CHAPTER 24

PHYSIOLOGY OF HEARING

Joseph R. Anticaglia, M.D.

INTRODUCTION

The basic function of the hearing mechanism is to gather, conduct and perceive sounds from the environment. Sound waves, propagated through an elastic medium, liberate energy in a characteristic pattern which varies in frequency and intensity.

The human voice and other ordinary sounds are composed of fundamental tones modified by harmonic overtones (refer to Chapter 23). Our hearing sensitivity is greatest in childhood, but as we get older, our perception of high tones worsens, a condition labelled "presbycusis." The frequency range of the human ear extends from as low as 16 Hz to as high as 30,000 Hz. From a practical standpoint, however, few adults can perceive sounds above 11,000 Hz.

The ear responds to alterations in the pressure level of sound. The amplitude of these sound pressure alterations determines the intensity of the sound. So great is the range of intensities to which the ear responds that a logarithmic unit, the decibel (dB), is commonly used to express the pressure level of sound. The subjective correlates of frequency and intensity are pitch and loudness.

The translation of acoustical energy into perceptions involves the conversion of sound pressure waves into electrochemical activity in the inner ear. This activity is transmitted by the auditory nerves to the brain for interpretation. Although there are many gaps in our understanding of the precise mechanism of hearing, the following presentation will emphasize the peripheral processes involved in hearing.

PERIPHERAL MECHANISM OF HEARING

Sound reaches the ear by three routes: air conduction through the ossicular chain to the oval window; bone conduction directly to the inner ear; and conduction through the round window. Under ordinary conditions, bone conduction and the transmission of sound through the round window are less significant than air conduction in the hearing process. An example of bone conduction occurs when you tap your jaw. The sound you perceive is not coming through your ears but through your skull. Sound perception via air conduction is the most efficient route and it encompasses the external and middle ear conducting system which will be discussed in more detail.

Conduction of Sound

External Ear. Anatomically, the ear can be divided into an external portion (outer ear), an "air-filled" middle ear, and a "fluid-filled" inner ear (Fig. 24-1). The outer ear consists of the auricle and the external auditory meatus, or canal. The auricle is an ornamental structure in man. Neither does it concentrate sound pressure waves significantly, nor does it function in keeping foreign bodies out of the ear canal. The two ears give us "auditory localization" or "stereophonic hearing," namely, the ability to judge the direction of sound. One explanation is that sound waves arriving at the two auricles have a slight time lag, differing in intensity and timbre since in the far ear the sound must travel a greater distance.

The external auditory canal is a little more than an inch in length and extends from the concha to the tympanic membrane. The skin of the cartilaginous portion of the ear canal secretes wax, which helps maintain relatively stable conditions of humidity and temperature in the ear canal. The ear canal protects the tympanic membrane and acts as a tubal resonator so that the intensity of sound pressure waves are amplified when they strike the tympanic membrane.

The tympanic membrane (TM, eardrum) separates the external ear from the middle ear. This almost cone-shaped, pearl-gray membrane is about a half-inch in diameter. The distance the eardrum moves in response to the sound pressure waves is incredibly small, as little as one billionth of a centimeter.[1] Besides vibrating in response to sound waves, the eardrum protects the contents of the middle ear and provides an acoustical dead space so that vibrations in the middle ear will not exert pressure against the round window.

Middle Ear. Medial to the eardrum is the special air-filled space called the middle ear. It houses three of the tiniest bones in the body: the malleus (hammer); the incus (anvil); and the stapes (stirrup). The handle of the malleus attaches to the eardrum and articulates with the incus which is connected to the stapes. The malleus and the incus vibrate as a unit, transmitting the sound waves preferentially to the stapedial footplate, which moves in and out of the oval window. Below and posterior to the oval window is the round window whose mobility is essential to normal hearing.

Fig. 24-2 shows the two intratympanic muscles, the tensor tympani and the stapedius. The tensor tympani extends from the canal above the eustachian tube to the handle of the malleus. It moves the malleus inward and anteriorly, and helps maintain tension on the eardrum. The stapedius muscle inserts on the posterior aspect of the neck of the stapes. It pulls the stapes outward and posteriorly.

The two muscles are antagonistic in their action, but contract only when stimulated by rela-

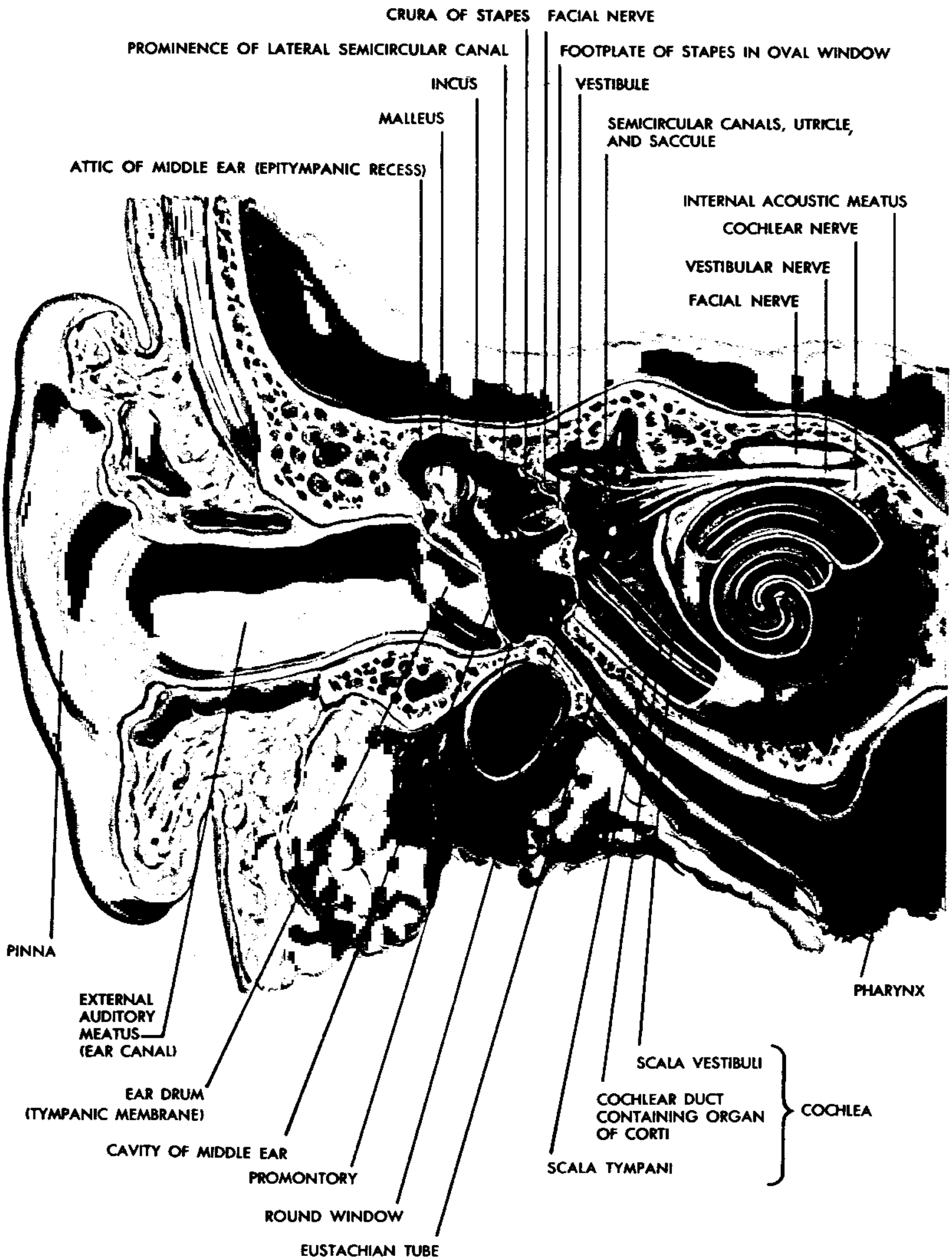
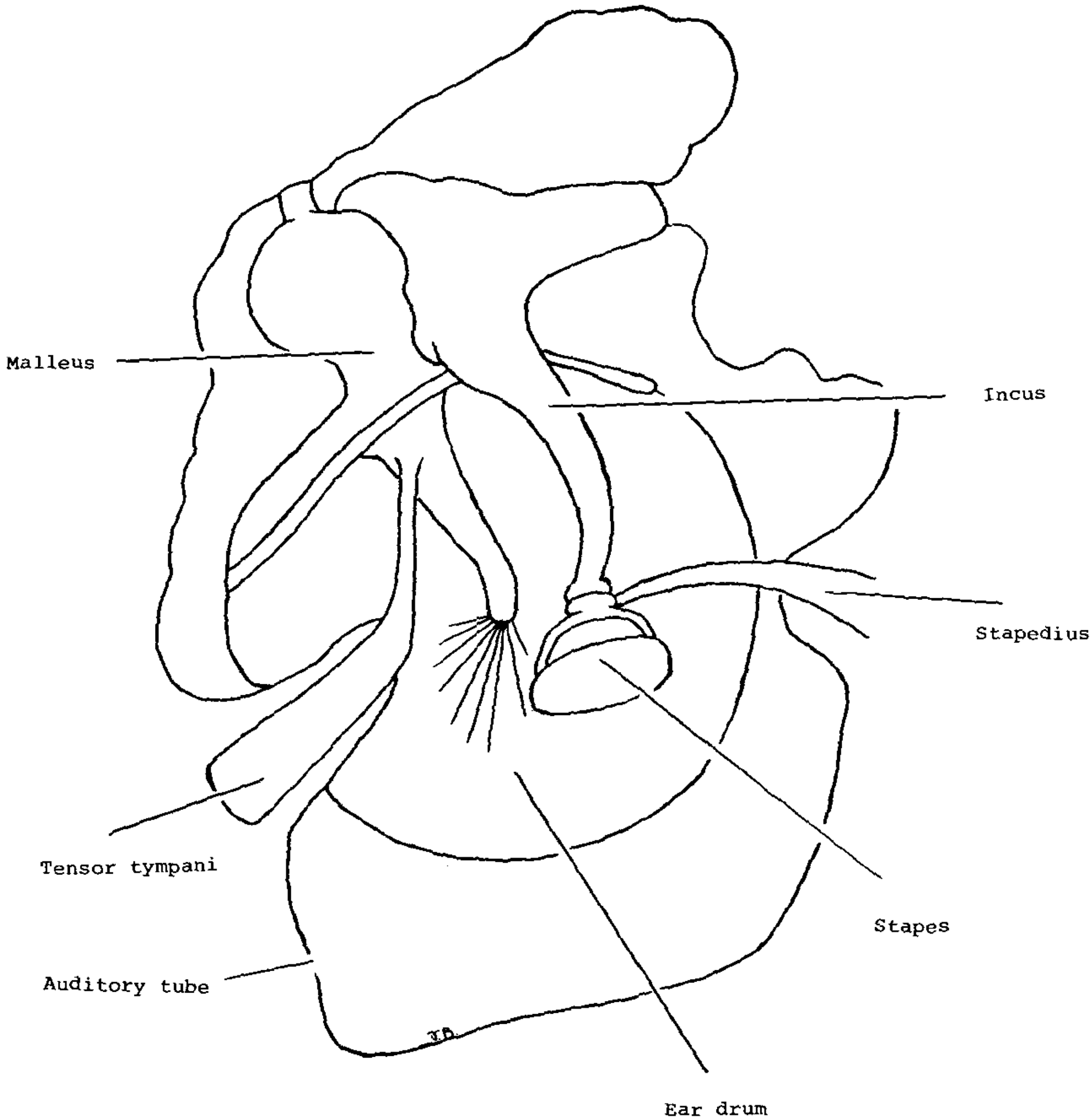


Figure 24-1. Pathway of Sound Conduction Showing Anatomic Relationships.



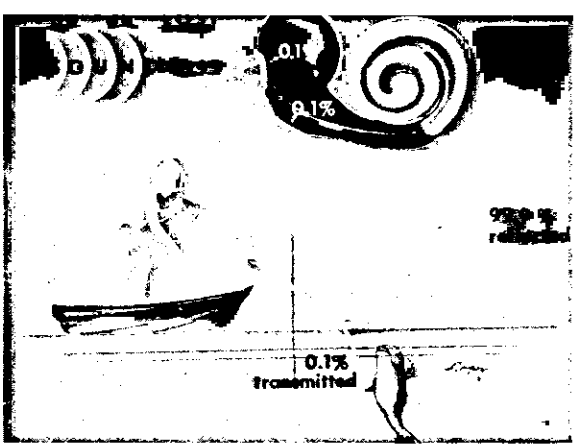
Lockart R., Hamilton G., Fyfe F.: Anatomy of the Human Body. London, Faber & Faber, 1959, p. 463.

Figure 24-2. Intratympanic Muscles Viewed from the Medial Wall. Faber & Faber, London.

tively loud sounds. Contraction of the muscles causes rigidity of the ossicular chain with a resultant decrease in the conduction of sound energy to the oval window. A limited protective function has been ascribed to this reflex contraction of the muscles although the aural reflex does not react fast enough to provide complete protection against sudden and explosive sounds. Also, exposure to steady state noise for long periods of time would cause the muscles to adapt or fatigue to the auditory stimulus.[2]

The "eustachian or auditory" tube connects the anterior wall of the middle ear with the nasopharynx. It is about an inch and a half in length and consists of an outer bony portion (one third of the tube which opens into the middle ear) and an inner cartilaginous part (two thirds of the tube which opens into the throat). The lumen of the bony part is permanently opened while that of the cartilaginous portion is closed except during certain periods such as swallowing, yawning, or blowing the nose. To hear optimally, the atmospheric pressure on both sides of the eardrum should be equal. The act of swallowing, for example, forces air up the middle ear and thus equalizes the atmospheric pressure on either side of the tympanic membrane.

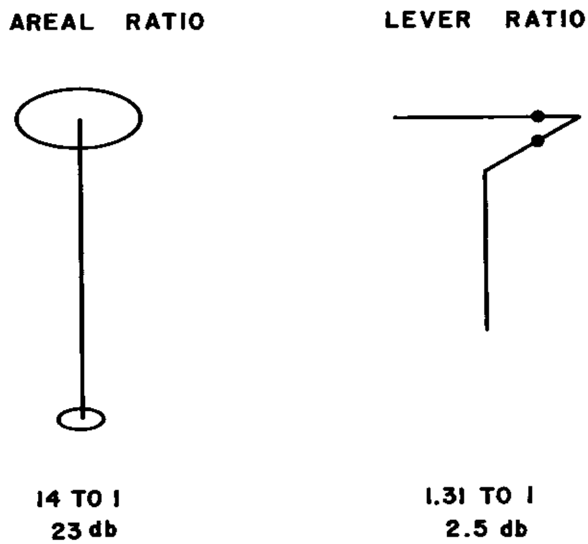
Yet, the fundamental problem that the middle ear must resolve is that of "impedance matching." In other words, the ear must devise a mechanism of converting the sound pressure waves from an air to a fluid medium, without a significant loss of energy. This is a noteworthy accomplishment since only 0.1% of airborne sound enters a liquid medium whereas the other 99.9% is reflected away from its surface. Stated differently, the intensity of vibration in the fluid of the inner ear is 30 decibels less than the intensity present at the eardrum (Fig. 24-3). The middle ear has two arrangements to narrow this potential energy loss.



Lawrence M., cited by De Wéese D., Saunders W.: Textbook of Otolaryngology. St. Louis, C. V. Mosby Company, 1968, p. 270, ed. 3; Courtesy of Dr. Merle Lawrence, Ann Arbor, Michigan.

Figure 24-3. Loss of Sound Energy at the Air-Water Interface.

First is the "size differential" between the comparatively large eardrum and the relatively small footplate of the stapes. The eardrum has an effective areal ratio which is 14 times greater than that of the stapedial footplate. This hydraulic effect increases the force of pressure from the eardrum onto the footplate of the stapes so that there is approximately a 23 dB increase of sound intensity on the fluid of the inner ear. The "lever action" of the ossicles amplifies the intensity of sound as it traverses the middle ear by about 2.5 dB. Thus, the impedance matching mechanism of the middle ear is not perfect, but accounts for a 25.5 dB increase in the intensity of sound pressure at the air-liquid interface (Fig. 24-4).



Lawrence M.: How we hear. JAMA 196:83, Copyright 1966, American Medical Association, Chicago, Ill.

Figure 24-4. Impedance Matching Mechanism of the Middle Ear which Minimizes Energy Loss as Sound Is Transferred from Air to Fluid Medium. Journal of the American Medical Association.

Perception of Sound

Inner Ear. The labyrinth or inner ear is a complex system of ducts and sacs which houses the end organs for hearing and balance. It consists of an outer bony and an inner membranous labyrinth. The center of the labyrinth, the vestibule, connects the three semicircular canals and the cochlea. A watery fluid, perilymph, separates the bony from the membranous labyrinth while inside the membranous labyrinth are fluids called endolymph and cortilymph.

The cochlea resembles a snail shell which spirals for about two and three-quarter turns around the bony column called the "modiolus" (Fig. 24-5). There are three stairways or canals within the membranous cochlea: the "scala vestibuli;" the "scala tympani;" and the "scala media

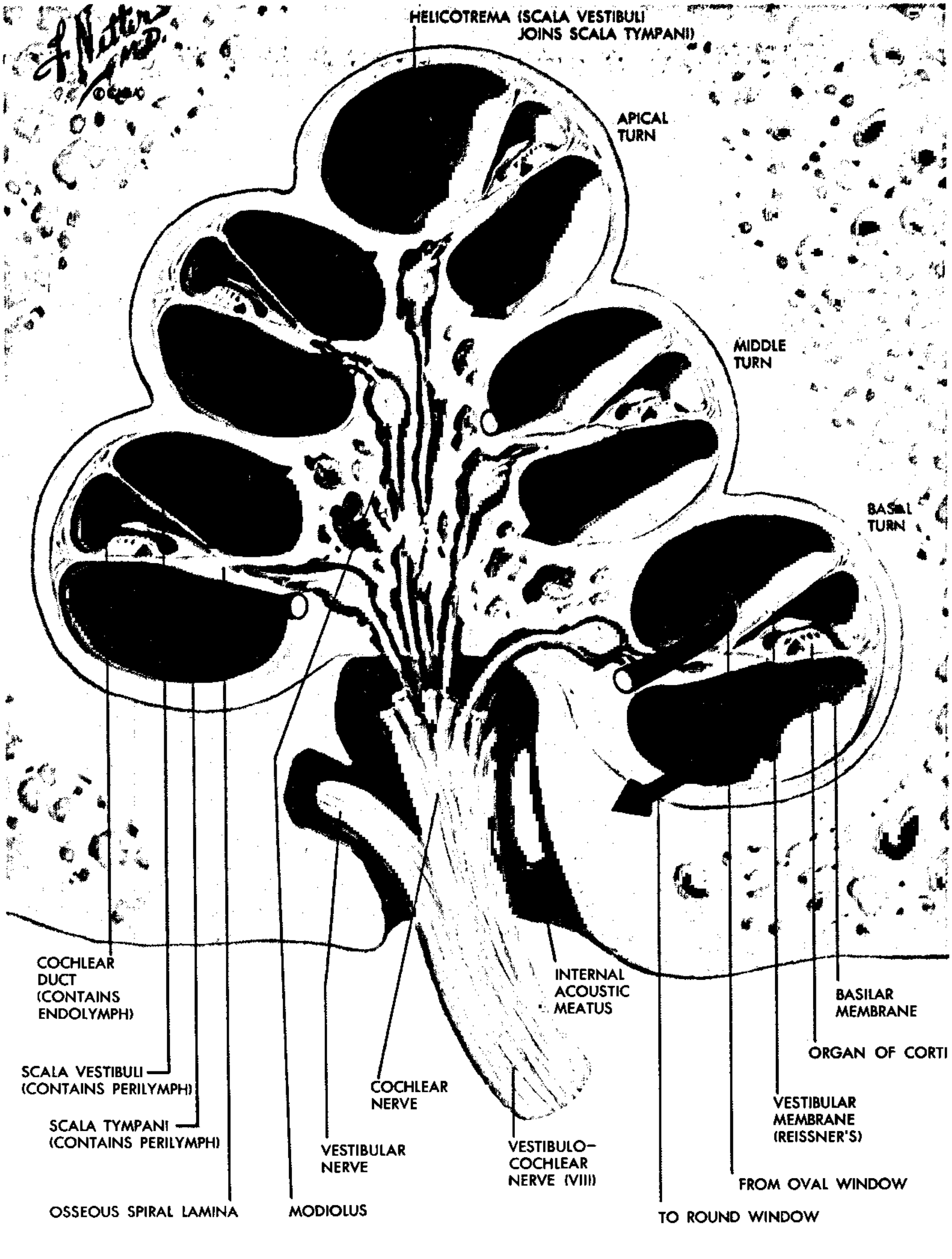


Figure 24-5. Cross Section of Cochlea.

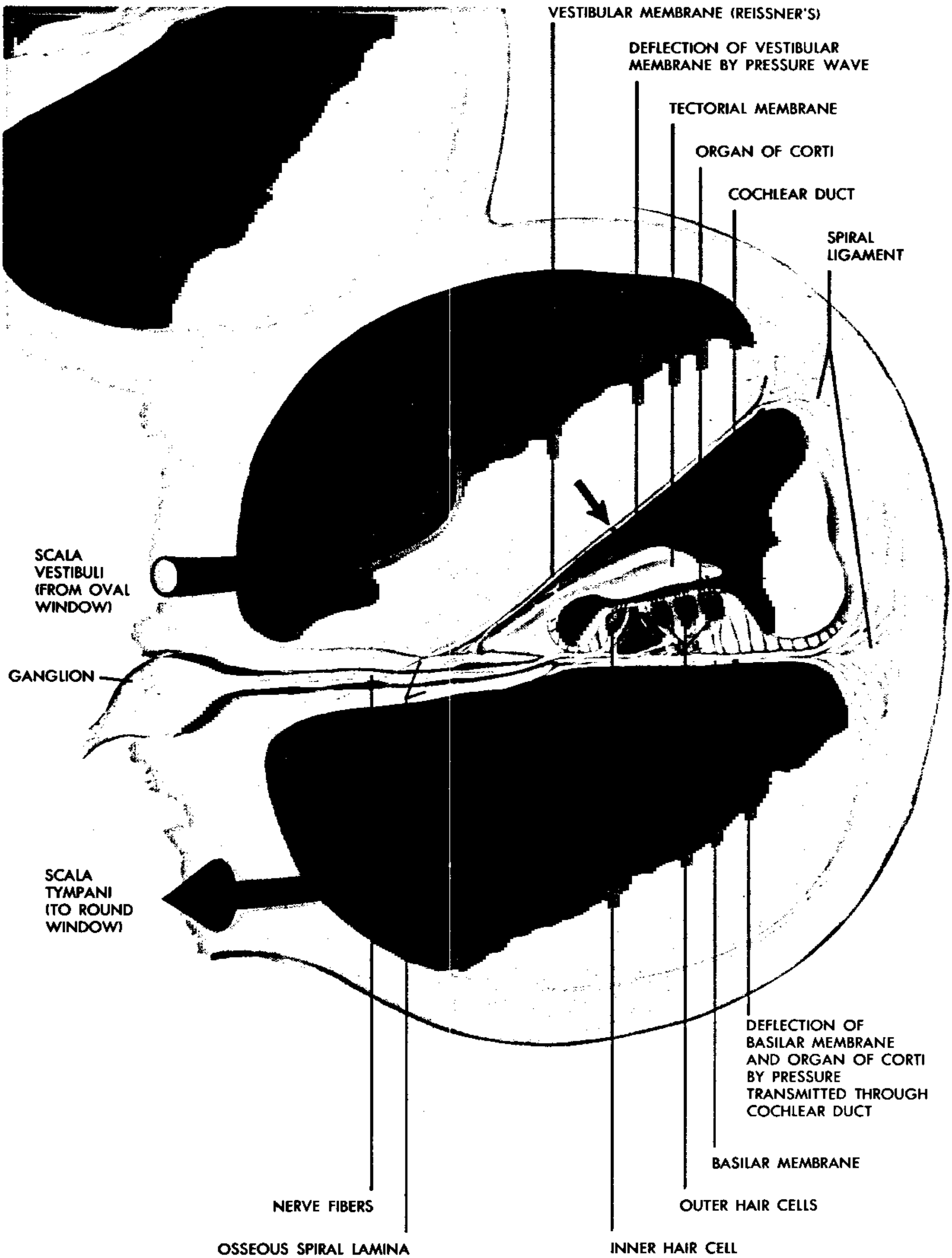


Figure 24-6. Transmission of Sound across the Cochlear Duct Stimulating the Hair Cells.

or cochlea duct." A bony shelf, the "spiral lamina," together with the basilar membrane and the spiral ligament, separate the upper scala vestibuli from the lower scala tympani. The third canal, the scala media, is cut off from the scala vestibuli by Reissner's vestibular membrane.

The scala media is a triangular-shaped duct within which is found the organ of hearing, namely, the "organ of Corti." The basilar membrane, narrowest and stiffest near the oval window, widest at the apex of the cochlea, helps form the floor of the cochlear duct. On the surface of the basilar membrane are found phalangeal cells which support the critical "hair cells" of the organs of Corti. The hair cells are arranged in a definite pattern with an inner row of about 3,500 hair cells and three to five rows of outer hair cells numbering about 12,000. The cilia of the hair cells extend along the entire length of the cochlear duct and are imbedded in the undersurface of the gelatinous overhanging tectorial membrane (Fig. 24-6).

Inner Ear Fluids. The vestibular and tympanic canals contain perilymph and communicate with each other through a tiny opening at the uppermost part of the cochlea, the "helicotrema." The perilymph has a high sodium concentration and a low potassium content whereas the opposite is true of endolymph. Since the transmission of neural impulses should be impossible in the high concentration of potassium found in endolymph, it has been shown that the fluid which bathes the organ of Corti — Cortilymph — has a different ionic content than that of endolymph, and furnishes a suitable medium for the normal functioning of the hair cells and neural endings of the organ of Corti.[3, 4]

The tectorial membrane appears to maintain a zero potential compared to the scala tympani while the endolymph has a positive potential and the organ of Corti, a negative potential. The positive resting potential of the endolymph has been labelled the "endocochlear or DC potential." A change in the resting potential of the endolymph results from acoustical stimulation so that the scala media is negative relative to the scala tympani, "summating potential."

In short, the fluids of the cochlear duct supply nourishment to Corti's organ, a system of removing waste products, an appropriate medium for the transmission of neural impulses, and a means of eliminating noise that its own blood supply would produce.

Transmission of Sound Waves in the Inner Ear. The two openings afforded by the oval and round windows are essential for sound pressure waves to pass through the cochlear fluids. The movement of the stapedial footplate in and out of the oval window moves the perilymph of the scala vestibuli (Fig. 24-7). This vibratory activity travels up the scala vestibuli, but causes a downward shift of the cochlear duct with distortion of Reissner's membrane and displacement of endolymph and Corti's organ. The activity is then transmitted through the basilar membrane to the scala tympani. When the oval window is pushed inward, the round window acts as a relief point and bulges outward.

Transduction. The conversion of mechanical energy of sound into electrochemical activity is called transduction. The vibration of the basilar membrane causes a pull, or shearing force of the hair cells against the tectorial membrane. This "to and fro" bending of the hair cells activates the neural endings so that sound is transformed into an electrochemical response. It remains to be clarified whether an electrical and/or chemical process stimulates the neural endings.

Travelling Waves. In general, the hair cells at the base of the cochlea transmit high frequency sounds while those at the apex especially respond to low frequency tones. This results in the travelling wave phenomena in which there is a specific point of maximum displacement of the basilar membrane beyond which the wavelength and the amplitude become progressively smaller in character. High pitched sounds travel a short distance along the basilar membrane before they die out; the opposite occurs with low pitched sounds.

Nerve Conduction. Each nerve fiber connects with several hair cells, and each hair cell with several nerve fibers. The hair cells stimulate auditory neural endings and nerve fibers which stream out through small openings in the spiral lamina into the hollow modiolus (Fig. 24-5). The cell bodies of the nerve fibers form the spiral ganglia whose axons make up the cochlear (auditory) division of the eighth cranial nerve. The movement of the hair cells sets up action potentials, and coded information from both ears are sent to the cochlear nuclei and thereafter to the temporal lobe of the brain where cognition and association takes place. Fig. 24-7 summarizes the peripheral mechanism of hearing.

CLASSIFICATION OF HEARING LOSS

Loss of hearing can be classified into the following categories: 1. Conductive impairment; 2. Sensorineural impairment; 3. Mixed (both conductive and sensorineural); 4. Central impairment; 5. Psychogenic impairment.

Conductive Hearing Loss

Any condition which interferes with the transmission of sound to the cochlea is classified as a conductive hearing loss. Pure conductive losses do not damage the organ of Corti nor the neural pathways.

A conductive loss can be due to wax in the external auditory canal, a large perforation in the eardrum, blockage of the eustachian tube, interruption of the ossicular chain due to trauma or disease, fluid in the middle ear secondary to infection, or otosclerosis, that is, fixation of the stapedial footplate. A significant number of conductive hearing losses are amenable to medical or surgical treatment.

Sensorineural Hearing Loss

A sensorineural hearing loss is almost always irreversible. The sensory component of the loss involves the organ of Corti and the neural component implies degeneration of the neural elements of the auditory nerve.

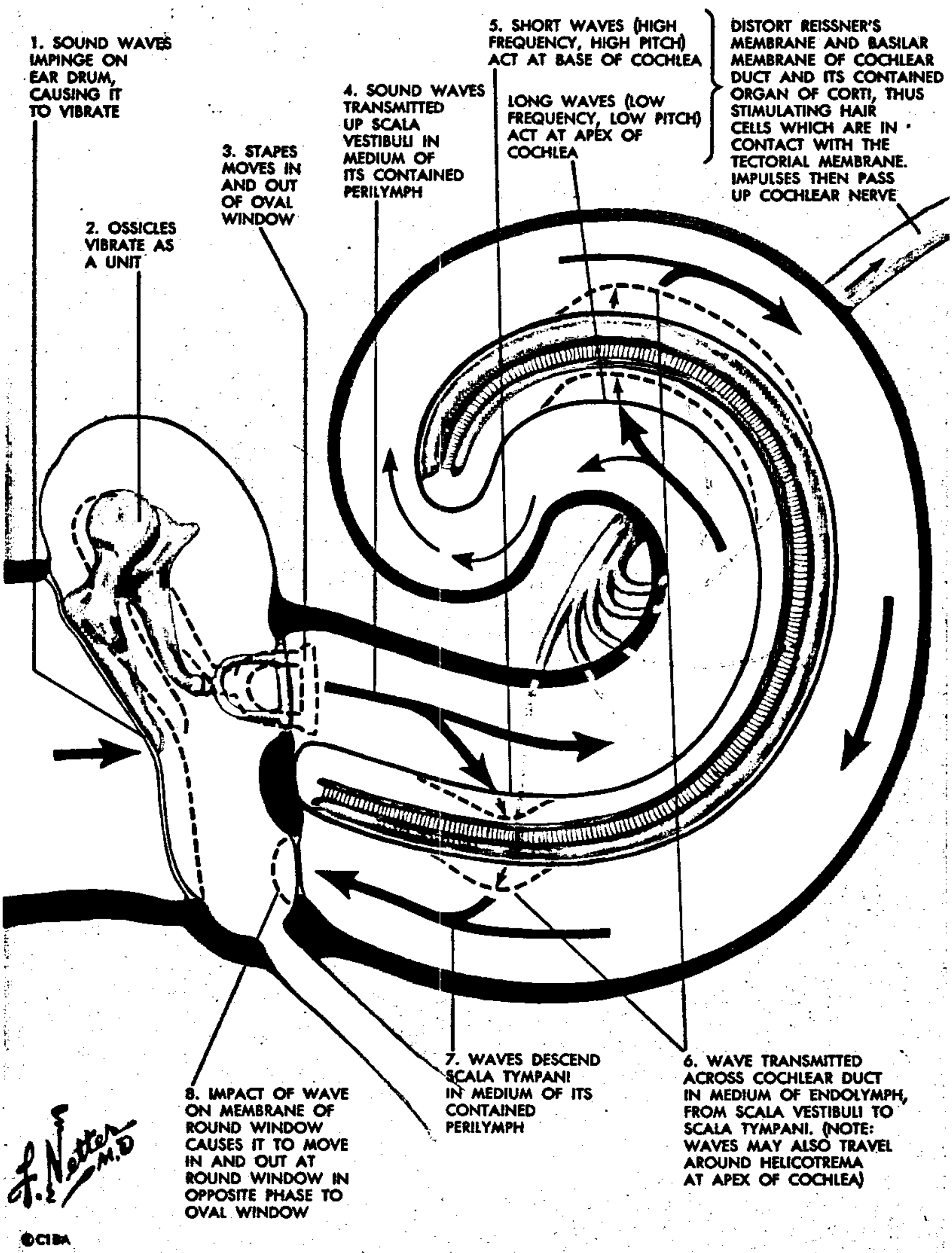


Figure 24-7. Transmission of Vibrations from Drums through Cochlea.

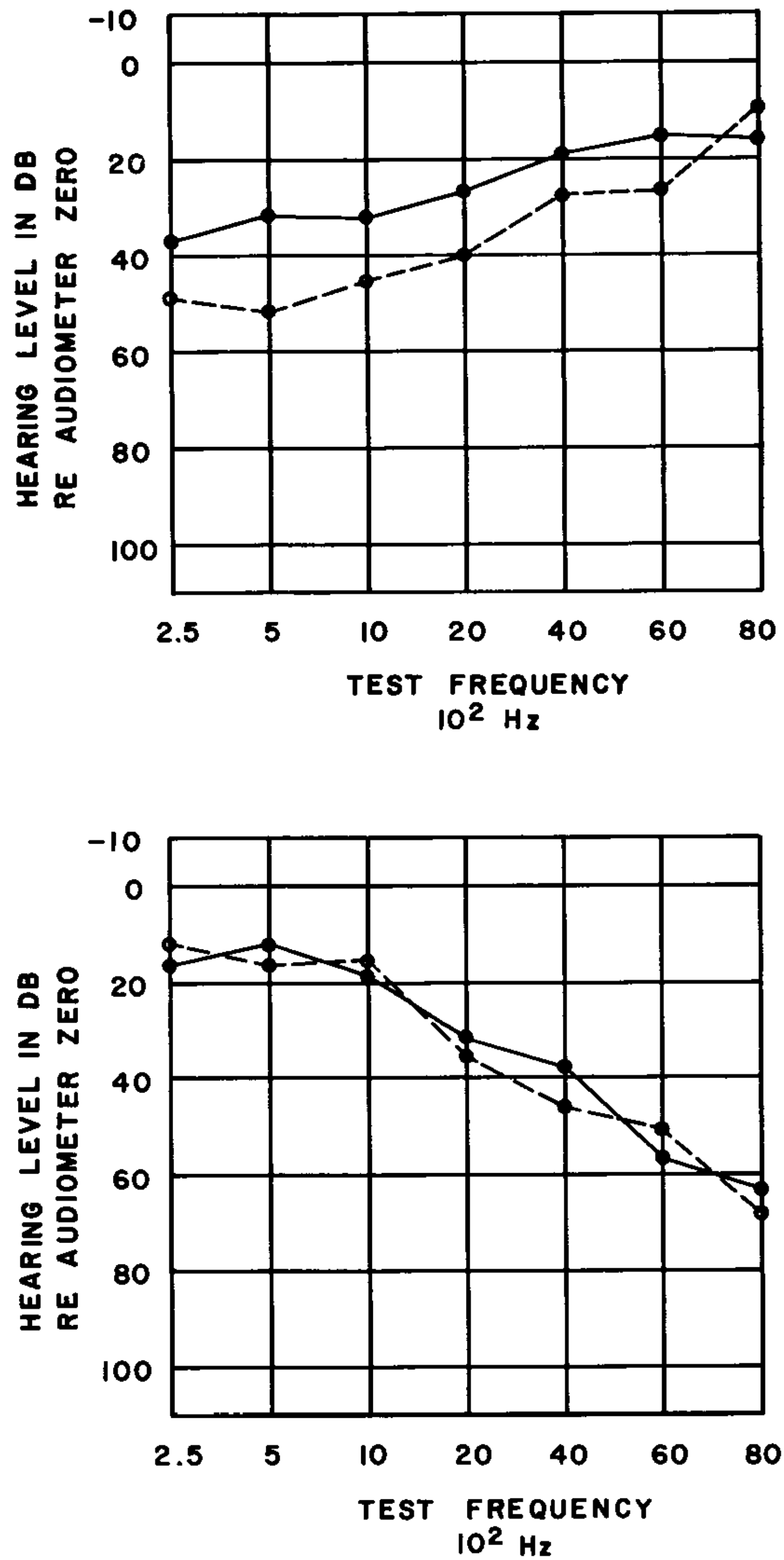


Figure 24-8. Audiograms Showing A) Conductive and B) Sensorineural Types of Hearing Loss.

Exposure to excessive noise causes an irreversible sensorineural hearing loss. Damage to the hair cells is of critical importance in the pathophysiology of noise-induced hearing loss. Invariably, degeneration of the spiral ganglion cells and the peripheral nerve fibers accompany severe injury to the hair cells.

Sensorineural hearing loss may be attributed to various causes, including presbycusis, viruses (e.g., mumps), some congenital defects, and drug toxicity (e.g., streptomycin).

Mixed Hearing Loss

Mixed hearing loss occurs when there are components and characteristics of both conductive and sensorineural hearing loss in the same ear.

Central Hearing Loss

A central hearing loss implies difficulty in a person's ability to interpret what he hears. The abnormality is localized in the brain between the auditory nuclei and the cortex.

Psychogenic Hearing Loss

A psychogenic hearing loss indicates a "non-organic" basis for an individual's threshold elevation. Two conditions in which such a loss may occur are malingering and hysteria.

AUDIOMETRY

The pure tone audiometer is the fundamental tool used in industry to evaluate a person's hearing sensitivity. It produces tones which vary in frequency usually from 250 Hz to 8,000 Hz at octave or half-octave intervals. The intensity output from the audiometer can vary from zero dB to 110 dB, and is often marked "hearing loss" or "hearing level" on the audiometer.

Zero dB or zero reference level on the audiometer is the average normal hearing for different pure tones and varies according to the "standard" to which the audiometer is calibrated. Zero reference levels have been obtained by testing the hearing sensitivity of young healthy adults and averaging that sound intensity at specific frequencies at which they were just perceptible. It is to be differentiated from the 0.0002 microbar references for the sound pressure level measurements. If a person has a 40 dB hearing loss at 4,000 Hz, it means that for the individual to perceive a tone the intensity of that tone must be raised to 40 dB above the "standard."

The audiogram serves to record the results of the hearing tests. A graphic description of the faintest sound audible is obtained by plotting the intensity against the frequency. Examples of audiograms which indicate conductive and sensorineural losses are shown in Fig. 24-8. In conductive hearing losses, the low frequencies show most of the threshold elevation, whereas the high frequencies are most often involved with the sensorineural losses.

The recording of an audiogram is deceptively simple, yet for valid test results, one must have a properly calibrated audiometer, an acceptable test environment to eliminate interfering sounds, and a qualified audiometrician. When a marked hearing loss is encountered, bone conduction audiometry and more sophisticated hearing tests are often helpful in diagnosing the site and cause of the hearing loss.[5] For more details concerning appropriate American National Standard Institute (ANSI) standards and the objectives of a good audiometry program, refer to the preferred reading list.

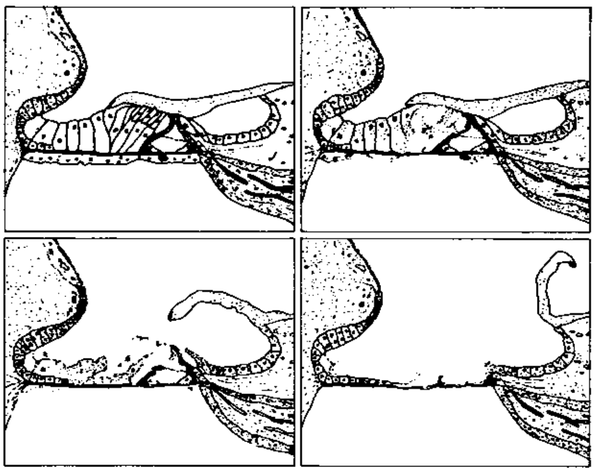
EFFECTS OF EXCESSIVE NOISE EXPOSURE

Since the ear does not have an overload switch or a circuit breaker, it has no option but to receive all the sound that strikes the eardrum. In industry, excessive noise constitutes a major health hazard. Such exposure can cause both auditory and extra-auditory effects.

Auditory Effects

Noise induced hearing loss (NIHL) can happen unnoticed over a period of years. At first, excessive exposure to harmful noise causes auditory fatigue or a temporary threshold shift (TTS). This shift refers to the difference in one's hearing sensitivity measured before and after exposure to sound. It is called "temporary" since there is a return of the individual's pre-exposure hearing level after a period of hours away from the intense sound.

However, repeated insults of excessive noise can transform this TTS into a permanent threshold shift (PTS). In fact, studies substantiate that the hearing sensitivity of factory workers in heavy industry is poorer than that of the general population. Fig. 24-9 depicts the stages of destruction



Lawrence M.: Auditory problems in occupational medicine. Arch. Environ. Health 3:2888, Copyright 1961, American Medical Association, Chicago, Ill.

Figure 24-9. Stages of Destruction of the Organ of Corti. (A) The normal organ of Corti. (B) A stage of hair cell degeneration following the first subtle changes within the cytoplasm of the cells. The internal hair cell remains intact. (C) Both inner and outer hair cells are gone, and the supporting structures are degenerating. (D) In the final stages, the entire organ of Corti is dislodged, leaving a denuded basilar membrane, which may become covered with a simple layer of eipthelial cells. (Arch. Environ. Health) of the organ of Corti in a laboratory test animal that was overstimulated by loud continuous noise.

Many factors influence the course of NIHL. The overall "decibel level" of the noise exposure is obviously important. If a noise exposure does not cause auditory fatigue, then such exposure is not considered harmful to one's hearing sensitivity.

Another consideration is the "frequency spectrum" of the noise. Noise exposure which has most of its sound energy in the high frequency bands is more harmful to a worker's hearing sensitivity than low-frequency noises.

Another factor is the daily "time distribution" of the noise exposure. In general, noise which is intermittent in character is less harmful to hearing than steady state noise exposure. As the "total work duration" (years of employment) of a worker to hazardous noise is increased, so too does the incidence and magnitude of his NIHL. However, no report of "total" hearing loss has been attributed to excessive noise exposure alone.[6]

Finally, the "susceptibility" of the worker to hazardous noise must be considered, since not every individual will suffer identical hearing impairment if exposed to the same noise intensity over the same time period. A small percentage of workers will be highly susceptible or, on the other hand, refractory to the degrading effects of noise.

The hearing loss from "acoustic trauma" should be differentiated from the insidious, irreversible sensorineural NIHL that results after months or years of exposure to excessive noise conditions. Acoustic trauma refers to the loss of hearing secondary to head or ear trauma, or after exposure to a sudden, intense noise such as that of firearms or explosions. A conductive type of hearing loss results when the trauma causes a perforated eardrum or disruption of the middle ear ossicles. The trauma can cause a sensorineural loss, but not infrequently, the hearing loss is temporary in nature. Besides causing hearing loss, hazardous noise levels can mask speech, be a source of annoyance, and occasionally degrade a worker's job performance.[7]

Extra-Auditory Effects

The extra-auditory effects of noise result in physiologic changes other than hearing. We are familiar with the reflex-like startle response of an individual to a loud, unexpected sound. Less commonly noted are the cardiovascular, neurologic, endocrine and biochemical changes secondary to intense noise exposure. Subjective complaints of nausea, malaise, and headache have been reported in workers exposed to ultrasonic noise levels. Vasoconstriction, hyperreflexia, fluctuations in hormonal secretions, disturbances in equilibrium and visual functions have been demonstrated in laboratory and field studies. These changes have been for the most part transient in character, and it remains to be clarified whether such noise exposure has long lasting ill effects on the organism.[8]

SUMMARY

The important function of the hearing mechanism is to convert the mechanical energy of sound pressure waves into an electrochemical response. Excessive noise exposure can tax the physiologic limits of the hearing mechanism and cause an irreversible, sensorineural hearing loss. Noise is just one of many causes of hearing loss, so that a relevant medical history and a detailed history of a worker's previous employment will eliminate many false conclusions concerning the cause of a worker's loss of hearing.

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