
DIAGNOSTIC AUDIOLOGY

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1991

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CHAPTER 8

Diagnosis of Middle Ear Pathology and Evaluation of Conductive Hearing Loss

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For the majority of patients, otologic pathology producing conductive hearing impairment (external and middle ear abnormalities) can be conclusively diagnosed with a good history and careful physical examination. Audiometry in these cases, in particular the comparison of air versus bone conduction pure tone thresholds, quantifies degree of suspected hearing impairment. Other procedures, such as immittance audiometry, serve to confirm the otologic diagnosis but do not provide new or essential clinical information. This application of audiometry is by no means unimportant and still may contribute to decisions regarding surgical or medical management, especially when the physical examination reveals no obvious abnormality. Probably the most important example of this latter application is differentiation of stapes fixation (as in otosclerosis) versus disruption of the ossicular chain, and quantification of the resulting conductive hearing impairment, by the pattern of findings for pure tone and immittance audiometry (illustrated in cases to follow).

Clinical experience suggests six other, somewhat unique, contributions of audiometry in patients with external/middle ear pathology. These are (1) screening for middle ear dysfunction in children by nonphysicians (Harford, Bess, Bluestone, and Klein, 1978; Holte & Margolis, 1987), (2) detection of middle ear dysfunction in newborn infants or uncooperative, difficult-to-examine children by nonotologists (Paradise, Smith, and Bluestone, 1976), (3) documenting pre- versus posttherapy (surgical or medical) changes in auditory status (Wehrs, 1976), (4) describing the mechanical dysfunction caused by middle ear pathology (Jerger, 1970; Jerger, Anthony, Jerger,

and Mauldin, 1974), (5) evaluation of auditory function in comatose patients who cannot provide a history or respond behaviorally for clinical techniques such as tuning fork tests (Hall, 1989; Hall et al., 1982), and (6) providing otherwise unavailable ear-specific information on sensory integrity in persons with severe bilateral apparently conductive hearing impairment, such as congenital aural atresia (Hall et al., 1986; Jahrsdoerfer and Hall, 1986; Jahrsdoerfer, Yeakley, Hall, Robbins, and Gray, 1985). The first four of these contributions rely almost entirely on a comprehensive battery of immittance measures (tympanometry for low and high probe-tone frequencies, ipsilateral and contralateral acoustic reflexes). The final two contributions require assessment with auditory evoked responses in addition, perhaps, to immittance measures. Both confirmatory and diagnostic applications of audiometry will be discussed here, but the emphasis will be on the latter.

Three general areas of knowledge are fundamental to consistently successful evaluation of conductive hearing impairment but are beyond the scope of this chapter. The first is an understanding of external/middle ear anatomy and physiology. This topic is addressed in greater detail in Chapters 4 and 5; however, a brief description of the salient features of external/middle ear function is presented before reviewing the auditory effects of dysfunction of this important portion of the hearing mechanism. The pinna, by virtue of its orientation (facing forward) and morphology (folds and depressions), modulates sounds, mostly in the high-frequency region, and thereby creates acoustic cues that facilitate localization.

The external ear canal protects the ear (by its length and cerumen), offers a channel for passage of sound to the middle ear, and also amplifies, via resonance effects, acoustic energy at approximately 3000 Hz.

The two major and related functions of the middle ear (tympanic membrane and ossicles) are to match low impedance of the vibrations in the air of the external ear canal to the relative high impedance of movement of cochlear fluids. Impedance matching and resultant amplification are accomplished in three well-known ways: (1) an intricate buckling movement of the tympanic membrane (amplification $\times 4$), (2) a lever action created by greater length of the manubrium of the malleus than the incus (amplification $\times 1.3$), and (3) most important, the ratio of area of the relatively large tympanic membrane to the relatively small stapes footplate (amplification $\times 35$). By means of these three properties, the middle ear functions as an acoustic impedance transformer and increases the amount of force transmitted from the external ear canal to the middle ear/inner ear junction (stapes footplate). This amplification is most pronounced in the auditory frequency region from 800 to 2000 Hz. Conductive hearing impairment resulting from external/middle ear pathology, therefore, may be practically defined as failure to collect or transmit sound energy from the environment to the organ of Corti. It is primarily a breakdown in the impedance matching mechanism somewhere between airborne sound arriving at the head and sound-related movement of cochlear fluid. Etiologies for this breakdown, however, are extremely varied, ranging from complete absence of the external ear and ear canal to the more subtle pathology of otosclerosis.

Second, a firm grasp of the acoustic immittance principles and practices is vital for maximum efficiency and effectiveness of audiometry in evaluation of conductive hearing impairment. Current concepts of immittance measurement are reviewed in Chapter 6 and also in several recent publications (Hall, 1985, 1987; Shanks, 1984; Van Camp, Margolis, Wilson, Creten, and Shanks, 1986).

Finally, the clinician must fully appreciate the complexities of air versus bone conduction pure tone audiometry in patients with suspected conductive hearing impairment. In the development of the audiology profession from about 1950 to 1965, pure tone techniques were essentially the only means available for audiometric evaluation of conductive hearing impairment. Immittance

measurement was not yet clinically feasible or commercially available, and measurement of auditory evoked responses, namely electrocochleography (ECoChG), was invasive and extremely limited in clinical application. It is, therefore, not surprising that this period produced a host of classic reports of air versus bone conduction audiometric findings in conductive hearing impairment. Among the important issues studied were the effect of procedural factors (Dirks, 1964; Hood, 1960; Jerger and Wertz, 1959; Studebaker, 1967; Ventry, Chaiklin, and Boyle, 1961), interaural attenuation (Zwislocki, 1953), masking (Dirks and Malmquist, 1964; Sanders and Rintelmann, 1964; Studebaker, 1962), the masking dilemma (Naunton, 1960), mechanical effects of middle ear pathology on cochlear function (Bekesy, 1960; Carhart, 1950; Huizing, 1960; Tonnendorf, 1964), the occlusion effect (Goldstein and Hayes, 1965), and the sensory acuity level (SAL) test. Contemporary clinicians will find the information provided in these publications to be as useful today as their predecessors did over 20 years ago when they were published.

This chapter continues with a discussion of the differential diagnosis of external/middle ear pathology from the otologist's perspective. Then essential clinical procedures for comprehensive audiometric evaluation of external/middle ear dysfunction and conductive hearing impairment are summarized. This is followed by a review of typical patterns of audiometric findings in varied pathologies, along with brief reports of more challenging cases illustrating application of pure tone, immittance, and auditory brainstem response (ABR) audiometry in evaluation of middle ear pathology. The chapter concludes with guidelines for efficient and effective audiometric evaluation of conductive hearing impairment using the wide array of behavioral and electrophysiologic auditory procedures currently available. A glossary of audiologic and medical terminology is appended.

CLINICAL EXAMINATION

A diagnosis is based on the patient's history, symptoms, and clinical signs, and the results of laboratory tests and special diagnostic procedures (including audiometric data). Differential diagnosis is a comparison of this information among diseases that the patient *might* have in order to determine which disease the patient *does* have. The purpose for establishing a diagnosis is to provide

a rational basis for selecting appropriate treatment and to estimate prognosis.

HISTORY

When a patient presents with the complaint of hearing loss or fullness in the ear, he or she should be questioned about other pertinent symptoms that may be valuable in reaching a diagnosis. For example, if there is a sensation of fullness, is it unilateral or in both ears? When did it start? Was it preceded by a common cold? Was it first noticed after flying, swimming, diving, or taking a shower? Did the patient "clean" the ear with a cotton swab just before the fullness became apparent? Are there other symptoms, such as earache, itching, tinnitus, discharge (of fluid from the ear), or fever? Did the symptoms start during pregnancy? Has the patient had previous surgery on the ear? Was there exposure to loud sounds or blasts, or any use of firearms? Has the patient taken any potentially ototoxic drugs? In order to consistently obtain an adequate history, the clinician must take the necessary time to put the patient at ease, and ask the appropriate questions. A good history is the first major step toward an accurate diagnosis of external/middle ear disease.

PHYSICAL DIAGNOSIS

Inspection of the External (Outer) Ear

The diagnosis can sometimes be made simply by closely viewing the external ear. Diseases involving the external ear are summarized in Table 8-1. An *absent auricle* (anotia) or a small, malformed auricle (microtia) often leads to the diagnosis of congenital disease of the ear. The presence of other associated maxillofacial deformities may allow the clinician to identify a syndrome. Likewise, obvious bleeding, lacerations, contusions, hematomas, or burns of the auricle and surrounding structures are evidence of *trauma*. More specifically, Battle's sign (bluish postauricular discoloration or ecchymosis) indicates a temporal bone fracture. Temporal bone fracture, as we will demonstrate, may be associated with varied types and degrees of hearing impairment. Facial nerve paresis or paralysis, a possible component of temporal bone fracture, is easily recognized when the patient is conscious but is frequently overlooked in comatose or paralyzed patients.

Redness, swelling, loss of auricular convolutions, weeping auricular skin, and sometimes dis-

TABLE 8-1. DISEASES OF THE EXTERNAL AUDITORY CANAL IN DIFFERENTIAL DIAGNOSIS

<i>Congenital</i>	<i>Acquired</i>
Atresia	Cerumen impaction
Collapsing external canal	Foreign body
	Infections
	Bacterial otitis externa
	Fungal (otomycosis)
	Parasitic
	Granulomas or polyps, or both
	Osteomas or exostoses
	Benign tumors
	Traumatic lesions
	Burns
	Hematomas
	Lacerations
	Fractures
	Scarring

charge or residual crusting are signs of a *diffuse external otitis or perichondritis* of the auricle, which may in turn cause collapse of the external ear canal. Diffuse redness and lateral displacement of the auricle, with or without purulent discharge from the meatus, with obliteration of the postauricular sulcus and generalized postauricular edema, are signs of acute mastoiditis. Swelling and redness over the sternocleidomastoid region indicate a Bezold's abscess. *Tumors of the auricle* that produce hearing impairment are generally large enough to be easily detected by physical inspection. Finally, the clinician should inspect visually for postauricular and endaural (within the meatus of the ear canal) incision scars that would suggest the nature of previous surgery.

Otoscopy of the Outer Ear

Atresia of the external auditory canal (EAC) is easily recognized. It may be associated with auricular deformities, preauricular skin tags, or a perfectly normal auricle. EAC *stenosis* with normal skin lining the canal is a congenital deformity that should be distinguished from a collapsing meatus. The latter consists of a slitlike meatus that opens on gentle posterosuperior retraction of the auricle and allows the examiner to insert the otoscope speculum. The remainder of the ear is usually normal. This condition is fairly common in elderly patients or in those who have undergone surgery via a postauricular incision. An unusually large meatus leading to a posterior cavity is evidence of previous surgery (radical or modified radical mastoidectomy).

Impacted cerumen (ear wax) is probably the most common cause of conductive hearing impairment

It is also the most annoying physical finding encountered by the otologist or audiologist because its removal is time consuming and requires a great deal of dexterity, particularly when it is found in children. Freshly secreted cerumen is soft and yellow. Cerumen becomes harder and turns a dark brown or black color if allowed to remain in the ear canal over time. It may appear dry and flaky and become covered with hair and dandruff, particularly in elderly men. On the other hand, cerumen may appear soft and creamy after swimming or bathing. Cerumen is only secreted in the cartilaginous (outer, hair-bearing) portion of the ear canal and normally tends to migrate and fall out of the canal spontaneously. Any cerumen accumulation in the bony (innermost) portion of the ear canal usually is due to the patient "cleaning" the ear with a cotton swab or some other equally inappropriate object. Patients often ask what they should do about earwax when they are advised not to clean their ears with, for example, a Q-tip. For these patients, it is safe to recommend insertion of a few drops of baby oil.

Foreign bodies in the EAC are common in children but are also observed in adults. Children are notorious for inserting beads, pebbles, milk teeth, and parts of toys in their ears. With adults, however, the object is most often left in the ear canal following insertion of a softer foreign body (cotton tip of a swab, paper tissue, detached pencil eraser) or the plastic cap of a ball point pen, as the patient attempts to scratch or clean the ear canal.

Osteomas or exostoses are occasionally seen in the ear canal during otoscopy. They are covered with thin, shiny, smooth skin and are located deep inside the bony portion of the canal. In some cases, an exostosis is large enough to occlude the ear canal lumen (opening). *Acute otitis externa* presents in the form of a boil or abscess in the hair-bearing skin of the outer portion of the EAC. When it is diffuse, the entire canal is swollen, red, wet, and extremely tender on moving the auricle or pressing on the tragus. Insertion of the otoscope into the meatus causes the patient severe pain. The tympanic membrane, when it can be visualized, usually appears normal. *Otomycosis* is diagnosed when a mold is found in the ear canal. Filaments and spores may also be present. Discharge is scanty.

Otoscopy of the Middle Ear

Middle ear pathologies are listed in Table 8-2. In *acute bacterial otitis media*, the EAC is normal

TABLE 8-2. DISEASES OF THE MIDDLE EAR IN DIFFERENTIAL DIAGNOSIS

CONGENITAL
Absence or malformation of the ossicular chain
ACQUIRED
Infections and their sequelae
Serous otitis media (acute or chronic)
Purulent otitis media (acute or chronic)
Tympanic membrane perforation
Erosion or necrosis of lenticular process of incus
Polyps
Cholesteatoma
Tympanosclerosis
Atelectasis of middle ear space
Barotrauma
Trauma
Insertion of foreign object or manipulation resulting in ossicular chain disruption or tympanic membrane perforation, or both
Iatrogenic
Blast injuries
Temporal bone fractures resulting in hemotympanum, ossicular disruption, cerebrospinal fluid otorrhea
Burns
Tumors
Benign (adenomas, glomus tympanicum)
Malignant
Granulomas
DISEASES OF THE OTIC CAPSULE
Otosclerosis

with no tenderness when the auricle is manipulated or when the speculum is inserted. With otoscopic inspection, the eardrum appears red and is sometimes bulging. Blood vessels are seen coursing over the manubrium. At a later stage, blood vessels become prominent over the entire tympanic membrane. The usual landmarks of the eardrum (the umbo and the light reflex) are lost. If untreated, the tympanic membrane may rupture, allowing pus to flow out. The tear in the eardrum is usually small and the purulent discharge is occasionally pulsatile. As the disease progresses, there is less discharge, and within days after the spontaneous rupture, the tear in the eardrum closes. At this stage, the eardrum is thinner and there may still be an air-fluid level in the middle ear.

The onset in *acute nonsuppurative otitis media* is sudden, and the tympanic membrane is thin, bluish, and retracted. Bubbles or air-fluid levels are readily visible through the eardrum. *Barotrauma*, caused by sudden changes in ambient pressure (associated with decompression, diving, and flying), is almost identical to this condition. *Chronic*

serous otitis media (glue ear) is characterized by a bluish-grey or yellowish tympanic membrane, which is thick, opaque, and, in some cases, retracted.

Chronic purulent otitis media (chronic ear) is an infected middle ear space that drains through a tympanic membrane perforation. The perforation is usually large but could be as small as a pin point. The perforation edges are well defined, smooth, and occasionally slightly thickened. The layer of tympanic membrane skin curls inward along the perforation edge to blend with the medial mucosal lining of the tympanic membrane. Middle ear mucosa is hyperemic, thickened, and wet. Pus may be observed in the most dependent area of the middle ear. Red fleshy polyps occasionally emerge from the perforation. At times, they are so large as to occlude the entire external canal. These polyps are wet, foul smelling, and bleed easily. Removing the polyps by simply pulling on them is ill advised since they may be attached to the ossicles.

A *dry tympanic membrane perforation* is easily recognized. It almost always occurs in isolation and in the pars tensa portion of the tympanic membrane. The perforation may be central, marginal, or total. The round window niche is frequently observed, and in some cases the stapes, stapedius tendon, and incudostapedial joint are visible. The manubrium of the malleus may also be adherent to the promontory. The perforation edges are well defined and smooth. *Tuberculous otitis media* should be suspected if multiple perforations are present.

Perforations of the pars flaccida suggest the possibility of *cholesteatomas*, particularly if shiny skin appears to grow into the middle ear space. This ingrowth may appear as a simple pocket or at times as a tract leading medially and superiorly toward the attic. This invagination or tract could be lined with shiny, pearly white flakes and may be filled with keratin debris and foul-smelling necrotic material and pus. In a large cholesteatoma, the normal anatomy of the eardrum may be completely replaced by these white pearly flakes and keratin debris. Polyps and pus are often present, and the foul smell is overbearing.

Tympanosclerosis (adhesive otitis media) is the result of long-standing middle ear infection. It is characterized by hyaline degeneration of the tympanic membrane and middle ear space, which leads to calcification and sometimes new bone formation. Such calcium deposits may be seen as white plaques in the substance of the eardrum. The eardrum may be extremely thin, transparent, and so severely retracted that it is wrapped around the ossicles. The retraction could easily be mis-

taken for a large perforation when it is plastered to the promontory. This is referred to as *atelectasis of the middle ear*.

Trauma to the middle ear can cause the eardrum to rupture, resulting in an irregular or stellate perforation and fresh blood in the ear canal and middle ear space (*hemotympanum*). This pattern of findings is usually the result of insertion of a foreign body accidentally into the ear (cotton swab or bobby pin), or it may be iatrogenic, secondary to attempted removal of cerumen or foreign bodies from the EAC. Blasts can produce the same clinical picture and associated inner ear symptomatology. A slap to the ear can trap air under pressure in the ear canal and cause the tympanic membrane to rupture. Occasionally, the tympanic membrane remains intact but the ossicular chain becomes disrupted.

Longitudinal fractures of the temporal bone produce a laceration of the EAC skin with fresh bleeding, exposure of bony fragments, tears in the tympanic membrane, and occasionally cerebrospinal fluid leak. When the eardrum is intact, or after it heals, hemotympanum is manifested by a blue, opaque discoloration of the tympanic membrane. Other signs that may be present are facial nerve paralysis, nystagmus, and ataxia. Many of these patients may be comatose and may have other severe brain injuries. Evidence of dysfunction in longitudinal fractures may be first discovered by the otologist or audiologist during an auditory evoked response assessment. It is imperative to remember that such ear injuries are potential ports of contamination to the brain and can lead to meningitis. Therefore, extreme care should be taken in cleaning blood and debris from the EAC, and sterile instruments and technique must be used during the procedure. Some otologists prefer to defer the cleaning procedure for a week or two so that healing of the tympanic membrane and lacerations can occur first. Under no circumstances should the ear be irrigated or a caloric test (ice water calorics or electronystagmography) be performed until the lacerations and tympanic membrane have completely healed. Also, conductive hearing impairment in these patients should be evaluated audiometrically only after resorption of the hemotympanum.

Tumors of the EAC are occasionally mistaken for polyps. They are fleshy, red, bleed easily, and may be associated with chronic otitis media and purulent discharge. Pulsating red masses observed behind the eardrum are usually *glomus tympanicum tumors*.

Finally, *otosclerosis*, a disease of the otic capsule, may produce a physical sign that can be observed with the otoscope. *Schwartz's sign* is a reddish discoloration of the eardrum secondary to an active otosclerotic focus that causes the blood vessels of the promontory to dilate and engorge with blood.

Additional Diagnostic Procedures

For the majority of patients, a diagnosis is made from the history and physical examination. Diseases of the external ear are obvious and easily diagnosed. Difficulties arise when the differential diagnosis narrows down to certain conditions that require additional clinical information. An example of this difficulty is diffuse otitis externa with a tender postauricular lymph node versus swollen purulent ear versus an acute mastoiditis. In such conditions, radiograms of the mastoids and possibly audiologic evaluation, when feasible, help the clinician in reaching a diagnosis.

Other clinical entities exist for which information from the history and physical examination does not consistently lead to a diagnosis. For example, the eardrum may appear normal, even for an experienced clinician, yet tympanometry may indicate middle ear effusion. Cholesteatoma may bridge the ossicular chain and mask a disruption in the ossicular chain by conducting sound waves from the tympanic membrane to the inner ear. Such patients may have little or no conductive hearing impairment with the cholesteatoma in place, but with its removal there may be a conductive hearing impairment of up to 50 dB HL. The same difficulty in correctly diagnosing middle ear disease may be present for the patient with a normal eardrum and an ossicular chain disruption. The most frequent lesion causing this finding is necrosis of the lenticular process of the incus following chronic middle ear disease. A serious (50–60 dB HL) conductive hearing impairment accompanies the disease. When the eardrum is ruptured, the clinician may be able to visualize the pathology.

High-resolution computed tomography is useful in the description of otologic pathology in temporal bone fracture, including definition of ossicular chain disruption. Finally, with chronic adhesive otitis media, the eardrum is so thin and retracted that the contour of the eroded incus or incudostapedial joint may be perceived through the membrane. Occasionally, the long process of the incus is completely eroded and the eardrum wraps around the capitulum of the stapes in what

appears to be a natural tympanoplasty type III effect. Such patients may have normal hearing.

AUDIOLOGIC ASSESSMENT

AURAL IMMITTANCE

Immittance measurement at the outset of an audiometric assessment is the most effective means of differentiating conductive versus sensory hearing impairment, as shown schematically in Figure 8-1. In 1970, Jerger observed that "impedance audiometry represents an invaluable diagnostic tool in clinical audiology. It has become, in our clinic, a routine part of the audiologic assessment of every patient. We frankly wonder how we ever got along without it" (Jerger, 1970, p. 322). Almost 20 years later, immittance measures remain invaluable and a routine component of audiologic assessment.

Rigorous definition of normal immittance findings is crucial for successful application of immittance measures in ruling out middle ear pathology. These normal immittance criteria that must be rigidly adhered to are (1) ear canal volume within normal limits, (2) normal type A tympanograms (not A_s or A_d) bilaterally, and (3) acoustic reflexes clearly and consistently present at expected stimulus levels (95 dB HL or better) bilaterally for both ipsilateral (uncrossed) and contralateral (crossed) conditions. With these immittance findings and pure tone audiometry evidence of hearing impairment, the deficit is almost always considered to be sensory and subsequent audiometric assessment directed toward differentiation of cochlear versus retrocochlear dysfunction, and complete definition of auditory status for one or the other site of lesion (see Chapters 9, 10, and 11). Measurement of acoustic reflex decay is a standard component of the immittance battery and, therefore, the first procedure administered for differentiation of sensory versus neural auditory dysfunction. *Bone conduction pure tone measurement in patients with unequivocally normal immittance findings (with the exception of abnormal acoustic reflex decay) is generally superfluous, that is, noncontributory toward the diagnosis or description of the hearing impairment, and a waste of valuable clinical time.*

Of course, this normal acoustic immittance pattern occurring in combination with normal pure tone hearing sensitivity does not necessarily imply normal function of the peripheral and cen-

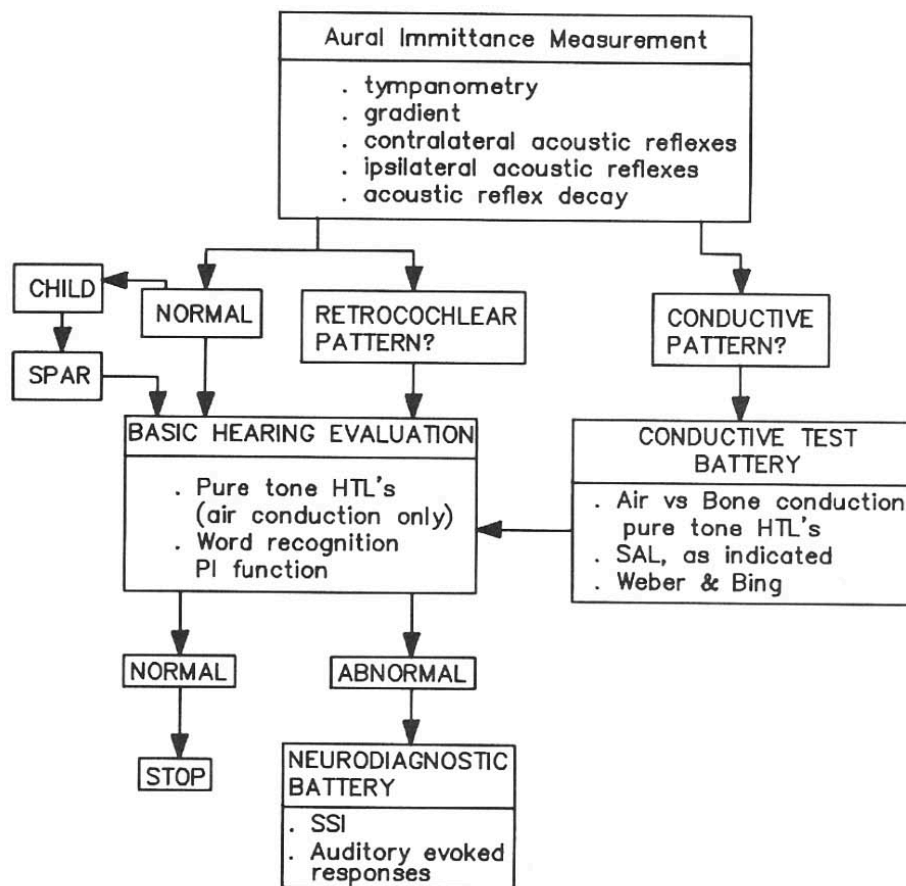


Figure 8-1. Audiologic test strategy for evaluation of conductive hearing impairment.

tral auditory system. With this combination of results, especially for patients with auditory complaints (e.g., difficulty understanding speech under adverse listening conditions), a history of central nervous system disease or insult (e.g., head injury, stroke, multiple sclerosis), or neurologic and neurotologic signs or symptoms (e.g., dizziness, tinnitus), there is a high index of suspicion for central auditory nervous dysfunction. More extensive analysis of acoustic reflex activity (Hall, 1983; Stach and Jerger, 1987) plus diagnostic speech audiometry (Hall, 1984) and assessment of auditory evoked responses (Hall, 1990) are the procedures of choice in these patients. The following general remarks will be limited to clinical evaluation of patients with suspected external/middle ear pathology and associated conductive or mixed hearing impairment. Specific patterns of audiometric findings for patients with various middle ear pathologies will be presented and discussed in a subsequent section.

Since Jerger published his classic paper in 1970 on clinical immittance audiometry experiences with over 400 patients, the unique diagnostic

value of immittance audiometry has been repeatedly reported in the literature and demonstrated clinically (Jerger, 1970; Jerger and Hayes, 1976; Jerger et al., 1974; Jerger, Jerger, and Mauldin, 1972). Analysis of acoustic immittance patterns is the most sensitive audiometric means of identifying middle ear dysfunction and, in addition, of differentiating among mechanical bases of this dysfunction. "Individually . . . each measure has serious limitations. In combination, however, they yield patterns of great diagnostic value" (Jerger, 1970, p. 322). There are numerous detailed descriptions of correlations among immittance patterns and types of underlying middle ear pathology. What follows herein is a summary of the salient immittance findings for types of pathology encountered most often clinically. In Table 8-3, characteristic audiometric patterns are summarized for commonly encountered types of otologic pathology.

Tympanometry

Major tympanogram types according to the Jerger (1970) classification system are illustrated in Figure 8-2. Other approaches for tympanogram analysis have been proposed (Bluestone, Berry,

TABLE 8-3. SUMMARY OF GENERAL PATTERNS OF AUDIOLOGIC FINDINGS ASSOCIATED WITH MIDDLE EAR PATHOLOGIES

Pathology	Audiologic procedure		
	Pure tone audiometry (db HL)	Tympanometry*	Acoustic stapedial reflexes
Serous otitis media	20-30 dB rising CHL	Type C or B	Absent
Acute otitis media	30-50 dB flat CHL	Type B	Absent
Early otosclerosis	Normal hearing to <20 dB CHL	Type A _s	Reverse direction
Otosclerosis	25-60 dB rising CHL or MHL; Carhart's notch	Type A _s	Absent
Complete ossicular chain discontinuity	60 dB flat or sloping CHL	Type A _d	Absent
Ossicular chain discontinuity with functional connection	30-40 dB flat or sloping CHL	Type A _d	May be present
Tympanic membrane perforation: peripheral	Within normal limits	Abnormally large EAC volume	CNE
Tympanic membrane perforation: central	Normal to 25 dB rising CHL	Abnormally large EAC volume	CNE
Tympanic membrane ventilation tube (patent)	Within normal limits	Abnormally large EAC volume	CNE
Cholesteatoma	25-60 dB CHL	Variable	Usually absent
Aural atresia	>60 dB flat or rising MHL or CHL	CNE	CNE

CHL, conductive hearing loss; MHL, mixed hearing loss; EAC, external auditory canal; CNE, cannot evaluate.

* Tympanogram types are described in text and Figure 8-2.

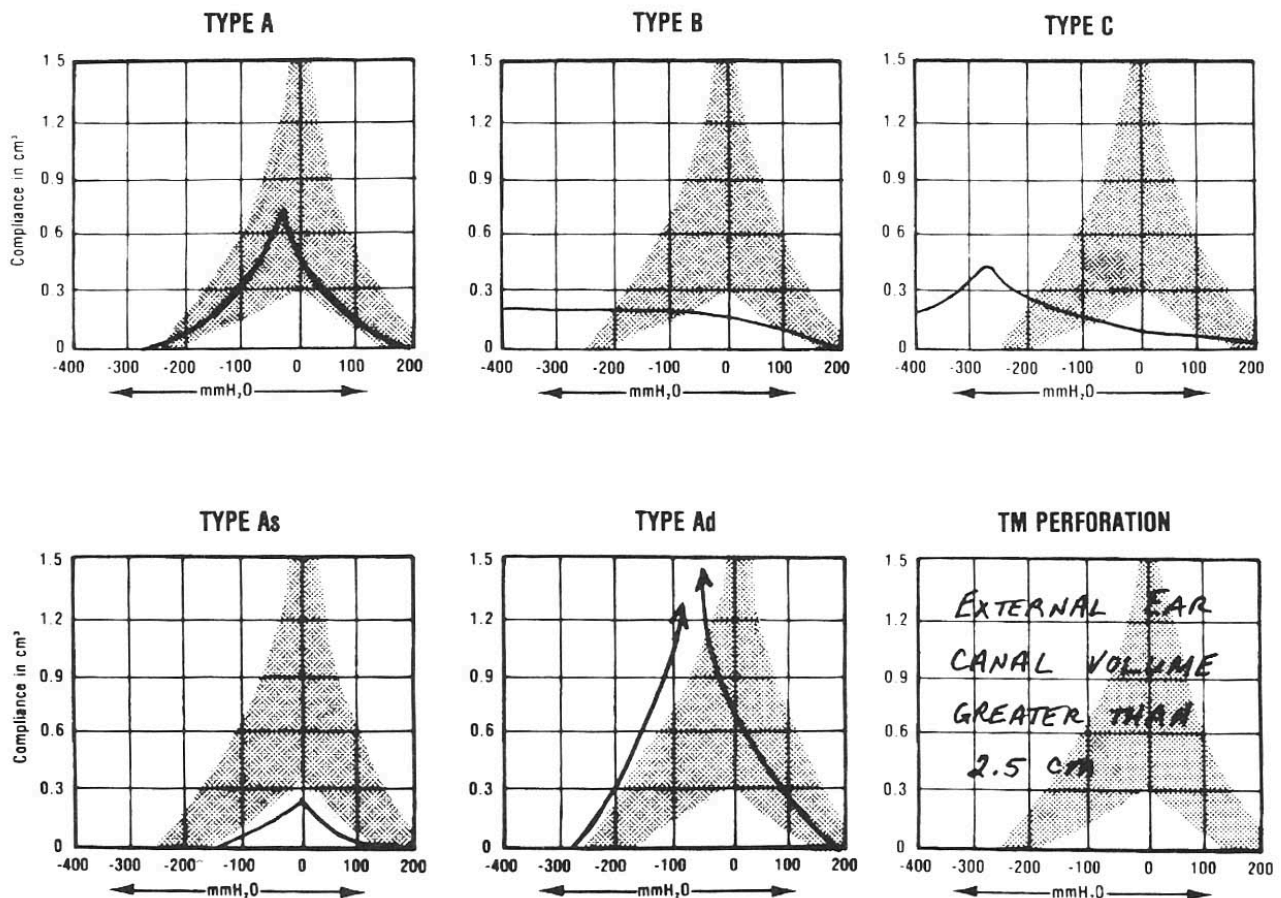


Figure 8-2. Clinically popular tympanogram classification system proposed by Jerger (1970).

and Paradise, 1973; Cooper, Hearne, and Gates, 1982; Feldman, 1976; Van Camp et al., 1986). Among these, the clinical use of multifrequency (e.g., 660 versus 220 Hz probe tone) and multi-component (e.g., susceptance and conductance versus admittance) tympanometry has been consistently endorsed in the United States and Europe. Some evidence exists that multiple-frequency, multicomponent, and phase-angle tympanometry offer diagnostically useful information in a rather selective category of patients with low-impedance (high-admittance or -compliance) middle ear pathology, such as ossicular chain disruption (Van Camp et al., 1986); however, findings from the combination of a complete medical history, otologic examination, routine immittance measurement, and pure tone audiometry rarely fail to confirm accurate diagnosis of this pathology, as will be demonstrated soon with actual cases. Probably as a consequence, Jerger's relatively simple tympanogram classification schema continues to be the most widely accepted clinically.

The clinician applying this system is advised to follow the test protocol originally described by Jerger, specifically plotting the tympanogram for an approximately 220 Hz probe-tone frequency as air pressure in the EAC is varied in a positive-to-negative direction. It is possible for tympanogram interpretation by the Jerger criteria to be invalidated if the other protocols are used. Probe-tone frequency, the rate and direction of ear canal pressure change, and, of course, the property measured (admittance, susceptance, or conductance) exert clinically important influences on tympanogram measurement (Shanks and Wilson, 1986; Van Camp et al., 1986; Wilson, Shanks, and Kaplan, 1984). It would, therefore, be unwise to routinely record tympanograms with a test parameter not employed by Jerger et al., such as a negative-to-positive versus positive-to-negative ear canal pressure change, and then to interpret the results with their classification system.

Acoustic Reflexes

Acoustic reflexes are usually not observed when recorded with the measuring probe in an ear with middle ear pathology because ossicular chain or tympanic membrane abnormalities obscure detection at the eardrum of the relatively small alterations in middle ear immittance that occur with stapedial muscle contraction. (See Hall, 1984, for a review of acoustic reflex anatomy and physiology.) Acoustic reflexes are almost

invariably abnormal for the cases in which they are present. These include minor middle ear pathology, early fixation of the ossicular chain, and, rarely, disruption of the ossicular chain with some type of functional connection between tympanic membrane and stapes. With even subtle middle ear dysfunction in the probe ear, such as the negative pressure associated with eustachian tube obstruction and early serous otitis media, acoustic reflex thresholds are markedly elevated (intensity levels of 95 dB HL or greater are required to elicit reflex activity). Early fixation of the ossicular chain characteristically produces an unusual acoustic reflex waveform. With stimulus presentation, a curious decrease occurs in middle ear impedance (referred to as *negative deflection* of the meter and X-Y plotter pen), rather than the usual increase in impedance (or stiffness). In these cases, hearing sensitivity may still be normal and there may be little or no air-bone gap. If the gap between the stapes and a more lateral portion of the middle ear system (e.g., tympanic membrane or malleus) is bridged in some atypical way, patients with ossicular chain disruption may show acoustic reflex activity. The fortuitous ossicular connection can be made with fibrous adhesions or other material, which serve to transmit stapedius muscle-related changes in middle ear immittance from the stapes to the tympanic membrane. Despite this functional connection, there is usually a maximum (60 dB HL) conductive component to the hearing impairment. As an aside, patients with ossicular chain disruption and other concomitant middle ear pathology, such as cholesteatoma or fluid, may have less hearing impairment because there is a means for mechanical transmission from eardrum to stapes footplate.

PURE TONE AUDIOMETRY

Comparison of hearing threshold levels for air-versus bone-conducted pure tone signals — measurement of the air-bone gap — is the traditional approach for audiometric description of conductive hearing loss associated with middle ear pathology. As noted earlier, the many technical issues of air versus bone conduction pure tone audiometry, among them placement of the bone vibrator, masking criteria and methods, and calibration, are fully addressed in hundreds of articles and scores of basic audiology textbook chapters and will not be reviewed here.

Acoustic Impedance Versus Audiogram Configuration

One aspect of pure tone audiometry in conductive hearing loss that is rarely noted, but diagnostically useful, is the relationship between middle ear pathology and audiogram configuration. Over 40 years ago, long before impedance measurement became incorporated into the basic audiometric test, Johansen (1948) described the hearing loss configurations that were correlated with different components of middle ear impedance. The acoustic impedance components, namely resistance (friction), reactance (mass and stiffness, or its reciprocal compliance), and their dependence on frequency of sound, are now familiar to audiologists. The audiometric correlates are illustrated in Figure 8-3. Resistance abnormalities, which are not common, affect a wide range of mid frequencies in the audiogram. The effect of a mass abnormality is decreased high-frequency hearing sensitivity, whereas abnormally increased stiffness, most commonly found clinically, produces a low-frequency hearing deficit. Thus, a simple inspection of air conduction pure tone audiometry configurations can provide some indica-

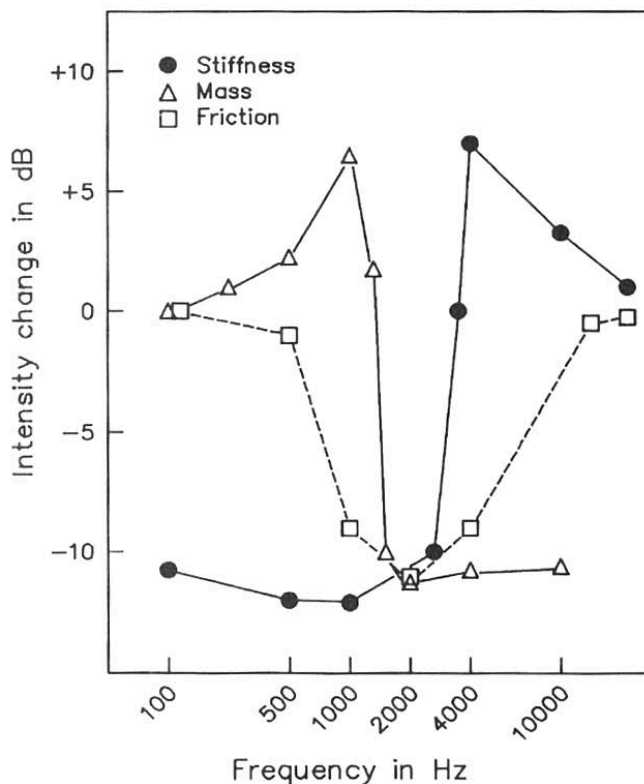
tion of underlying middle ear pathology and confirm otologic and immittance findings.

Masking Dilemma

One of the most challenging problems in clinical audiology is the *masking dilemma* (Jerger and Wertz, 1959; Naunton, 1960), which is presented most often by a person with a moderate to severe bilateral, apparently conductive hearing loss. This audiometric pattern is illustrated with a forthcoming case report of congenital atresia (case 17). The dilemma is that the intensity level of masking noise necessary to effectively mask the non-test ear (e.g., overcome the conductive component on the non-test ear and reach the cochlea at an intensity level about 10 dB higher than any crossed-over test signal) exceeds interaural attenuation (minimally 40–50 dB) and can cross over (via bone conduction) to mask hearing on the test ear. The most vivid demonstration of this potential problem is provided by unmasked air versus bone conduction findings for patients with a total unilateral sensory hearing impairment (a "dead ear"). Unmasked air conduction thresholds are usually in the 50 to 60 dB range, and bone conduction thresholds (obtained with the bone vibrator on the mastoid of the ear in question) may be in the 10 to 20 dB range (Chaiklin, 1967). Both air and bone conduction thresholds in the dead ear are due to cross over to the normal non-test ear.

Three simple techniques can help to resolve this dilemma in some cases. The first two are the audiometric Weber and Bing tests. If a patient with bilaterally symmetric and apparently conductive hearing impairment consistently lateralizes low-frequency pure tone signals to one ear (the Weber test) or shows no occlusion effect (Goldstein and Hayes, 1965) for these signals on one ear (the Bing test), it is likely that this ear has a greater conductive component, and unmasked bone conduction thresholds originate from this ear. Admittedly, these tests generally do not unequivocally resolve the masking dilemma, but the information can be very valuable clinically. A third simple approach to dealing with the masking dilemma is the use of insert earphone cushions (ER-3A) for both air conduction pure tone stimulation and masking during bone conduction stimulation. If these cushions are inserted to maximum recommended depth within the ear canal, they do contribute to increased interaural attenuation for lower frequency (below 1000 Hz) tones. This may

Figure 8-3. Relationship of aural acoustic impedance and audiogram configuration. (From Johansen, H. [1948]. Relation of audiograms to the impedance formula. *Acta Oto-laryngologica* [Stockholm] Suppl. 74.)



reduce the likelihood of stimulus cross over to the non-test ear as well as cross over of the masking noise at high-intensity levels to the test ear. However, minimal interaural attenuation benefit comes from so-called tube-phones for higher frequencies, particularly at more typical insertion depths. Also, insert cushions cannot, of course, be used in patients with aural atresia (anotia). This discussion of the masking dilemma presumes that immittance audiometry was consistent with bilateral conductive hearing impairment. If immittance audiometry shows normal tympanometry and acoustic reflex activity on one ear, a sensory hearing impairment can be presumed and there will be no masking dilemma. Two additional approaches for solving the masking dilemma — the SAL test and ABR — will now be presented.

Sensory Acuity Level

The SAL test is perhaps the most underused and underappreciated of clinical audiometric procedures. Rainville introduced in 1955 a new, but rather complicated, technique for assessing sensory function that did not rely on conventional bone conduction audiometry. Several years later, Jerger and Tillman (1960) and then others (Burke, Creston, Marsh, and Shutts, 1965; Jerger and Jerger, 1965; Jerger and Tillman, 1960; Keys and Milburn, 1961; Michael, 1963; Tillman, 1963) provided comprehensive investigations of a clinically feasible modification of the Rainville technique. The two major limitations of conventional bone conduction audiometry motivating the SAL test were the problem with calibration of bone conduction stimuli and difficulties associated with adequately masking the non-test ear, that is, the masking dilemma.

In 1963, Jerger and Jerger described the SAL test protocol, systematically studying the possible effects of certain procedural variables on SAL outcome, including occlusion effect, force of bone vibrator application, effect of noise duration, linearity of masking, effect of correlated noise in non-test ear (i.e., masking level difference phenomena), and the acoustic reflex. Importantly, they found that SAL and conventional bone conduction audiometry hearing threshold levels were equivalent.

In brief, the SAL procedure is as follows: Air conduction pure tone hearing threshold levels for signals of 500, 1000, 2000, and 4000 Hz are first measured for each ear without any masking. Then a masking noise is presented via a bone vibrator

placed on the forehead. The intensity level of the noise is 2 volts root-mean-square. Clinically, this level can be approximated by presenting masking noise by bone conduction at the output limit for the audiometer, usually about 55 dB HL for 500 Hz and 60 or 65 dB HL for 1000, 2000, and 4000 Hz. The average shift in air conduction thresholds produced by this noise level at each of the four pure tone stimulus frequencies is determined for a group of normal-hearing subjects, prior to application of SAL testing in a clinical facility. The SAL test cannot, unfortunately, be performed on audiometers that do not permit presentation of a masking noise via the bone vibrator.

The fundamental calculation in SAL testing is comparison of the air conduction hearing threshold level (HTL) shift from the quiet test condition to the bone conduction masking condition in a patient versus the normal expected shift, derived as noted above. If the patient's hearing loss is entirely conductive, his or her shift will equal the normal shift, since the masking noise will have full effect on sensory hearing. Normal bone conduction is presumed. On the other hand, if the hearing loss is sensory, masking noise will not affect hearing until it exceeds the patient's HTL. An approach that facilitates an understanding of SAL is to consider the difference between the patient's noise-produced HTL versus the normal noise-produced HTL as an estimation of air-bone gap, not bone conduction hearing level. If subtraction of the normal air conduction threshold in noise (e.g., 55 dB) from a patient's threshold in noise, for example 65 dB, produces a difference of only 10 dB, then there is a 10 dB air-bone gap. The remainder of the hearing loss is sensory. That is, bone conduction hearing is 10 dB better than air conduction hearing. SAL threshold symbols (diamonds) are plotted at an intensity level 10 dB better than those for air conduction as initially measured in quiet. If the difference for patient versus normal threshold is 60 dB, on the other hand, the air-bone gap is 60 dB (i.e., SAL symbols are plotted at the dB HTL 60 dB better than the air conduction HTL in quiet). A useful form for calculating SAL outcome clinically is shown in Figure 8-4.

Concerns about validity and clinical accuracy of the SAL test expressed in the early 1960s (Goldstein, Hayes, and Peterson, 1962; Martin and Bailey, 1964; Tillman, 1963) were essentially allayed by the findings of Jerger and Jerger in 1965; however, two practical limitations are sometimes encountered in clinical application of SAL in pa-

Patient: _____		Age: _____		Sex: _____		
Date of test: _____		Tester: _____				
Test Frequency (Hz)						
Ear	Condition (HTLs in dB)	500	1000	2000	4000	SRT
Right	Quiet					
Right	Noise					
	SAL shift norm					
	Noise - SAL norm (air bone gap)					
Left	Quiet					
Left	Noise					
	SAL shift norm					
	Noise - SAL norm (air bone gap)					

Figure 8-4. Worksheet for computing SAL.

tients with moderate to severe hearing impairment. For patients with severe conductive loss, air conduction threshold levels may be shifted beyond the output limits of the audiometer by the bone conduction noise. An air-bone gap of as much as 60 dB can be confirmed, but the actual sensory hearing level cannot be determined. Also, if a patient showing no shift in air conduction threshold levels with noise has HTLs worse than the intensity level of the noise (e.g., 55-60 dB), one cannot be certain that a pure sensory loss exists, that is, no air-bone gap. Nonetheless, the authors have found SAL testing to be a very effective approach for differentiating between largely conductive versus sensory types of deficits in patients with severe bilateral hearing impairment. Clinical application of SAL testing is illustrated by case reports in the following section.

AUDITORY BRAINSTEM RESPONSE

With infants or young children, and patients of all ages who are uncooperative or for whatever reason untestable with traditional audiometry, it may be impossible to use earphones or bone vi-

brators in behavioral assessment of auditory sensitivity. The SAL test cannot be used in these patients and, furthermore, does not always provide an estimate of air conduction HTL. According to prevailing opinion, ABR by air conduction and bone conduction is similarly limited by masking problems. Acoustic cross-over levels of 50 to 75 dB have been reported for click stimuli (Humes and Ochs, 1982; Reid and Thornton, 1983). Bone conduction ABR is additionally constrained by maximum stimulus output levels of 50 to 60 dB HL.

Numerous clinical reports describe the use of ABR in defining conductive hearing loss (Arlinger and Kylen, 1977; Cornacchia, Martini, and Morra, 1983; Finitzo-Hieber, Hecox, and Cone, 1979; Fria and Sabo, 1980; Hall, 1984; Hall, Morgan, Mackey-Hargadine, Aguilar, and Jahrsdoerfer, 1984; Hooks and Weber, 1984; Jahrsdoerfer et al., 1985; Mauldin and Jerger, 1979; McGee and Clemis, 1982; Weber, 1983) and several attempts to assess sensory function with an ABR SAL-type procedure (Hicks, 1980; Webb and

Greenberg, 1983). To date, the major emphasis seems to have been on applying ABR in accordance with the principles and constraints of behavioral audiometry rather than exploiting the unique advantages of auditory evoked response methodology. For instance, Weber (1983) states that with air conduction ABR, "as with any audiometric procedure, a masking noise must be administered to the non-test ear when the poorer ear is being evaluated" (p. 344), and "because a bone-conducted signal reaches each cochlea with about the same intensity, masking of the non-test ear is essential if information about the individual ear is desired. . . . This masking dilemma in patients with a marked conductive hearing loss is not unique to ABR testing" (p. 348). A similar theme is reiterated by others (Finitzo-Hieber, Hecox, and Cone, 1979).

Although ABR permits assessment of monaural auditory sensitivity with earphone and bone vibrator transducers, even in infants and young children with middle ear pathology, this advantage would appear quite markedly reduced in patients with a serious conductive component to the hearing impairment. That is, if effective masking were necessary and yet could not be applied without the likelihood of overmasking, then ear-specific information would be impossible with air and bone conduction ABR, just as it is with traditional threshold audiometry. The recent commercial availability of insert transducers may, as noted before, offer a partial practical solution to the overmasking problem in behavioral and ABR assessment in some patients. Insert transducers are not useful for patients with complete aural atresia or stenotic ear canals. Furthermore, with the limitation in maximum stimulus output, bone conduction ABR provides little information for patients with a mild to moderate sensory component.

Masking is not invariably necessary in ABR assessment of serious bilateral conductive hearing impairment, or ABR measurement in general. In the authors' experience, ABR assessment can often define the extent of the conductive loss and residual sensory function for each ear (Hall, 1989; Hall et al., 1984; Jahrsdoerfer et al., 1985). A clear wave I in the ipsilateral recording, usually at a high-stimulus intensity level, by air or bone conduction ensures an ear-specific ABR. A delayed wave V component at a high-stimulus intensity level, with no evidence of a wave I, indicates the need for contralateral masking to rule out a cross-over response.

Success in obtaining ear-specific information is highest for infants and young children but can be

enhanced in all patients by following a test protocol that includes four major components: (1) an earlobe or, whenever possible, ear canal or promontory inverting electrode (versus mastoid placement) serves to augment wave I amplitude. In fact one of the first clinical applications of ECoChG was evaluation of conductive hearing impairment (Lempert, Wever, and Lawrence, 1947); (2) a high-pass filter setting (low-frequency cutoff) of 30 Hz (versus 150 or 300 Hz) encompasses the substantial low-frequency energy that characterizes the ABR. Low-frequency spectral content may be greater, in fact, for the bone conduction ABR, since the frequency response of the transducer is predominately below 1500 Hz; (3) stimulus (click) rate of no greater than 21 per second and occasionally 11.1 per second or even slower; (4) a quiet (natural sleep or sedated) patient, and (5) simultaneous multichannel recordings for air and bone conduction stimulation.

The initial overall objective of these technical maneuvers is to produce a clear wave I for the ipsilateral recording channel (versus no wave I for the contralateral channel), which confirms that the response is arising from the ear stimulated. Masking the non-test ear is superfluous if a wave I is identified in the stimulus ipsilateral waveform. The response cannot, in this case, be due exclusively to stimulation of the non-test ear. A secondary objective is to record a distinct and reliable wave V component that can then be followed as stimulus intensity is decreased in estimation of ABR threshold. With a reliable wave I for at least one intensity level, it is then safe to conclude that waveforms for lower-intensity stimuli are also ear specific, even if a wave I cannot be confidently identified. Guidelines for application of bone conduction ABR are presented in a recently published textbook (Hall, 1990). The application of these ABR techniques in eliciting clinically useful bone conduction responses is illustrated (case 17) in a section that follows.

PATTERNS OF AUDIOMETRIC FINDINGS IN EXTERNAL AND MIDDLE EAR PATHOLOGY

The following illustrative cases highlight findings for a basic audiometric test battery that typify common types of external or middle ear disorders or pathology. The emphasis is on audiometric patterns that permit differentiation of one disorder

der from the next and contribute to accurate interpretation of findings. As noted at the outset of this chapter, a careful history and thorough physical examination often lead to the correct diagnosis. Certainly, this would be true for most of the following cases; however, the patients first presented to audiology. It is often the audiologist's responsibility to conduct the initial assessment and ensure that the patient receives proper hearing health care. Atypical patterns of findings for middle ear pathology are discussed in the next section.

CASE 1: IMPACTED CERUMEN

A 76-year-old woman presents with the chief complaint of long-standing difficulty in hearing, especially understanding speech in the presence of background noise, but recent onset of increased difficulty in all listening conditions. As shown in Figure 8-5, she had a moderate to severe, sloping mixed hearing impairment bilaterally. Speech audiometry performance was fair for word recognition and synthetic sentence identification (SSI) at a high-intensity level (80 dB HL) but very poor at even a loud conversation level (60 dB HL). Immittance audiometry showed flat type B tympanograms in each ear.

Initial review of audiometry might suggest otitis media. The important audiometric finding for this patient was abnormally small EAC volumes, as estimated from immittance audiometry (Shanks and Lilly, 1981). This prompted otoscopic inspection, which confirmed that cerumen occluded each external auditory meatus. Removal of the cerumen by an otolaryngologist produced a mild to moderate sloping sensory hearing impairment suggested by the bone conduction HTLs in Figure 8-5. The finding of a medically treatable external ear disorder influenced the alternatives for audiologic management of this elderly patient. Otoscopy before immittance audiometry is advisable.

CASE 2: COLLAPSING EAR CANALS

A 69-year-old woman noted a slight decrease in hearing for the left versus right ear. Pure tone audiometry (Figure 8-6) showed a mild high-frequency conductive component bilaterally. Speech audiometry was good for phonetically balanced (PB) word recognition and SSI. SSI scores were higher than PB word scores for low to moderate (40–60 dB) intensity levels. Tympanometry was normal. At this juncture, the presence of a slight conductive component and normal tympanograms would suggest stapes fixation, although this pathology produces a hearing impairment with a rising configuration.

The presence of normal ipsilateral acoustic reflex threshold levels and elevated contralateral acoustic reflexes, especially for higher-stimulus frequencies, led to the suspicion of collapsing ear canals. The immittance probe prevented ear canal collapse, whereas the standard audiometric earphone cushion used for contralateral reflex assessment caused ear canal collapse just as it did during pure tone audiometry. The relative poorer performance for PB word recognition, which depends most on hearing sensitivity in the 2000 Hz region versus SSI materials, which reflect hearing in the 750 Hz region, was consistent with the high-frequency deficit. Elevated contralateral versus ipsilateral acoustic reflexes, a horizontal pattern, are also a sign of brainstem pathology (Hall, 1985). The air-bone gap would not be consistent with this pathology. In addition,

repeat air conduction audiometry following insertion of an otoscope speculum produced HTLs equivalent to those for bone conduction shown in Figure 8-6. A collapsing ear canal produces an apparent hearing deficit of 10 to 15 dB, which is greatest at 2000 Hz (Ventry et al., 1961). It is more often encountered in newborn infants and the elderly than other patient age groups. The possibility of collapsing ear canals should be considered in patients of any age with large "lop" ears.

CASE 3: PATENT VENTILATION TUBES

Ventilation (aeration or pressure equalization) tubes were inserted bilaterally into the tympanic membranes of a 5-year-old boy with recurrent otitis media. Audiometric findings obtained 1 year later (shown in Figure 8-7) confirmed that the ventilation tube for the right ear remained in the tympanic membrane and was patent. EAC volume of 2.3 cc, as estimated by immittance measures (Hall, 1987), greatly exceeded normal expectations for a child (no more than 1.0 cc) and the value for the other canal (0.60 cc). Positive ear canal pressure (+200 decaPascal [daPA]) was relieved by swallowing. *A finding of patent ventilation tube (or tympanic membrane perforation) precludes tympanometry and acoustic reflex measurement (Hall, 1987).* It is improper to perform tympanometry and report a type B tympanogram, since pressure against the eardrum is, in effect, not being varied. Air conduction pure tone HTLs were well within the normal range, although bone conduction testing showed a consistent air-bone gap. Word recognition (PB-K lists) was excellent.

For the left ear, the pattern of immittance findings (normal EAC volume, negative pressure of -300 daPA, and absent acoustic reflexes with probe left) and a mild conductive hearing impairment, with only fair word recognition at a conversational intensity level, indicated extrusion or occlusion of the ventilation tube and apparent recurrence of serous otitis media. The tube was found lying deep within the ear canal on otoscopic inspection with an operating room microscope. The presence of the ventilation tube in the right tympanic membrane was verified otoscopically.

By tympanometric measurement, no distinction exists between a patent ventilation tube and tympanic membrane perforation. History and otoscopic inspection can differentiate between the two. Also, a patent ventilation tube typically causes little or no deficit in hearing sensitivity, although an air-bone gap, as in this case, may be present whereas a very mild conductive hearing component (e.g., 20 dB) at the least is usually found in tympanic membrane perforation.

CASE 4: SCARRED TYMPANIC MEMBRANE

A 55-year-old Spanish-speaking woman complained of recent difficulty understanding what her grandchildren said to her. She reported a history of repeated ear infections and tympanic perforations as a child, but no problems with her ears since then. Tympanometry with a 226 Hz probe tone showed abnormally high compliance (>1.60 cc) bilaterally (Figure 8-8).

Because of the sharp tympanogram peak, it was difficult to remain at maximum compliance and maintain a steady compliance meter during acoustic reflex measurement bilaterally. An attempt to measure acoustic reflexes was abandoned for this reason with probe left. Pure tone audiometry showed a mild, gently sloping sensory hearing impairment bilaterally. Bone conduction threshold assessment was mandated by the abnormal immittance findings. There

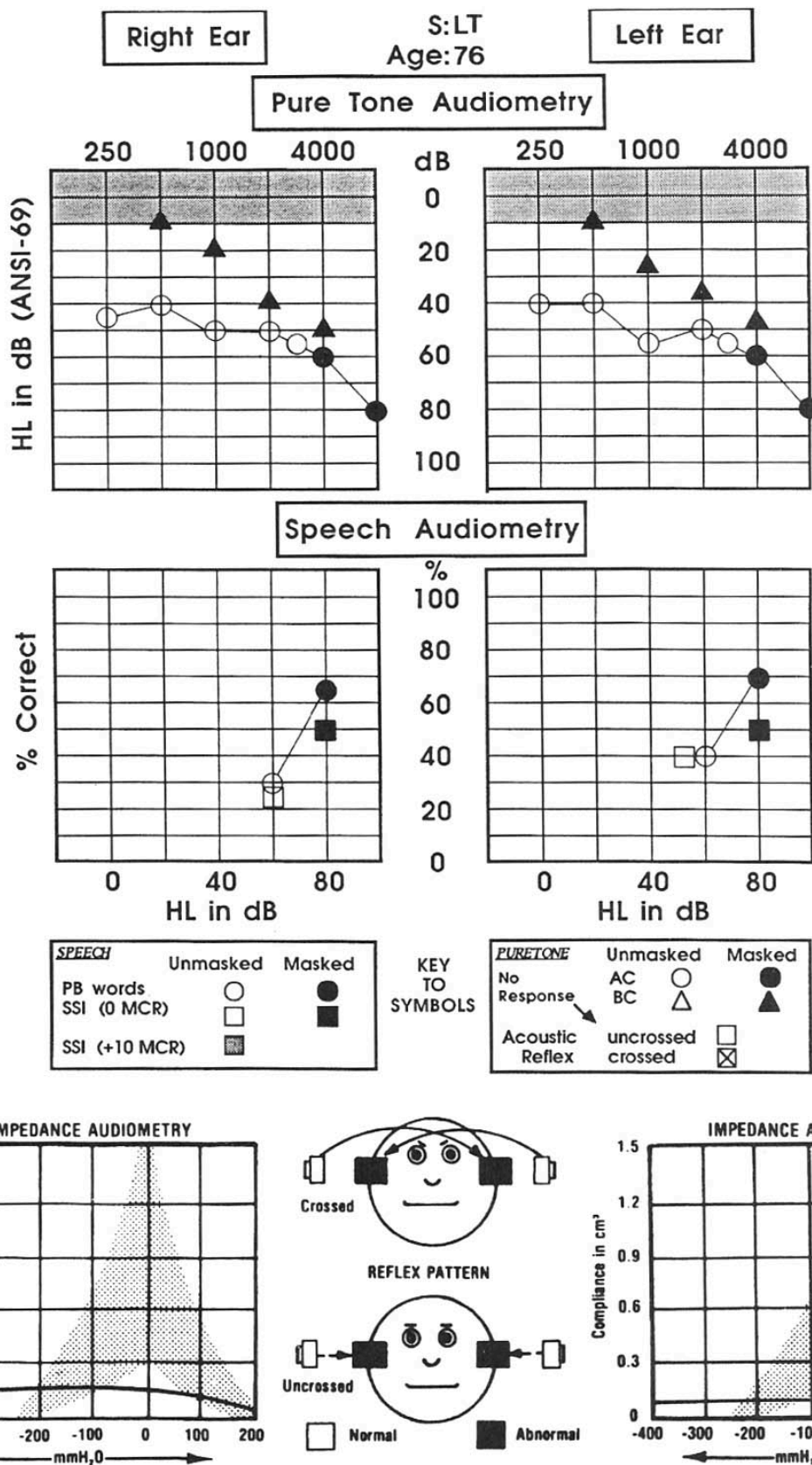


Figure 8-5. Audiometric findings for a 75-year-old woman with presbycusis in combination with impacted cerumen within external auditory canal (case 1).

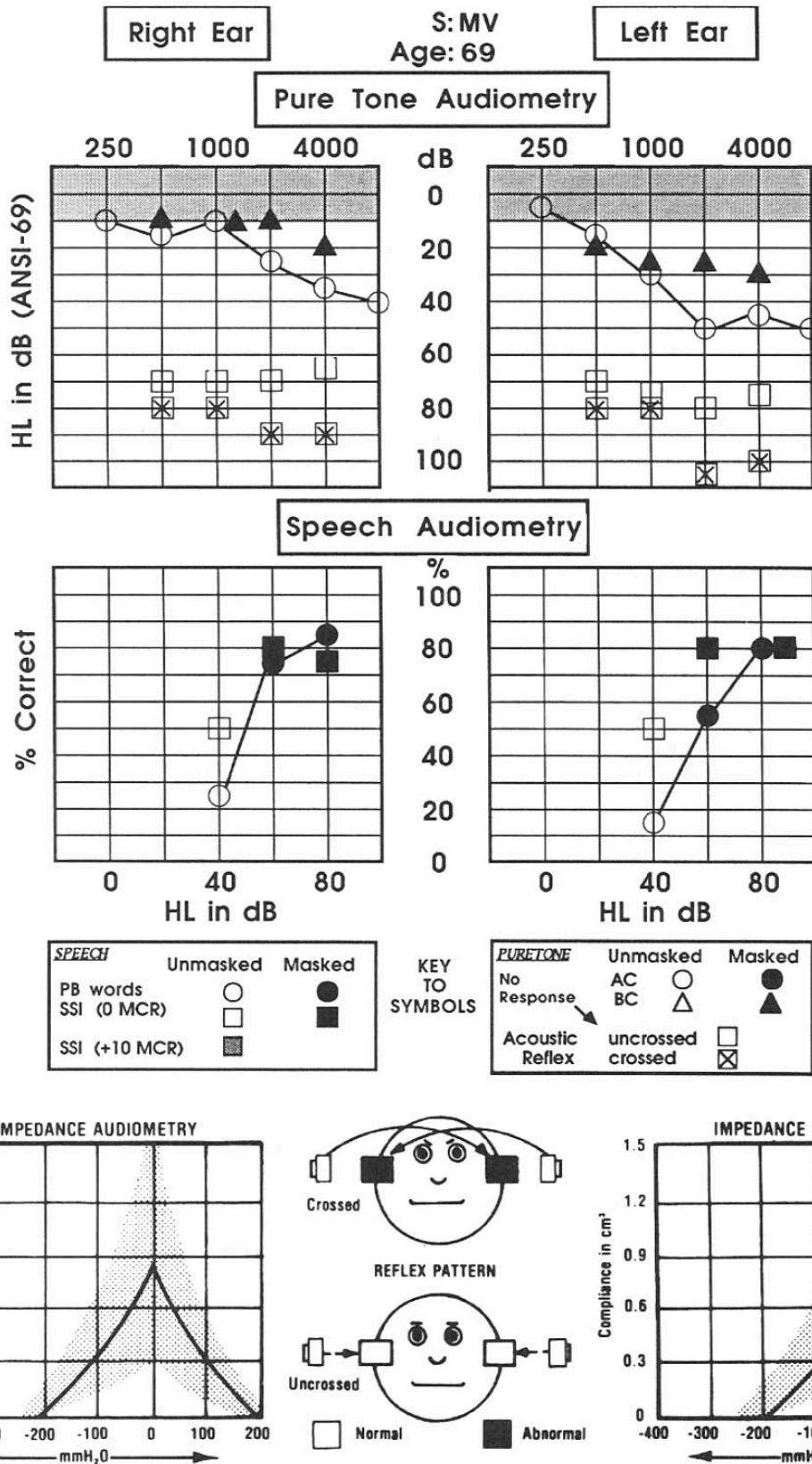


Figure 8-6. Audiometric findings for a 69-year-old woman with collapsing ear canals (case 2).

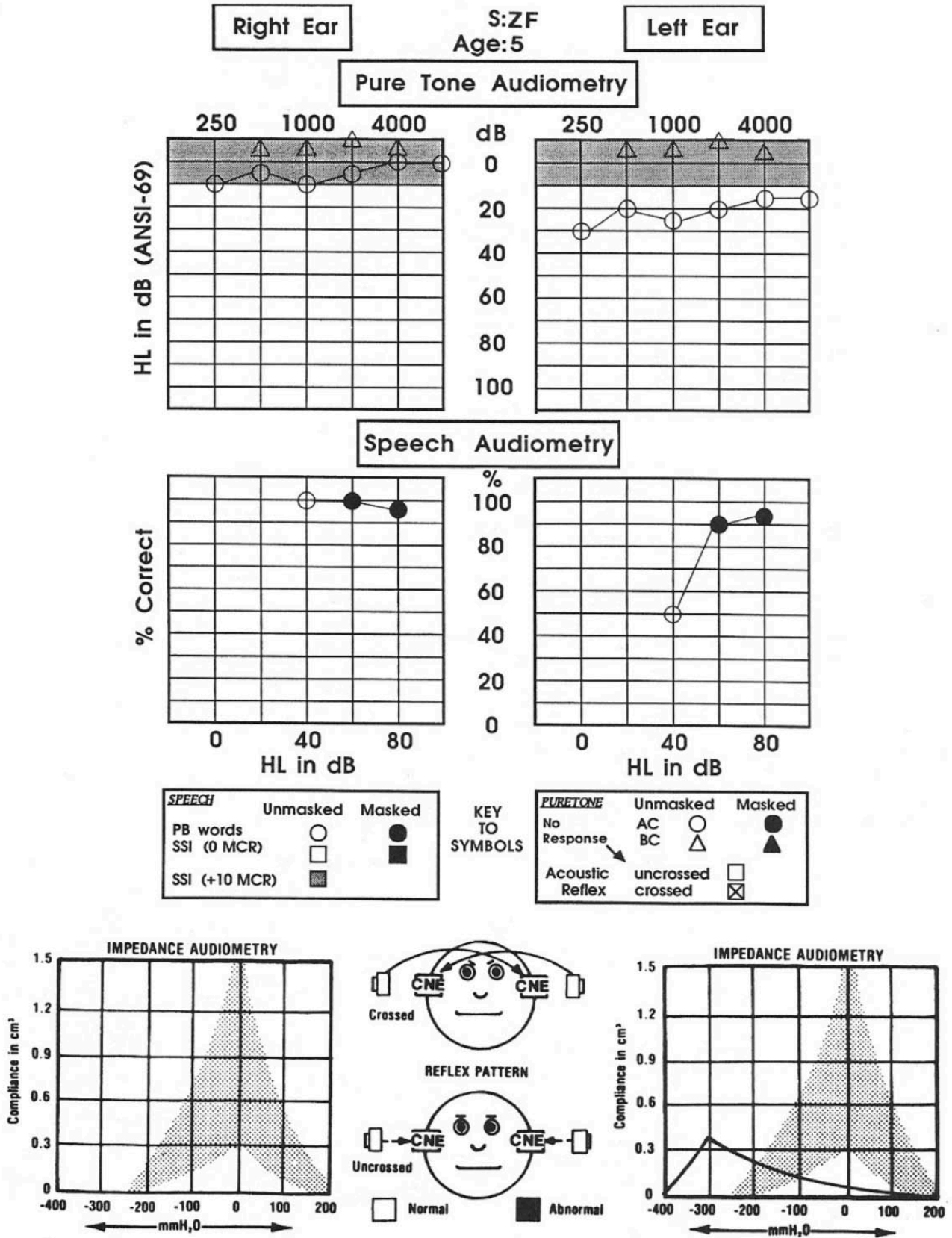
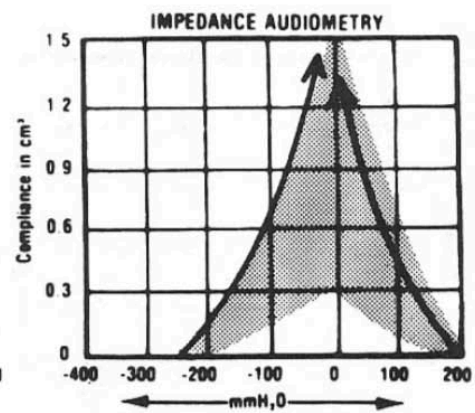
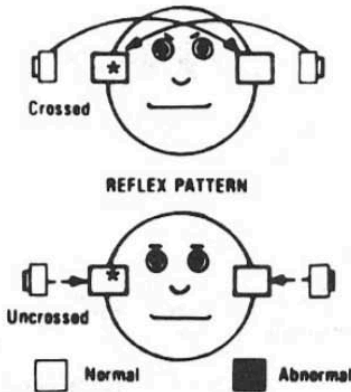
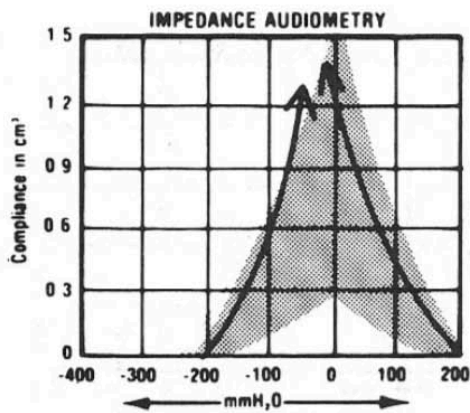
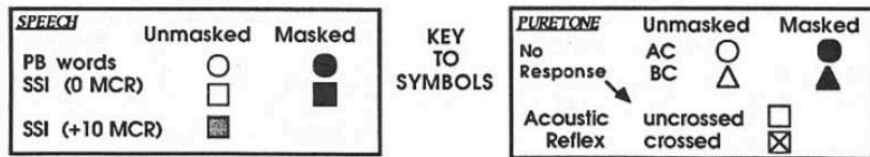
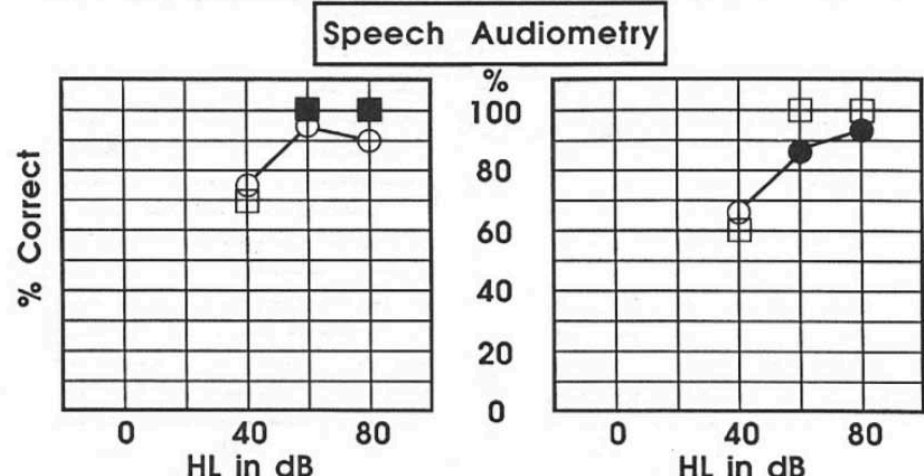
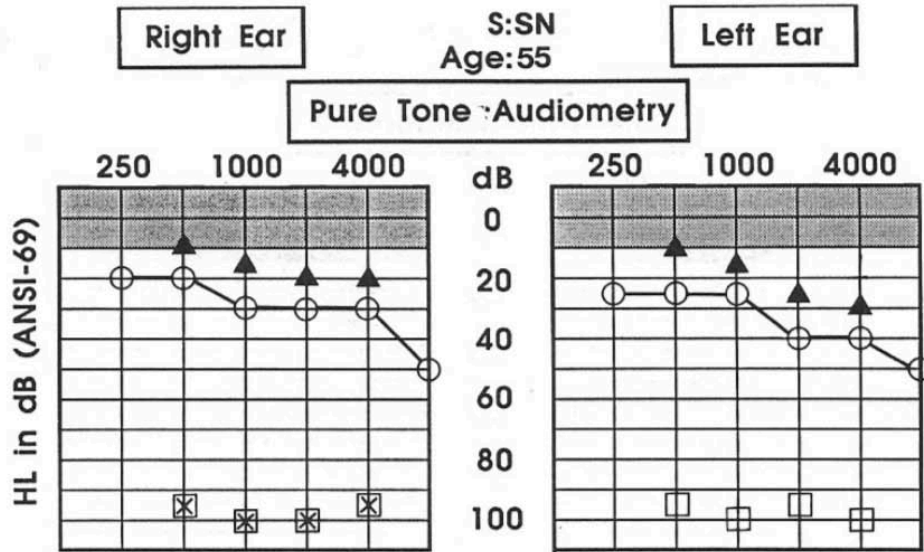


Figure 8-7. Audiometric findings for a 5-year-old boy with patent ventilation tubes (case 3).



* Excessive Meter Movement

Figure 8-8. Audiometric findings for a 55-year-old woman with scarred tympanic membrane (case 4).

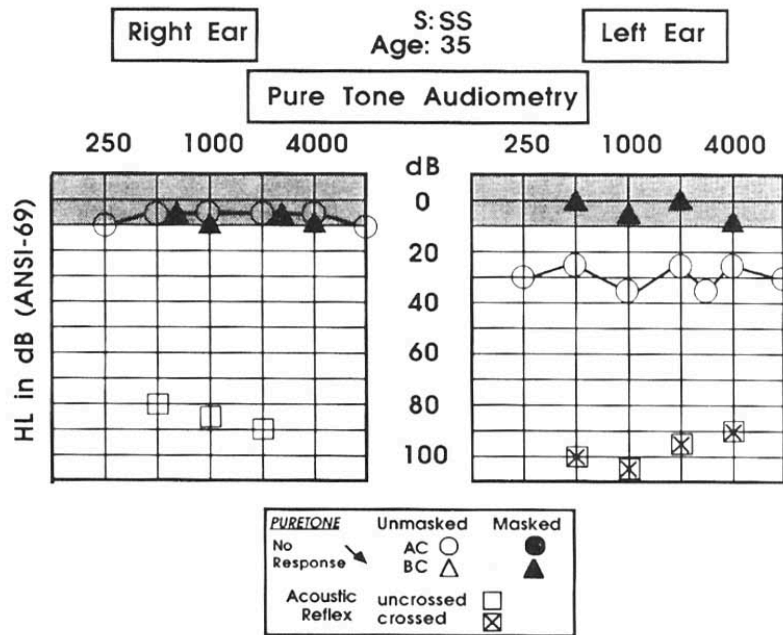


Figure 8-9. Audiometric findings for a 35-year-old woman with perforation of tympanic membrane (case 5).

was no air-bone gap. Selected Spanish word recognition (not standardized) and Spanish language SSI performance were excellent for higher-intensity levels but slightly reduced at conversational level (40 dB HL).

Highly compliant tympanograms suggest the possibility of ossicular chain discontinuity; however, in this case the history and pure tone audiometry are consistent with tympanic membrane dysfunction instead. The pattern of sensory impairment may have been related to presbycusis. Otoscopic examination revealed monomeric membranes and scarring of tympanic membranes bilaterally. The patient was not interested in evaluation for possible amplification at the time of the evaluation.

CASE 5: TYMPANIC MEMBRANE PERFORATION

A 35-year-old woman reported bleeding and slight difficulty hearing on the left ear after she was slapped in the ear during an altercation. Auditory function was normal on the right ear (Figure 8-9). Despite repeated efforts, a seal could not be maintained for tympanometry on the left ear, although an ear canal volume of greater than 2.5 cc was temporarily recorded before pressure was relieved spontaneously. Pure tone audiometry revealed a mild conductive hearing impairment. Acoustic reflexes were present with probe in the normal right ear, although the crossed reflexes were elevated by the degree of conductive component with stimulus left. Audiometric Weber lateralized to the left ear at 500 and 1000 Hz. Otologic examination of the left ear showed a large perforation in the posterior-superior portion of the tympanic membrane.

CASE 6: EUSTACHIAN TUBE DYSFUNCTION

The patient was a 3-year-old child with nasal congestion. Type C tympanograms were evident bilaterally when pressure was varied in a positive-to-negative direction (Figure 8-10). Note, however, that with a negative-to-positive change in ear canal pressure, there was a positive pressure shift in the tympanogram peak that altered interpretation according to the Jerger system. Acoustic reflexes were pres-

ent but at elevated stimulus intensity levels under all conditions (right and left ears, crossed and uncrossed). Hearing sensitivity was borderline normal in each ear. The child was referred for medical management of serous otitis media.

CASE 7: PURULENT OTITIS MEDIA

The patient was an 8-year-old child with a history of chronic ear infections and drainage from both ears, which were managed medically by his pediatrician. Parents and teachers noted reduced attention and poorer school performance. Immittance audiometry (Figure 8-11) showed ear canal volumes within normal limits and flat type B tympanograms bilaterally with absent acoustic reflexes. There was a moderate conductive hearing impairment by pure tone audiometry. SAL testing confirmed normal sensory function bilaterally. Word recognition was good at a high-intensity level but very poor (30–40%) at a quiet conversational level. Otologic management was strongly urged. The child's teachers were informed of the audiometric findings and the need for preferential classroom seating.

CASE 8: FACIAL (SEVENTH CRANIAL) NERVE PALS

A 46-year-old male oil rig worker reported a recent onset of facial weakness on the left side. The diagnosis was Bell's palsy. He also acknowledged some difficulty in understanding conversational speech. Immittance measurement showed normal tympanometry. Acoustic reflexes, however, were not observed with probe in the left ear. With probe in the right ear, reflexes were at expected intensity levels. Pure tone audiometry (Figure 8-12) indicated a bilateral hearing deficit in the 3000 to 4000 Hz frequency region. There was no air-bone gap and the audiometric Weber was referred to midline. Maximum speech audiometry performance was excellent for words and SSI materials, but roll over was noted on the left ear.

This pattern of findings is consistent with facial nerve palsy on the left side involving the branch to the stapedius muscle. The vertical acoustic reflex pattern on the left is typical of a peripheral disorder, usually middle ear dysfunction. In this case, the normal tympanogram, midline Weber, lack of air-bone gap, and crossed acoustic reflexes at a reduced sensation level with sound left all argument con-

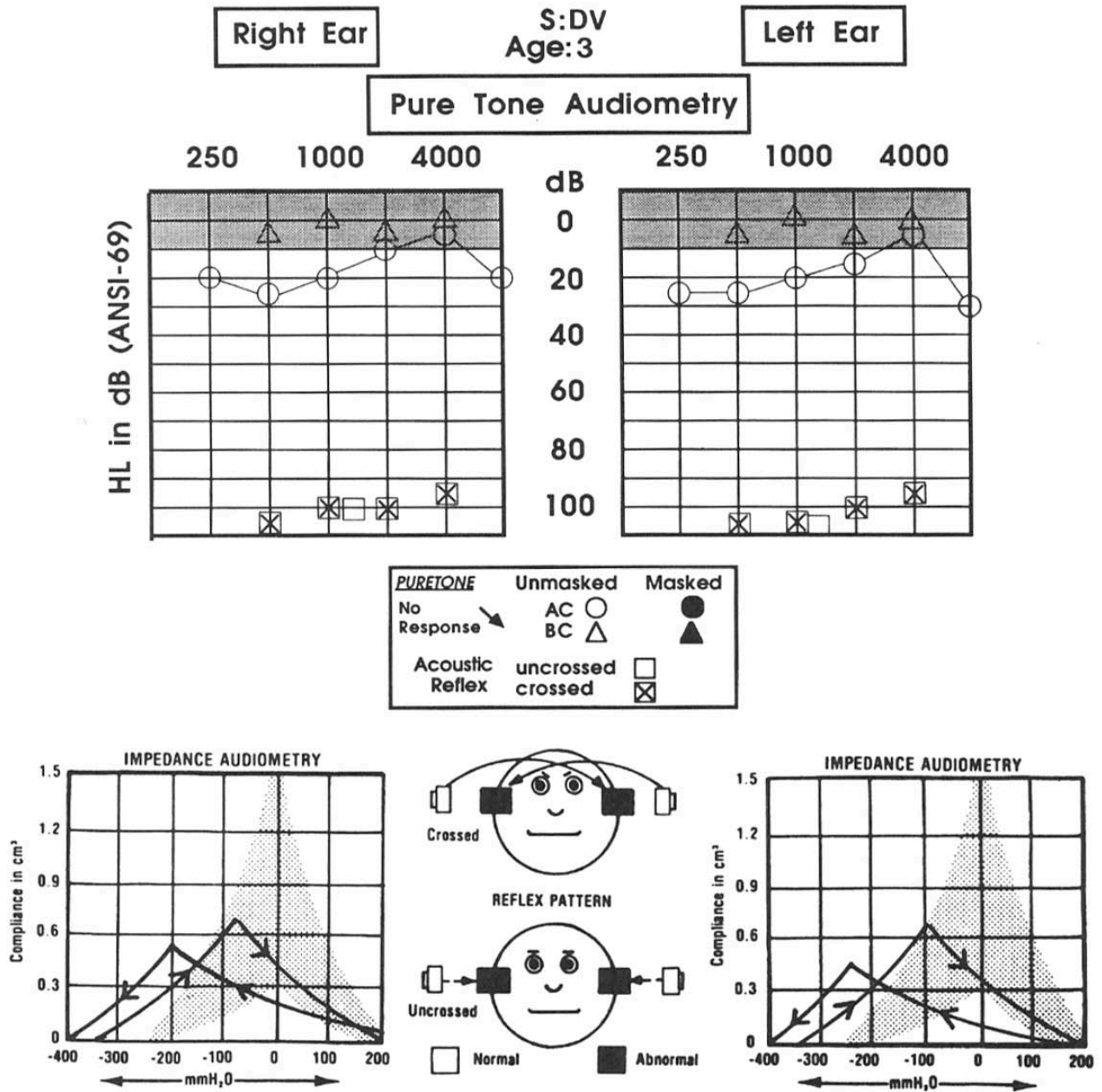


Figure 8-10. Audiometric findings for a 3-year-old child with eustachian tube dysfunction (case 6).

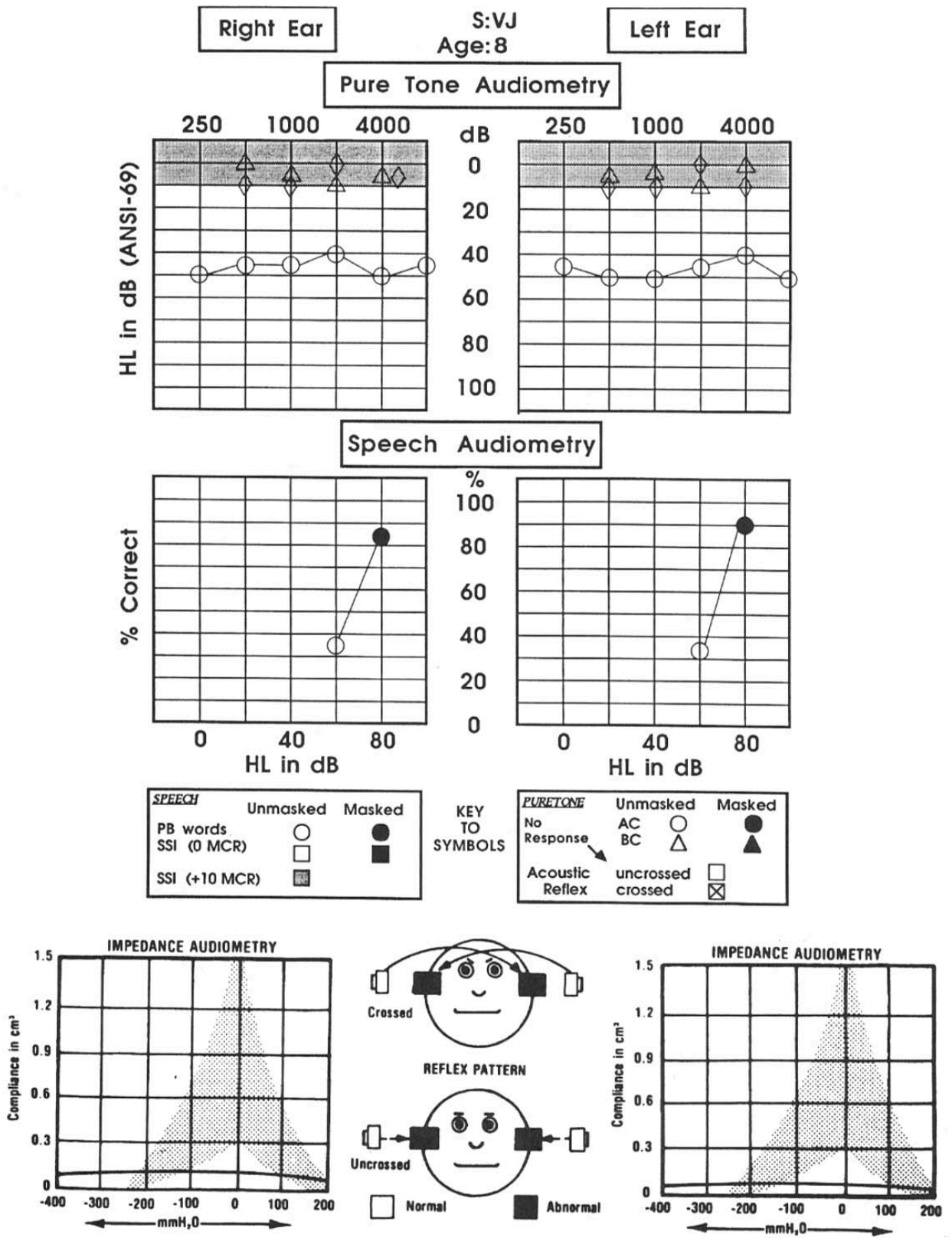


Figure 8-11. Audiometric findings for an 8-year-old child with purulent otitis media (case 7).

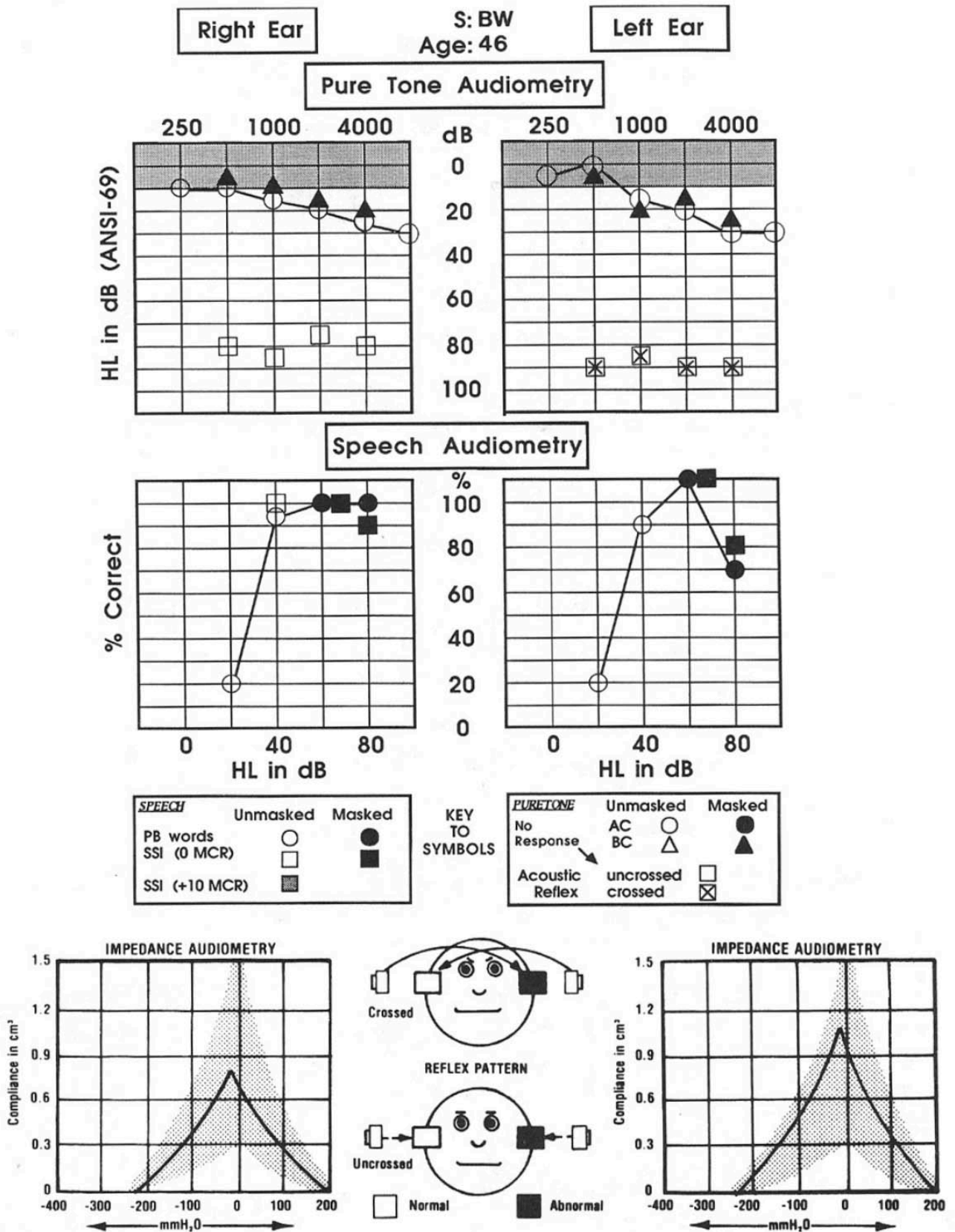


Figure 8-12. Audiometric findings for a 46-year-old male with facial (seventh cranial) nerve palsy (case 8).

vincingly for a seventh nerve etiology versus conductive hearing component. The patient's reported difficulty hearing the relatively high-pitch voices of his wife and children was a reflection of the high-frequency hearing loss. Roll over in speech understanding in ears with no acoustic stapedial reflex activity and normal middle ear function is sometimes found in facial nerve palsy (Hall, 1985).

CASE 9: DISCONTINUITY OF THE OSSICULAR CHAIN

A 26-year-old man was involved in an automobile accident that resulted in head trauma and bilateral hearing impairment. Audiometric findings are illustrated in Figure 8-13. Tympanograms were type A_d bilaterally, and acoustic reflex activity was not observed for maximum (110 dB HL) stimulus intensity levels. Pure tone audiometry showed a severe, slightly sloping, apparently conductive hearing impairment bilaterally. At these air conduction HTLs, there was a masking dilemma. Masking levels needed to overcome the air conduction impairment (greater than 70 dB HL) also exceeded interaural attenuation and could cross over to the test ear. SAL testing (refer back to data displayed in Figure 8-4) confirmed that the loss was entirely conductive bilaterally. Speech audiometry at a high-intensity level was necessarily unmasked, although insert earphones were used to reduce the likelihood of cross over (i.e., to increase interaural attenuation). Surgical otologic management was planned.

CASE 10: OTOSCLEROSIS

A 48-year-old woman reported a gradual, progressive hearing impairment bilaterally, which she first noticed during her college years. Her mother had experienced a similar decrease of hearing. Tympanograms were type A_s bilaterally (Figure 8-14). Acoustic reflexes were not present. Pure tone audiometry showed a primarily conductive hearing impairment bilaterally, with a notch for bone conduction thresholds at 2000 Hz. Speech audiometry performance was excellent at high-intensity levels. The audiometric Weber test produced a midline response. SAL test results generally confirmed the conductive component, but thresholds were better than for conventional bone conduction at 2000 Hz (Tillman, 1963). This case provides a classic illustration of otosclerosis producing stapes footplate fixation and a conductive component. Two other otosclerotic patterns of pure tone audiometry are essentially no air-bone gap (early otosclerosis) and a serious mixed or largely sensory impairment (partially or primarily cochlear focus for otosclerosis).

CASE 11: "CORNER AUDIOGRAM" WITH AIR-BONE GAP

A 3-year-old boy reportedly had normal hearing sensitivity until he contracted meningitis. Within the following month, he had experienced recurrent upper respiratory infections. Audiometric assessment (Figure 8-15) at 1 month after the onset of meningitis showed a "corner audiogram" configuration. There was an apparent air-bone gap for low-frequency stimuli (250 and 500 Hz), suggesting a conductive component to the hearing impairment. Immittance measurement, however, yielded normal tympanograms and evidence of acoustic reflex activity for low-frequency pure tone stimuli. Sensitivity prediction by acoustic reflex was consistent with a severe sensory hearing impairment bilaterally (Hall, 1987). The bone conduction audiometric responses were vibrotactile rather than auditory (Nober, 1964).

AUDIOLOGIC FINDINGS IN ATYPICAL CONDUCTIVE HEARING IMPAIRMENT

CASE 12: OTITIS MEDIA

DIAGNOSED BY SCHOOL NURSE

A 14-year old girl was referred to the audiology service by the school nurse with the diagnosis of right middle ear effusion and approximately 60 dB conductive hearing impairment. Audiometric findings are illustrated in Figure 8-16. Immittance audiometry immediately cast doubt on the suspicion of middle ear effusion. Tympanograms were normal (type A), and the diagonal acoustic reflex pattern was consistent with a severe sensory impairment on the right ear, rather than a conductive component. Hearing sensitivity was normal on the left. For the right ear, air and bone conduction audiometry confirmed the presence of a severe, high-frequency sensory hearing impairment (the patient's shadow curve is also indicated). Audiometric Weber was lateralized to the left ear. Word recognition performance on the right was nil, due to the high-frequency deficit, and the SSI score was 50 percent (reflecting the 60 dB loss at 750 Hz). Without using masking in pure tone testing, the school nurse inferred a conductive hearing impairment on the right ear. Our findings indicated a sensory deficit, and a subsequent ABR confirmed cochlear site of lesion. The patient deferred amplification.

CASE 13: COCHLEAR OTOSCLEROSIS

IN A POSTSTROKE PATIENT

A 64-year-old woman recovering from a stroke was referred for audiological assessment by a physical medicine and rehabilitation specialist because she was experiencing difficulty understanding speech. Since the stroke, she experienced left-sided paresis of upper and lower extremities. She reported a long-standing hearing impairment for which she had taken fluoride 15 years previously in order to prevent further loss of hearing.

Audiometric findings are shown in Figure 8-17. Immittance audiometry (not shown) yielded shallow type A (A_s) tympanograms bilaterally. Acoustic reflexes were observed at abnormally elevated levels in each ear but were characterized by negative deflections (i.e., an increase versus decrease in compliance with stimulus presentation). There was a severe, flat, primarily sensory hearing loss on the right and a mild to moderate deficit on the left. Audiometric Weber lateralized to the ear with greater conductive component (right). Speech audiometry showed good word recognition but only fair performance for the SSI materials. Speech understanding was relatively poorer on the ear (left) with better hearing sensitivity.

This is an example of otosclerosis with a primarily cochlear focus (Paparella and Shumrick, 1980), although the negative deflection acoustic reflexes are evidence of stapes footplate involvement. Audiologic management was not straightforward. There was central auditory system involvement. Speech understanding was poorer for the ear with better auditory sensitivity (right), presumably because it was contralateral to the cerebral hemisphere involved in the stroke (left). The patient relocated to another city and was referred for amplification.

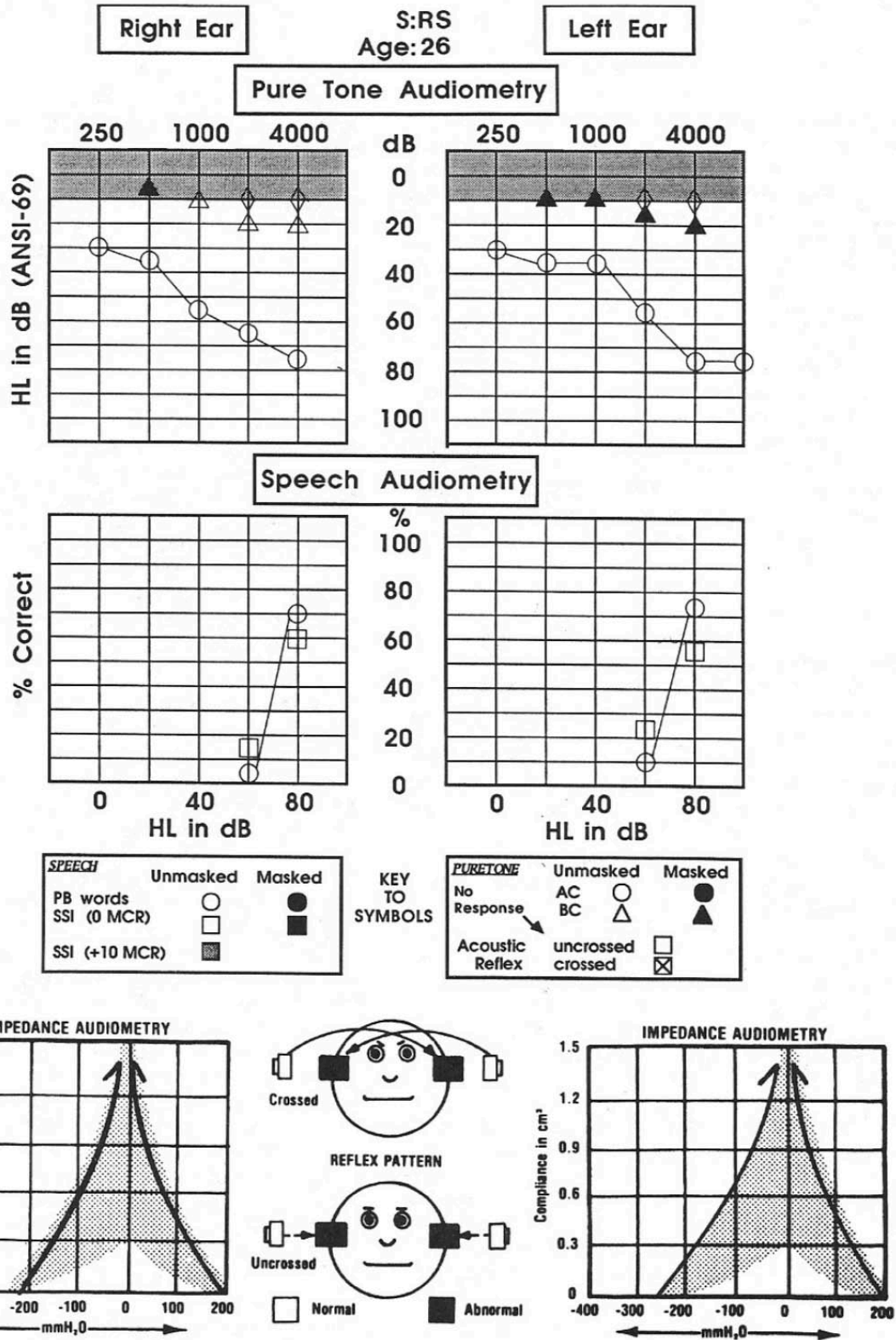


Figure 8-13. Audiometric findings for a 26-year-old man with discontinuity of the ossicular chain (case 9).

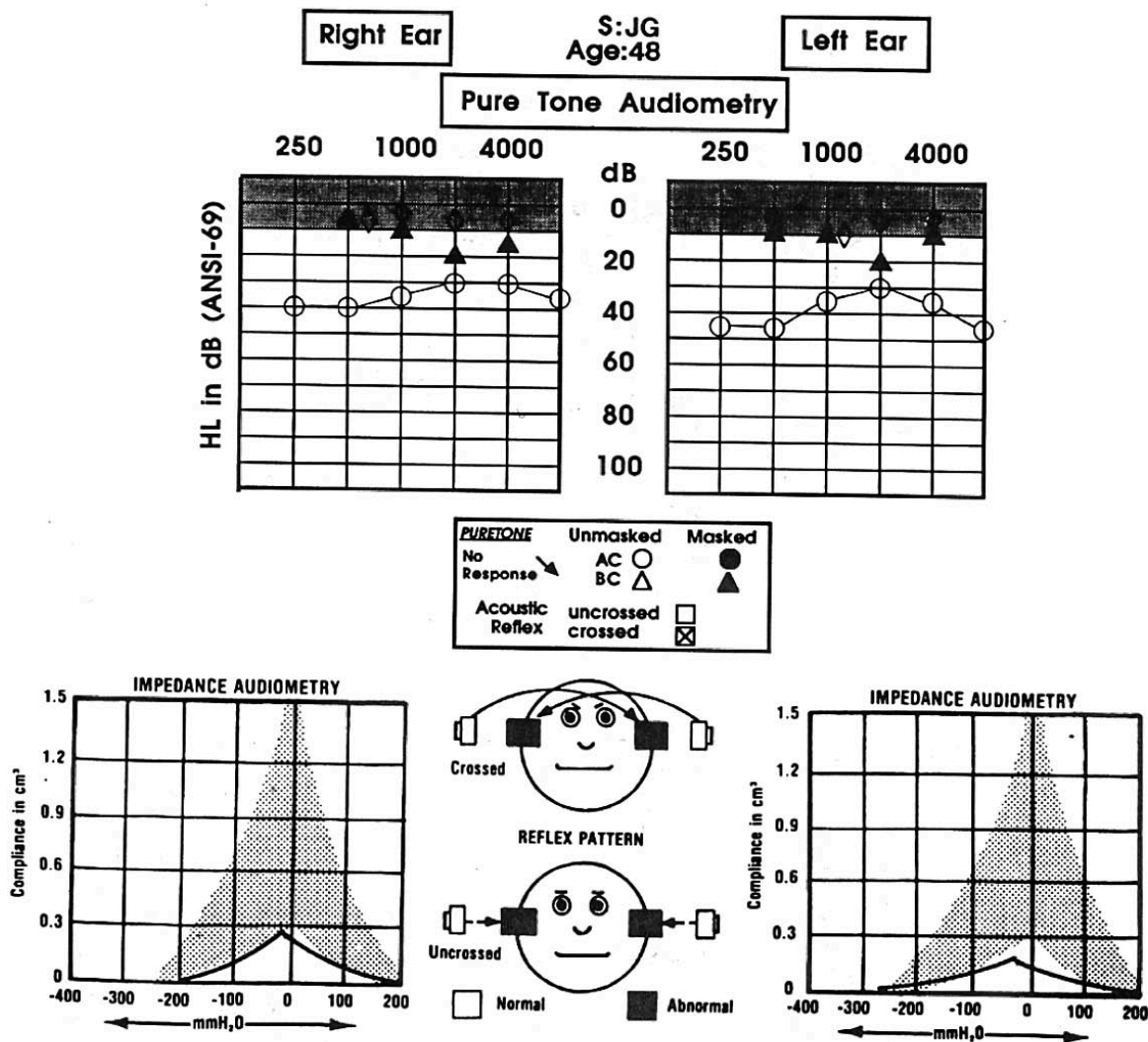


Figure 8-14. Audiometric findings for a 48-year-old woman with otosclerosis (case 10).

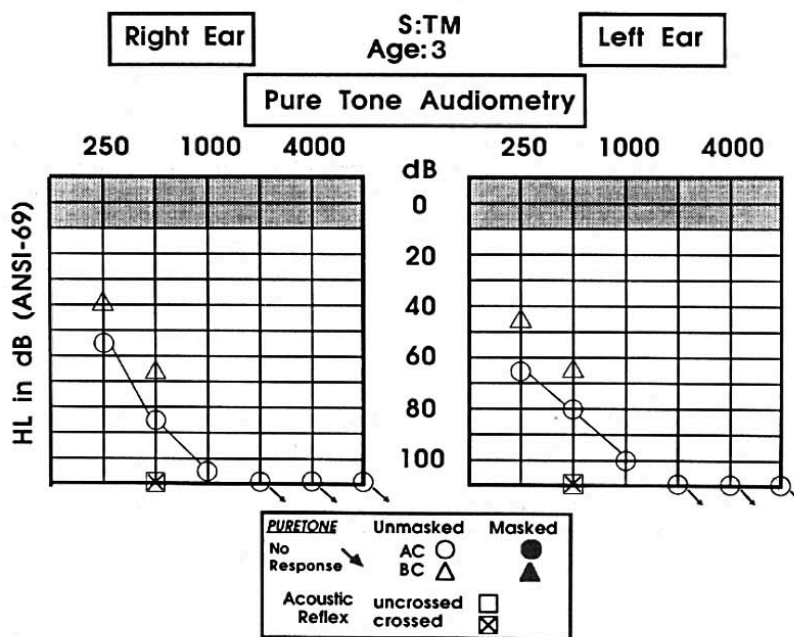


Figure 8-15. Audiometric findings for a 3-year-old boy with coner audiogram (case 11).

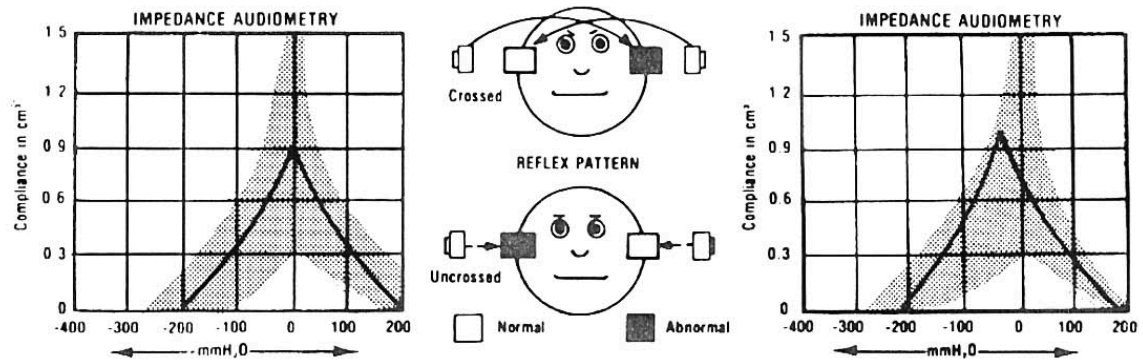
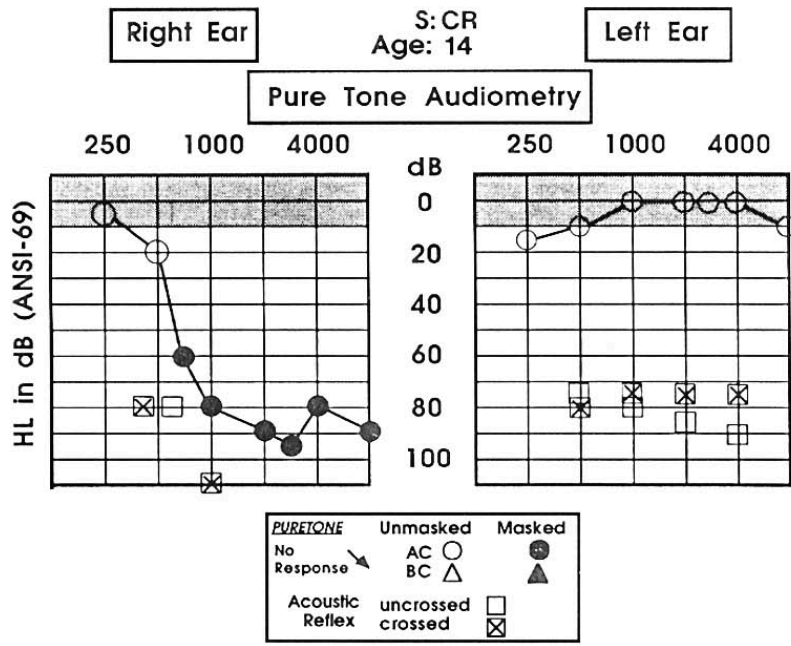


Figure 8-16. Audiometric findings for a 14-year-old girl with suspected middle ear effusion on the right ear (case 12).

CASE 14: POSTSTAPEDECTOMY CHANGES IN SENSORY STATUS

The patient was a 55-year-old woman employed by the local school district. Twenty years previously she had a right ear stapedectomy with a revision five years later. Since then, hearing has reportedly fluctuated in the right ear. She noted mild dizziness and unsteadiness, particularly when getting up from a sitting or supine position. Physical examination of the right ear showed evidence of the previous surgery (chorda tympani nerve adherent to the under surface of the tympanic membrane and curettage of the posterior bony ear canal wall). The left ear examination was normal. The patient underwent a left stapedectomy with oval window drill out, fracture of the crura, disarticulation of the incudostapedial joint, incision of the stapes tendon, and insertion of a platinum Teflon Fisch prosthesis.

Pre- and postoperative audiograms are presented in Figure 8-18. Before surgery, there was a severe, primarily conductive hearing impairment on the left ear and a mild high-frequency sensory loss on the right ear. Three weeks after surgery, audiometric assessment showed a high-frequency, apparently sensory deficit on the left. By 4 months post-operative, however, hearing was bilaterally symmetric.

As this case illustrates, poststapedectomy improvement in hearing loss does not necessarily occur within the initial weeks after surgery. The audiologist would be wise to refrain from any comment to the patient about postoperative changes in hearing until it is clear that hearing has stabilized. In addition, the dynamic interaction of the middle ear and cochlea and the possibility of surgery-related changes in cochlear function (e.g., fistulas, noise-induced damage) can produce temporary or permanent bone conduction deficits that may or may not reflect long-term sensory status.

CASE 15: SUSPECTED MALINGERER

A 9-year-old boy was referred from another audiologist because of inconsistent audiometric findings on two previous test sessions. Hearing sensitivity was described as normal on the left. For the right ear, however, one test showed normal hearing whereas the next showed inconsistent evidence of sensory hearing impairment (Figure 8-19). Immittance measurement was consistent with normal middle ear function on the left (type A tympanogram with uncrossed acoustic reflexes at expected intensity levels).

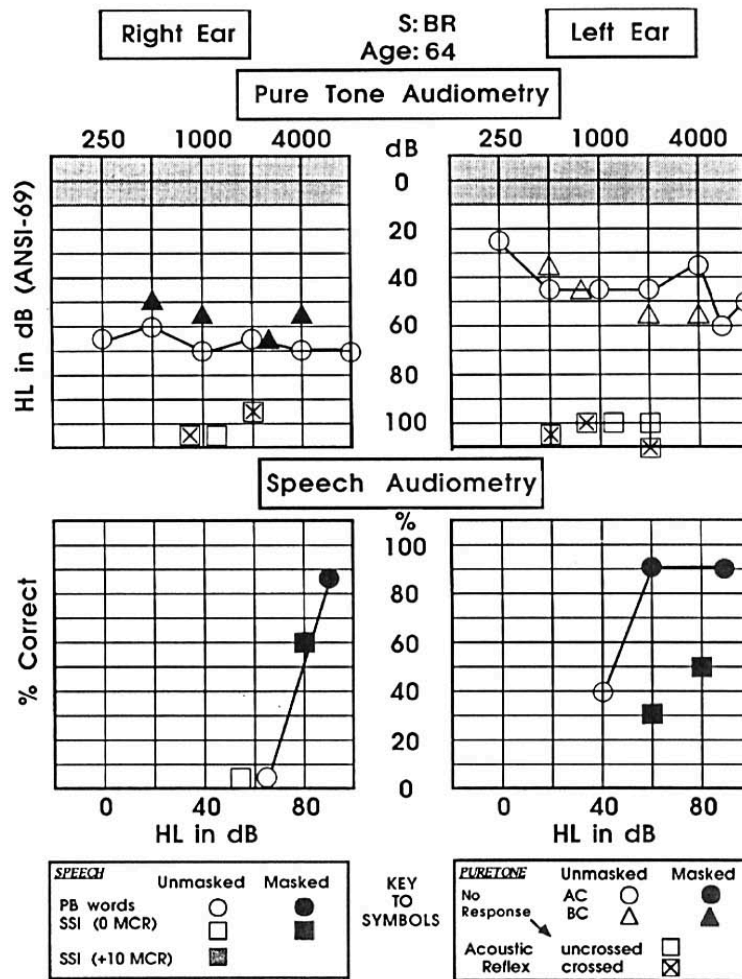


Figure 8-17. Audiometric findings for a 64-year-old poststroke patient with cochlear otosclerosis (case 13).

There was a type A_s tympanogram and no acoustic reflex activity with probe in the right ear, and no acoustic reflex activity for the contralateral mode with sound right. This pattern is consistent with at least a moderate conductive hearing impairment on the right.

Pure tone audiometry confirmed this expectation (Figure 8-19). Pure tone audiometry and speech threshold measures were in close agreement. SAL results were similar to bone conduction thresholds but slightly better at 2000 Hz. The audiometric Weber lateralized to the right ear at 500 and 1000 Hz. Speech audiometry performance was excellent by word recognition bilaterally but slightly depressed for SSI materials on the right ear because of the greater low-frequency hearing deficit.

On questioning, we determined that the first audiologist had done tympanometry but no reflex testing and concluded that the middle ear function was normal. On the first test, she had found normal masked right bone conduction thresholds and rejected the possibility of decreased air conduction thresholds. As the test session wore on, patient and tester became frustrated. The patient was asked to return and on this occasion provided inconsistent evidence of a possible sensory impairment. The mother became disenchanted with the test process and sought a second opinion. The patient was referred for otologic management of an apparent ossicular chain fixation following our audiometric assessment.

CASE 16: OSSICULAR DISCONTINUITY WITH FUNCTIONAL CONNECTION

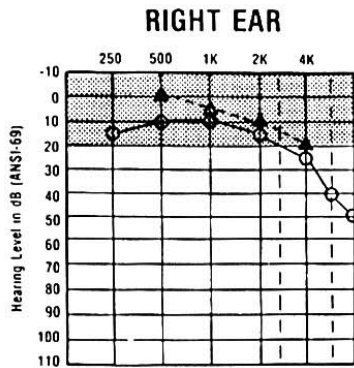
A 30-year-old man noticed decreased hearing on the left after head trauma. Audiometric assessment (Figure 8-20) showed a normal tympanogram on the right and a type A_d tympanogram on the left ear. Acoustic reflexes were present in each test condition but consistently elevated in the contralateral mode with sound left (probe right) and not observed for several stimulus frequencies with the probe in each ear. Hearing sensitivity was essentially normal on the right ear. There was a moderate to severe sloping conductive hearing impairment on the left ear. Audiometric Weber was referred to the left.

Otologic diagnosis was ossicular chain discontinuity on the left, but with a functional connection along the chain. The audiometric pattern is consistent with discontinuity, except for the presence of acoustic reflexes with probe left. This case illustrates the importance of bone conduction pure tone assessment when any immittance aberration exists (e.g., A_d tympanogram or elevated acoustic reflex thresholds). Presumption of a sensory impairment on the right ear on the basis of an A-type tympanogram and the presence of acoustic reflexes, along with a sloping high-frequency configuration, would have led to a serious error in audiometric interpretation.

CASE 17: CONGENITAL AURAL ATRESIA BILATERALLY

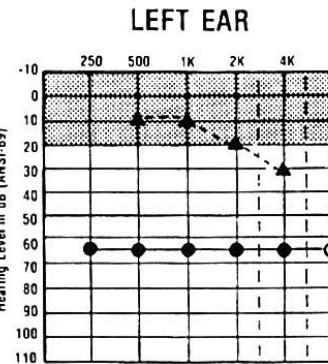
The patient was a 6-year-old girl with congenital aural atresia. A resident of a Middle East country, she was in the

OTOSCLEROSIS
2 WEEKS PRE-OPERATIVE

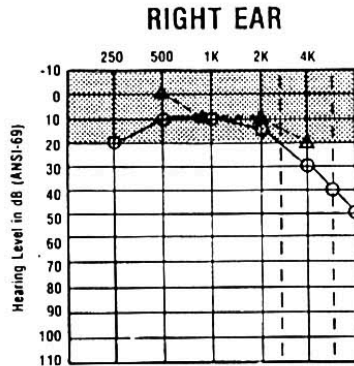


Summary	
Right Ear	Left Ear
12 dB PTA	65 dB
10 dB ST	65 dB
96 % PB _M	92 %
% SS _M	%

□ ← WEBER → □
 ——— H_x

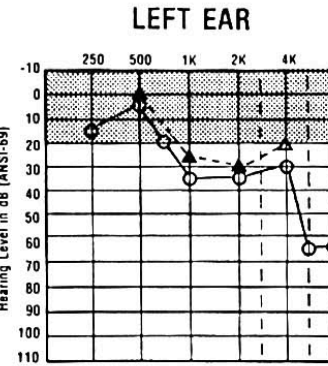


3 WEEKS POST-OPERATIVE

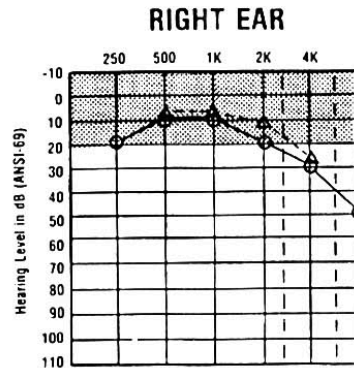


Summary	
Right Ear	Left Ear
12 dB PTA	25 dB
15 dB ST	20 dB
96 % PB _M	92 %
% SS _M	%

□ ← WEBER → □
 ——— H_x



4 MONTHS POST-OPERATIVE



Summary	
Right Ear	Left Ear
11 dB PTA	15 dB
15 dB ST	10 dB
92 % PB _M	92 %
% SS _M	%

□ ← WEBER → □
 ——— H_x

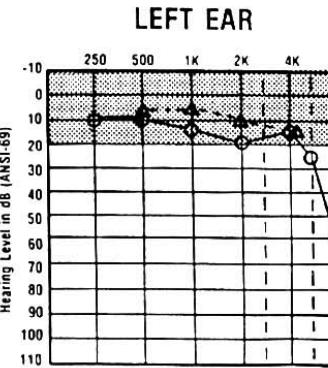


Figure 8-18. Serial pre- and poststapedectomy audiograms (case 14).

United States for evaluation and possible surgical repair of the otologic defect. Pure tone audiometry is illustrated in Figure 8-21. There was a moderate to severe, apparently bilateral and conductive sensitivity loss. The results of SAL audiometry suggested normal bone conduction thresholds in each ear. Unmasked word recognition performance with selected Arabic vocabulary presented by live voice was 100 percent at 90 dB HL but only 20 to 30 percent at 50 dB HL.

ABR assessment was carried out in the operating room under general anesthesia, immediately before high-resolution, thin-slice computed tomography of the temporal bone (Jahrsdoerfer and Hall, 1986). As shown in Figure 8-22, with air conduction stimulation at maximum intensity level (95 dB HL), there was a reliable ABR wave I and V component for the right and left ears with the vertex to

ipsilateral mastoid array. Simultaneous recording with a contralateral mastoid electrode pair, however, revealed no wave I component for either stimulus ear. An ABR wave V was observed for air conduction stimulation at intensity levels down to 75 dB on the right and 85 dB on the left. Bone conduction ABR waveforms for this patient are shown in Figure 8-23. Again, a clear and reliable wave I component was observed with the ipsilateral mastoid electrode array, but the contralateral array failed to yield a wave I. An ABR was recorded for bone conduction stimulation levels down to 10 dB on the right and 20 dB on the left.

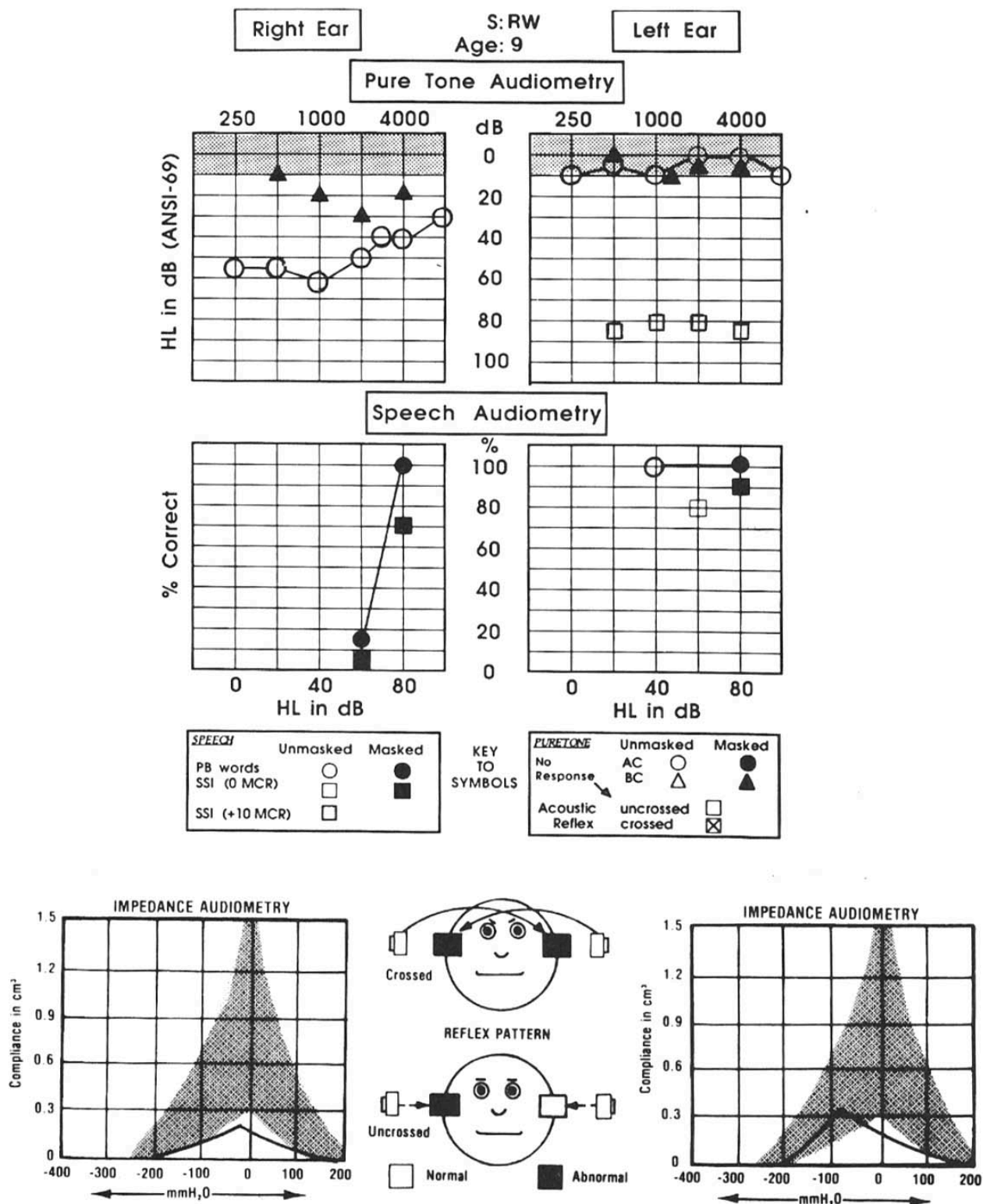


Figure 8-19. Audiometric findings for a 9-year-old boy with previously inconsistent test results classified as a malingerer (case 15).

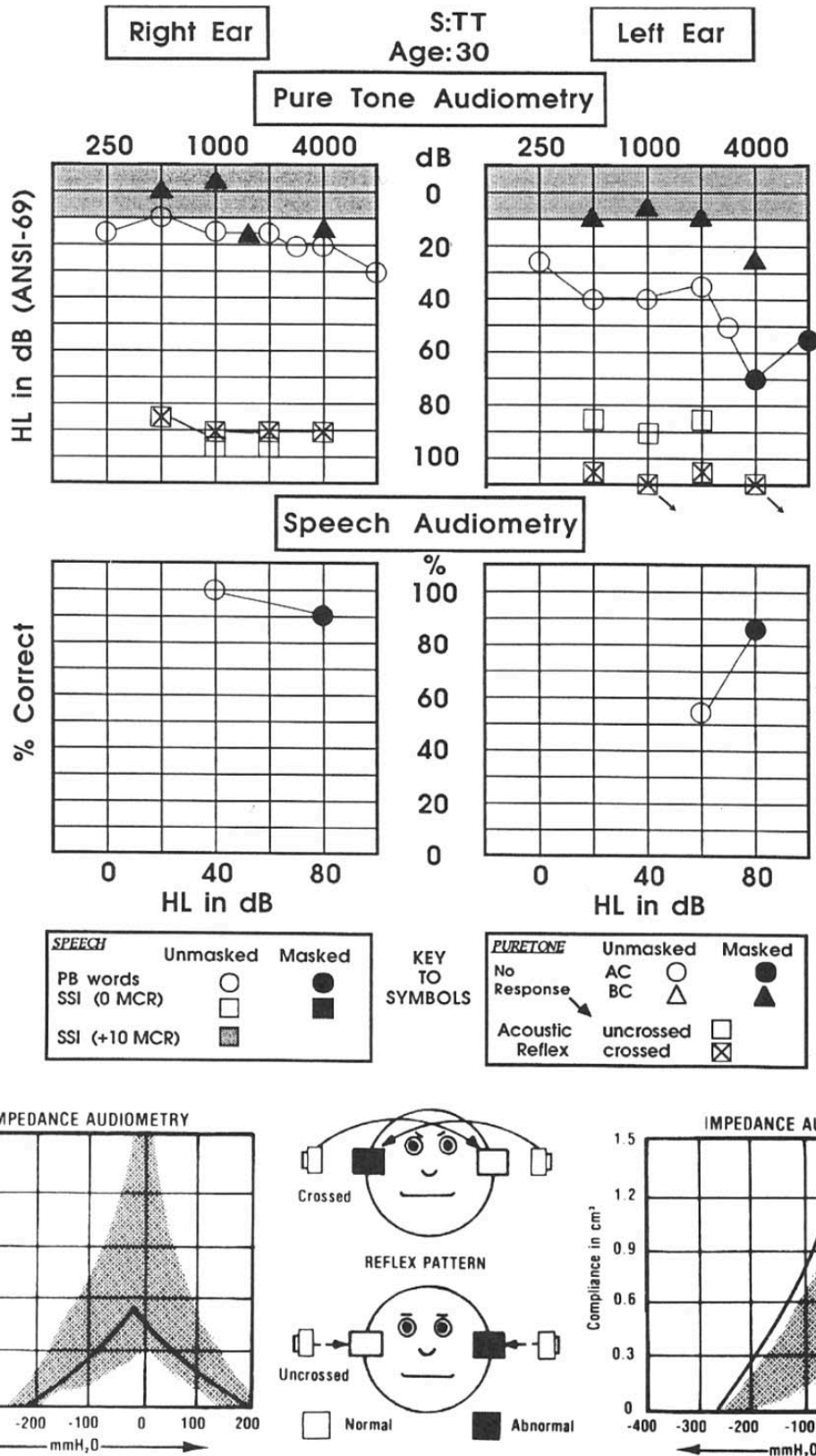


Figure 8-20. Audiometric findings for a 30-year-old man with ossicular chain discontinuity and functional connection (case 16).

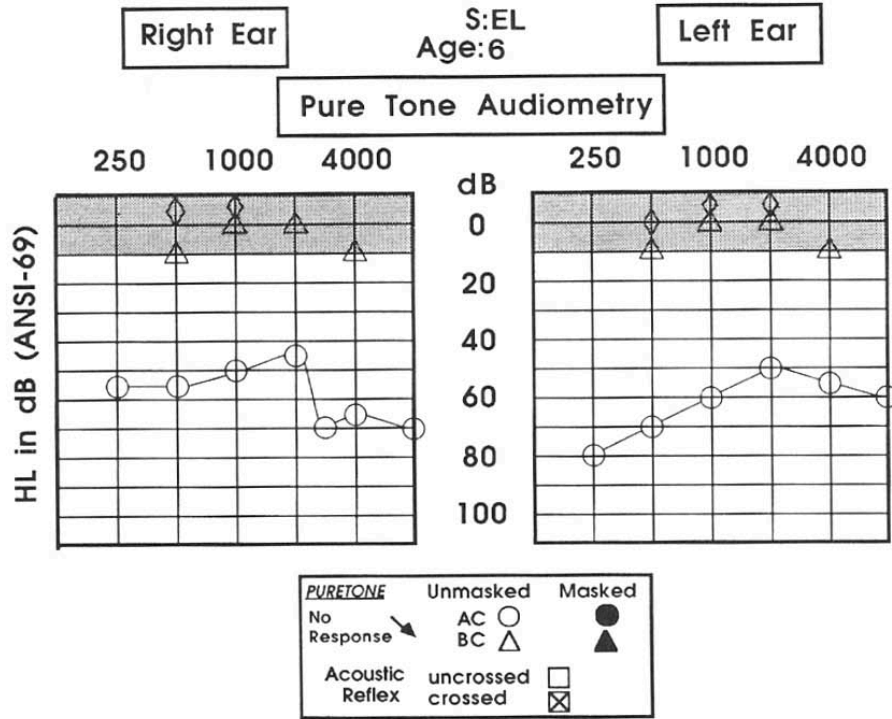


Figure 8-21. Preoperative pure tone audiometry findings for a 6-year-old girl with bilateral aural atresia (case 17).

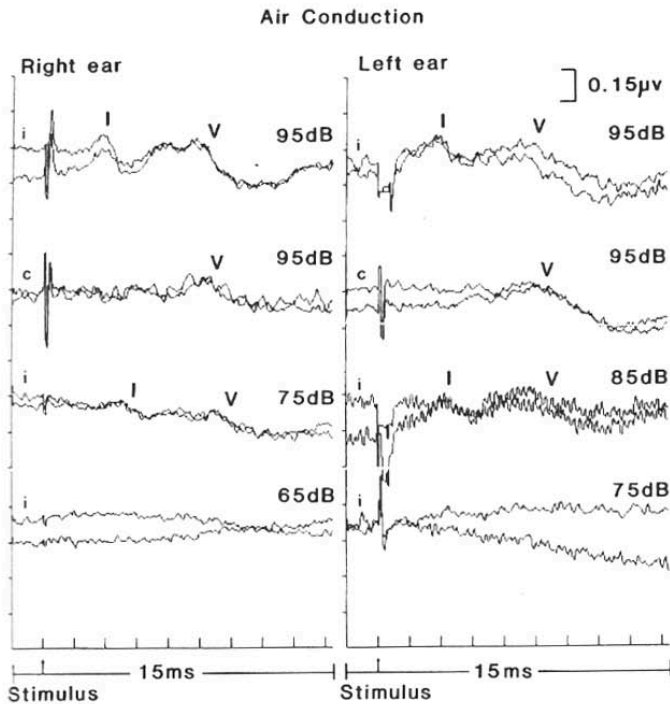


Figure 8-22. ABR waveforms for air conduction stimulation (case 17). Note presence of wave I component in ipsilateral but not contralateral electrode array.

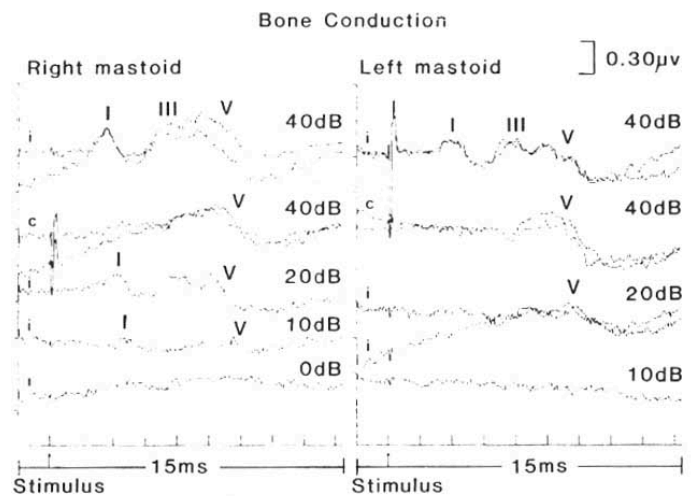


Figure 8-23. ABR waveforms for bone conduction stimulation (case 17). Note presence of wave I component in ipsilateral but not contralateral electrode array.

On the basis of these ABR results and computed tomography scanning, the otologist chose to operate on the right ear. That is, auditory sensitivity for air and bone conduction by ABR appeared to be better for the right than left ear; sensory sensitivity was within normal limits; and the computed tomographic scan showed less anatomic aberration for the right temporal bone. Postoperative pure tone average on the right ear at 3 months was 30 dB HL and speech awareness threshold was 15 dB HL.

CASE 18: SUSPECTED CONGENITAL CHOLESTEATOMA

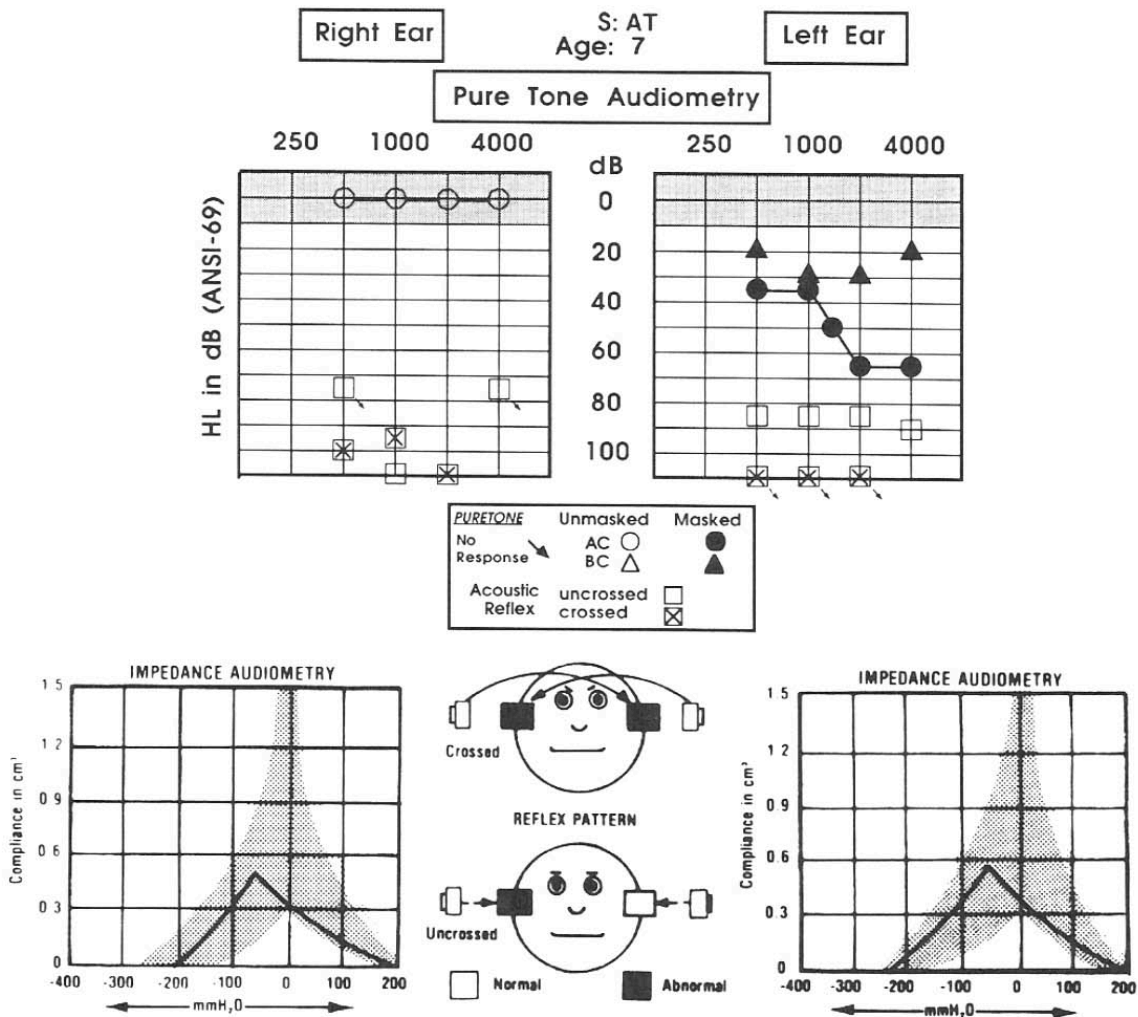
A 7-year-old boy was referred to the audiology service by the child development center for evaluation of hearing with reported speech-language delay and academic difficulties in school. He had a 4- to 5-year history of otitis media. He had failed a hearing screening in the center. With immittance measurement (Figure 8-24), tympanograms were normal bilaterally, but the acoustic reflex pattern was consistent with middle ear dysfunction on the left ear. Pure tone audiometry showed normal hearing sensitivity on the right and a mild to moderate sloping mixed hearing loss on the left ear. Audiometric Weber at 1000 and 2000 Hz was lateralized to the poorer left ear. Word recognition was good on the right and excellent on the left ear. Otologic consultation was initiated.

Physical examination of the ears revealed retraction pocket enclosing debris. An attic cholesteatoma in the left middle ear was suspected and surgery was scheduled. Exploratory tympanotomy showed that the retraction pocket did not extend into the epitympanum. Adhesions found around the lenticular process of the incus were removed. Hearing sensitivity was normal on postoperative audiologic evaluation.

CASE 19: CHRONIC EAR

The patient was a 6-year-old boy with extensive history of middle ear pathology. Tympanostomy (ventilation) tubes were first placed bilaterally at 6 months of age. Six other sets of tubes were placed since then. At age 2 years, a mastoidectomy and tympanoplasty were performed on the right side. The patient had been hospitalized 22 times for ear infections that resisted medical treatment. On audiologic assessment (Figure 8-25), each ear was free of tubes. Immittance measurement showed type B tympanograms and reflexes were not detected. There was a conductive component for frequencies through 4000 Hz. Hearing was best at 2000 Hz. Hearing sensitivity was decreased bilaterally above 4000 Hz. In the absence of bone conduction thresh-

Figure 8-24. Audiometric findings for a 7-year-old boy with congenital cholesteatoma (case 18).



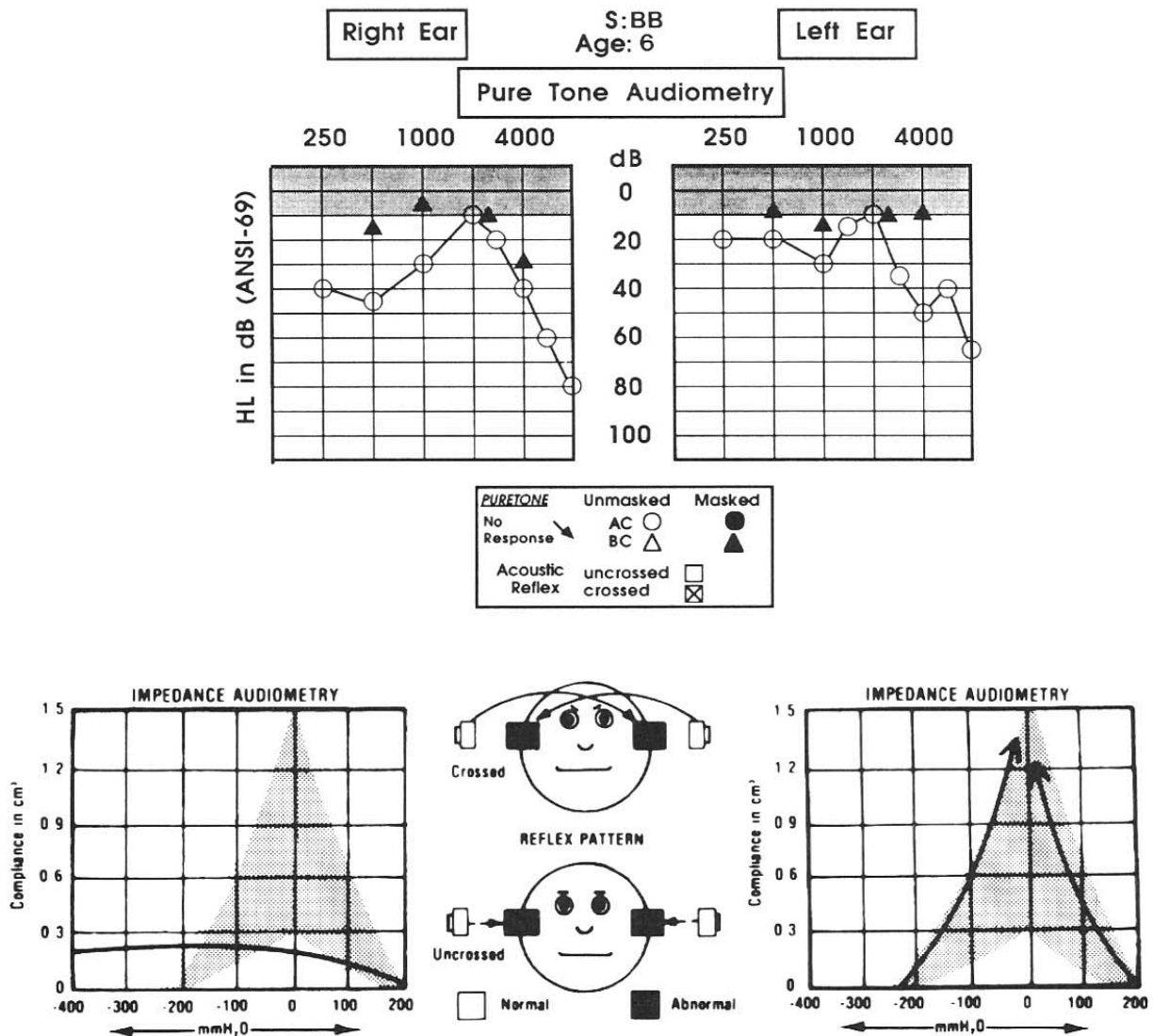


Figure 8-25. Audiometric findings for a 6-year-old boy with chronic middle ear disease (case 19).

olds, it was not clear whether it was conductive or sensory in nature.

Some investigators suggest that a mixed or sensory deficit at 4000 Hz or for higher frequencies may be a component of chronic otitis media (Dommerby and Tos, 1986); others theorize that bone conduction sensitivity is depressed by mechanical changes in middle ear functioning (Walby, Barrera, and Schuknecht, 1983). In any event, bone conduction thresholds may not always represent permanent sensory status. Furthermore, as this case illustrates, chronic otitis media may produce an unusual audiogram configuration.

AUDIOLOGIC MANAGEMENT OF PATIENTS WITH MIDDLE EAR PATHOLOGY

Patients with middle ear pathology constitute an important interface between audiologists and phy-

sicians, usually otolaryngologists or pediatricians. The audiologist may contribute to management of patients with middle ear pathology in four ways. The first is *detection of middle ear pathology* in patients entering the health care system through the audiologist, whether the setting is a school, a hearing center, a hearing aid dispensary, a nursing home, a hospital audiology clinic, or a newborn intensive care unit. Audiologists must have technical skills necessary for evaluating middle ear function and the clinical skills necessary for recognizing and describing varied types of middle ear pathology. *It is imperative that audiologists refer such patients for appropriate medical management before initiating audiologic management.* With most hearing-impaired patients with middle ear dysfunction, immediate audiologic management is at the very least inappropriate and, in many cases, contraindicated. Among them are pa-

tients with excessive cerumen, otitis media, tympanic membrane perforations and draining ears, and audiometric evidence of otosclerosis. These conditions are potentially medically and surgically treatable. The audiologist who fails to make a proper medical referral is not only doing the patient a disservice, but also risking medicolegal action and jeopardizing the credibility and reputation of audiologists in general as competent hearing health care professionals. Proper medical referral following detection of middle ear pathology should be viewed with the same seriousness as medical referral of patients with audiologic signs of retrocochlear pathology. Likewise, unexplained and, particularly progressive, sensory hearing loss warrant medical referral. Medical or surgical treatment, or both, may be effective for various etiologies causing sensory deficits (e.g., Ménière's disease or autoimmune pathology).

The second contribution is accurate *description of middle ear function* based on audiometric findings. Even the most complete and well-administered hearing evaluation is of little clinical value if findings are not promptly and accurately reported to the appropriate persons or acted on by the managing audiologist. Pure tone audiometry often permits classification of hearing impairments as conductive, sensory, or mixed. With the inclusion of immittance audiometry to the basic test battery, however, further and more precise description of auditory status is possible. Often, immittance measurements are carried out only to confirm the presence of middle ear dysfunction in patients with air-bone discrepancies by pure tone audiometry. This practice grossly underuses the diagnostic power of aural immittance. As indicated at the outset of this chapter, if the hearing evaluation begins with immittance measurement, it is usually possible to determine quickly and confidently whether hearing impairment has or does not have a conductive component. Bone conduction pure tone audiometry may not be necessary. If evidence of middle ear dysfunction by aural immittance exists, the pattern of tympanometry and acoustic reflex findings often leads to a precise description of the nature of the middle ear dysfunction and narrows down the possible underlying diseases. Then, suspicion of a conductive component by abnormal immittance is confirmed by careful comparison of air versus bone conduction pure tone audiometry.

A key factor in fully exploiting assessment of auditory status with this test battery approach is the precision with which results are described.

Unfortunately, precision is in fact discouraged with the somewhat more traditional approach for assessing audiologic status and reporting audiologic findings.

It is generally accepted that one cannot diagnose ear disease with audiometric findings. As noted in the beginning of this chapter, the results of a hearing evaluation may contribute, but the medical diagnosis is based also on the history, physical examination, and sometimes other information (e.g., laboratory or radiologic procedures). According to the traditional test report philosophy, therefore, the audiologist should carefully avoid any semblance of "making a diagnosis." Terminology used in reports is purposefully vague to accomplish this goal. Rather than endorsing this philosophy, the authors strongly submit that audiologists should take full advantage of the diagnostic power offered by certain procedures, such as immittance measurement, and report findings with as much precision and detail as possible. The principle that "a diagnosis is not made by audiologic tests" is not discarded with this more aggressive reporting style. Terminology and phrases are, in fact, specifically selected to avoid this inference.

Numerous examples of report writing "buzz words" and audiologic descriptors were provided among the case reports presented here. A brief review of audiologic data for an additional patient may serve to illustrate the unfortunate consequences of inadequate audiologic assessment and vague description of hearing status, followed by immediate audiologic management without proper medical referral. A self-referred young female adult was evaluated at a hearing center. She noted a gradual decrease in hearing over the previous 5 or 6 years. Pure tone audiometry showed a mild, flat hearing loss bilaterally. There was a slight air-bone gap in each ear, but the hearing loss was described as "sensorineural." Aural immittance was not done and there was no medical referral. The patient then underwent evaluation for hearing aid use and was fitted with amplification. She recently came to the first author's attention when she was referred for audiologic evaluation on a routine postoperative visit to an otolaryngologist following laryngeal surgery. The hearing impairment had progressed to the moderate to severe range. Immittance findings of type A tympanograms and absent acoustic reflexes, in combination with an air-bone gap and evidence of Carhart's notch, suggested otosclerosis. In contrast to the initial audiologic report,

findings for the second evaluation were described directly. Unfortunately, without the potential benefit from medical/surgical intervention over the preceding unknown number of years, the patient's hearing sensitivity had decreased markedly. An otologic referral was made. Pending any medical treatment, a hearing aid reevaluation was essential.

The third contribution of audiologists in patients with middle ear pathology is *pre- versus postoperative evaluation* of hearing status. The audiologic evaluation unequivocally and rather dramatically affects medical management and communicative or medical outcome in several types of patients. Among these are newborn infants with hearing impairment and patients with retrocochlear pathology. In persons with middle ear pathology, audiometric findings often are the most important factor in determination of surgical candidacy. The implication here is quite clear. Inaccurate or incomplete audiometric data may potentially rule out surgery for a patient who could in fact benefit. This outcome would result, for example, from incorrectly describing poor bone conduction thresholds or failing to report any valid bone conduction thresholds because of technical limitations (e.g., masking dilemma). Perhaps worse, audiometry may lead to surgery for a patient with little or no chance for benefit, and furthermore put the patient at risk for total loss of hearing on the operated ear. This outcome, of course, results from overestimation of the air-bone gap and, particularly, the assumption of relatively good bone conduction sensitivity (often because of inadequate contralateral masking) in an ear with a significant sensory component. Clearly, the most important single factor in preoperative evaluation is accurate and valid description of air versus bone conduction thresholds. In cases posing masking problems, the audiologist is obligated to first use all procedures at his or her disposal to obtain accurate and complete air and bone conduction pure tone audiometry results and then to employ a conservative approach to interpreting these results.

In addition to this concern about preoperative test accuracy, the audiologist must be cautious in commenting on postoperative audiologic findings. Some clinical audiologists probably do not devote an appropriate amount of time to patient counseling. For many patients, hearing test results are not adequately explained, and little or no guidance on dealing with their hearing impairment is provided. For patients who have under-

gone middle ear surgery, however, less explanation of hearing status by the audiologist is often better. The potential for long-term postoperative improvement in bone conduction hearing was vividly illustrated above by case 14. Remarks on the audiologic effects of surgery are certainly premature until repeated postoperative tests have demonstrated stable hearing status. Even then, the audiologist is advised to leave detailed explanations of audiometric test findings to the surgeon who is in a position to interpret audiometric findings in the context of intraoperative and postoperative physical findings. As a general rule, the audiologist who anticipates working closely with otolaryngology colleagues is advised to initiate an open discussion on policies for audiologic management of mutual patients, including counseling of patients after hearing testing.

The fourth, and final, contribution of the audiologist is *management of patients* with middle ear pathology that is not amenable to medical or surgical management. Although such patients are traditionally considered good candidates for amplification, multiple factors add to the challenge of audiologic management, including audiogram configuration, a draining ear, and fluctuations in hearing sensitivity. The patient with a serious conductive component often requires a creative and flexible management approach.

ACKNOWLEDGMENTS

Susan M. Tompkins, M.Ed., Charlotte H. Prentice, M.S., Deborah S. Wilson, M.S., and Lisa Sells contributed to selection of cases.

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- blue") behind ear (postauricular) in mastoid region, indicating temporal bone trauma.
- Bing test** Comparison of auditory function (e.g., audiometric hearing threshold or tuning fork response) with ear unoccluded versus occluded, usually for lower-frequency signals (e.g., 500 Hz). Enhanced hearing sensitivity in the occluded state is consistent with normal middle ear function. No occlusion effect is a sign of conductive impairment.
- Carhart's notch** Decrease in bone conduction hearing sensitivity at 2000 Hz in otosclerosis, named after describer audiologist Raymond Carhart. Carhart (1950) actually described typical bone conduction sensitivity reductions of 5 dB at 500 Hz, 10 dB at 1000 Hz, 15 dB at 2000 Hz, and 5 dB at 4000 Hz in this patient group.
- Cerebrospinal fluid** Clear liquid filling the ventricles of the brain and communicating with inner ear fluids via the cochlear aqueduct.
- Cerumen** Ear wax.
- Cholesteatoma** Secondary acquired cholesteatoma, occurring in infected ear or previously infected ears, is migration of squamous epithelium into the middle ear from the external meatus through a tympanic membrane perforation.
- Echymosis** Hemorrhagic (discharge of blood into) areas of the skin, producing a "black and blue" spot.
- Edema** Swelling of tissue due to fluid collection, often in response to injury.
- Glomus tympanicum** Tumor found in middle ear space consisting of blood vessels.
- Granuloma** Tumor of epitheloid cells.
- Hematoma** Collection of blood.
- Hemotympanum** Collection of blood behind eardrum in middle ear space, usually after head trauma.
- Iatrogenic** Disease or dysfunction inadvertently caused during management of a medical problem (e.g., laceration of ear canal walls during cleaning by a physician).
- Interaural attenuation** "Acoustic insulation" offered by the head. Signal intensity levels of 50 to 60 dB are required before pure tone signals presented via air conduction with conventional supra-aural earphone cushions cross over from the test ear to the non-test ear. Interaural attenuation for bone conduction signals is practically 0 dB (Chaiklin, 1967).
- Masking** Presentation of a sound, usually white noise or narrow-band noise, to the non-test ear to eliminate its participation in perception of a signal due to acoustic cross over (Sanders and

GLOSSARY

- Acoustic (stapedial) reflex** Reflexive contraction of the stapedius muscle elicited by an acoustic signal (pure tone or noise) of usually greater than 70 to 75 dB HL (Hall, 1985).
- Anotia** Congenital absence of external ear (auricle).
- Atelectasis** In otology, the collapse (retraction) of the tympanic membrane across the middle ear space and against the promontory (lateral wall of the inner ear).
- Auricle** Outer, visible portion of the ear (includes the pinna).
- Battle's sign** Bluish discoloration ("black and

- Rintelmann, 1964; Studebaker, 1967).
- Masking dilemma** Intensity level necessary to mask an ear when impaired hearing exceeds air conduction interaural attenuation (usually 40–50 dB) and masks the test ear (Naunton, 1960).
- Mastoidectomy** Surgical removal of contents of mastoid bone (within temporal bone just behind the ear) to eradicate infection.
- Meatus** External auditory canal opening or channel.
- Microtia** Abnormally small or malformed auricle (outer ear).
- Necrosis** Death of tissue or bone (with destruction).
- Occlusion effect** Improvement in bone conduction hearing when the ear is occluded. Effect is on the order of 15 to 25 dB for lower frequencies (250 and 500 Hz), 10 dB or less at 1000 Hz, and negligible above 1000 Hz (Goldstein and Hayes, 1965).
- Osteoma** Benign bony tumor (arising from the ear canal walls).
- Otitis externa** Infection or inflammation of the external ear.
- Otitis media** Inflammation of the middle ear (see *Serous otitis media* and *Purulent otitis media*).
- Otosclerosis** Bony disorder (otospongiosis) of otic capsule (bony labyrinth) producing conductive and sometimes cochlear hearing impairment.
- Purulent otitis media** Bacterial infection in middle ear space characterized by pain, fever, reddish tympanic membrane, and, with exudation, tympanic membrane bulging.
- Schwartz's sign** Dilated blood vessels (reddish appearance) on promontory in active otosclerosis.
- Sensory acuity level (SAL)** Test of bone conduction hearing in which the air-bone gap is determined by subtracting air conduction hearing thresholds for pure tone or speech signals recorded with no masking from those recorded with masking noise (approximately 50–60 dB HL) presented to the forehead via bone conduction (Jerger and Jerger, 1965).
- Serous otitis media** Acute nonsuppurative otitis media characterized by sudden appearance of nonpurulent effusion (fluid) in the middle ear.
- Sign** Results of tests that are used in differential diagnosis (e.g., an air-bone gap in pure tone audiometry).
- Suppurative otitis media** Follows spontaneous rupture of tympanic membrane (after purulent otitis media). Small perforation of tympanic membrane is observed in pars tensa portion.
- Symptom** Patient complaint or description of a physical problem that is used in differential diagnosis (e.g., tinnitus).
- Tinnitus** Spontaneously occurring sensation of sounds (e.g., ringing, buzzing, clicking) that sometimes is localized to one or both ears.
- Toynbee test (maneuver)** Patient swallows with nostrils occluded (e.g., pinched). With the expected effect, negative pressure in the nasopharynx is transmitted via the eustachian tube to the middle ear space. This effect is observed otoscopically as tympanic membrane retraction or tympanometrically as negative pressure compliance peak.
- Tympanic membrane** Ear drum.
- Tympanogram** Measure of middle ear mobility (e.g., compliance or admittance) as a function of changes in air pressure in the ear canal.
- Valsalva test (maneuver)** Patient attempts to inflate middle ear space by blowing air against occluded nostrils and closed mouth. The expected effect on tympanic membrane (outward movement) and middle ear pressure (increase) can be observed otoscopically or recorded with tympanometry.
- Ventilation tube, P.E. tube, or grommet** Pressure-equalization device inserted in tympanic membrane.
- Weber's test** Audiometric or tuning fork procedure in which the patient lateralizes a pure tone signal (low frequency) presented via bone conduction to the forehead.