence between 3000 and 5000 Hz. More than 80 per cent of patients with tinnitus will have an identifiable bandwidth of 2400 Hz or less.

In his audiometric study of 200 patients, Reed (1960) found that the tinnitus associated with conductive hearing loss tended to be of lower frequency than that associated with sensorineural hearing loss. He also noted that low-frequency tinnitus was generally of greater magnitude than high-frequency tinnitus. The band width, however, of low-frequency tinnitus was similar to that of high-frequency tinnitus and had no apparent relationship to diagnosis, type of deafness, site of origin, or subjective severity. There also was no correlation between the objectively determined loudness of the tinnitus and the diagnosis, type of deafness, site of origin, or degree of hearing loss. There was some correlation between the subjective description of tinnitus and the measured band width, with those complaining of "steam escaping" having the broadest band width, those complaining of "ringing" having the narrowest band width, and those complaining of "buzzing" having an intermediate band width. A correlation also was found between objective band width and objective loudness, with those patients suffering from narrow-band tinnitus having the greatest intensity. In most patients, tinnitus is constant but may be intermittent and pulsate, with fluctuations in intensity and quality depending on the cause.

Although many tinnitus sufferers to not complain of hearing loss, the vast majority will, indeed, have an identifiable hearing loss. The perceived pitch of tinnitus associated with sensorineural hearing loss often lies within a region approximately one-third down the sloping audiometric curve, and there appears to be some correlation between the degree of hearing loss and the intensity of the tinnitus, although this may not be constant. There also is some lack of agreement as to whether the frequency of maximum hearing loss and that of the tinnitus are similar. Certainly there appears to be some relationship between the frequency of hearing loss and tinnitus in cases in which an etiology, such as noise-induced hearing loss with the auditory defect and tinnitus both of high frequency, can be identified. The lack of total understanding of tinnitus is evidenced by these confusing factors and also by its multiple and varied explanations. The literature primarily concerns itself with clinical and theoretical impressions, having little factual basis. The many therapeutic approaches that have been offered and now exist are based, at least in part, on one or more of these explanations. The

lack of a scientific foundation for these impressions can be confusing and may be explained by the fact that tinnitus is primarily a subjective complaint for which objective indicators are usually sparse, and also because a proven animal model for tinnitus is lacking.

Classification of Tinnitus

There are almost as many proposed classifications for tinnitus as there are treatments. The site-of-lesion classification for tinnitus shown in Table 1 may be helpful in patient evaluation. Another simple classification offered by Wegel (1921) and the two major categories proposed by Fowler (1944) are quite similar and appear to have the best pathophysiologic and potential therapeutic rationale. These classifications for tinnitus contain two major categories (Table 2) and are the ones used in this chapter:

1. *Vibratory tinnitus* - real sounds, mechanical in origin, arising within or near the ear.

2. *Nonvibratory tinnitus* - neural excitation and conduction from anywhere within the auditory system to the auditory cortex, without a mechanical basis.

Table 1. Classification of Tinnitus by Site of Lesion

External Auditory Canal

Foreign body, cerumen impaction, infection
    Otitis externa (acute, chronic, fungal, necrotizing)
Tumors
    Benign (osteoma, exostosis)
    Malignant (squamous cell carcinoma, basal cell carcinoma, ceruminoma)
Atresia
    Congenital (osseous, nonosseous)
    Traumatic

Tympanic Membrane

Perforation
Atelectasis

Middle Ear

Effusion
    Blood, cerebrospinal fluid, purulent, serous, mucoid
Ossicular
    Fixation, discontinuity
Tumor
    Cholesteatoma, glomus tumor, facial nerve neuroma, hemangioma, carcinoma
Vascular
    Aberrant vessels (internal carotid artery, dehiscent jugular bulb)
Neuromuscular
    Myoclonus (palatal, tensor tympani, and stapedial muscles)

Cochlea

Virtually any disorder of the cochlea resulting in a sensorineural hearing loss

Retrocochlear

Internal auditory canal and cerebellopontine angle
    Acoustic neuroma, cholesteatoma, hemangioma, facial nerve neuroma, meningioma, vascular loops on auditory nerve
Central nervous system
    Tumors, inflammatory and vascular lesions

Miscellaneous

Patulous eustachian tube
Temporomandibular joint dysfunction
Head trauma
Extracranial aneurysms
Arteriovenous malformations
Venous hum.

The vibratory form of tinnitus may be further subdivided into subjective (heard only by the patient) and objective heard by interested observers). Nonvibratory tinnitus can be subdivided into central and peripheral (tympanic and petrous) in origin. Due to its inherent nature, the nonvibratory form of tinnitus is always subjective, although cochlear omissions may be detected at the round window membrane in patients suffering from this condition.

Vibratory tinnitus generally has a more easily identifiable cause than nonvibratory tinnitus, and therefore a rational basis for treatment is available. A number of pathologic states are known to produce enough mechanical energy to stimulate the auditory system, resulting in perception of sound (vibratory tinnitus). These pathologic states are usually vascular or neuromuscular in nature or are due to an abnormally patent eustachian tube. If these entities produce enough mechanical energy to be heard by others, they become objective. If not, they remain subjective.

Nonvibratory tinnitus, although more common than vibratory tinnitus, is less well understood. Reed (1960) reported an audiometric study of 200 cases of nonvibratory tinnitus and described the symptom as one in which the exact causes have eluded otologists and effective cures have been equally elusive. Proposed pathophysiologies for nonvibratory tinnitus include paresthesias of the auditory nerve, autonomic imbalance resulting in vasospasm, irritation of the tympanic plexus, hypersensitivity of the chorda tympani nerve, sludging of blood, increased tension by middle ear muscles on inner ear fluids, and intracellular edema of the organ of Corti pushing hair cells into contact with the tectorial membrane. Hilding (1953) suggested that detachment of the outer border of the tectorial membrane may increase hair cell tension, resulting in tinnitus.

Table 2. Major Categories of Tinnitus

1. Vibratory
- A. Vascular disorders
  - Arteriovenous malformation
  - Aneurysm
  - Venous hum
  - Dehiscent jugular bulb
  - Persistent stapedial artery
  - Eagle's syndrome
  - Glomus tumor
  - Hypertension
- B. Neuromuscular
  - Palatal myoclonus
  - Stapedial muscle spasm
  - Tensor tympani muscle spasm
  - Temporomandibular joint dysfunction
- C. Miscellaneous
  - Patulous eustachian tube
  - Local inflammation or infection

2. Nonvibratory
- A. Peripheral
  - External auditory canal
  - Middle ear
  - Cochlea
- B. Central
  - Auditory nerve
  - Brain stem
  - Central nervous system.

Vibratory Tinnitus

Local or regional vascular disorders with associated increase in blood flow and turbulence may have tinnitus as the first or most prominent clinical symptom. Arteriovenous malformations, acute and chronic middle ear inflammation, thyrotoxicosis, severe anemia, pregnancy, aberrant vessels (internal carotid, persistent stapedial artery, dehiscent jugular bulb), vascular neoplasms, and aneurysmal dilatations or partial stenoses of major vessels can all create enough mechanical energy to result in auditory perception of sound. The tinnitus in such cases is usually pulsatile and synchronous with the heartbeat. Vibratory tinnitus secondary to a venous anomaly (cephalic murmur) may, however, have a more constant, humming characteristic. Mechanical blockage of the vessels supplying the anomaly (eg, turning the head, pressure over the carotid artery) adds diagnostic evidence, as it usually diminishes or totally relieves the tinnitus.

Clonic muscular contractions of the tensor tympani, stapedius, tensor veli palatini, or levator veli palatini muscles may result in a clicking sound that can be heard by both the patient and the examiner. This sound usually has a faster rate than the pulse (averages

between 40 and 200 contractions per minute) and occasionally can be altered by conscious effort by the patient. Movement of the tympanic membrane or palate may be observed and recorded by tympanometry or electromyography, respectively. These recordings are found to be synchronous with the tinnitus. Psychogenic disorders, multiple sclerosis, cerebrovascular disorders, and intracranial neoplasms have all been implicated as underlying causes for this neuromuscular problem.

The eustachian tube, which is normally closed, may become abnormally patent or patulous. The nasopharyngeal air turbulence of respiration may then be transmitted through the patent eustachian tube, resulting in another form of vibratory tinnitus. Otoscopy and tympanometry will reveal respiratory-associated tympanic membrane movements. This condition may also result in autophony (abnormal awareness of one's own voice) and is generally seen following a large weight loss with decreased body fat (chronic illness or dieting), in patients on birth control pills or other forms of estrogens, and during the postpartum period. Placing the head in a dependent position may cause local venous engorgement and closure of the eustachian tube, with resultant temporary cessation of the symptoms.

Nonvibratory Tinnitus

A number of disorders have been associated with nonvibratory tinnitus, and a large majority have associated hearing loss at the cochlear level (Table 3). The understanding of this symptom is far less complete than that of vibratory tinnitus. Mawson (1963) divided nonvibratory tinnitus into tympanic, petrous, and central, depending on its proposed origin. Of 200 cases studied audiometrically Reed (1960) identified 75 per cent as being cochlear in origin, 4 per cent of conductive origin, 18 per cent of central nervous system origin, and 3 per cent of vascular origin. Observations that tinnitus persists in a certain number of patients despite destruction of the inner ear and/or section of cranial nerve VIII supports the concept of central tinnitus. Central tinnitus is usually described by the patient as being general in location, whereas peripheral tinnitus can frequently be localized to one or both ears. Several known causes of central tinnitus include the presence of space-occupying lesions, inflammation, and vascular abnormalities. In the absence of these entities, a proposal for the central origin of tinnitus is related to the "gate theory" of Melzack and Wall (1965). This theory suggests that the efferent pathways control the presynaptic mechanisms so that the ease with which cochlear information, for example, can penetrate the brain stem is determined both by the effect cochlear activity and by the central cortical processing through a feedback system. Alterations in the system may allow central input out of usual proportion to peripheral input.

A neurogenic etiology for tinnitus of tympanic origin is the basis for the tympanosympathectomy advanced by Lempert (146). According to Trowbridge (1949), "there is an intimate nerve connection between the neural elements of the middle ear and those of the internal ear". He stated that it is through these inner ear connections with the trigeminal, glossopharyngeal, and sympathetic nerves of the middle ear that tinnitus aurium can be produced by pathologic changes within the middle ear and neighboring structures. Trowbridge further stated:

It seems to make little difference whether the active lesion is located in the middle ear or in the neighboring structure, since the impulse which produces tinnitus is transmitted or relayed from the middle ear to the inner ear by the tympanic plexus. Apparently, the tympanic plexus acts as a central nerve plexus, receiving impulses through its connections with the fifth nerve, the ninth nerve, and the carotid sympathetic fibers. There impulses are manifested either by tinnitus or by aural pain. Presumably, the intimate connection of the tympanic plexus with the inner ear through the ramus vestibuli and the ramus cochlearis creates two phases in aural response to stimulation. Impulses of a minimal nature traverse the tympanic plexus without initiating a reaction of pain but are adequate to stimulate a reaction of tinnitus in the sensitive cochlear neuromechanism. Finally, if the stimulation becomes more intense, a reaction of pain occurs through stimulation of the tympanic plexus itself. The initial phase of minimal irritation is a subthreshold phase, resulting in tinnitus, and the secondary phase is a suprathreshold phase, resulting in pain.

Table 3. Processes Known to Cause or Be Associated with Nonvibratory Tinnitus

Presbycusis
Trauma
    Head trauma
    Acoustic trauma
Medications
Conductive hearing loss
Tumor
    Cranial nerve VIII
    Temporal lobe
Ménière's disease
Vitamin deficiency
Trace mineral deficiency
    Copper
    Iron
    Zinc
Metabolic disorder
    Hypothyroidism
    Diabetes mellitus
Labyrinthitis
    Allergic
    Viral
    Bacterial
    Spirochetal
Bell's palsy
Circulatory disturbance
    Hypertension
    Asud
Otosclerosis

This proposed pathophysiologic process could explain the tinnitus which is associated with Costen's syndrome. This syndrome is poorly understood and included temporomandibular

joint dysfunction with associated pain, limitation of motion, headache, tenderness over the muscles of mastication, joint crepitus and, in about 25 per cent of cases, the aural symptoms of tinnitus, hearing loss, otalgia, and disequilibrium. Electromyography of the masseter and temporalis muscles will frequently be abnormal, and occasionally tympanic membrane retraction can be demonstrated with jaw clenching, presumably due to tensor tympani contraction. Treatment directed toward the joint dysfunction itself usually relieves or reduces the aural symptoms.

In a study designed to elucidate the etiology of idiopathic nonvibratory tinnitus, Sasaki and co-workers (1981) hypothesized that functional cochlear integrity is necessary to the suppressive influences of the central nervous system on the auditory neurons. With cochlear ablation, they summarized that there would be an alteration in afferent auditory signals, reducing the suppressive influences of the central nervous system and allowing increased neuronal activity higher in the auditory system. These investigators used C-2-deoxyglucose as a method of mapping cellular metabolism in the brain. Following cochlear ablation in the guinea pig, they found early reduction in neuronal activity with subsequent return of activity by 12 to 48 days. This activity was felt to be a neurophysiologic correlate of tinnitus. If such is the case, patients with tinnitus are not too dissimilar from patients who have suffered injury to other sensory nerves and, subsequent to healing, experienced pain. This pain is felt to result from spontaneous discharge from deafferented secondary auditory neurons.

These findings are somewhat in conflict with the "leaky" hair cell theory of Davis (1954), however, which proposed that injured hair cells have an increased spontaneous resting rate ("leak"). Increase of the spontaneous "leak" associated with alteration of the central regulatory mechanism according to Davis's theory would result in tinnitus. It is apparent that the questions surrounding nonvibratory tinnitus remain largely unanswered.

Patient Evaluation

The evaluation of a patient with tinnitus begins, as does the evaluation of any symptom or complaint, with a thorough history. Attempts should be made to ascertain the age of onset, mode of progression, family history, and association with other audiovestibular symptoms (eg, hearing loss, aural fullness, vertigo). Subjective information should be gathered regarding the location of the tinnitus (general, unilateral, bilateral), its pitch (high, low), the relative complexity (single sound, multiple sounds), the pattern (steady, pulsatile, clicking, blowing), apparent intensity (loud, soft), level of irritation (slight, moderate, severe), effect of ambient noise (increasing intensity, decreasing intensity), and duration (continuous, intermittent). This information is important for future and perhaps epidemiologic considerations, but it must be noted that the patient's subjective descriptions often have no correlation with the acute acoustic properties of the tinnitus, underlying cause of the tinnitus, or site of lesion. It may be that the failure to establish such a correlation is the result of inadequacies in the majority of previous tinnitus descriptions. These have been mainly verbal, such as "the roar of the ocean", the ringing of a bell, or the buzzing of an insect. There may be no promise for isolating the site of lesion or for making a diagnosis in such an approach, yet the potential for better understanding of the complaint and the treatment does exist.

Additional history should include information regarding aural discharge, head trauma, noise exposure, and exposure to ototoxic drugs. The patient should be asked about any

apparent precipitating evens and about signs and symptoms of any disorders known to have tinnitus as an associated symptoms (See Table 3). A Minnesota multiphasic personality inventory and psychologic evaluation as proposed by House (1978) may have prognostic as well as diagnostic value.

A complete otoneurologic and head and neck examination should be performed, with special emphasis on auscultation of the mastoid tip, ear, skull, and neck. Inspection of the auricle, external auditory canal, and tympanic membrane, along with tuning fork tests and pneumatic massage of the tympanic membrane, are also necessary. Vital signs should be assessed as a screen for cardiovascular and hypertensive disease. Tewfik (1974) described phonoencephalography using a stethoscope for sound pickup and a phonocardiographic apparatus for amplification in a systemic search for vibratory basis for the tinnitus. This, as of yet, has not been widely accepted.

Many diagnostic modalities are available to study patients suffering tinnitus (Table 4). In cases in which the tinnitus is objective or in those patients whose history and physical examination suggest a mechanical basis for the tinnitus, specific studies should be undertaken to identify the responsible pathologic process. Angiography will confirm the presence of vascular lesions. Tympanometry helps to identify subtle changes in tympanic membrane stiffness due to respiratory influence through a patent eustachian tube or to myoclonus of the stapedial muscle, tensor tympani muscle, or muscles of the palate. Electromyography will confirm myoclonus of the palatal muscles. Once identified, the cause for myoclonus should be sought (eg, psychogenic, multiple sclerosis, cerebrovascular disorders, intracranial neoplasms).

Audiometric assessment plays a substantial role in the evaluation of the patient suffering nonvibratory tinnitus because of the poor correlation of the subjective complaint with actual acoustic properties. This assessment should begin with pure-tone audiometry and include Békésy audiometry to ensure that hearing is sampled at all possible ranges. Although atypical, patients whose octave and semioctave interval audiometry is normal may indeed, have a localized hearing loss disclosed only by Békésy audiometry. Attempts at matching the frequency of the patient's tinnitus to the identified hearing loss may produce a reasonable approximation by a narrow-band noise in the same frequency range. These patients are usually unaware of hearing loss, as are many patients with high-frequency hearing loss and tinnitus. The basis for this apparent paradox of hearing loss without awareness of it is that the bulk of listening involves speech that does not require the entire hearing range. In other cases, such as music, the listener may have control of the spectral composition of the signal and be able to compensate through the use of tonal controls, or he or she may simply be uninterested or inattentive to spectral imbalances. This, if hearing in the speech range is adequate, then self-estimates of hearing will often be normal.

The discovery of a hearing loss warrants a full evaluation for site of lesion and determination of etiology. Management then proceeds as though hearing loss were a coexistent complaint. Speech discrimination should be tested in and out of noise. Tone decay tests should be performed, as well as alternate binaural loudness balance (ABLB), tympanometry (stapedial reflex, reflex latency and reflex decay, tympanic membrane compliance), short increment sensitivity index (SISI), and brain-stem response audiometry.

Table 4. Laboratory Examination for Tinnitus

Audiologic	
Audiometry	Pure-tone air and bone conduction
Speech discrimination score	
Speech reception threshold	
Brain-stem response audiometry	
Impedance audiometry	Acoustic reflex
	Acoustic reflex decay
Tinnitus matching	
Electronystagmography	
Radiologic	
CT Scan	Intravenous contrast enhanced scan
	Air contrast scan of internal auditory canal
Tomography of temporal bones	
Angiography	Digital subtraction angiography
Jugular venography	
Hematologic	
MHA-TP (FTA-ABS)	Syphilis
Thyroid functions	Hyperthyroidism or hypothyroidism
Blood count (CBC)	Anemia
Allergic	
Allergic evaluation	Inhalants
	Food
	Environmental
Miscellaneous	
Electromyography.	

Further investigation into known causes of tinnitus should be dictated by the index of suspicion aroused by the history, physical examination, and audiometric profile. Electronystagmography will give insight into the status of the vestibular system. X-rays of the petrous apex may be helpful in diagnosing cerebellopontine angle lesions, and computerized tomography will help demonstrate most intracranial lesions. Laboratory data should include serologic tests for syphilis, complete blood count, serum thyroxine levels, and a glucose tolerance test.

In an attempt to classify tinnitus, objective tinnitus matching is needed in addition to the subjective information gained in the history. The major problem in matching tinnitus is to have a sufficient variety of stimuli available to the patient so that a match may be found.

Basic audiometers will provide tones at octave or half-octave intervals and at least one form of noise. More complex models are likely to have, in addition, several noises (eg, white, complex or sawtooth, speech spectrum, narrow band), warble tones (in which the fundamental frequency is variable by as much as 5 per cent in a systematic temporal way), and continuously variable tones or noise (as in variations of Békésy audiometers). Some recent reports have employed devices as complex as music synthesizers to broaden the possible matches to an almost infinite variety.

Given the stimuli available, the next problem is to determine a technique for matching. If the tinnitus is unilateral or different in each ear, a method similar to the ABLB test may be used. If the tinnitus is equal bilaterally, the technique becomes an approximation of a monaural loudness balance in which the tinnitus alone is compared with the tinnitus plus matching noise both for loudness and spectral content. An easier approach is to make the criterion for description less stringent by seeking a noise that will mask the tinnitus. It must be understood, however, that describing tinnitus in terms of a noise that will mask it is unlike any matching procedure, either in terms of the task or interpretation of the result. Matching presumes that the tinnitus is identical to the matched stimulus, within the capabilities and limitations of the observer. A tinnitus masker is at least the equivalent of the tinnitus and is, most likely, greater than the tinnitus in both spectral content and level. As an extreme example, it is likely that white noise at some level will mask any tinnitus, either of low or high frequency. Thus, the specification of tinnitus in terms of a masker is only an approximation whose closeness depends on the capabilities for manipulating the spectrum of the masker. The task of obtaining increasingly finer approximations of the minimal spectrum necessary to just barely mask the tinnitus is not unlike the procedure for obtaining critical bands of noise that will just mask a tone.

As has been stated, several investigators have begun the quantification of tinnitus. A somewhat surprising finding is the comparatively low level of tinnitus sufficient to make it a complaint. Of those tested, 87 per cent match to noise at a 20-dB sensation level or less. Such figures are important for determining the levels necessary for one successful mode of treatment (masking) and have a direct bearing on another mode (biofeedback) based on the assumption that tinnitus is a stress-related symptom.

Treatment

Patients who have compensated for their tinnitus in a healthy and socially acceptable manner need little or no treatment, whereas those uncompensated patients need to be brought to a compensated level. Several therapeutic modalities for the uncompensated patients have been proposed, not all of which are mutually exclusive and none of which offer a panacea. Some patients may respond to one or more of these approaches; others will respond to none. Treatment modalities include surgery, medicines, masking (including amplification of ambient sounds), biofeedback, hypnosis, psychotherapy, and galvanic stimulation of the end-organ. It must be remembered that patients with tinnitus do have a perplexing problem and, even if a treatable etiology is not uncovered, these patients should not be abandoned. Simple measures such as reassurance and understanding should not be forgotten as part of the art of medicine.

Surgical Therapy

Several surgical procedures have been advocated for the treatment of tinnitus. Surgery definitely plays a role in the treatment of most causes of vibratory tinnitus, as well as space-occupying cerebellopontine angle lesions, temporal lobe neoplasms, and conductive hearing loss. Vibratory tinnitus is usually relieved following removal of the underlying disease process; although while surgery for the tinnitus of cerebellopontine angle lesions, temporal lobe neoplasms, and conductive hearing loss may be successful, the prognosis is somewhat less optimistic. An example is the tinnitus frequently associated with otosclerosis. Oval window surgery will correct this problem in up to 75 per cent of cases, but the results are not predictable.

Operations on the endolymphatic sac, ultrasound directed toward the inner ear, cryotherapy to the horizontal semicircular canal, sodium chloride crystals placed at the round window membrane, stellate ganglion sympathectomy, and intermittent repetitive sacculotomy have all given relief to 30 to 50 per cent of patients suffering tinnitus from Ménière's disease. Labyrinthectomy and section of cranial nerve VIII in patients with tinnitus sacrifice hearing, and the results are variable and generally poor. Tympanosympathectomy as advocated by Lempert (1946) has also failed to stand the test of time.

Chemical tympanosympathectomy has been suggested using lidocaine, procaine, alcohol, and ethyl-morphine hydrochloride in the form of subcutaneous injections into the tympanic promontory. Approximately 50 per cent of patients with nonvibratory tinnitus so treated in the Soviet Union (Tsyganov, 1968) have experienced relief with this treatment modality. The placement of tympanostomy tubes has been advocated by some; however, in the absence of altered middle ear physiology, the benefit obtained by this procedure is unclear.

Based on the theory that tinnitus is the result of inner ear vasospasm, temporary chemical and permanent surgical sympathectomies have been tried. Golding-Wood (1960) reported that seven of 30 patients with tinnitus who did not have Ménière's disease experienced improvement of tinnitus following sympathectomy. Temporary percutaneous block of the stellate ganglion can be achieved with local anesthesia. Occasionally, following one or more blocks, permanent relief can be obtained. In other cases in which temporary relief is followed by recurrence, surgical sympathectomy may be indicated.

Translabyrinthine avulsion of the cochlear division of cranial nerve VIII in patients suffering idiopathic non-vibratory tinnitus in a non-hearing ear is successful in about 60 per cent of cases.

Medical Treatment

An infinite variety of medications has been advocated by various authors for the treatment of tinnitus. Several drugs are directed toward interrupting proposed pathophysiologic mechanisms for the production of tinnitus, while others are used to help the patient better tolerate his malady. Agents aimed at increasing blood flow have been advocated, based on the theory that ischemia to the end-organ or even, perhaps, the central auditory system is plausible etiology for tinnitus. These medications include adrenergics, adrenergic-blocking

agents, antiadrenergics, cholinomimetics, anticholinesterase agents, cholinolytics, smooth muscle relaxants, plasma polypeptides, and vitamins. In a review of the relative efficacy of these drugs and their potential benefits and potential side effects, Snow and Suga (1975) concluded that papaverine hydrochloride, a smooth muscle relaxant, was the drug of choice for increasing cochlear blood flow.

Vitamins - primarily A, C, B_2, and nicotinic acid - have been advocated for their beneficial effects on the vascular system as well as their beneficial effects on the well-being of the individual as a whole. Trace metals such as zinc and copper have been used for similar reasons.

Antihistamines and decongestants have been used when eustachian tube dysfunction was felt to be etiologically involved, and tranquilizers aimed at decreasing associated anxiety have been widely used. Magnesium sulfate, barbiturates, meprobamate, and reserpine have all been tried for their depressant effect on the reticular formation in the central nervous system.

Local anesthetics, especially the derivatives of para-aminobenzoic acid (eg, procaine) and the aminoacyl amide group (eg, lidocaine, lignocaine) have been advocated for the treatment of tinnitus, based on their ability to decrease sensory activity at a central level. Intravenous (IV) administration of these agents has been shown to reduce hyperactivity of conducting tissue. When administered in this manner, they provide temporary diminution of tinnitus in the majority of patients. Since the tinnitus almost always returns to the pre-administration level within hours to days, there is very little therapeutic efficacy in this approach for long-term therapy. Melding and associates (1978) and Shea and Harell (1978), however, have used the information gained from the intravenous administration of procaine for diagnostic purposes. Those patients that respond well to test doses of IV procaine are considered to have tinnitus based on an increased sensory activity and are given Tegretol, an anticonvulsant found to be effective in epilepsy and trigeminal neuralgia. The majority of patients so treated have obtained relief from their tinnitus. It must be cautioned, however, that Tegretol has been associated with irreversible aplastic anemia. Because of this serious side effect, Mysoline and Primidone, which are closely related to phenobarbital, have been tried, with varying degrees of success. None of these described medical treatments have proved, *over time,* to provide more than random relief.

Recently, tocainide hydrochloride (the oral analogue of lidocaine) has been reported to benefit up to 85 per cent of patients who experience relief from tinnitus with intravenous lidocaine. The strength of this information, however, is diluted somewhat by the fact that up to 40 per cent of tinnitus sufferers benefit from placebo administration.

Masking

According to Vernon and Schleuning (1978), masking for the relief of tinnitus was mentioned as early as 400 BC. The fact that one tone can be masked by another was formally demonstrated by Wegel and Lane (1924). Masking tinnitus by amplifying environmental noise or by the introduction of artificial noise has been employed intermittently ever since. It is a frequent observation that the amplified environmental noise or artificial noise is less objectionable to the patient than his tinnitus and that the tinnitus is most troublesome during

periods of quiet. A simple form of tinnitus masking is that provided by setting the FM radio dial between stations, allowing broad-spectrum static to be generated and providing the patient with a tinnitus-free interval prior to sleep. The hearing aid which, in the presence of a hearing loss, may be recommended for speech perception and discrimination, will at the same time introduce masking as a function of amplifying environmental noise. The procedure is conceptually straightforward.

An additional step is to define a noise that will mask the tinnitus and select a device that will produce this masking noise and is simple and portable enough to be used by the patient. It is well known that a loud tone masks tones of higher frequency more easily than tones of lower frequency, and that the degree of masking increases as the frequency of the masking tone approximates the frequency of tinnitus. For tinnitus in the range of spectral capabilities, and using the hearing aid loudspeakers and acoustic modifications, which may be made through earmold and speaker-earmold connectors, it is necessary only to modify the output of the hearing aid or masking generator to produce as little noise as necessary to overcome the tinnitus. Those familiar with the tailoring of frequency response will understand that the leap from the concept to its technical implication is not easy. There also is a need for a device to mask tinnitus outside the range of currently available hearing aid loudspeaker reproduction.

At the present state of the art, tinnitus maskers are small-noise generators capable of delivering a constant or varied signal at low frequencies (500 to 3000 Hz) or high frequencies (2000 to 20.000 Hz). Volume control can adjust output between 45 and 90 dB sound pressure level. Tinnitus instruments combine frequency-specific amplification with masking for patients with significant hearing loss.

Feldman (1971) described five different patterns that can be identified when attempting to mask tinnitus. Type I is the *convergence type* and is found in patients with high-frequency hearing loss and high-pitched tinnitus. The tinnitus masking curve and the hearing sensitivity threshold curve slowly merge from low to high frequencies. These two curves meet at the frequency corresponding to the pitch of the tinnitus and coincide at higher frequencies. Type II is the *divergent type,* in which hearing threshold curves and masking threshold curves separate when going from low to high frequencies. In type III (the *congruence type*) a narrow-band noise will mask tinnitus at a low sensation level. Type IV, the *distant type,* is masked only by noise well above threshold. In type V, the *resistance type,* the tinnitus cannot be masked by external stimulus.

An unusual finding arising from the systemic study of noise in masking tinnitus is that cessation of masking produces continued suppression of tinnitus for periods sufficiently long to be considered significant to the patient. This post-masking suppression of tinnitus was first described by Josephson (1931) and termed "residual inhibition" by Feldman (1971), who reported the first formal study on the effect. Vernon and Schleuning (1978) indicated that this effect may last as long as 30 to 40 minutes following the cessation of masking. Not all patients experience residual inhibition of tinnitus, and those that do may experience it for varying lengths of time. Residual inhibition appears to be a favorable prognostic sign for future successful tinnitus masking. Contralateral masking does not appear to produce this phenomenon.

Masking is not without limitations. In those patients with little or no residual hearing in the range of the tinnitus, it may be impossible to mask the tinnitus by external means. There is some indication that masking may be achieved by introduction of the masker into the contralateral ear when the ear affected by tinnitus cannot be masked by an external source. If the tinnitus is in the frequency range necessary for speech discrimination, however, the introduction of a masker may interfere with or further decrease understanding in the functional ear. It has been proposed that patients in whom tinnitus is particularly difficult to mask may be suffering from tinnitus of a central origin.

Experience to date suggests that masking devices will help 10 to 15 per cent of tinnitus sufferers. For this reason, this device should be considered, but judicious patient selection is required for the most rewarding results.

Biofeedback

In many cases the disability attributed to tinnitus is more closely related to the patient's psychological reaction to the tinnitus than to the intensity of the tinnitus itself. From this rather universal observation, the role of biofeedback has emerged as a modality in the treatment of tinnitus. Biofeedback has been used in the treatment of other stress-related disorders for over 30 years. Those patients who experience extreme irritation from their tinnitus respond best to this form of therapy. The use of biofeedback to alleviate tinnitus does not confront a patient's symptom directly, as does the use of masking. Rather, it is based upon the assumption that tinnitus is a stress-related symptom, and that a generalized reduction in stress can lead to a reduction in the patient's adverse reaction to it. Although not explored in reports on the use of biofeedback, it would appear that a reduction in the magnitude of tinnitus (as measured by masking, for example) is not necessary to obtain a report of reduced patient discomfort.

The primary focus of biofeedback is on relaxation achieved through teaching the patient to control certain functions overtly. This requires a learning process in which the patient gains voluntary control over heretofore involuntary functions, thereby normalizing disturbed body functions. Six to eight sessions of 30 minutes each are usually required if benefit is to be expected. The most common form of feedback involves information gained from electromyography, electroencephalography, and monitoring vital signs and skin temperature readouts of appropriate sensing devices regarding muscular tension and vasospasm. These techniques are used in the broader context of what might be described as a psychotherapeutic situation. The patient is not only taught to relax but is also encouraged to relate his or her state of relaxation to the stress of experiences in daily living. In the case of the patient with tinnitus, this observation of his or her ongoing state of relaxation-tension includes the symptom of tinnitus. The patient is encouraged to document the relationship in the form of a daily journal. Recognition of the following three facts is presumed to lead to the alleviation of discomfort: (1) the patient can control his or her state of relaxation; (2) increased relaxation reduces stress and its symptoms, and (3) tinnitus is such a symptom.

Several points should be made in regard to patients involved in reports of biofeedback therapy. To date they have been the most resistant patients to other means of treatment, having undergone and failed to find relief in such techniques as hypnosis and acupuncture. The efficacy of biofeedback has been judged on a psychological level and not in terms of

objective indices, such as pre- and post-treatment tinnitus matching or masking measures, even though there are reports of total disappearance of tinnitus during biofeedback therapy. Many of these patients have other stress-related symptoms, such as migraine headaches, and have been receiving some form of chemotherapy, such as tranquilizers. Last, the degree of relief may be related generally to stress and not particularly to tinnitus. Thus, a patient may feel better or have a better understanding of the factors associated with stress without necessarily having the tinnitus disappear.

Biofeedback draws on a body of knowledge in which the otologist has little, if any, formal education. The resources of a psychologist are undoubtedly necessary for the design of a biofeedback program and for the evaluation of pre- and post-therapy effects, which may range from the disappearance of tinnitus to simply a less irritative response to a reasonably constant source of irritation.

The results of biofeedback for the treatment of tinnitus are encouraging, as they demonstrate more than chance relief from this perplexing symptom. Up to 80 per cent of patients treated by biofeedback gain improvement and up to 15 per cent are totally relieved.

Miscellaneous Therapies

Other treatments have been tried for tinnitus with varying degrees of success. Among these are the galvanic stimulation of the end-organ, hypnotherapy, counseling, support groups, and holistic therapy. During the galvanic stimulation of the cochlea in attempts to learn more about auditory rehabilitation of the deaf, it was noted that tinnitus occasionally decreased. Follow-up studies indicated that this suppression of tinnitus occurred during positive pulse stimulation, without affecting acoustic perception or hearing. When negative current was applied, auditory sensation occurred. Direct current appears to relieve tinnitus but also appears to be harmful to neural tissue. To date, the most practical use of this information has been with the cochlear implant, which improves tinnitus in over 50 per cent of patients. This avenue of investigation seems promising for the patient with tinnitus but currently remains experimental.

Because few therapeutic approaches to tinnitus exist that can reliably result in alleviation of the problem, careful explanation of the symptom along with reassurance add immeasurably to the art of patient care. If the physician or audiologist cannot fulfill these needs, support groups and counseling may be beneficial. In some regions, the holistic approach to patients with tinnitus, including extensive general health appraisal and diet and hair analysis, has helped elucidate underlying causes of tinnitus. For the severely disturbed and pre-suicidal patient, psychotherapy may be necessary.