Improvements in imaging technology have made it possible to evaluate the human auditory system in a manner that would have bewildered our predecessors. Conventional radiographs have been augmented by the development of computed tomography (CT) to provide highly detailed images of osseous and soft tissue structures in and about the ear. The same is true for magnetic resonance imaging (MRI), particularly when gadolinium contrast enhancement is used. Both of these technologies are evolving rapidly, and new MRI pulse sequences, stronger magnets, and improved image reconstruction techniques hold the promise for further improvements. Other imaging techniques, particularly positron emission tomography (PET) and single photon emission computed tomography, provide the clinician and investigator with critical data that have made it possible to attribute specific auditory tasks or sensations to discrete regions of the brain. This chapter is designed to provide a guide so that these resources can be used optimally.

**ESTABLISHING A DIAGNOSTIC FRAMEWORK**

To use imaging techniques efficiently, it is essential to establish a diagnostic framework for the patient with tinnitus. Two broad categories for these patients are (1) those who have subjective tinnitus, the false perception of sound in the absence of an environmental source, and (2) those who have objective tinnitus, in which the sounds are real but possibly not perceptible to an observer-clinician. Some causes of objective tinnitus are shown in Table 18-1.

### Table 18-1. Objective Tinnitus

<table>
<thead>
<tr>
<th>Pulsatile Tinnitus</th>
<th>Nonpulsatile Tinnitus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neoplasms (typically vascular in nature)</td>
<td>Palatal myoclonus</td>
</tr>
<tr>
<td>Glomus tumors or paragangliomas (chemodectoma, paragangliomas)</td>
<td>Spasm, fasciculations, or fibrillations of tensor tympani or stapedius muscles</td>
</tr>
<tr>
<td>Glomus tympanicum, glomus jugulare, glomus jugulotympanicum</td>
<td>Spontaneous otoacoustic emissions</td>
</tr>
<tr>
<td>Hemangioma</td>
<td>Patulous eustachian tube</td>
</tr>
<tr>
<td>Facial nerve hemangioma, cavernous hemangioma</td>
<td></td>
</tr>
</tbody>
</table>
**Clinical Imaging in Patients with Subjective Tinnitus**

Most patients with subjective tinnitus have a partial or complete loss of hearing. This deficit may range from mild loss at high frequencies to total deafness. Although the literature describes a small portion of patients with tinnitus as having normal hearing, we raise the question as to whether this normal ability to perceive stimuli represents a decline from a prior level. A patient with a threshold of 20 dB HL at 4 kHz would be considered to have normal hearing. However, if this threshold represents a decline from 10 dB HL, this patient has lost hearing. The audiogram is typically measured from 125 to 8,000 Hz in octave steps. A patient could have normal thresholds at these discrete frequencies, but losses could go undetected between test frequencies or frequencies above 8,000 Hz. If hearing loss is an important factor in the pathogenesis of tinnitus owing to plastic changes in the central part of the auditory system caused by deafferentation, then these seemingly minor changes may be important.

Studies from our laboratory suggest strongly that subjective tinnitus is the consequence of plastic transformations in the central auditory pathways that arise as the result of auditory deafferentation. We further suggest that the transformed centers and pathways that contain aberrant connections (including connections to nonauditory systems) perceive sound when, in fact, there is no source of sound. Restated, tinnitus may be the result of neural plasticity gone awry. Thus, tinnitus may be the auditory system analog to neuropathic pain syndromes in which the intensity of the pain is proportional to the degree to which somatosensory pathways have been transformed by an alteration in the afferent input to the somatosensory system. Therefore, imaging of patients with subjective tinnitus is largely the same as the imaging of hearing loss. This much broader topic, which is outside the scope of this chapter, has been well reviewed from an imaging perspective.

Among patients with subjective tinnitus, the question “Does this patient have a vestibular schwannoma?” arises frequently, particularly among patients who complain of tinnitus confined to a single ear. The emphasis on the early diagnosis of these tumors has increased with the development of surgical techniques that preserve hearing and gamma knife radiation therapy, which may obviate the need for surgery. On the basis of the history, clinical examination, and audiologic assessment, it is usually possible to differentiate patients with retrocochlear lesions from those with cochlear pathology. In making this determination, the auditory brainstem response (ABR) is often particularly helpful. For those patients in whom the index of suspicion is particularly high, a stacked ABR should be performed. This technique appears to be more sensitive than conventional ABR testing in the detection of small tumors. In patients with retrocochlear lesions or a high index of suspicion pointing to a tumor, MRI with gadolinium enhancement is the diagnostic test of choice. A small intracanalicular vestibular schwannoma is shown in Figure 18-1. For those patients who are unable to undergo MRI, CT with air or positive contrast must suffice.

![Figure 18-1. T1-weighted magnetic resonance images of a small intracanalicular vestibular schwannoma before (left) and after (see arrow, right) the administration of gadolinium. Image courtesy of Rohit Bakshi, MD.](image_url)
CLINICAL IMAGING OF PATIENTS WITH OBJECTIVE TINNITUS

In patients with objective tinnitus, the sounds are real. Therefore, it is important to clarify the mechanism that underlies the generation of the sound and to determine how these sounds are transmitted to the cochlea. When evaluating patients with objective tinnitus, it is important to differentiate those with pulsatile tinnitus from those whose tinnitus is not pulsatile. Bruits or other sounds emanating from arteriovenous malformations and tumors, for example, may be heard by the examiner under appropriate conditions. Every effort should be made to determine whether the bursts of sound coincide with cardiac systole.

Pulsatile Tinnitus For most patients with pulsatile tinnitus, the sounds are generated by turbulence in arteries or other vessels with rapid blood flow. The turbulence creates pressure waves in the vascular system that are transmitted through bone or blood to the cochlea. In the cochlea, these fluctuations in pressure are converted into neural impulses that are experienced as sound. Virtually any condition that creates intravascular turbulence can result in the symptom of objective tinnitus. During the physical examination of patients with pulsatile tinnitus, it is important to auscultate the portion of the head identified by the patient as the site of his or her sounds. The most common conditions causing objective tinnitus are listed in Table 18-1.

Because the prevalence of pulsatile tinnitus and the prevalence of atherosclerotic cardiovascular disease increase with age, many patients with pulsatile tinnitus will be found to have stenotic arteries. An example of this condition is illustrated in the angiogram shown in Figure 18-2. Stenosis of any of the intracranial arteries causes turbulence at and distal to the site of the stenosis. The mechanical energy created by the turbulence stimulates the cochlea. The bifurcation of the carotid artery is a particularly common site for these lesions. The intradural portion of the carotid artery is in close proximity to the cochlea. This facilitates transmission of vibrations from any carotid lesion to the cochlea. Carotid stenosis at other sites, particularly at the siphon, and other lesions of the carotid, such as fibromuscular dysplasia, may also cause pulsatile tinnitus. Stenotic lesions in other intracranial arteries, including the vertebrobasilar system and its branches, may be associated with pulsatile tinnitus.

Other vascular lesions, such as dissections or aneurysms, may also be associated with audible pulsations.

Arteriovenous malformations are also a site where turbulent blood flow may give rise to audible pulsations. The malformation may be found at virtually any intracranial location. Intracerebral malformations and dural malformations are particularly common. Occasionally, a head injury will cause a traumatic arteriovenous malformation in which arteries communicate with dural or other venous structures.

In rare instances, an aberrant internal carotid artery, a persistent stapedial artery, or an abnormality of the jugular bulb will be responsible for pulsatile tinnitus.

Some highly vascular tumors, as exemplified by the glomus jugulare tumor shown in Figure 18-3, may be the source of pulsatile tinnitus. Again, flow turbulence produces the sound. These lesions may be seen during an otoscopic examination. Other tumors associated with pulsatile tinnitus are listed in Table 18-1.

Other conditions that may be associated with pulsatile conditions include valvular heart disease, particularly aortic stenosis and mitral regurgitation, and a hyperdynamic state associated with severe anemia or thyrotoxicosis. Patients with thyrotoxicosis may

FIGURE 18-2. Carotid stenosis with an ulcerated plaque (arrow) as a cause of pulsatile tinnitus. Turbulent blood flow at the site of the stenotic internal carotid artery generates sound waves that are transmitted to the cochlea. A loud bruit may be heard, and a thrill may be palpable. Image courtesy of Rohit Bakshi, MD.
also have atrial fibrillation, making it more difficult to link the audible sounds to the pulse.

Patients with pulsatile tinnitus should have a radiologic evaluation to determine the site and severity of the lesion. Because of the diversity of the lesions that may give rise to audible pulsations, no single procedure suffices for all patients. The results of the history and physical examination usually serve to orient the physician to the appropriate test. Diagnostic procedures designed to identify vascular lesions are changing rapidly with the advent of CT and magnetic resonance angiography. The degree of invasiveness, and hence the risk associated with these investigations, is substantially lower than that for catheter-based angiography, the gold standard for image quality. However, rapid improvements in software and the introduction of magnets with higher field strength into clinical practice are closing this quality gap. Unless one is familiar with the procedures performed by the imaging specialists, a personal contact to establish an appropriate plan is likely to lead to the best result for any given patient.

**Nonpulsatile Tinnitus** Nonpulsatile objective tinnitus is less common. The major causes are shown in Table 18-1. Palatal myoclonus is characterized by rapid rhythmic contractions of the muscles in the soft palate. These are often associated with similar time-locked contractions of other muscles in the pharynx, mouth, and lower face and, occasionally, other muscles subserving the neck and eyes and even muscles associated with respiration. These contractions result in the opening and closing of the eustachian tube, the source of the sound perceived by these patients. On examination, contractions of the affected muscles should be visible, and auscultation may allow the examiner to hear the sounds perceived by the patient. Infarcts of the pons or other lesions that interrupt the pathways connecting the inferior olivary nucleus and the red nucleus cause this rare disorder. MRIs may show the lesion and the characteristic combination of hypertrophy and degeneration of the inferior olivary nucleus.

**FUNCTIONAL IMAGING OF THE AUDITORY SYSTEM**

**Normal Central Part of the Auditory System**

It is essential to understand the functional anatomy of the normal auditory system to understand the results of altering this complex system. Although there are numerous and detailed descriptions of the anatomic and physiologic features of the central part of the auditory system, few studies have sought to identify all of the human brain regions that are activated by simple stimuli.

We used PET to map the central auditory pathways in a group of normal young adults using methods specifically designed to minimize the impact of scanner and ambient noise. This strategy uses water, labeled with radioactive oxygen, to measure cerebral blood flow, which is a surrogate measure of neural activity. By comparing cerebral blood flow images in two states, for example, scans done during auditory stimulation minus scans done in the absence of sound, it is possible to identify brain regions that respond to the stimulus. By superimposing these results on an anatomic representation of the brain, typically an averaged MRI, it is possible to deduce
the anatomic sites activated by the stimulus. Thus, when we stimulated the right ear, we identified a network of sites in the left brainstem that extended from the level of the cochlear nuclei, superiorly to the level of the medial geniculate body, and bilaterally to both primary auditory cortices, as shown in Figure 18-4. By using stimuli of different frequencies (0.5 and 4.0 kHz), we found the expected tonotopic relationships in the primary auditory cortex with low-frequency sounds activating lateral sites and higher-frequency sounds activating the same lateral site but also a more medially located site, confirming and extending prior studies, including an earlier PET study confined to image planes that included only the superior temporal gyrus.6

These studies have served several important purposes. First, they identify the neural sites most active during the processing of simple auditory stimuli that are free of linguistic content. Second, they serve as a basis for understanding the effects of various conditions that have the potential to affect neural activity in the central part of the auditory system, most notably, hearing loss and tinnitus.

**Central Part of the Auditory System in Patients with Hearing Loss and Tinnitus**

Our studies have focused on two groups of patients with tinnitus. The first involved patients with sensorineural hearing loss who localized their subjective tinnitus to the right ear.7 In this investigation, we stimulated the tinnitus ear with 2 kHz tones and applied the same stimulus to control subjects, all of whom had normal hearing. The stimuli activated a larger region of the temporal lobe in the patients with hearing loss and tinnitus compared with the controls, as shown in Figure 18-5. The additional regions activated by the stimuli were observed in the anterior portion of the superior temporal gyrus and a second site slightly posterior and inferior to the primary auditory cortex. This confirmed numerous studies in experimental animals that have shown expansion of brain regions responding to auditory stimuli after partial deafferentation.8

In a second series of studies, we studied patients with gaze-evoked tinnitus (GET).9 This unusual phenomenon is usually the consequence of surgery to treat a cerebellopontine angle tumor and is characterized by tinnitus that either appears or increases in loudness, pitch, or both with gaze directed away from the primary position.10,11 These patients had all been treated for vestibular schwannomas, had tinnitus in the operated ear that was present in the primary gaze

![FIGURE 18-4. Sites of neural activation during right ear stimulation with a 4.0 kHz tone at 90 dB SPL. The results of the analysis of positron emission tomographic images (white “blobs” in the book, colored on the CD) are superimposed on idealized T1-weighted magnetic resonance images in the transaxial plane. Numbers (z = mm) indicate distance above and below the commissural plane. Images are shown with the right side of the brain on the left side of the figure. See Lockwood and colleagues5 for additional details.](image-url)
position, and reported an increase in subjective loudness with sustained horizontal gaze. Hearing in the unaffected ear was normal or nearly normal. In this study, we stimulated either the right or the left ear and sensorineural hearing loss. The data in this image set were produced by subtracting the sites activated by 2 kHz tones in normal controls from sites activated by the same stimulus delivered in an identical manner to the patients. Sites of additional activation in the patients are seen in the anterior portion of the superior temporal gyrus and a second site posterior and inferior to the primary auditory cortex. See Lockwood and colleagues for additional details.

FIGURE 18-5. Expansion of neural sites activated by tonal stimuli delivered to the right ear in patients with subjective tinnitus reported in the right ear and sensorineural hearing loss. The data in this image set were produced by subtracting the sites activated by 2 kHz tones in normal controls from sites activated by the same stimulus delivered in an identical manner to the patients. Sites of additional activation in the patients are seen in the anterior portion of the superior temporal gyrus and a second site posterior and inferior to the primary auditory cortex. See Lockwood and colleagues for additional details.

as shown in parts D, E, and F of Figure 18-6. Thus, it is evident that deafferentation accompanying a deafened ear expands the locus of the sites of neural activity when the remaining normal ear is stimulated.

The results of these studies show that the combined effects of hearing loss and tinnitus have profound effects on the central part of the auditory system. We are unaware of any PET studies of patients with hearing loss without tinnitus. Hearing loss may be caused by a variety of disorders that can be diagnosed by appropriate radiologic examinations.

RESEARCH IMAGING OF PATIENTS WITH TINNITUS

Research techniques, particularly PET and functional magnetic resonance imaging (fMRI), have made it possible to perform studies that have expanded our understanding of tinnitus. PET uses a measure of cerebral blood flow as an index of neural activity, and fMRI uses a change in the redox state of hemoglobin. Both allow a superimposition of a site of change on an anatomic reference, usually the participant’s own structural MRI or a standard MRI obtained by averaging multiple, usually T1-weighted, scans from normal individuals. PET has an advantage in that the imaging system is associated with almost no noise and is relatively insensitive to small movements of participants. fMRI examinations are usually less expensive and yield functional images of a single subject with greater ease. However, MRI systems, particularly those with magnets with high field strength that are best suited to fMRI applications, are noisy and highly sensitive to movement, including small movements of the brainstem caused by arterial pulsations. Thus, it is appropriate to consider each of these approaches as complementing the other.

We have used PET to study three different groups of patients with tinnitus. In the first of these, we examined patients who had unilateral tinnitus that was reported to come from the right ear. In this study, we stimulated either the right or the left ear and again observed the bilateral cortical activation in the control subjects and in the two patients with right vestibular schwannomas after stimulation of the left ear and in five patients with left vestibular schwannomas after stimulation of the right ear, as shown in parts A to C of Figure 18-6. Parts B and C suggest strongly that the volume of brain reactive to the stimuli is larger in the patients than in the controls, an impression that is confirmed by subtracting the control scan activations from those observed in the patients,
decreased in parallel to changes in the reported loudness of tinnitus. This site, shown in Figure 18-7, was confined to a cortical region immediately adjacent to the primary auditory cortex. Because cochlear stimulation in patients with tinnitus and normal controls produced bilateral activation of auditory cortical sites, we interpreted the unilateral nature of this brain region as evidence to support a noncochlear, that is, central auditory, origin of tinnitus. This finding has been supported by data from two other studies, one involving patients with GET and another in which we administered intravenous lidocaine. In both studies, we found unilateral changes in neural activity associated with changes in tinnitus loudness.

FIGURE 18-6. Expansion of neural sites activated by 0.5 kHz tonal stimuli to the unaffected ear of patients with gaze-evoked tinnitus. These “glass brain” statistical parametric maps are generated by projecting the three-dimensional data set onto two dimensions, in this case, in the transaxial plane. All sites portrayed are in and around the primary auditory cortex and depict sites of neural activity revealed by the various comparisons. For all images, the front of the brain is on the right side of the figure and the right hemisphere is on the top. A depicts the activations observed in the controls, right ear stimulation, compared with rest (no sound). B depicts the same stimulus minus rest condition for two patients treated for right-sided vestibular schwannomas and stimulated in the left ear. C shows the analogous comparison for the patients with left-sided tumors. D to F show the results of subtracting the expected activations in the normal group from the activations in the patients [contrast form = patient(active − rest) − control(active − rest)]. D and E show data from subjects with right and left vestibular schwannomas, respectively. F is similar to E (left vestibular schwannoma), except after omission of subject 3, the only patient in the group with preserved hearing. Reproduced with permission from Lockwood AH et al.

FIGURE 18-7. Sites of neural activity associated with tinnitus. Positron emission tomographic images of patients with tinnitus altered by jaw clenches were obtained. Tinnitus-soft images were subtracted from tinnitus-loud images as described in Lockwood and colleagues. The sagittal and coronal statistical parametric maps were produced as described in Figure 18-6. The arrows shown on the sagittal projection identify the plane of the coronal plane images shown in the lower panel, where y distances in millimeters are referenced to the anterior commissural plane. Reproduced with permission from Massachusetts Medical Society, Lockwood AH et al.
In the brainstems of the patients with GET, we found evidence for abnormal activity during lateral gaze, as shown in Figure 18-8. We interpreted this as evidence of an abnormal interaction between neural centers controlling eye movements and those mediating auditory perception. Neural firing rates increase as needed to execute a lateral eye movement. Apparently, some of this increased activity is transmitted through aberrant connections to neurons in the auditory system. This aberrant activity may explain the shifts in the apparent pitch and loudness of the tinnitus associated with eye movements that are experienced by many of these patients.

The study of patients with GET yielded the additional important result shown in Figure 18-9. When we scanned normal participants, who did not have GET, during full lateral gaze, we found a reduction of activity in auditory cortical areas. This appears to be a physiologic correlate of “crossmodal inhibition.” Thus, during the execution of a visually based task, auditory sites are inhibited. There was no inhibition of neural activity in auditory cortical sites when the patients with GET executed the same visual task, that is, there was a failure of crossmodal inhibition in the patients with GET. This lack of inhibition may cause the increase in the loudness and possibly the pitch of the tinnitus that these patients report.

In a final study, we administered lidocaine intravenously to patients with tinnitus. To our initial surprise, almost half of our patients reported an increase, rather than the expected decrease, in the perceived loudness of their tinnitus. This response is well described in the anesthesia literature. This fortuitous circumstance worked to our advantage by allowing us to separate the global effects of lidocaine on neural function from those associated with tinnitus. This ability to differentiate specific from nonspecific effects is particularly important in drug studies because drugs frequently have effects other than those that form the focus of the research. In the tinnitus-focused analysis of these data, we found a unilateral focus in the auditory cortex, similar in location to the site of activity
shown in Figure 18-7, in which neural activity increased and decreased in parallel with the perceived loudness of the participant’s tinnitus. Other sites of activity, possibly related to the direct effects of the drug on the brain, were found in the drug-focused analyses.

Other noninvasive imaging techniques have been used to study patients with tinnitus. In a novel approach, Melcher and colleagues used fMRI to evaluate neural activity in the inferior colliculi of patients who lateralized their tinnitus to one ear. In their study, they measured the increment in activity produced by a masking noise. In normal controls, they found symmetric increases in activity in response to the external sound. In the patients, the increase was asymmetric, with the largest increase confined to the inferior colliculus ipsilateral to the stimulated ear. The investigators concluded that as a result of the spontaneous neural activity associated with tinnitus, neural activity in the contralateral inferior colliculus had already plateaued at its maximum. Therefore, the additional stimulation provided by the external noise failed to increase the level of neural activity in the inferior colliculus contralateral to the stimulated ear.

The technique of magnetoencephalography has also been applied to investigate patients with tinnitus. Magnetoencephalographic systems measure the small magnetic fields generated by neuronal activity and have high spatial and temporal resolution. In their study, Muhlneckel and colleagues mapped the loci of magnetic sources generated by tones of differing frequencies in controls and patients with tinnitus. In the patients, they found a displacement of the site responsive to a tone, the frequency of which was matched to the frequency of the tinnitus in a given patient. The magnitude of the displacement from the expected site of activation was significantly related to the subjective strength of the tinnitus, that is, patients with loud tinnitus had the largest displacements. These data provide additional evidence for reorganization of the central auditory pathways in patients with tinnitus. These and other studies, not reviewed, have provided evidence of tinnitus-associated excess neural activity in auditory pathways, plastic transformations of central auditory pathways that appear to be the result of tinnitus and hearing loss or both, and aberrant connections between portions of the brain classically associated with the auditory and nonauditory neural systems. We anticipate that these initial observations will lead the way to additional studies that will further define the pathophysiologic mechanisms that produce tinnitus, with the hope that improved knowledge will be translated into effective therapies.

**CONCLUSIONS**

In the clinical approach to patients with tinnitus, it is essential to determine through a comprehensive history and physical examination whether patients have objective or subjective tinnitus. Among patients with objective tinnitus, particularly those with pulsatile tinnitus, radiologic evaluations are likely to identify the source of the sound and indicate a course of action. Among patients with subjective tinnitus, audiologic assessment, including electrophysiologic tests, helps differentiate patients with retrocochlear lesions who require radiologic evaluation from patients with cochlear disease. Radiologic testing in patients with subjective tinnitus should be focused on determining the cause of hearing loss.

**ACKNOWLEDGMENTS**

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**REFERENCES**

EDITORIAL COMMENTARY

Wackym and Friedland describe the bedside neurotologic examination essential in the evaluation of the patient with tinnitus. They call attention to the important role of vestibular tests in the examination, particularly cerebellar tests and the head thrust and shake, Valsalva’s maneuvers, and hyperventilation, and they outline the findings that lead to the audiologic assessment, electrophysiologic tests, and imaging necessary for a definitive diagnosis. The importance of auscultation of the head and neck in pulsatile tinnitus and palatal myoclonus, stapedial or tensor tympani muscle spasm, and the patulous eustachian tube is noted, as well as the need for direct observation of the oropharynx and nasopharynx in myoclonic tinnitus.

Wackym and Friedland point out that although the combination of pulsatile tinnitus and conductive hearing loss is most commonly due to eustachian tube dysfunction, it may be due to a middle fossa encephalocele or dehiscence of the jugular bulb. They emphasize computed tomography for the diagnostic erosion of the jugular foramen caused by glomus jugulare tumors and magnetic resonance imaging for posterior fossa lesions.

Henry emphasizes that tinnitus does not share the same psychophysical characteristics as external acoustic stimuli. Most notably, masking of tinnitus differs from the masking of sound in that frequency dependence in tone-on-tone masking is usually not found in tinnitus. Interestingly, he points out that residual inhibition occurs in 80 to 90% of patients who receive appropriate acoustic stimulation.

Although great individual variation limits generalizations about the psychoacoustic profile of tinnitus, Henry calls attention to the fact that in large populations of patients with tinnitus, 75% have pitch matches above 4 kHz and 55% have pitch matches above 6 kHz. Patients with tinnitus can generally provide repeat loudness matches within a few decibels, but repeated pitch matching tends to produce variance over several octaves. He calls attention to the as yet unexplained merging and parallel categories of patients with tinnitus on the basis of their loudness match functions compared with their hearing levels across frequencies.

Henry has been working and continues to work on computer automation of tinnitus testing procedures, in part to achieve much needed standardization of tinnitus assessment.

Newman and Sandridge summarize the currently most used questionnaires for assessing the qualitative and quantitative nature of tinnitus and point out that qualitative questionnaires have formed the basis for items in the quantitative questionnaires. The effects of tinnitus on sleep and hearing are more frequently found through questionnaires than emotional problems and the effects on one’s general health. Quantitative questionnaires are necessary because psychoacoustic measures do not fully characterize the impact of tinnitus on the individual. Quantitative questionnaires can serve as outcome measures in before and after treatment comparisons for an individual patient or in clinical trials. The Tinnitus Handicap Inventory, the Tinnitus Handicap Questionnaire, and the Tinnitus Reaction Questionnaire have the test–retest reliability over short time intervals to serve as outcome measures, at least for therapy requiring short intervals. Newman and Sandridge call attention to the need for an internationally recognized minimal outcome measure for clinical trials and other research on tinnitus. Indeed, such a standard would be very welcome.

Lockwood and colleagues review the progress with positron emission tomography (PET) and functional magnetic resonance imaging in imaging evidence of tinnitus in various parts of the brain. PET has the advantages of producing almost no noise and being relatively insensitive to small movements, such as those from arterial pulsations in the brainstem. They used subtraction techniques to good advantage in patients with tinnitus who can alter the tinnitus with jaw clenching and patients with gaze-evoked tinnitus and by administering intravenous lidocaine to subjects with tinnitus. They demonstrated unilateral changes in the neural activity associated with changes in tinnitus loudness.

Lockwood and colleagues point out that with magnetoencephalography, Muhlnickel and colleagues have demonstrated displacement of the site responsive to a tone, the frequency of which was matched to the frequency of the tinnitus, providing additional evidence of the reorganization of the central auditory pathways with tinnitus.

James B. Snow Jr