Chapter 175: Conductive Hearing Loss

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Conductive hearing loss occurs when the outer or middle ear structures fail to optimally capture, collect, or transmit sound from the environment to the cochlea. Medical and surgical advances, particularly in microsurgical instrumentation, prosthetics, and technique, have made most types of conductive hearing loss amenable to treatment with a high probability of success. To fully appreciate the pathogenesis of conductive hearing loss and, more important, to recognize the advantages and possible limitations of certain rehabilitative methods, the modern otologist must be intimately familiar with the regional anatomy. Further, the otologist must develop a working understanding of the physiologic mechanisms that enable the remarkably efficient transfer of sound energy through the outer and middle ear structures to the cochlear fluid system.

This chapter provides a basis for the development of such understanding. Many comprehensive presentations of the regional gross and microscopic anatomy exist, and there is no lack of physiologic literature; we have included some of these as reference material at the end of the chapter. Moreover, we recognize that the practicing otologist is usually not engaged in the advancement of either anatomic or physiologic science, but in the application of such sciences. Unfortunately, some of the scientific information that is most important to good medicine and surgery is often lost in the arithmetic that is used in its explanation. The purpose here is to give the scientific and analytic findings a meaning that is directly useful to practitioners in the surgical and medical management of conductive hearing loss. Our presentation helps translate the mathematical relationships into visualizations that can more directly guide such surgical and medical intervention.

Another purpose of this chapter is to briefly review the diagnosis and management of entities that cause conductive hearing loss. The chapter begins with an overview of disease and dysfunction, and then discusses the anatomy, physics, and physiology of the normal ear and how they relate to a dysfunctional conductive system. We have included the phylogeny of the ear so that the surgeon will be as mindful of the embryologic origins of the altered tissues as he or she is of the physics of the structure being repaired.

Anatomic Considerations

The auricle has only a limited effect on the transmission of sound waves. Nevertheless, depending on the degree of incidence to which it is positioned to the head, as well as the fine contour of the cartilaginous framework, sound volume and quality may be modified.

The pinna begins to form at about the fourth week of embryologic development. Here tissue aggregates are noted at the distal end of the first branchial groove. These tissue groupings are referred to as *hillocks* and *number six.* Over the next 4 months the form of the mature auricle is attained, with distinct clefts, folds, and angulations that are characteristic of the pinna. While variations do appear in the literature, the first hillock is associated with the tragus, the second and third with the helical root, the fourth and fifth with the antihelix, and the sixth with the antitragus.

Concomitant but independent development during this period results in the formation of the external auditory canal and the middle ear space.

The external auditory canal develops from the dorsal segment of the first branchial groove. Medial to this, the first pharyngeal pouch is growing laterally as the tubotympanic recess, and during the second gestational month the ectoderm of the groove juxtaposes the endoderm of the recess. Mesoderm will be noted to efface both surfaces and act as a barrier. A solid meatal plate develops as a cord of epithelial cells grows into the primitive external auditory canal. Beginning in the fifth month of gestation the solid epithelial cord begins to cannulize as the tympanic ring, fused and elongating, begins to mature. By the seventh month a patent external auditory canal is noted. The external ear canal develops at about this time, when the middle, outer, and inner ear structures are developed. Abnormalities of the pinna and external auditory canal result from first branchial groove developmental anomalies.

The first pharyngeal pouch forms the tubotympanum and begins at approximately the third week of embryologic development. Endodermal tissue of the dorsal end of the pouch will eventually form the middle ear space and mastoid compartment. The growth of four sacci will define the pneumatized space and envelop the ossicles. Interfaces will form mesenteric folds, which are responsible for the transmission of blood vessels. Although the middle ear cleft is developed by week 30, the hypotympanum will continue to develop. The antrum is pneumatized by birth, with continued growth of air cells into the mastoid until age 19 (Schuknecht and Gulya, 1986). Bone fixation in the epitympanum may result from incomplete pneumatization.

The ossicular chain is related to the gill arch system of lower animal forms. The mammalian malleus and incus are arch derivatives originally found to be part of the jaw apparatus, and the stapes may be traced to the reptile columella auris arising from the blastem of the hyoid bar.

Early mesenchymal material at 1 month of gestation is recognized as the initial growth source for the ossicular mass. The ossicular chain undergoes endochondral bone formation. By week 15, the ossicles have achieved adult size and ossification begins.

Because the developing fetus is subjected to a multiplicity of potential developmental interruptions, abnormal development at specific gestational stages will result in external or middle ear defects and contribute to an eventual conductive hearing loss.

Pathologic Dysfunction

Despite a number of entities that may contribute to dysfunction of the sound-conducting mechanisms, there are fortunately only a limited number of dysfunctional pathophysiologic processes that cause a conductive hearing loss, including the following:

1. Obstruction of the external auditory canal.

2. Perforation of the tympanic membrane.

3. Fixation of the ossicular chain with or without fixation of the drum.

4. Discontinuity of the drum-ossicular chain mechanism.

5. Significant negative middle ear pressures.

6. Congenital abnormalities.

Obstruction

The normal external auditory canal has an average diameter of 6 mm and an average length of 25 mm; it has a resonance frequency of approximately 3500 Hz, and its reverberatory contribution to sound transmission is approximately 10 dB near the resonance frequency. There is normal variation in the length of the external auditory canal, resulting in altered resonance frequencies; shorter canals have frequencies above 3500 Hz. *Partial* obstruction may distort the quality of the perceived sound without demonstrating a measurable effect on sound pressure level. *Complete* obstruction may cause a conductive loss of as much as 30 dB. Variable loss of hearing levels may be observed as the obstructing process causes immobility of the tympanic membrane, as well as direct occlusion of the canal.

Several different types of processes can obstruct the external auditory canal and cause a conductive hearing loss; these are outlined on the next page.

Box: Conductive hearing loss - pathophysiologic conditions

Infection

Otitis externa
Acute otitis media
Furunculosis
Otomycosis
Malignant otitis
Myringosclerosis
Keratosis obturans
Herpes zoster oticus
Chondritis
Perichondritis
Cellulitis
Erysipelas
Bullous myringitis
Tympanosclerosis
Serous otitis media
Chronic otitis media
Cholesteatoma
Tuberculous otitis
Syphilis

Trauma

Burns
Foreign body
Barotrauma
Cauliflower ear
Auricular hematoma
Laceration with clot
Laceration without clot
Avulsion
Longitudinal fracture
Transverse fracture
Mixed fracture
Penetrating wounds

Congenital - hereditary

Anotia
Microtia
Cup ear deformity
Treacher Collins syndrome
Mohr's syndrome
Otofacial cervical syndrome
Otopalatal-distal syndrome
Pierre Robin syndrome
Pyle's disease
Crouzon's disease
Apert's disease
Goldenhar's syndrome
Turner's syndrome
Osteogenesis imperfecta
Osteopetrosis
Paget's disease
Achondroplasia
Marfan's syndrome
Mucopolysaccharidoses

Tumor

Basal cell carcinoma
Squamous cell carcinoma
Melanoma
Hidradenoma
    Adenoma
    Pleomorphic adenoma
    Adenoid cystic adenocarcinoma
Osteoma
Sebaceous cell carcinoma

Paraganglioma
Neurofibroma
Rhabdomyosarcoma
Exostosis
Hemangioma
Hemangiopericytoma
Lymphangioma
Leukemia
Multiple myeloma

Systemic - metabolic

Cerumen
Keloid
Wegener's granulomatosis
Relapsing polychondritis
Fibrous dysplasia
Eosinophilic granuloma: Hand-Christian-Schüller disease
Polyarteritis nodosa
Sarcoidosis

Iatrogenic

Surgical failure
Traumatic cleaning

Idiopathic

Otosclerosis.

Acute and chronic otitis media or externa can result in inflammatory tissue that can completely occlude the meatus. For example, a granulation tissue polyp can occur in response tomany forms of suppurative ear disease and present in the external auditory canal. With complete obstruction a conductive hearing loss results. The treatment, obviously, depends on proper diagnosis of the specific cause of the granulation tissue, with therapy directed against that cause. Similarly, chronic inflammatory changes, whether from chronic external otitis or after surgery, can lead to membrane formation lateral to the tympanic membrane, or to dramatic thickening of the tympanic membrane with conductive hearing loss. Once an obstructive membrane has formed, surgery is necessary to correct it.

Probably the most common cause of conductive hearing loss in adults is complete blockage of the external auditory canal by a **cerumen impaction.** This can be easily removed in nearly all cases by any of a number of mechanical techniques, depending on the consistency of the cerumen, physician preference, and patient tolerance.

Aural **foreign bodies** as a cause of conductive hearing loss are essentially restricted to children and the mentally disturbed. Their safe removal requires delicacy, good equipment and illumination (otomicroscope), and patient cooperation or general anesthesia.

Rarely, **tumors** entirely occlude the external auditory canal before diagnosis, and conductive hearing loss prompts the patient to seek medical advice. Malignant tumors are extremely rare in this regard, since their other symptoms are usually evident earlier. Treatment varies with the specific neoplasm.

Benign tumors include both neoplasms and nonneoplastic growths. **Keratosis obturans** and **external auditory meatus cholesteatoma** (EAMC) are the most prominent examples of the latter. In these rare conditions desquamated keratin debris accumulates in the external auditory canal and, when completely obstructive, causes conductive hearing loss. In both conditions failure of the normal epithelial migration, which normally propels desquamated keratin from the tympanic membrane laterally out the external auditory canal, appears to be at least in part involved with the pathogenesis. Sismanis et al (1989) have recently reviewed these two related entities and present some features that help differentiate them. Opinion is divided regarding treatment for these lesions, and since cases are few, it is difficult to assess results. General opinion favors meticulous removal of the accumulated debris at monthly intervals initially until accumulation slows and intervals can be lengthened. Long-term follow-up is important to assess bone erosion; surgical contouring of the external auditory canal or canal wall down mastoidectomy are occasionally necessary.

Exostoses are the most common solid tumors of the external auditory canal; they occur most commonly in men with a history of repeated swimming in cold water, are multiple and bilateral, and appear as smooth, rounded nodules of bone covered by normal skin and attached by a broad base to the osseous canal. They are usually asymptomatic unless impending occlusion of the external auditory canal results in keratin accumulation and infection. If they are large or symptomatic, excision is appropriate.

Osteomas are less common pedunculated bony lesions that are usually single and commonly occur along the external auditory canal suture lines. Small lesions can either be excised by chiseling or drilling across the pedunculated attachment, or observed at annual intervals to determine whether there is enlargement, which would favor removal. Osteomas that are large when initially seen should be removed before near-total occlusion makes removal much more difficult.

Even more rare are **solid benign tumors** that arise in the external auditory canal and produce conductive hearing loss by totally occluding it. These include adenoma and ceruminoma; treatment is wide local excision.

Perforation of tympanic membrane

A defect in the tympanic membrane will result in the loss of airborne sound-receiving surface area and may also cause sound distortion at its torn edge. Clearly, the size, location, and nature of the perforation will affect the degree of hearing loss. Certain perforations will not present sound waves to both the oval window and the round window concordantly. Conversely, in cases where the phase difference between the oval and round windows is lost, a more significant hearing loss may make itself manifest. The hearing loss associated with perforations, however, tends to be affected by low-frequency sound. Studies in both animal models and human clinical cases, however, demonstrates that a pattern of loss can occur in all frequency ranges and that the configuration of the perforation will affect the degree of loss

(Tonndorf and Khanna, 1970). Moreover, the status of the ossicular chain with a tympanic membrane perforation will affect the degree of loss (Austin, 1990). Furthermore, Austin notes that in a particular case of tympanic membrane perforation and ossicular disruption, which had an average loss of about 38 dB, 19.5 dB of the loss could be ascribed to the absence of the hydraulic lever, 7.3 dB of the loss could be ascribed to loss of the catenary lever, and 5 dB of the loss could be attributed to phase cancellation between the round and oval windows.

Ossicular fixation

A decrease in the mobility of the ossicular chain due to fixation creates a clinical situation wherein the mass and stiffness of the middle ear transformer mechanism are increased. With fixation the ossicular complex will have greater resistance to sound energy forces in both its compliance-dominated low frequencies and in its inertia-dominated high frequencies; a greater sound energy force will be required to transmit a given sound pressure level. Depending on the underlying pathologic process, the tympanic membrane may show reduced compliance when evaluated by pneumatic otoscopy or by tympanometry.

Normal ossicular motion can be restricted by physical fixation, such as occurs with abnormal bone with otosclerosis, hyalinized connective tissue with tympanosclerosis, or scar tissue after surgery or infection, or may be passively restricted by pressure on parts of the ossicular chain from middle ear structures.

Otosclerosis is treated fully in Chpater 170.

Tympanosclerosis represents submucosal hyalinization that occurs exclusively in the tympanic membrane (myringosclerosis) or that involves the mesotympanum and epitympanum as well, often encompassing the ossicles. Prerequisites for its formation appear to be infection and healing. In the tympanic membrane the risk for development of tympanosclerosis increases when otitis media is treated with the placement of ventilating tubes, although rarely is hearing affected by tympanosclerosis restricted to the tympanic membrane. The conductive hearing loss that results from tympanosclerotic fixation of ossicles is difficult to treat successfully. Medical therapy is ineffective in reversing the process, and surgical treatment has limited success for long-term improvement in hearing. Vascularity is poor, and there is a strong tendency for the process to recur after excision.

Cholesteatomas cause conductive hearing losses by several mechanisms, but direct pressure of the cholesteatoma sac on the ossicular chain often contributes to the loss. Similarly, glomus tympanicum and occasionally glomus jugulare tumors can impinge on the stapes and produce a conductive hearing loss. Rare middle ear tumors, such as meningiomas and schwannomas, can do the same. Even normal structures in abnormal locations, such as a jugular bulb with no bony covering projection high into the middle ear cavity, can impair stapes motion.

Ossicular discontinuity

Because the incudostapedial joint is suspended in the middle ear and relies on the passage of blood vessels along the incus long process and suprastructure of the stapes, this joint is at great risk of being devascularized when infection or trauma occurs. When the

incudostapedial joint is separated completely, a maximal conductive hearing loss will occur when the tympanic membrane is intact. With a perforation the phase difference between the oval and round windows may be preserved, and a maximal conductive hearing loss will not be made manifest. In contradistinction to a fixed system, the tympanogram or pneumatic otomicroscopic examination may show abnormally high compliance and excessive excursion. In general, ossicular dissociation presents as a flat conductive loss; however, Anderson et al (1964) have shown that when the incudostapedial joint space is maintained by soft tissue fibrous attachments, the low-frequency component of sound will be effectively transmitted, whereas high-frequency conduction may be lost.

Negative pressure

Goode (1980) notes that negative middle ear pressure ≤ 100 mm H_2O) will result in a reduction of tympanic membrane excursion and states as an axiomatic note that "the worse the negative pressure, the worse the hearing". As an extension, atelectasis will gradually result in the loss of middle ear volume, and pressure will need to be increased to maintain a given level of hearing threshold.

Congenital malformation

Because they are derived from different embryologic tissues, the auricle, external auditory canal, and middle ear space with ossicles may each develop without full maturation of the other sites (Anson and Donaldson, 1981). Conductive hearing losses that result from congenital aberrations may range from mild to severe, with maximum losses occurring in cases of bony atresia.

Pathologic Lesions

The box outlines the common pathologic lesions that may interrupt the conduction of sound from the environment into the middle ear and to the footplate and cochlea. From a mechanistic perspective it should be appreciated that the previously noted pathophysiologic dysfunctions will result in a conductive hearing loss and that perhaps with the exception of otosclerosis, an exogenous source contributes to the loss.

Nature of Sound and Physiology of Conductive Sound Transfer

Wave mechanics and rigid-body dynamics are two ways in which the mechanical transfer of energy can be categorized; our study of the conduction processes of the ear uses both viewpoints. The collection and direction of airborne sound energy by the outer ear and the conversion process at the tympanic membrane lend themselves to a description using wave mechanics. Once the airborne sound pressure has been converted into forces on the rigid bodies of the ossicular chain and its supporting structures, the mechanical transfer of sound is well described in terms of classical rigid-body dynamics. Separation of the conduction processes permits us to see clearly why the structures have the shapes that nature gave them, and it shows what parts of the structures are susceptible to functional compromise either by disease or by surgery.

We retain our perspective of providing a *feel* for the transmission of sound in this section. We hope that we can provide a better understanding of the underlying rigorous mathematics by using simple examples of the meaning of each equation; further, as we cite some of the better-known physiologic studies, we hope that we can enhance their meaning to the clinical practice.

Wave character of sound transmission

Sound is mechanical vibration within the range of approximately 20 Hz to 20 kHz. The transmission of sound energy in an acoustic medium is accurately described by a partial differential equation known as the *wave equation:*

$$(d^2y)/(dx^2) = k^2(d^2y)/(dt^2)$$

The wave motion described by differential equation is representative of many physical situations; the reader has no doubt been exposed to bobbing corks and ripples in water or to the coil motion of a Slinky, both examples being analogous to the transmission of sound. Another analogy described by equation 1 is the "stadium wave" (of football fame).

The motion analogy has been developed with the assumption that the source of the stadium wave is stationary; that is, it is initiated by a fan sitting at the far end of the row of seats. To test your understanding of sound motion and pressure, consider now that the source of the wave is moving, and that multiple pulses are initiated. Let the initiator of the motion be a vendor who walks much slower than the propagation velocity of the wave, and let him pulse every fan as he walks by the fans in the row of seats. Each spectator in the row will feel the pulse *transferred* to him by his neighbour, and he will also feel the pulse initiated on him by the vendor. As long as the vendor's speed is less than the propagation speed of the disturbance, each successive spectator will feel an increasing stackup of pulses and a corresponding increase in crowding. However, when the vendor increases his walking speed to the point where he exceeds the propagation speed of the wave, he will reach a point where the pulse that he imparts on the fan will be *ahead* of the propagating stackup of crowding. At such a point an abrupt change in crowding occurs, going from a maximum to a minimum in the space of one spectator. Assuming that the vendor reaches some top speed, then the view from across the stadium will show the vendor out ahead of an intense stadium wave that hits successive spectators with a great shock. A jet plane does the same thing when it "breaks the sound barrier".

The stadium wave analogy enables a clear visualization of the distinct motion in sound and how these distinct motions relate to pressure and acoustic impedance. The stadium wave is initiated by a spectator raising his hands above his head, lowering them, and signaling for the adjacent spectator to do the likewise. If you get the visual image, you will see *two* motions: a disturbance of any fan raising his hands up and down in his chair and a second motion associated with the changing of the place of the disturbance. The y in the equation represents the disturbance, specifically the position of any fan's arms up or down. The x in the equation represents the location of any fan (or his seat) in the row of seats. The t in the equation is time. The equation says that the *second differential* relationship between the disturbance and its location along the seats is equal to the *second differential* relationship between the disturbance and time - all to within a constant, k^2.

There are many individual solutions to the wave equation. The stadium wave in equation 1 is characterized by a wave function that describes a pulse, that is, the propagation of a single disturbance event. Another solution to the wave equation is:

$$y(x, t) = A \sin (2pft - 2 pf(x/c))$$

In the stadium wave analogy this equation corresponds to each fan continuing to move his hands with a constant harmonic motion once he has received the signal from his neighbor. A is the amplitude of how high or low the individual spectator swings his arms about some midpoint. In this equation the motion of the disturbance *and* the motion of the wave are sinusoidal in nature, and they are both dependent on the frequency of the disturbance, f. The disturbance motion (analogous to the hand waving) is the portion related to 2pft, and the propagation motion is identified by 2pfx/c. The x/c has physical meaning as the distance along the seat divided by the speed of propagation. In describing sound waves, equation 2 is descriptive of the transmission of a continuous pure tone of sound having a frequency of f and a molecular disturbance whose amplitude is A; c is the speed of sound in a particular medium.

Equation 1 also is descriptive of a stadium wave in which the spectators move from side to side instead of up and down in their seats at the appointed time. When the disturbance is viewed from across the stadium, a moving wave disturbance still appears, but this time the stadium wave is seen as a ripple *in the direction of the motion of the wave.* In terms of the wave equation, the motion term y is measured in the *same* direction as x, that is, longitudinally. Sound is transmitted by such *longitudinal* wave motion, and in equation 2 y is the position of any molecule that oscillates back and forth about a point located at a distance *x* from the point of the initial disturbance. The time of the observation is identified by the variable, *t.*

Sound pressure and particle velocity

The relationship between sound pressure and the wave equation has been a source of confusion to even the best researchers in this field (Békésy, 1960; Pickles, 1982). The difficulty arises because it seems that sound pressure should be directly related to *displacement* (such as with a piston). Remembering, however, that the wave equation identifies two motions, a *disturbance* motion and a *propagation* motion, it is easy to visualize why pressure relates to particle velocity. Like the spectators participating in the stadium wave, there is a crowding of mechanical bodies at the exact point where the wave is propagating. If the whole row of fans moved in unison, there would be no wave propagation and no opportunity for such crowding. However, once the wave propagation has been created, the intensity of the crowding at the point of disturbance is dependent on how fast and how far the individual spectator moves when it is his appointed time to pass on the disturbance. In sound-carrying media, molecules are like spectators, and crowing is pressure. It is not surprising that sound pressure should be related to local motions of particles. In elementary physics the relationship between molecular *velocity* and static pressure is well accepted. That the dynamic pressure of an acoustic wave should relate to particle disturbance velocity is in perfect keeping with the laws governing static gas pressures. Carrying the description back to solution equation 2 of the wave equation, the velocity of the disturbance is the first

(partial) derivative of *y(x,t)* with respect to time:

$$p = K2\ (dy(x,t)\ /dt)$$

which, when applied to equation 2 solvers as:

$$p = K2\ 2pfAcos(2pft - 2pf(x/c))$$

where p is pressure, K2 is at most a conversion constant between descriptors of pressure and A is the amplitude of the local disturbance. The magnitude of the pressure, pa, is the combination of the first several terms on the right-hand side of the equation:

$$Pa = K2\ 2pfA$$

The presence of the frequency term f in the amplitude shows that to maintain a given pressure, a source of low-frequency sound must swing with greater amplitude than a source of high-frequency sound.

Acoustic impedance

Air and perilymph have very different acoustic impedances or characteristics with regard to the way they transmit sound. Whenever two media that have different acoustic impedances are in contact, there is both reflection and transmission as the sound energy wave passes from one to the other. If sound in air had to pass directly into the fluid of the perilymph, more than 99.9% of the acoustic energy would be lost to reflection (Wever and Lawrence, 1964). The primary purpose of the conductive structures of the middle ear is to efficiently transfer acoustic energy from air to the fluids encased within the cochlea. In the literature the middle ear is referred to as functioning as an *impedance-matching transformer*. We will return to the middle ear impedance-matching transformer presently, and we will dissect its functional attributes in a surgically meaninful way. First, we have to understand what is meant by acoustic impedance.

Returning to the stadium wave analogy, the speed of propagation of the stadium wave is a function of the mass and resilience of the spectators, and it is easy to see that such a mechanical system of humanity has a characteristic speed for "passing on" the disturbance. In the old days of collegiate ball, when there were east and west bleachers, there was a definite limit to the extent of a stadium wave. If the bleachers were walled on their ends, then the last spectator in the row would "pass on" the disturbance to a brick wall, and the stadium wave (and possibly the fan) would be terminated. In terms of physical mechanics, the wall possesses high impedance, and the fan low impedance. The resulting impedance mismatch causes the reflection or destruction of most of the energy being transmitted in the stadium wave.

When acoustic energy propagates through a medium, it is affected by characteristics of the medium, primarily the bulk, modulus of elasticity (springiness) and density in fluid media, and also by the physical boundaries of the medium. It is convenient to formally characterize such effects in measurable, cause-and-effect ways. *Specific* acoustic impedance is easy to describe because it expresses a ratio between pressure (a cause) at a point in an

acoustic medium and the effective particle velocity (an effect) at that point. Its mathematical expression is given by:

$$\mathbf{Zs = p/u}$$

While equation 6 is conceptually easy to understand, it has historically not been easy to measure. A more useful, and directly measurable, expression for acoustic cause and effect deletes the word *specific.* Acoustic impedance is defined at a given surface as the ratio of the effective sound pressure averaged over that surface to the effective volume velocity through it. Its mathematical representation is given by:

$$\mathbf{Za = p/U}$$

The given surface may be a theoretical surface or a physical surface such as a piston or a membrane. The term U represents volume velocity, which is made up of the area of the given surface multiplied by the average speed of a particle on that surface.

Impedance-matching transformers and middle ear

Impedance-matching transformers permit the propagation of energy from a medium with one characteristic impedance to a medium with another. Returning to our much-used stadium analogy one last time, if the fan next to the wall had somehow obtained a very long lever to pry the foundation, then a great excursion of his motion on his long end would have resulted in a very great force at the foundation - conceivably enough to move the wall. The energy (being the product of force multiplied by distance) would be the same at the spectator end and at the foundation end of the lever. The impedance-matching transformations of the middle ear are much like the stadium lever: a large tympanic membrane to capture the soft disturbances of airborne sound and a small oval window interfacing the perilymph.

The operative middle ear transformer is identified in contemporary literature as having three components: (1) the area ratio of the tympanic membrane to the oval window, (2) the lever ratio formed by the effective lengths of the manubrium of the malleus and the long process of the incus, and (3) a contribution due to the changing of the surface shape of the tympanic membrane. There is disagreement regarding the exact numbers in the impedance transformation rations, but it is on the order of 185:1 and it is dominated by the area ratio component (Pickles, 1982). More important than knowing the exact ratio is knowing how each element in the middle ear transformer works and the susceptibility of each to disease, surgery, and postsurgical scar.

We begin with the tympanic membrane. The tympanic membrane lies in an oblique plane at the medial termination of the auditory canal. Unlike most of its textbook presentations, the membrane is almost a continuation of the posterior wall of the external canal. The tympanic membrane is supported on its anulus by the tympanic ring, and the membrane provides partial support to the malleus, enveloping the manubrium. The membrane is remarkably compliant because of the curvature in its surface - the so-called catenary waves seen in sections transverse to the axis of the malleus. The compliance permits the membrane to undergo the large excursions necessary to transmit low-frequency sound energy. If the membrane were flat, or even if it were a conical shape *without the catenary-like cross-*

sectional curves, then very small displacements would cause very high membrane stresses. The surface curvature avoids the generation of such stresses. Conversion of air pressure to a force on the ossicular chain is obtained by an integration of localized pressures on the membrane surface in much the same way that a sail imparts the integrated force from the wind on the mast of a boat. The integration process is evident in the very well known holographic studies of Khanna and Tonndorf (1972), wherein the dramatic bull's-eyes are less telling of the total force transmitted than are the relatively few numbers of fringes crossing the manubrium of the malleus. The latter are indicative of the presence of a rocking motion in the manubrium of the malleus at the acoustic frequencies studied. The *effective* area of the tympanic membrane is a way to compare the force exerted by the tympanic membrane as if it were a flat-topped piston; the effective area of the tympanic membrane is about 65% of its total anatomic area of 85 mm52^2 (von Békésy and Rosenblith, 1951).

The bones of the ossicular chain move as *rigid bodies,* a term we introduced earlier without much definition. In a solid body, sound has two possible ways of being transferred: by the vibration of the body as a whole or by conduction through the body by wave mechanics. Generally, if the solid body has dimensions that are much, much shorter than the wavelength of sound, the solid body will transmit mechanical motion as gross movement of the entire body (Timoshenko and Goodier, 1951). Nonetheless, the *rigid-body motions* of the ossicular chain in the transmission of sound are almost imperceptibly small, about the diameter of a hydrogen molecule at the threshold of sound, and well within 0.1 mm - the thickness of a hair - at 120 dB.

Rigid bodies have three physical attributes that are analogous to those we described in the media-carrying wave motion; mass (or density), compliance, and dissipation. Two of the attributes - mass and compliance - provide intermediate storage during the energy transfer process, and their effects are frequency dependent. The dissipative attribute provides no such intermediate energy storage, and its effect is independent of frequency.

In more rigorous terms, the dissipation can be frequency dependent or frequency dependent, depending on whether it represents coulomb (dry) friction or viscous friction. In the ear most dissipation comes from energy loss in the cochlea, and the error in neglecting its effect on the middle ear for expository purposes here is acceptable.

Rigid bodies, unlike simple particles, can have spin or rotation in addition to a translatory movement, a situation that requires some broadening of our understanding of their motions. For all practical purposes a single particle is so small that it is indifferent to rotation; it is without a head or tail; it is a point of mass. A rigid body has an identifiable size, so much so that it may be considered a collection of particles. The total amount of mass and *how that mass is distributed* are important to the way that a rigid body moves in response to a force and to *where that force is applied.* The property of *inertia* is a description of how the combination of mass and mass distribution exists in a rigid body. Just as mass may be considered a measure of how a particle resists translatory motion in response to a force, inertia may be considered a measure of how a rigid body resists rotational motion in response to a torque. In real-world, three-dimensional space, rigid bodies can have linear motions responsive to forces in three perpendicular directions - the usual *x, y,* and *z*; they also can have rotations responsive to torques about these three axes. Stated more formally, rigid bodies

have six "degrees of freedom" of motion; three *translational* and three *rotational.*

The malleus and the incus are rigid bodies whose primary motions are rotational. Their peculiar shapes give them an inertia that favors particular rotations *at particular frequencies.* Alterations to their mass, and more particularly alterations to their inertia, whether by disease or surgery, will affect the way that sound is transmitted through them. *And the effects will be different for different frequencies of sound.*

The malleus and the incus each have a large mass and long process. Joined, the two bones form a structure that looks like the Greek letter *pi,* with the manubrium of the malleus forming one leg of the letter and the long process of the incus forming the other leg. Atop the crossbar of the *pi* lies the head of the malleus and the body of the incus, and extending rearward from the crossbar is the short process of the incus with its posterior ligament. Extending forward from the crossbar is the anterior process and the anterior ligament of the malleus. In the transmission of forces in the acoustic frequencies, the two bones move substantially as a unit, rotating the *pi* about its crossbar. Anatomically, the axis of rotation extends approximately along a line extending from the tip of the short process of the incus forward through the anterior ligament of the malleus (Barany, 1938). The malleus articulates with the incus in the saddle-shaped surface of the incudomalleal joint, and the incudomalleal joint permits the faithful transfer of rotary motion from the malleus to the incus much as a universal joint in a car permits the transfer of rotation from the transmission to the driveshaft. The lengths of the manubrium of the malleus and the long process of the incus about the axis of rotation give rise to the so-called lever ratio of the ossicular chain. There is disagreement as to the exact number for the functional ratio of such a lever effect, which is not surprising, since (1) the force of the tympanic membrane is distributed along the manubrium of the malleus and (2) the incudomalleal joint permits flexing of the incus with respect to the malleus. The tendency to rotate about the centers of mass of the bones is frequency dependent, with rotation being favored by higher-frequency stimuli.

Helmholtz (1885) described the ability of the saddle joint to release the motion of the malleus and protect the remainder of the ossicular chain and oval window in cases of large pressure variations. His account of the action is so elegantly stated that it is repeated here:

The joint between the anvil and hammer is a curved depression of a rather irregular form, like a saddle. In its action it may be compared with the joints of the well-known Breguet watchkeys, which have rows of interlocking teeth, offering scarcely any resistance to revolution in one direction but allowing no revolution whatever in the other. Interlocking teeth of this kind are developed upon the under side of the joint between hammer and anvil. The tooth on the hammer projects towards the drumskin, that of the anvil lies inwards; and, conversely, towards the upper end of the hollow of the joint, the anvil projects outwards, and the hammer inwards. The consequence of this arrangement is that when the hammer is drawn inwards by the handle, its bites the anvil firmly and carries it with it. Conversely when the drumskin, with the hammer, is driven outwards, the anvil is not obliged to follow it. The interlocking teeth of the surfaces of the joint then separate, and the surfaces glide over each other with very little friction. This arrangement has the very great advantage of preventing any possibility of the stirrup's being torn away from the oval window, when the air in the auditory passage is considerably rarefied.

It is frequently stated that Helmholtz's description of the ossicular motion incorrectly indicated that the malleus and incus would move out of phase with one another, as if they performed their sound-transmitting actions like two gears mating at the joint interface. Whet he in fact discovered was an action that would permit the *pi*-shaped malleus-and-incus unit we have just described to *fold* about the incudomalleal joint as if the joint were a hinge in the crossbar of the *pi.* The protective mechanism he described is anatomically possible, and it would be favored to act *at very low frequencies* where the inertia of the malleus and incus permit translation of the malleus *without* an accompanying movement at the tip of the long process of the incus. The swinging ossicular motion described by Barany is the way that energy in the audio frequency range gets from the surface of the tympanic membrane to the stapedial footplate. Helmholtz probably identified the way that the inner ear is protected from low-frequency, high-pressure disturbances.

Transfer functions and complex numbers in description of impedance

Contemporary physiologic description of middle ear performance frequently employs the tools of "transfer function" analysis. Transfer function analysis has very strong foundations in the analysis of electrical networks, and it is a convenient way to characterize the causes and effects of a system in a way that treats everything between the input and the output as a "black box". The benefits of transfer function analysis lie (1) in its compatibility with measurable, experimental cause-and-effect studes and (2) in its predictive abilities for the performance of systems that have wide-operating frequency ranges. The disadvantages of such presentations to the practicing otologist are that (1) the mathematics and the models are steeped in electrical engineering analogies and (2) more important, the models fail to relate to the operable parts within the "black box". We present this small section to help ease the first of the drawbacks.

We have indicated that impedance, whether used in describing the wave motion of sound through an acoustic medium or in describing rigid-body motions, has three distinct components relating causes to effects. Two of the components were shown to provide for intermediate storage of energy as it passes through, and their influences were noted to be frequency dependent. The third component provides for energy dissipation and is independent of frequency. Because impedance has components that can be broken down as frequency dependent and frequency independent, it is convenient to represent it in mathematical terms as a *complex* number, that is, as a number having two independent parts. The x and y axes of complex numbers are termed, respectively, the *real* axis and the *imaginary* axis. The term have no physical meaning; the frequency-dependent components - which are quite real - are measured along the imaginary axis.

The impedance of mechanical structures looks very much like acoustic impedance (differing only by multiplying the numerator and denominator by area) and is given by the ratio of force divided by velocity:

$$\mathbf{Zs = F/V}$$

The right-hand side of equation 8 can be looked on as "measurable" causes and effects; the left-hand side of equation 8 really describes the storage and dissipative *physical*

elements:

8 $$\mathbf{Zs = R + i(2pfM - (S/2pf))}$$

The *R* term represents energy-dissipative, frequency-independent actions such as the friction in ligaments and joint surfaces. The *i* term is the mathematical representation of square root of negative 1 and indicates that the terms in the parentheses are plotted along the imaginary complex number axis. The *2pf* terms represent angular frequency in radians; *M* is mass, and *S* is the stiffness or spring constant of the structure. A careful reading of equation 9 reveals that the two energy storage parts have inverse and out-of-phase effects on impedance. Such a relationship within the energy storage elements gives a structure the capacity for *responance*, a condition whereby the storage elements - the springs and masses - transfer energy back and forth in an oscillatory manner. The fact that there is always dissipation in the real world limits the size of such oscillations. Nonetheless, the resonances can greatly affect conduction at their characteristic frequencies.

Equation 9 (and those that resemble it in the contemporary literature) have important medical and surgical implications that are not obvious from only an intuitive grasp of impedance and sound transmission. The structures of the middle ear - ossicles, tendons, ligaments, and membranes - all have characteristic springiness and mass distributions, and their interfaces have energy-dissipative (resistive) attributes. Clearly, the impedance of the entire middle ear can be altered by disease, surgery, and scar formation. What the mathematics demonstrates is that there will be frequency-dependent effects when the mass or mass distributions are altered and inverse frequency effects when the springiness is changed. The conductive alteration is therefore predictable to a large extent by understanding that adding mass will cause a dominance of mass-related impedance at high frequency; that is *high*-frequency loss will occur. Increasing the stiffness of the membranes will, on the other hand, cause *low*-frequency loss. When the terms within equations begin to look more complicated than those of equation 9, it only means that the behavior of more elements (for example, the volume epitympanic space or a suspensory ligament) have been included to make the mathematical model more consistent with observed behavior.

Other components of conductive system

Some additional components need explanation, including the simple, but important, effects of the pinna and the external canal, the middle ear space, the stapes, and the role of the intratympanic muscles.

The pinna captures sound pressures from the environment, bounces those sound waves from its numerous folds, and guides the sound pressures to the bowl of the concha, where they are focused down the external auditory canal. The outer ear structures provide resonance and some directionality cues for the incoming sound. The canal itself behaves much like an organ pipe with one closed and one open end and, like the organ pipe, has a resonance for sound whose wavelengths are four times the 25 mm effective length of the canal. At sea level, sound has a nominal speed of 347 m/sec, which gives a fundamental resonance of about 3500 Hz. The diameter of the canal, about 6.5 mm, has little effect on the resonance. If it were much smaller, it would reinforce sound frequencies of the third and fifth harmonics: 10.500 and 16.500 Hz (if the latter could be heard!). Occlusions in the outer ear do not

change the resonance reinforcements significantly; they reduce the amount of sound energy that can reach the tympanic membrane.

The middle ear space provides a cushion of air that influences the compliance of the tympanic membrane as if it were a spring acting in parallel with the membrane. The function of the middle ear space is comparable to the enclosure of a high-fidelity speaker, particularly that of a "woofer". Changes to the volume in the middle ear space or changes in the static pressure within the space will change the compliance, generally decreasing the sensitivity to low-frequency sound. The volume of the middle ear normally provides for the delicate structures of the middle ear to function unimpeded from the tympanic membrane to the oval window. The isolation of the bone chain from other anatomic structures is important, and the ossicles are sensitive to any disturbance, much as a phonograph needle's isolation protects its sensitivity. When structural bridges occur from the floor or walls of the middle ear space to the ossicles, as can happen in scar formation from middle ear surgery, sounds that normally reach the cochlea through bone conduction can be disturbingly amplified.

We have, until now, neglected the role of the stapes, the smallest bone in the body. The capitulum of the stapes articulates in a ball-and-socket joint on the lenticular process of the incus. The footplate of the stapes is contiguous with the membrane of the oval window. The stapes has a very small mass compared with that of the first two bones of the ossicular chain, and its effect on the frequency-dependent transmission of sound is normally small. However, the stapedial linkages are tremendously important; ankylosis of the footplate or disarticulation at the incudostapedial joint can occur and will compromise the entire conductive system.

The intratympanic muscles - the tensor tympany and the stapedius - are usually treated together in discussions of middle ear physiology because they are "active" elements that are capable of introducing energy into the ossicular chain, and because they have been demonstrated to alter the frequency response of the ossicular chain. Their exact functions are still largely unknown. The two muscles have different embryologic origins and different neural excitations. The stapedius muscle provides a rearward tensioning of the incudostapedial joint (which has no apparent influence on the rest of the ossicular chain), and such tensioning will actively and perhaps selectively suppress the transmission of low-frequency sound through the joint much like tightening a guitar string will raise its pitch. The tensor tympany inserts on the anteromedial portion of the neck of the malleus and can directly influence the effective compliance of the tympanic membrane, an action that can alter the sensitivity of the entire conductive system. It has been suggested that the tensor tympany also serves as a "weak spring", achieved by the resting tonus of the muscle (Anderson, 1976).

Understanding small

Before we leave the physiology of conductive loss, it is approriate to dwell on the minute sizes of the physics we have just described. It is difficult to think small. Usually, the otologist's first introduction to the middle ear under the surgical microscope presents a new world of unfamiliar landmarks that, in time, become old friends. Those macroscopic structures account for the nearly infinitesimal movements that should also be appreciated with the same comfort.

The pressure disturbances of the *loudest* sounds are only about 30 Pa; normal atmospheric pressure is more than 100.000 Pa. On the other end of the loudness scale, pressure disturbances of just audible sound at 1000 Hz are only 0.00003 Pa, and displacement amplitudes are about 10^{-11} m, 50 times *less* than the distance between atoms in metals (Sears et al, 1987). In relationship to things that we can see, the surgical microscope provides comfortable magnification at about 10x, enough for the stapes to fill the field. Not even the most powerful optical microscope, however, can resolve objects much smaller than a few hundred nanometers, that is, 10^{-7} m, and the most powerful optics would fail to perceive the motion of threshold sound by a factor of more than 10.000 times. Yet it is common (and good) practice on installation of middle ear prosthetics to "nudge" them very *slightly* as a test of the integrity of the conductive repair. The motion of that gentle nudge is several millions of times more excursion than the piece will see in the rest of its normal service.

Summary

Most causes of conductive hearing loss are examined in great detail in their respective chapters, as are the options regarding auditory rehabilitation. Toward this end, we have reviewed the problem regarding the transmission of sound when a conductive block occurs in a manner that we believe will enable the physician to view, with a broader perspective and greater clarity, the underlying mechanisms that contribute to conductive hearing loss from a mechanistic approach. Optimal management of patients with this type of hearing loss is dependent on an understanding of these mechanics.