9 AUDITORY FUNCTION

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Physiology and Function of the Hearing System

The hearing system, also called also the auditory system, consists of the outer ear, middle ear, inner ear, and central auditory nervous system. The overall function of the hearing system is to sense the acoustic environment thus allowing us to detect and perceive sound. The anatomy of this system has been described in Chapter 8, *Basic Anatomy of the Hearing System*. The current chapter describes the function and physiology of the main parts of the hearing system in the process of converting acoustic events into perceived sound.

In order to facilitate perception of sound, the hearing system needs to sense sound energy and to convert the received acoustic signals into the electro-chemical signals that are used by the nervous system. A schematic view of the processing chain from the physical sound wave striking the outer ear to the auditory percept in the brain is shown in Figure 9-1.

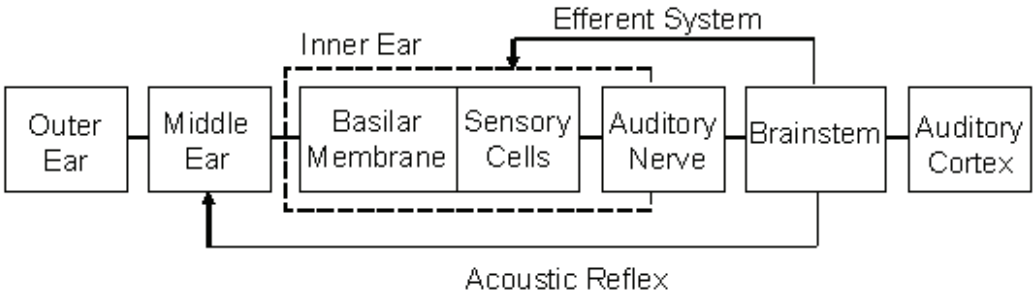

Figure 9-1. A schematic view of the hearing system.

The hearing system shown in Figure 9-1 has two functions: sound processing and hearing protection. Sound processing by the hearing system starts when the sound wave arrives at the head of a person. The head forms a baffle that reflects, absorbs, and diffracts sound prior to its processing by the hearing system. The first two sound processing elements of the hearing system are the outer and middle ears that form together a complex mechanical system that is sensitive to changes in intensity, frequency, and direction of incoming sound. Acoustic waves propagating in the environment are diffracted, absorbed, and reflected by the listener's body, head, and the pinnae and arrive through the ear canal at the tympanic membrane of the middle ear. After the acoustic wave strikes the eardrum, its acoustic energy is converted into mechanical energy and carried across the middle ear. At the junction of the middle ear and the inner ear, the mechanical energy of the stapes is transformed into the motion of the fluids of the inner ear and thence into the vibrations of the basilar membrane. The motion of the basilar membrane affects electro-chemical processes in the organ of Corti and results in generation of electric impulses by the array of the hair cells distributed along this membrane. The electrical impulses generated by the hair cells affect the inputs to the nerve endings of the auditory nerve and are transmitted via a network of nerves to the auditory cortex of the brain where the impulses are converted into meaningful perception.

A secondary function of the hearing system is to provide some protection for the organ of Corti and the physical structures of the middle ear from excessive energy inputs and subsequent damage by modulating the

reactivity of the mechanical linkages. The anatomy of the outer ear also protects the tympanic membrane from harmful effects of wind, dust, and changes in temperature and humidity while the muscles of the middle ear provide some protection of the inner ear and organ of Corti. The text of this chapter covers the sound transmission function of the hearing system but the main protective structures of the ear also will be discussed as they are mentioned.

The Outer Ear

Directional properties of the hearing system

As sound waves arrive at a listener's head, the energy of sound entering the ear is affected by the presence of the human body and by the acoustic properties of the outer ear. Some sounds are attenuated and reflected away by the barriers caused by the head structures while others are reflected toward the ear canal and even amplified by the ear cavities. The shape of the head and of the upper torso and the locations and shapes of the two pinnae serve as a direction cueing system that modifies incoming sound depending on the location of the sound source. The difference between the sound arriving at the listener and the sound that enters the ear canal is called the Head Related Transfer Function (HRTF) and it varies as a function of the direction from which sounds arrive and with the frequency of the sound (see Chapter 5, Audio *Helmet-Mounted Display Design* and Chapter 11*, Auditory Perception and Cognitive Performance*).

The directional system of the human head operates throughout full three-dimensional spherical space and is sensitive to a wide range of acoustic frequencies. Directional cues generated by the hearing system generally can be divided into binaural and monaural cues, depending whether they involve both ears or just one ear. The two main binaural cues are the interaural time difference (ITD) and the interaural intensity difference (IID).

If a sound source is located in the median sagittal plane (midline, dividing right and left sides) of the head, the two ears receive approximately the same acoustic signal. However, if the sound is approaching the head from one side, the ear closest to the sound source will receive the sound earlier and with greater intensity than the other ear. ITDs are the differences in the onset of sound and are equivalent to phase differences in the case of continuous periodic sound with no perceived onset. IIDs are caused by the absorption and reflection of incoming sound by the body and head structures and creation of an "acoustic shadow" affecting the ear farther away from the sound source. The ITD cues operate most effectively at low auditory frequencies whereas the IID cues are most effective at high frequencies but fail at low frequencies since these sound waves diffract around the human body.

Binaural cues are supported by monaural cues resulting from the specific positions and shapes of the two pinnae. The pinna has the shape of an irregular funnel that is attached to the head at an angle of 15 to 30° (see Chapter 8, *Basic Anatomy of the Hearing System*). Its main function is to collect sound and to channel it to the ear canal. However, the frontal orientation of the pinna favors sounds coming from the front and helps to differentiate between the sounds arriving from various locations along the front to back axis. In addition, the configuration of the ridges and depressions on the surface of the pinna provides a complex system of resonating cavities and reflecting surfaces that differently affects sounds arriving from various locations along both the vertical axis and the horizontal plane. The effect of pinna reflection on the same sound arriving from different vertical directions is shown in Figure 9-2. Depending on the angle of sound arrival, different ridges of the pinna are involved in sound reflections causing angle-dependent changes in the overall acoustic spectrum of the sound entering the ear canal (Batteau, 1967; Hebrank and Wright, 1974; Lopez-Poveda and Meddis, 1996; Roffler and Butler, 1968).

The relatively small dimensions of the pinna and its features compared to the wavelengths of sound perceived by humans cause the directional function of the pinna to operate primarily in the mid- and high-frequency regions of perceived sound (Wright, Hebrank, and Wilson, 1974). Low-frequency sounds have wavelengths longer than the dimensions of the pinna and are easily diffracted. As a result, the locations of low-frequency sound sources are difficult to localize using pinna mechanisms, that is, along top-down and front-back axes.

The brain uses monaural cues for sound localization on the vertical plane and both monaural and binaural cues for sound localization on the horizontal plane. Both binaural and monaural cues are additionally enhanced by different positions of both pinnae on the head and in relation to the torso, which contributes to the three-dimensional directional characteristic of the human head by causing time delays and intensity changes for both direct and body-reflected sounds entering the ears. In addition, the different patterns of pinna convolutions in the left and right ear of each human affect the directional properties of the head, creating a very unique and non-transferable HRTF for each individual. The detailed discussions of the directional properties of the human auditory system and the limitations of the specific directional cues are presented in Chapter 11, *Auditory Perception and Cognitive Performance*.

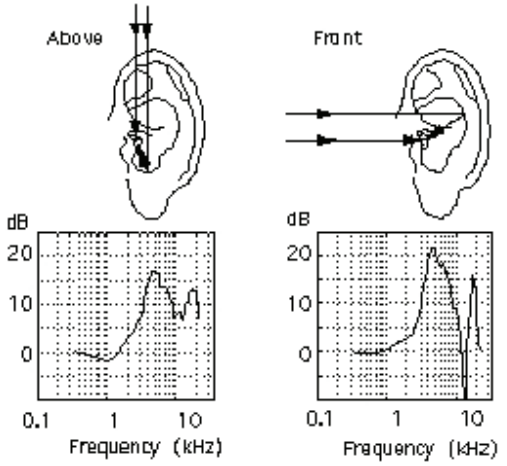

Figure 9-2. Sound spectra at the ear canal of the same sound arriving from two different directions (Duda, 2000).

Selective amplification of sound

After the pinna collects, modifies and channels sound toward the ear canal, the sound is further altered by the resonances of the concha and ear canal. As the sound enters the concha and the ear canal, the sounds of some frequencies are relatively amplified while others are correspondingly suppressed resulting in distinct spectral shaping of the incoming sound by the outer ear. The function of selective amplification of sound by external ear cavities is to enhance the sounds that are important to human behavior and speech communication.

The cavity of the ear canal forms a tube that acts as a ¼ wavelength resonator. This type of resonator enhances sounds of certain frequency and damps others depending on the relationship between the wavelength of sound and the length of the resonator. A ¼-wavelength resonator increases sound pressure at the blocked end of a tube for sound waves that have a wavelength four times the length of the tube and any odd whole number multiple of this wavelength. The resonance frequencies of the ¼ wavelength resonator can be calculated as

$$f_n = \frac{(2n-1)c}{4L} \qquad \text{Equation 9-1}$$

where f_n is an n^{th} resonance frequency of the resonator, c is the speed of sound, L is the length of the tube, and n is the resonance frequency number.

The average effective length of the ear canal (which is the ear canal plus some of the depth of concha) is approximately 30 mm. This means if the ear canal were a hard walled tube with uniform cross sectional diameter, the average ear canal would increase the relative sound pressure at the tympanic membrane at approximately 2833 hertz (Hz) (assuming standard temperature and pressure conditions such that c = 340 meters/second [m/s]). However, the ear canal is lined with soft tissue, does not have a uniform cross-sectional diameter, and varies in

size based on age, gender, and genetic factors. Therefore, the specific resonance characteristics of the ear canal vary with people and age. The specific amplification effects of the ear canal and other parts of the external ear measured by Shaw (1974) are shown in Figure 9-3.

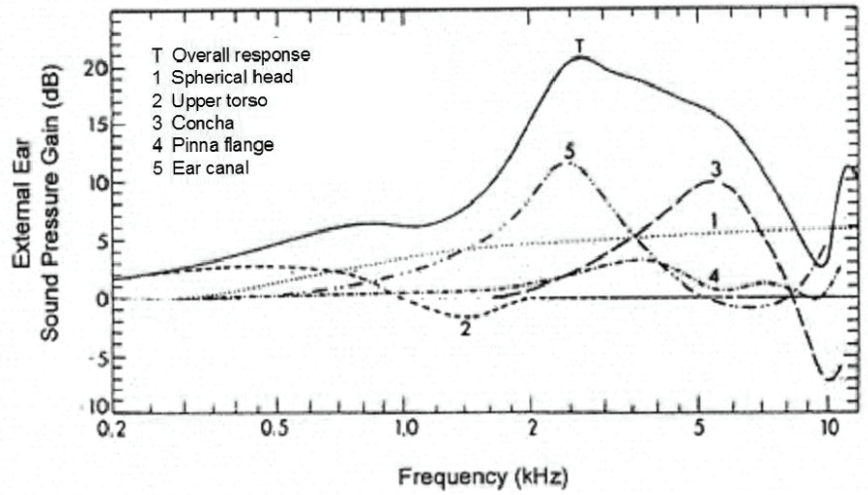

Figure 9-3. The sound pressure gain at the tympanic membrane due to the contribution of the outer ear, head, and body. Direction of sound arrival: azimuth: 45°; elevation: 0° (adapted from Shaw, 1974).

Examination of Figure 9-3 indicates that the resonance properties of the concha increase the sound pressure around 5,000 Hz, the helix and antihelix provide some lesser amount of amplification across a broader frequency range, and the largest resonance is provided by the ear canal. All together the peak of the resonance of the outer ear occurs between 2,000 and 3,000 Hz and is between approximately 15 and 20 decibels (dB). This is the frequency region that is most important for speech communication.

The functions presented in Figure 9-3 show amplification characteristics for sound waves propagation in the horizontal plane and arriving at a 45° angle relative to the front of the head. At this angle of arrival the overall sound pressure gain caused by the head and outer ear is the greatest. For other angles of arrival, the specific gain functions have slightly different shapes providing information approximately the direction of the incoming sound. Some examples of the overall gain functions for sounds propagating in the horizontal plane and arriving at different azimuth angles are shown in Figure 9-4.

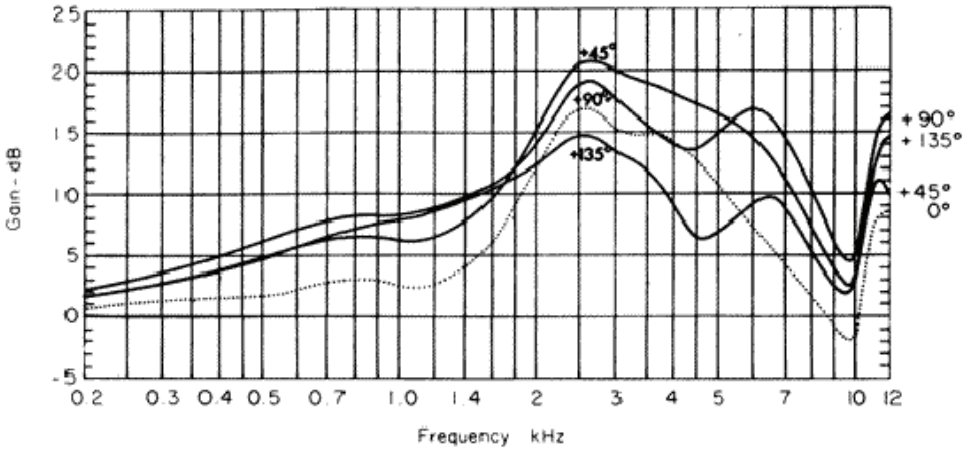

Figure 9-4. Sound pressure gain functions for sound waves propagating in the horizontal plane and arriving at different azimuth degree at the listener ear (Pickles, 1988).

In addition to the frequency-specific selective filtering of sound, the anatomical features of the ear canal (e.g. a long, curved, narrow tunnel) provide a protective barrier between the outside world and the middle ear structures. The length and shape of the ear canal serve to isolate the tympanic membrane from the changes in external temperature, humidity, and the effects of wind. They also make it difficult for dust, small flies, and other debris to reach the tympanic membrane. The skin of the ear canal contains hair follicles and glands that secrete the oily substances that protect the canal from drying and act to repel dust particles and insects (see Chapter 8, *Basic Anatomy of the Hearing System*). Even the shape, position and cartilaginous form of the pinna contribute to hearing system protection by providing a cushion against physical impact to the head.

The Middle Ear

Acousto-mechanic transduction

The primary function of the middle ear is to act as an impedance matching element between the air-filled outer ear and the fluid-filled inner ear. Impedance is the opposition of a system to the energy flow through the system, e.g., to a change in the velocity of motion, and defines the ability of the system to store and transfer energy. Impedance is a vector quantity that has two parts, resistance (real part) and reactance (imaginary part), that are responsible for the transfer and storage of energy, respectively. The transfer of energy from one system to another is most efficient when both systems have the same impedance (Emanuel and Letowski, 2009).

When sound waves reach the tympanic membrane their acoustic energy is converted into physical vibrations of the ossicular chain, which is attached at one end to the tympanic membrane to the membrane covering the oval window of the inner ear at the other, providing an anatomical bridge between the outer and inner ears. Without this conversion of sound energy into mechanical energy, the amount of energy delivered to the inner ear would be significantly less than the amount of energy arriving at the tympanic membrane.

When the tympanic membrane vibrates in response to changes in sound pressure in the ear canal, its vibration is confined primarily to the *pars tensa*, which constitutes approximately two-thirds of the membrane's surface (see Chapter 8, *Basic Anatomy of the Hearing System*). However, both parts of the tympanic membrane, the *pars tensa* and the *pars flaccida*, are responsible for the tension of the membrane. The tension of the tympanic membrane directly affects hearing sensitivity. If the membrane is too flaccid, most sound energy is absorbed by the membrane itself, that is, it goes into stretching the membrane. If the membrane is too tense, too much sound energy is reflected back into the environment. Therefore, the tympanic membrane must be at the appropriate tension for sound energy to be converted efficiently into the mechanical movement of the ossicles in the middle ear.

The middle ear is normally filled with air, and under normal operation conditions, the static air pressure in the middle ear is the same as the atmospheric pressure in the ear canal. Equal air pressure on both sides of the tympanic membrane is needed to establish proper tension on the membrane. The pressure in the middle ear cavity is maintained by the periodic opening and closing of the eustachian tube (auditory tube). This tube connects the middle ear with the nasopharynx (back of the throat) and can be opened or closed by the action of the tensor veli palatine muscles (muscles from the velum and palate). The tube is normally closed, but it pops open during yawning and swallowing. A swelling of the surrounded tissue can cause tube malfunction and consequently a lack of proper air pressure in the middle ear. If the air pressure in the middle ear cavity is significantly different from the pressure in the ear canal, this may cause over- or under-stretching of the tympanic membrane, which leads to inefficient sound transmission, pain and can also produce middle ear diseases.

Impedance matching

The acousto-mechanical transformation of the received sound energy serves to match the high acoustic impedance [1]of the fluid-filled inner ear with the low acoustic impedance of the air in which sound waves propagate and to optimize energy transfer between these two systems. In order to calculate the actual mismatch one needs to know the input impedance of the oval window and the impedance of the source from which the sound impinges on the window (Killion and Dallos, 1979). The acoustic input impedance of the oval window has been calculated by Zwislocki (1975) to be approximately 350,000 acoustic Ω [dyne · s/cm^5]. This value is based on Békésy's low frequency impedance data corrected for postmortem effects (Békésy, 1942, 1949, 1960). At higher frequencies this impedance is probably closer to 1,200,000 acoustic Ω measured for a cat's ear (Lynch, Nedzelnitsky and Peake, 1982). Both these impedances are much higher than the characteristic impedance of air, which is approximately 41.5 centimeter-gram-second system (cgs) rayls [dyne · s/cm^3] at 30°C temperature.

The specific acoustic impedance of air in the ear canal is the characteristic impedance of air normalized by the cross-sectional area of the canal (0.45 cm^2) and equal approximately 100 acoustic Ω (Shaw, 1974, 1997; Zwislocki, 1957, 1970, 1975). The power transmission index η describing power transmission from the ear canal to the tympanic membrane has been reported to be approximately η=0.75 and fairly similar across all mammals (Hemilä, Nummela and Reuter, 1995; Møller, 1974; Voss and Allen, 1994). Thus, assuming that both the input impedance of the tympanic membrane (Z_t) and the characteristic acoustic impedance of air in the ear canal (Z_{ec}) are resistive, the relationship between Z_t, Z_{ec}, and η can be written as:

$$\eta = \frac{4Z_t Z_{ec}}{(Z_t + Z_{ec})^2} \qquad \text{Equation 9-2}$$

If Z_{ec}=100 acoustic Ω, and η = 0.75, then the impedance of the tympanic membrane equals $Z_t = 3Z_{ec}$ = 300 acoustic Ω, and the impedance ratio of the inner ear fluid (cat's data) and the tympanic membrane is approximately 4000.

In order to ensure an efficient transfer of energy between the acoustic system of the ear canal and the hydraulic system of the inner ear, the middle ear must compensate for this mismatched impedance by increasing the pressure between the tympanic membrane and oval window by approximately 63 times ($63^2 \sim 4000$). This is equal to a 36 dB increase in sound pressure level (SPL). In other words, the pressure acting on the fluids in the inner ear must be 36 dB higher than pressure acting on the tympanic membrane to ensure the most efficient transfer of acoustic energy to the inner ear. This is the role of the middle ear transformer, consisting of the tympanic membrane, ossicles, and oval window membrane. Without the impedance matching function of the middle ear, more than 99.9% of the acoustic energy acting on the tympanic membrane would be reflected by the tympanic membrane back into the ear canal and not used. If the human middle ear matching function is not functioning properly, sound can only be transmitted via a shunt pathway (tympanic membrane to the air in the middle ear to the fluid of the inner ear), which results in the transmission of less than 0.1% of the input energy.

The impedance matching system of the middle ear consists of three separate mechanisms:

1. Area ratio transformation
2. Ossicular chain lever action
3. Catenary lever action

[1] Acoustic impedance is defined as the ratio of effective acoustic pressure averaged over a given surface to effective volume velocity of acoustic energy flowing through this surface. The units for impedance are Pa-s/m^3 or dyne-s/cm^5, which are called the acoustic ohm (Ω).

All three mechanisms contribute to the overall pressure transformation, however, the first mechanism, the area ratio transformer, is the most essential to the impedance matching process. The presence of the last mechanism, the catenary lever action, is still somewhat controversial, and there is some disagreement in the literature regarding its contribution (Goode, 2006).

Area ratio transformation

The area ratio (pressure) transformer is the first and most effective of the three impedance matching mechanisms. It results from the difference in surface area between the tympanic membrane and the membrane covering the oval window. The principle of this mechanism is illustrated in Figure 9-5.

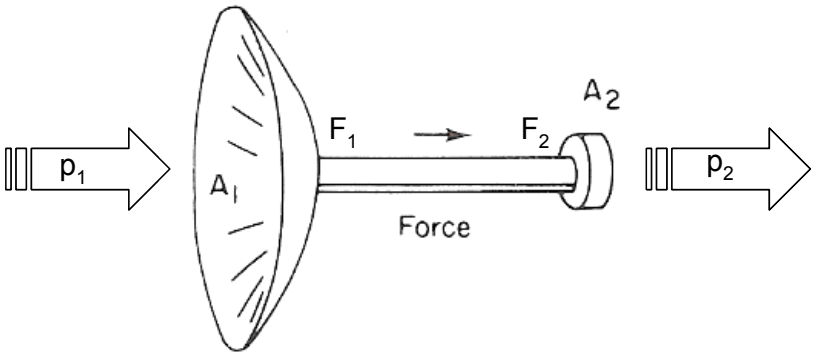

Figure 9-5. Schematic drawing of surface area mismatch between tympanic membrane and oval window membrane (adapted from Pickles, 1988).

In Figure 9-5, a pressure p_1 acts over the surface of the tympanic membrane and results in a force F_1. Assuming that the ossicular chain is a lossless system, the force F_2 acting on the oval window is equal to force F_1, that is, $F_1 = F_2 = F$. Since force (F), surface area (A), and pressure (p) are related by the equation p=F/A, then

$$F = p_1 \times A_1 = p_2 \times A_2 \qquad \text{Equation 9-3}$$

and

$$p_2 = p_1 \times \frac{A_1}{A_2}. \qquad \text{Equation 9-4}$$

Since the vibrating area of the tympanic membrane ($A_1 = 55mm^2$) is approximately 17.2 times larger than the vibrating area of the oval window membrane ($A_2 = 3.2mm^2$), this results in an increase in SPL at the oval window of approximately 25 dB.

Ossicular chain lever action

The second impedance matching mechanism of the middle ear, the ossicular chain lever action, involves the rotational motion between the malleus and stapes. This kind of motion is possible because the ossicles are fixed at the junction between the malleus and incus while being suspended in the middle ear cavity by the anterior ligament of the malleus (anteriorly) and the posterior ligament of the incus (posteriorly). This arrangement creates a central pivot point (fulcrum) and allows for the relative rotational motion of the malleus and stapes, thereby forming a lever mechanism. The principle of this mechanism is shown in Figure 9-6.

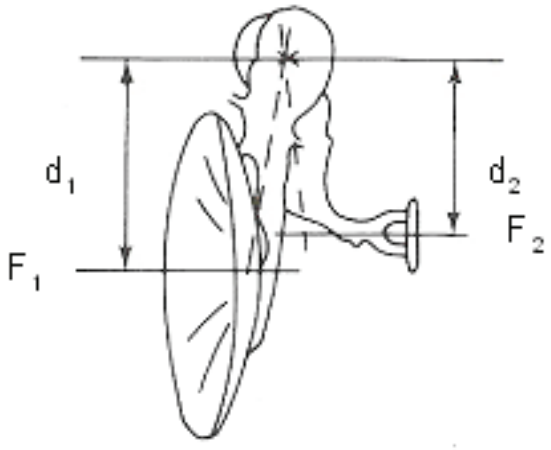

Figure 9-6. Schematic drawing of ossicular chain level action (adapted from Pickles, 1988).

In a lever system, a force F_1 applied at effort arm d_1 results in force F_2 acting on effort arm d_2, that is

$$F_1 x\ d_1 = F_2 x\ d_2. \qquad \text{Equation 9-5}$$

In the case of the ossicular chain lever, the forces F_1 and F_2 are the forces acting at the malleus and stapes and the distances d_1 and d_2 are the lengths of the malleus and stapes, respectively. Since the length of the malleus is approximately 1.3 times longer that the length of the stapes, this increases the force between the tympanic membrane and the oval window membrane by approximately 2 dB (Békésy, 1941; Wada, 2007). It should be noted that some authors recommend using 1:1.15 (1.2 dB increase) rather than 1:1.3 (2. dB increase) ratio in order to compensate for the fact that the malleus and the tympanic membrane act as a coupled system (Battista and Esquivel, 2003; Pickles, 1988).

Catenary lever action

The third impedance matching mechanism, the catenary lever action, curved membrane effect, or buckling effect of the tympanic membrane, was first explained by Helmholtz (1868), who observed that the umbo of the tympanic membrane is displaced less than the remaining surface of the tympanic membrane. Since the outside edge of the membrane is firmly attached to the annulus and curves medially to attach to the umbo, the displacement of the membrane between the annulus and umbo is larger than at the umbo (Khanna and Tonndorf, 1970; Tonndorf and Khanna, 1970). This creates a lever action which increases the force acting at the umbo by approximately 2 times or 6 dB (Rosowski, 1996). The principle of this mechanism is shown in Figure 9-7.

Overall transformation

The 63 (36 dB) ratio needed to compensate for the air-to-cochlea impedance mismatch is called in the literature the *ideal transformer prediction* (Rosowski, 1996) but is not fully realized by the middle ear system. The three impedance matching mechanisms together increase the sound pressure at the footplate of the stapes by approximately 40 to 45 times (32 to 33 dB) in comparison to the sound pressure acting on the tympanic membrane. This increase is approximately 3 to 4 dB short of completely making up for the impedance mismatch. However, the 32 to 33 dB effectiveness of impedance matching mechanisms agrees with physiological findings that completely disconnected ossicular chain causes a hearing loss (due to the air-bone gap) of approximately 32

dB (Rosowski, Mehta and Merchant, 2004), indicating that the transformer model described above adequately represents the real-world operation of the middle ear system.

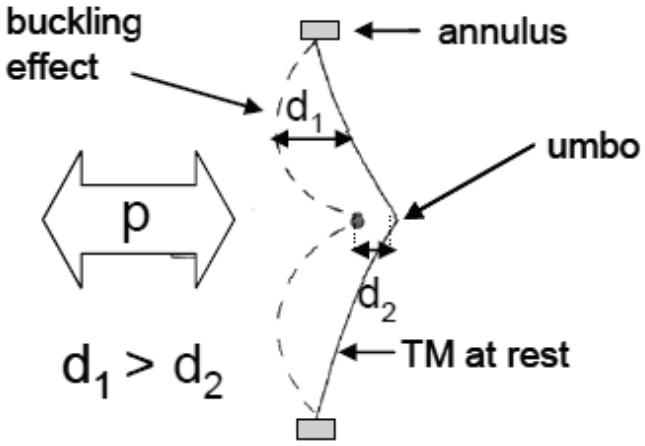

Figure 9-7. Schematic drawing of the catenary lever action of the tympanic membrane (TM); p-acoustic pressure, d–membrane displacement; (adapted from Pickles, 1988).

The impedance matching provided by the middle ear is most effective between approximately 500 and 3000 Hz but becomes less effective as the sound frequency is further away from this region (Battista and Esquivel, 2003; Nedzielnitsky, 1980; Puria, Peake and Rosowski, 1997). At low frequencies, the impedance of tympanic membrane becomes reactive and impedes the transfer of energy. Above 1000 Hz, the tympanic membrane changes its vibration pattern, resulting in a decrease in the area of the membrane contributing to the vibration (Tonndorf and Khanna, 1972). In addition, the ossicles vibrate less efficiently at frequencies above 2000 to 3500 Hz, affecting the lever mechanism and resulting in a decrease in energy transfer for higher frequencies (Battista and Esquivel, 2003). Sound transmission through the middle ear also can be affected by the air pressure in the middle ear cavity, abnormal inner ear impedance, and air coupling between the oval and round window membranes. The non-ideal operation of the middle-ear transformer at higher frequencies is, however, greatly ameliorated by the outer ear resonances. Thus, the combined effects of the outer and middle ear systems overcome their individual limitations, which would otherwise result in a large loss in the amount of energy transferred from the air to the inner ear fluid.

A natural way to assess the effect of the middle ear impedance transformer on sound transmission through the hearing system is to measure directly the input impedance of the tympanic membrane. Resistance and reactance of the tympanic membrane measured by Zwislocki (1975) for male and female populations are shown in Figure 9-8.

An examination of Figure 9-8 indicates that stiffness reactance (primarily due to the stiffness of the tympanic membrane) is the primary component in middle ear reactance. It offers the greatest opposition to the flow of energy for sounds below approximately 500 Hz and becomes negligible above approximately 800 Hz. Mass reactance (primarily due to the mass of tympanic membrane and ossicular chain) is negligible for the mid frequencies but is the primary contributor to reactance above approximately 5,000 Hz. Most importantly, the resistance of the middle ear (tympanic membrane), which affects energy transmission of most sounds within the auditory range of frequencies, varies between 200 and 400 acoustic Ω and is relatively independent of frequency across the entire range of measured frequencies (200 Hz to 8,000 Hz) (Shaw, 1997; Zwislocki, 1975). The average value of the middle ear impedance in this frequency range is approximately 300 acoustic Ω (Shaw, 1974), which agrees with the value calculated earlier in this chapter on the basis of energy reflected from the tympanic membrane.

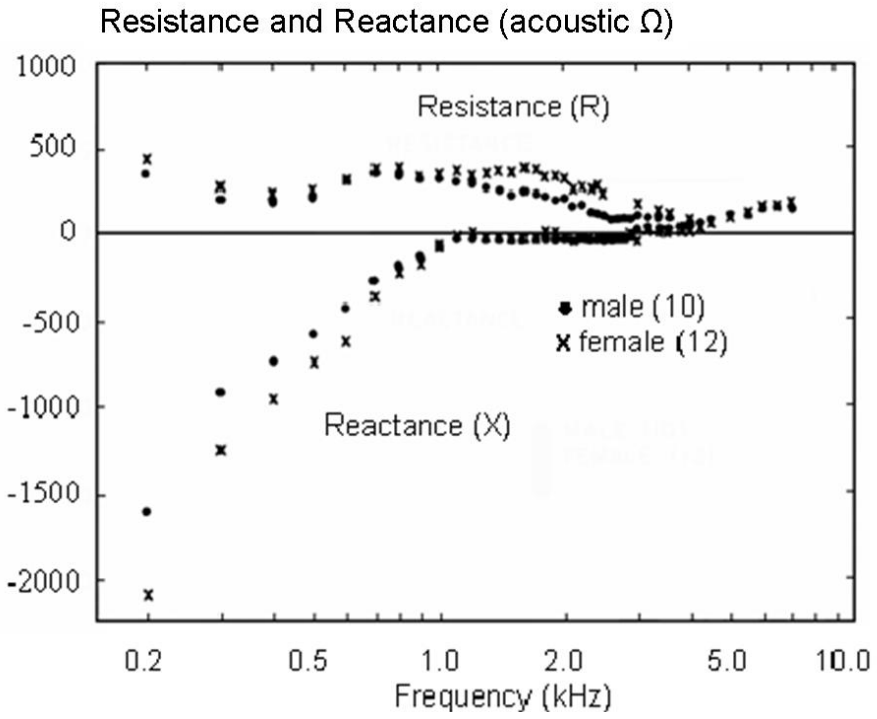

Figure 9-8. The effects of gender on resistance and reactance of middle ear impedance (adapted from Zwislocki, 1975).

Acoustic reflex

In addition to acousto-mechanical energy transformation and impedance matching, the middle ear also provides some limited protection to the inner ear against very strong stimulation. When very high acoustic pressures arrive at the tympanic membrane, the tensor tympani and stapedius muscles of the middle ear contract and temporarily stiffen the middle ear system, thereby decreasing the efficiency of energy flow through the middle ear. When the muscles are activated, the tensor tympani stiffens the tympanic membrane by pulling it toward the middle ear, and the stapedius muscle stiffens the stapes by rotating it away from its normal axis of action. This protective mechanism is known as the *acoustic reflex* or middle ear reflex and causes a 15- to 20-dB attenuation in the transmitted sound (Bess and Humes, 1990). While the role of the stapedius muscle in the acoustic reflex is generally accepted, the role of the tensor tympani has recently been questioned due to the long response latency of the tensor tympani action (approximately 100 ms) (Bosatra, Russolo and Semerano, 1997).

The SPL that triggers the acoustic reflex varies among people but is generally approximately 80-90 dB HL (above threshold of hearing). An important limitation of the acoustic reflex is that it primarily operates at low frequencies (below 4000 Hz), long latency of 35 to 150 ms (Møller, 1962), and that its contraction can be limited in duration to as little as a few seconds (e.g., at 4000 Hz). However, it is noteworthy that middle ear muscles are also activated before an onset of vocalization or chewing and remain contracted for the duration. Therefore, in addition to protecting inner ear from too intense low frequency external stimulation, the middle ear muscles may be protecting the inner ear from noise generating by the muscles activated during vocalization and jaw movements (Simmons, 1964). It is also possible that another goal of attenuation of low frequency energy by acoustic reflex is to improve audibility high frequency stimuli that are subjected to masking by low frequency stimuli (see Chapter 11, *Auditory Perception and Cognitive Performance*.

The Inner Ear

Cochlear mechanism

The cochlea of the inner ear is the system that converts the mechanical energy of the stapes motion into electro-chemical impulses that can be transmitted by the central auditory nervous system to the auditory centers of the brain. The first stage in this process is the conversion of the stapes' motion into motion of the fluids of the cochlea and the subsequent creation of a traveling wave moving along the basilar membrane. The induced movement of the basilar membrane affects the motion of the stereocilia of the outer and inner hair cells. The outer hair cells provide an amplification function, increasing the amplitude of the incoming sound wave, while the inner hair cells are sensory receptor cells, changing the mechanical motion of the stereocilia into the release of a neurotransmitter chemical that communicates with the auditory portion of the vestibulocochlear nerve. Therefore, the sound reception process in the inner ear is an active process that dissipates some of its energy in the form of otoacoustic emissions.

The complex process by which the cochlea breaks down the mechanical motion of the basilar membrane and translates it into a series of nerve impulses that can be transmitted, reassembled, and interpreted has been theorized for over a century but is still under investigation.

Traveling wave

The oval and round windows of the cochlea are both covered with elastic membranes that can bulge in and out of the cochlea. An inward motion of the stapes into the scala vestibuli causes movement of the incompressible perilymph from the scala vestibuli into the scala tympani. After the fluid passes through the helicotrema at the apex of the cochlea, the round window membrane bulges out to accommodate the increased amount of fluid in the scala tympani. A motion of the stapes away from the scala vestibuli causes perilymph to move from the scala tympani into scala vestibule and the membrane of the round window to consequently bulge inwards. The motion of the inner ear fluid caused by the inward and outward motions of the stapes creates a traveling (transverse) wave motion along the basilar membrane. The basilar membrane responds differently to sound stimuli of different frequencies, making the location where it reaches its maximum displacement depend on the frequency of the sound wave. There is a systematic shift in the point of maximal vibration from the apex toward the base as the frequency increases. Thus, the basilar membrane is said to be tonotopically organized. A view of a traveling wave moving along an uncoiled basilar membrane is shown in Figure 9-9.

The traveling wave was first described by Békésy (1953; 1955), who worked with cadaver ears and ear models. He found that the point at which the displacement of the basilar membrane was the greatest was dependent on the frequency of the sound wave and considered the traveling wave mechanism to be responsible for the sound analysis done by the cochlea. The theory of hearing, or of sound perception, based on this concept is called the traveling wave theory. There is however another competing theory of hearing, called the resonance theory, that views the basilar membrane as an array of sequentially tuned tiny resonators distributed along the membrane. This theory was originally proposed by Helmholtz (1885) and states that the tiny resonators of the basilar membrane are set directly into motion by sound pressure changes in the perilymph without needing the traveling wave to set them off. Both of these theories belong to a larger group of theories of hearing, called the place theories, which support the tonotopic organization of the basilar membrane. Place theories as well as the other group of theories of hearing, called periodicity theories, will be addressed in the frequency coding section of this chapter.

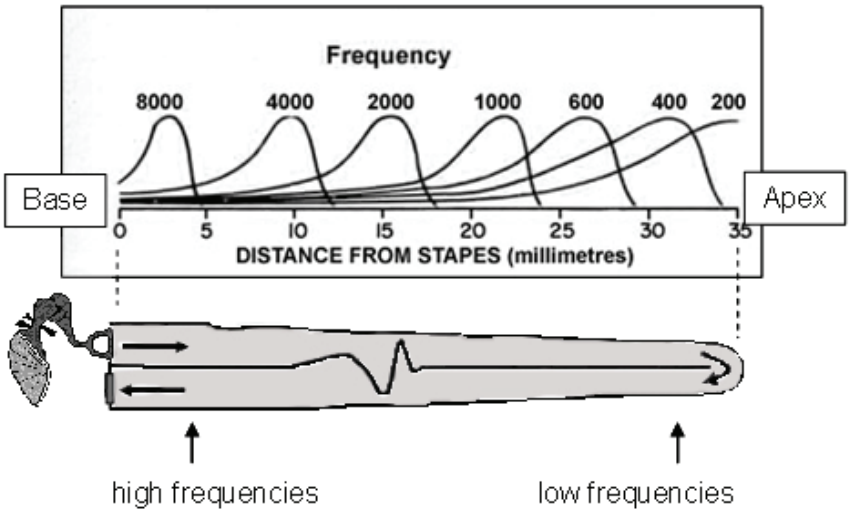

Figure 9-9. View of an uncoiled cochlea and the traveling wave (adapted from Bear, Connor and Paradiso, 2001).

There are a number of experimental studies which support either the traveling wave or the resonance theory of hearing, but there is still a lack of complete agreement in the literature as to whether the traveling wave is directly responsible for the basilar membrane motion or whether it is a secondary effect caused by the direct stimulation of basilar membrane's resonators by sound pressure propagating through the perilymph (Bell, 2004; Bell and Fletcher, 2004). Regardless of the theory behind the true manner in which the cochlea analyzes sound, the basilar membrane is the element responsible for sound analysis, and its tonotopic organization is mirrored within the auditory nervous system.

Current research, as well as some older studies (e.g., Gold, 1948, 1987; Gold and Pumphrey, 1948), supports the notion that the basilar membrane is not a passive element of the auditory system and that its action is amplified by an active mechanism in the cochlea called the *cochlear amplifier*. Thus, the fairly small motion of the basilar membrane caused by low and mid intensity sounds is amplified by the cochlea prior to the transmission of these signals from the cochlea to the vestibulocochlear nerve. The action of the cochlear amplifier has been attributed to the motility function of the outer hair cells, which expand and contract along their long axis in response to voltage changes across the cell membrane.

Electric potentials in the inner ear

The neural activity of the inner ear is dependent on electro-chemical processes and initial electric potentials between the fluids occupying the various structures of the inner ear. An electric potential is created when there is a difference in electric charge between two different locations. The area of higher charge is said to be positively polarized while the area of lower charge is said to be negatively polarized. In a biological system, such as the human ear, a difference in chemical charge between two areas is called a bioelectric potential. When the two polarized areas are connected, the charged particles move from one area to another. This occurs because of the electromotive force that is created by the difference in electrical charge. Common charged particles in the human ear include positively-charged potassium ions (K^+), negatively-charged chloride ions (Cl^-), positively-charged sodium ions (Na^+) and positively-charged calcium ions (Ca^{2+}). In the inner ear, the endolymph contains a large amount of potassium ions and the perilymph contains a large amount of sodium ions. A static bioelectric potential

that involves the separation of charged particles by a cell membrane is called a *resting potential*. The resting potential of the endolymph in the scala media, called the *endocochlear potential* (EP**)**, is + 80 millivolts (mV) in reference to the resting potential of the perilymph in the two other cochlear channels. The resting potential of the inner hair cells is -40 mV and of the outer hair cells is -70 mV compared with the perilymph (Jahn and Santos-Sacchi, 2001). Therefore, the difference in potential between the endolymph and the inner hair cell is 120 mV and between the endolymph and the outer hair cell is 150 mV. This 150-mV potential is a biological battery that supports all inner ear processes. It is a very efficient system that consumes only approximately 14 microwatts (μW) of power while carrying out the equivalent of approximately one billion floating-point operations per second (1Gflops) (Zhak, Mandal and Sarpeshkar, 2004).

A dynamic bioelectrical potential that involves the movement of charged particles from one area to another in response to a stimulus is called a *stimulus related potential*. There are three stimulus related potentials that are commonly observed in the inner ear in response to an auditory stimulus. These are the summating potential (SP), cochlear microphonic (CM), and compound action potential (CAP). The former two are generated by the hair cells and the latter is generated by the vestibulocochlear nerve

The SP is a direct current potential that causes a positive or negative change in the endocochlear potential for the duration of a signal. It is the driving force for moving the charged ions through the stereocilia and membrane separating hair cell from the surrounding endolymph.

Both the CM and CAP are alternating current potentials that vary in polarity based on changes in the phase of the signal. The CM is a pre-neural electric potential that mimics the incoming sound signal; it is considered to be a reflection of receptor currents flowing through the hair cells. The CAP is the actual event related potential (ERP) that is generated when the auditory nerve "fires" (transmits a signal) in response to a stimulus. The CAP results from the firing of the auditory portion of the vestibulocochlear nerve in response to the release of neurotransmitter from the hair cells. Various ERPs and their changes in time and space can be measured in the ecenral nervous system using electroencephalography (EEG). One example of such potentials is the mismatch negativity (MMN) potential generated in the auditory cortex and having a latency of 150 to 250 ms post-stimulus. The MMN is a negative, task independent neural potential generated in response to an infrequent change in a repetitive sound sequence.

Hair cell action

Up and down movement of the basilar membrane causes shearing force acting on the cilia of the hair cells of the organ of Corti. The shearing force is a result of different points of attachment of the basilar membrane and the tectorial membrane to the cochlear wall. The force bends the cilia to the left and to the right of the basilar membrane axis. The stereocilia of a hair cell have gradually changing height and are held together by tip-to-side links that cause the whole bundle to move together when stimulated. Tilting movements of the stereocilia affect the tension on the fiber in the tip link. When the stereocilia are bent toward the largest stereocilium, the tip-to-side links cause mechanically-gated ion channels in the stereocilia membranes to open. The opening of the ion gates allows positively-charged ions (K+) of potassium, which are the main cations in the endolymph, to flow from the positively-charged endolymph into the negatively-charged hair cell. As the fiber tension increases, the flow of ions into the hair cell also increases. When the stereocilia bundle is bent in the direction away from the largest stereocilium, the ion channels close and the excess of K+ in the cell is pushed out of the cell through a semi-permeable membrane via active pumping processes restoring natural negative polarization of the cell (Geisler, 1998). However, the effects of stereocilia bending and the in-and-out flow of K+ ions are different in the inner and the outer hair cells.

When the K+ ions enter the inner hair cell, they depolarize the content of the cell, that is, they change to zero the difference in electric potentials between the areas inside and outside the cell. As a result, when the gates are open, the cell becomes depolarized (excited), and when the gates are closed, the cell becomes hyperpolarized (inhibited). These actions are shown in Figure 9-10.

The change in the electric potential across the membrane of the inner hair cell opens voltage-dependent calcium (Ca+) channels in the cell membrane. The flow of Ca+ ions triggers a release of a chemical neurotransmitter into the synaptic cleft between the hair cell and the afferent nerve ending at the basal end of the cell. The release of the neurotransmitter excites the dendrite endings of the afferent neurons connected to the inner hair cells and results in the generation of action potential in the neurons. Thus, the change in the resting potential of the inner hair cell due to K+ ions influx results in generating a stimulus related potential at its synaptic juncture with the afferent nerve fiber and in subsequent firing of the fiber.

When K+ ions enter and leave the outer hair cells, these cells alternately contract and expand in response to the alternating current generated by the polarization and depolarization of the cell walls. Outer hair cells are anchored both at their top (at the base of the stereocilia) and at their bottom but are not firmly attached at their sides. Thus, the expansion and contraction of the outer hair cells pushes up on the reticular lamina and down on the support cells and basilar membrane. Active motions of outer hair cells (OHCs) increases the range of motion of the basilar membrane, thereby causing larger deflections of inner hair cells (IHCs), and "sharpens" the shape of the traveling wave motion along the basilar membrane.

Each hair cell is connected to both afferent and efferent neurons. When activated by a hair cell, the afferent neuron conducts stimulus related potential up to the central nervous system. The brain-controlled action of the efferent neuron is to release a neuro-inhibitor acetylocholine (Ach) in a synaptic juncture with the hair cell or with a respective afferent neuron to impede hair cell action.

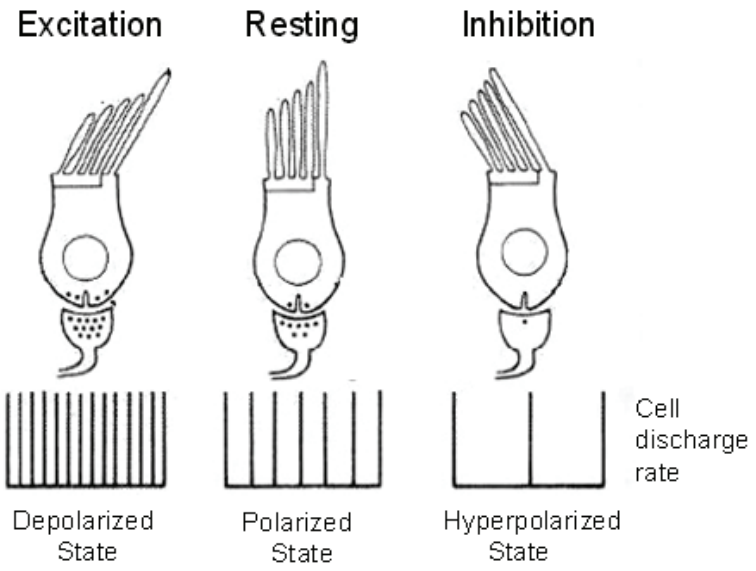

Figure 9-10. Inner hair cell response to bending of the stereocilia.

Cochlear amplifier and otoacoustic emissions

The amplification of basilar membrane's vibration caused by the motility of the outer hair cells is not linear and varies based on the intensity of the incoming signal. Low intensity signals are amplified more than high intensity signals. It is generally accepted that an active mechanism of amplification of the IHC responses by OHC motility operates in the range to 50 dB SL (Stebbins et al. 1979).

A by-product of non-linear activity of the outer hair cells is nonlinear distortion of the basilar membrane movements. The distortion products generated in the cochlea travel from the inner ear through the middle ear and into the ear canal in a transmission process that is the reverse to the process of hearing. They have a form of

cochlear echoes, more formally called otoacoustic emissions (OAEs), which can be observed in the ear canal. It is still unclear, however, exactly how the waves travel backwards along the cochlea to the oval window, that is, whether they are slow traveling waves along the basilar membrane or faster compression waves in the perilymph. The results of recent studies favor compression waves; however, the debate is still unsettled.

The OAEs produced by nonlinear actions of OHCs have been first reported by Kemp (1978) and can be measured in the ear canal using sensitive microphones and applying signal averaging techniques to the collected data. The presence of the OAEs in the outer ear canal is an evidence of an active amplification process within the cochlea.

There are two types of OAEs that can be observed in the ear: spontaneous OAEs and evoked OAEs. Spontaneous OAEs are present in approximately 50% of people with normal hearing and result from random processes ongoing in the inner ear. Evoked OAEs are the ear responses to external stimulation and are present in nearly 98% of the normal ears. They are usually evoked for audiologic assessment of the ear function with either a click stimulus or two simultaneous pure tones f_1 and f_2, whose ratio is between 1.1 and 1.3. The most prominent and commonly observed evoked OAE component is the cubic difference distortion product denoted as $f_{cd} = 2f_1\text{-}f_2$.

Auditory nerve: The link

The transduction of the mechanical actions of the shearing of the stereocilia to the electro-chemical signal transmitted by the nervous system begins when a neurotransmitter is released from the base of the inner hair cell. This neurotransmitter crosses the synaptic cleft and binds to specialized receptor sites located on the post-synaptic membrane of the peripheral processes of the nerve fibers connecting the inner ear with the brainstem. The bundle of nerves connecting the inner ear with the brainstem is called the auditory nerve. If sufficient neurotransmitter is released, the afferent nerve fibers will fire in response, sending an electric signal down the length of the auditory nerve towards the brainstem. The innervations of the inner and outer hair cells by the fibers of the auditory nerve are shown in Figure 8-18 of Chapter 8, *Basic Anatomy of the Hearing System*.

The auditory nerve (cochlear nerve, acoustic nerve) is a part of the vestibulocochlear (VIII) cranial nerve comprised of the vestibular nerve (not discussed in this chapter) and the auditory nerve, The auditory nerve is a bipolar nerve with cell bodies (collectively called the spiral ganglia) located within Rosenthal's canal in the cochlea. The peripheral projections (dendrites) of the cells synapse with the hair cells and the central projections (axons) synapse with other nerve cells located in the cochlear nucleus of the brainstem. The fibers of the auditory nerve that transfer information from the cochlea to the brainstem are mostly myelinated, i.e., covered with an insulating lipid membrane that wraps around the nerve fiber and acts as an electrical insulator.

The tonotopic organization of the basilar membrane is passed to the auditory nerve via a tonotopic arrangement of auditory nerve fibers. The fibers that originate in the low frequency area (apex) of the basilar membrane run in the center of the auditory nerve while the fibers that originate in the high frequency area (base) of the basilar membrane are in the periphery of the auditory nerve. Therefore, damage to the outside of the auditory nerve will result in high frequency hearing loss, while damage to the central core of the nerve trunk will cause low frequency hearing loss.

The nerve conduction velocity or the speed at which the neural impulse travels along the neural pathways depends on the size of the neuronal fiber (axon or dendrite) – larger fibers conduct impulses faster than smaller ones. In addition, nerves with myelin sheathing have faster conduction times than the un-myelinated ones. It is only at the gaps in the myelin (nodes of Ranvier) that electrically-gated ion channels will open and close in response to the impulse traveling down the nerve. When the impulse travels along an unmyelinated axon, the ion channels open and close along the entire axon, slowing the conduction time. The type I bi-polar neurons responsible for conveying sound information to the central auditory system have myelin coating most of their length and thus are relatively efficient conductors of nerve impulses. The size of the auditory nerve fibers, at least in children, is relatively uniform, suggesting that nerve conduction velocity in the auditory nerve is similar, which preserves the timing coding created at the level of the cochlea (Spoendlin and Schrott, 1989).

Frequency coding in the cochlea

There are a number of theories that attempt to explain how the motions of the hair cells work to code sound. In other words, trying to explain how the firing pattern created by the periodic release of neurotransmitter from the inner hair cells to the auditory nerve results in a signal that the brain can interpret as sound. Two major classes of the theories of hearing that try to explain how signal frequency is encoded by the cochlea are place theories and periodicity (frequency, time) theories.

Place theories of hearing

Place theories of hearing, such as the traveling wave theory and the resonance theory, assume that sound frequency is coded along the basilar membrane by the place at which the membrane vibrates the strongest in response to the acoustic stimulus. Recall that the physical properties of the basilar membrane change gradually along the length of the membrane and the high and low frequency vibrations reach their maximum amplitudes at the base and the apex of the membrane, respectively. The place along the basilar membrane that vibrates the strongest maximally stimulates the hair cells at that location and these, in turn, stimulate the auditory neurons at that location. However, since the vibration of the basilar membrane extends always over a certain finite area, it does not just activate a single discrete row of neurons but also those in the surrounding region. Therefore, one of the important properties of the basilar membrane treated as a place-dependent coder of signal frequency is frequency selectivity of nerve fibers located along the basilar membrane. This selectivity can be measured and expressed by tuning curves of various fibers.

A tuning curve represents the changes in the minimal response threshold of auditory fibers as a function of frequency. The tuning curve for a single auditory fiber resembles the letter “V” where the tip of the tuning occurs at the characteristic frequency (CF) of the fiber. The CF is the sound frequency at which a fiber has its lowest threshold, i.e., it fires in response to the sound at a fairly low intensity level compared to sounds with other frequencies. This same nerve will also fire in response to other frequencies, but the threshold will be much higher. Generally, the further away in frequency from the characteristic frequency, the greater the intensity is required for the nerve to fire. The tuning curves can be measured using both psychoacoustic and physiologic methods. Some examples of psychoacoustic tuning curves are shown in Figure 9-11.

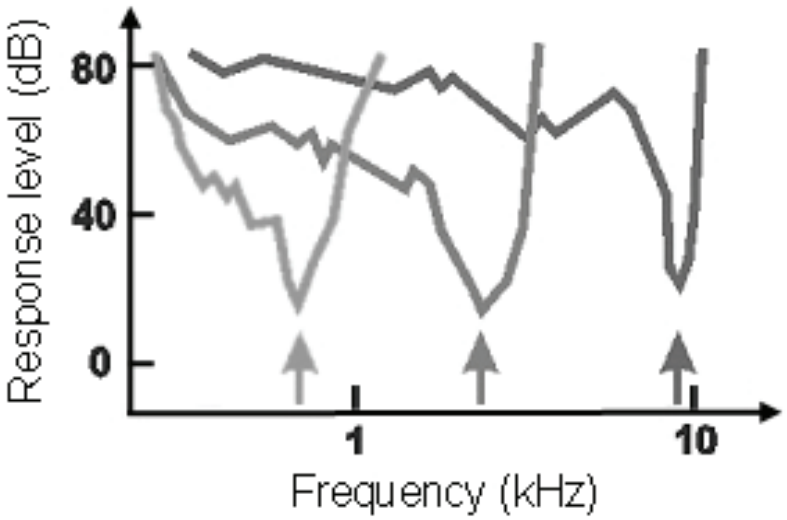

Figure 9-11. Psychoacoustic tuning curves.

Periodicity theories of hearing

Periodicity (frequency, time) theories of hearing, such as the telephone theory (Rutherford, 1886) or volley theory (Wever, 1949), state that the perception of sound depends on the temporal pattern of the sound wave and that the sound frequency is coded by the number of neural impulses per second that are fired by a group of nerve fibers along the basilar membrane. Periodicity theories claim that single nerve fibers of the basilar membrane need not respond to every successive wave of the sound stimulus but could respond only to every second, third, or fourth wave. Each wave is thought to excite a separate group of nerve fibers and the pattern of neural impulses reaching the brain by the successive "volley of impulses" represents the frequency of the sound.

Periodicity coding assumes that during the positive phase of a sound wave the stereocilia are sheared in one direction and in the negative phase of the wave, the stereocilia are shearing in the opposite direction. All nerve fibers have some amount of random, spontaneous firing. When stimulated, the firing rate increases and when inhibited from firing, the firing rate decreases. When the shearing of the outer hair cell stereocilia is in an excitatory direction, neurotransmitter is released and the receiving neuron is stimulated (assuming sufficient neurotransmitter). When the shearing is in the inhibitory direction, the firing rate decreases from the resting firing rate. Thus there is a distinction in the firing code between phases with positive polarity and phases with negative polarity. Sound waves containing a periodicity will have a firing rate that follows the period of the sound wave up to approximately 4,000 to 5,000 Hz (Kiang et al., 1965; Rose et al., 1967). Individual neurons cannot fire at such high rate, but groups of neurons acting together can, a phenomenon known as population coding.

Newer research indicates that place coding and periodicity coding actually work together in coding signal frequency. According to this concept the place coding mechanism acts like a series of tuned filters which divide the signal into pieces that are more easily transmitted in the form of periodicity codes (d'Cheveigne, 2005). The most common current view on frequency coding, holds that low frequencies up to approximately 400 Hz are coded by signal periodicity and high frequencies above approximately 4000 Hz by place of excitation. The frequencies in 400 to 4000 Hz range are coded by a combination of place and periodicity codes.

Intensity coding in the cochlea

Place coding and *periodicity coding* are the two ways in which sound frequency is coded into neural impulses. These theories, however, do not explain how the ear codes the intensity of the sound.

According to current literature, there are two basic ways in which the intensity of the sound can be coded in the cochlea. First, the more intense the stimulus, the larger the excitation of the basilar membrane and the larger the number of neural fibers that can fire simultaneously. Thus, a high intensity sound may stimulate a broad range of cells and the number of cells that fire may represent the intensity of the stimulus.

Second, auditory neurons, like all nerve fibers, fire spontaneously even in the absence of stimulation. There is evidence that auditory neurons can be grouped into three basic types on the basis of how frequently they exhibit this spontaneous activity and the range of sound levels that they respond to when stimulated (Lieberman, 1978). These different types of nerve fibers are called high, medium, and low spontaneous activity fibers. High spontaneous activity fibers have, as their name implies, a high rate of firing in the absence of stimulation and their firing rate increases in response to stimuli between approximately -10 and 30 dB SPL. At approximately 30 dB SPL, these fibers reach their saturation point and their response plateaus. Mid spontaneous activity fibers have a lower rate of firing in the absence of stimulation than do the high spontaneous activity rate fibers and their firing rate increases in response to somewhat louder stimuli between approximately 5 and 35 dB SPL. Low spontaneous activity fibers have the lowest rate of firing in the absence of stimulation and their firing rate increases in response to stimuli between approximately 25 and 40 dB SPL. It is hypothesized that the combination of these fibers working together allows for a large dynamic range of intensity to be coded by the cochlea and carried along the auditory nerve.

Bone Conduction

Sound waves and mechanical vibrations acting on the surface of the human head are absorbed by the soft tissue and bones of the head and propagate through the head's structures. They also induce complex physical vibrations of the skull that can be transmitted to the brain and sensory organs of the head. Both these mechanisms together can affect the hearing system and evoke auditory responses analogous to those caused by sound waves arriving at the outer ear of the hearing system. Note that all important head resonances have frequencies below transmission range of bone conducted stimuli to protect supporting them mechanisms from potential harmful effects of body vibrations and acoustic stimulation. They include, but are not limited to, eyeball resonance, jaw resonance, neck resonance, and head and shoulder vibrations

For the human communication purposes only bone vibrations created by directly applied physical vibrations (via a mechanical vibrator) have sufficient energy to be used as a carrier of information. Sound waves arriving to the surface of the head are either captured by the outer ear and delivered through the hearing system to the organ of Corti or are mostly reflected back into the environment due to the impedance mismatch between the impedance of the skull and impedance of surrounding air. Sound waves can only be heard through bone conduction when the arriving sound has very high intensity and the person's ears are occluded by hearing protectors or head mounted audio displays (audio HMD). However in such cases, perceived sound often constitutes the harmful noise that leaked to the hearing system by bone conduction pathways rather than a communication signal.

The first modern theory of bone conduction hearing was proposed by Herzog and Krainz in 1926 (Herzog and Krainz, 1926). According to this theory the bone conduction hearing is a combination of two phenomena:

1. Relative motion of the middle ear bones caused by head vibrations
2. Compression waves in the cochlea resulting from the transmission of vibrations through the skull

Two landmark publications on bone conduction by Békésy (1932) and Barany (1938) provided further evidence for and expanded this theory. They also provided clear evidence that air conduction and bone conduction mechanisms were two different hearing mechanisms resulting in the same excitation of the basilar membrane.

Current theory of bone conduction hearing is mostly based on the comprehensive studies by Tonndorf (1966; 1968) who expanded Herzog and Krainz' work and identified seven potential mechanisms that can contribute to human hearing. The four main mechanisms proposed by Tonndorf are:

1. Inertial Mechanisms
    a. Middle ear inertial mechanism involving relative and delayed movement of the ossicular chain in reference to the surrounding temporal bone (cochlear promontory).
    b. Inner ear inertial mechanism involving transmission of temporal bone vibrations on the inner ear fluids.
2. Compression Mechanisms
    a. Outer ear compression mechanism involving radiation of bone-conducted energy from the osseous walls of the ear canal back to the ear canal.
    b. Inner ear compression mechanism involving compression and decompression of the inner ear fluids by compression vibrations of the bony cochlea.

The most dominant of the above mechanisms seems to be the inner ear inertial mechanism (Stenfelt and Goode, 2005a) although several other mechanisms contribute as well. The effectiveness of the individual mechanism depends on the frequency of the signal, place and direction of vibration application, and the status of the outer ear. The two first factors affect the modes of vibrations of the skull whereas the third depends on the type and quality of the ear occlusion. The last factor affects dramatically the effectiveness of the outer ear compression

mechanism. Ear occlusion boosts the effectiveness of bone conduction in a low frequency range by as much as 30 dB at 100 Hz and 5 dB at 2000 Hz (Stenfelt, 2007).

The middle ear inertial mechanism and the inner ear compression mechanism seem to operate in the high frequency range above 1 kHz (Stenfelt, 2006; Stenfelt and Goode, 2005b). However, the contribution of the latter mechanism has been questioned recently due to a lack of convincing experimental evidence. For example, clinical findings for otosclerosis and semicircular canal dehiscence cases do not provide support for the presence of this mechanism for frequencies below 4 kHz (Stenfelt, 2007).

In summary, the bone conduction mechanisms are still not very well understood, although hearing through bone conduction has been known and used for centuries (Henry and Letowski, 2007). The process is linear and most likely cochlear (Steinfelt, 2007). However, some new theories of bone conduction hearing point to the movement of the otolith stones in the saccule of the inner ear (Lenhardt, 2002; Todd and Cody, 2000; Welgampola et al., 2003) or to the action of the cochlear and vestibular aqueducts that exchange perilymph between cerebrospinal cavity and the cochlea (Freeman, Seichel and Sohmer, 2000; Sohmer et al., 2000).

One of the limitations of the bone conduction hearing is limited spatial perception due to the lack of pinnae cues and high velocity of acoustic waves through the bones. However, it permits sound source lateralization within the head and for spatial perception of audio signals delivered through bone conduction audio HMDs if air conduction HRTFs are used (MacDonald, Henry and Letowski, 2006).

Vestibular System Function

In addition to the cochlea, the inner ear houses the organs of balance. They include three cristae ampullares, located in three bony channels called the semicircular canals, and two maculae, located in two connected sacks, called utricle and saccule, within the bony cavity of the vestibule, between the semicircular canals and the cochlea. The cristae ampullares convey information approximately angular acceleration of the head to the central nervous system. The maculae convey information about linear acceleration and head position relative to gravity. The utricular macula is oriented horizontally and the saccular macula is oriented vertically. Tilting head to the side stimulates saccular macula and titling head forward or to the back stimulates the utricular macula. All these sensory organs contain hair cells with their stereocillia responding to the head motion analogous to the way the inner hair cells in the cochlea respond to the acoustic signal. Depending on the head position and the direction of the head movement the endolymph flow in the semicircular canals and the vestibule stimulates the hair cells of the organs of balance. For example, the cilia of the maculae are embedded in the gelatinous membrane containing relatively heavy calcium carbonate (Na) otoliths (otoconia); movements of the head cause the otoliths to bend the cilia causing depolarization/hyperpolarization of the hair cells depending on the direction of movement.

The signals from the organs of balance are transmitted through the vestibular portion of the vestibulocochlear nerve to four vestibular nuclei (superior, lateral, medial, and inferior nuclei) within the brainstem and further to the brain (cerebellum). The fibers from the vestibular nuclei also crossover to the contralateral nuclei from which they project, among others, to oculomotor nuclei that drive eye muscle activity resulting in vestibule-ocular reflex that helps maintain fixation of the eyes on the object moving in relation to the head position. Thus, human balance involves a complex coordination between the vestibular system, visual system, proprioceptors (sensors in muscles and joints), and structures within the cerebellum, brainstem, and the whole cortex.

Central Auditory Nervous System Processing

Cochlear nucleus

Auditory nerve fibers arrive at the brainstem by forming synapses with large groups of neurons in the cochlear nuclei located in the border between the pons and medulla. The fibers from each ear terminate on the nucleus located on the same (ipsilateral) side of the brainstem from where most of the fibers cross to the opposite

(contralateral) side of the brainstem and either connects to contralateral superior olivary complex or ascends directly to contralateral inferior colliculus in the midbrain. Type I nerve fibers with large myelinated neurons are responsible for transporting the coded auditory signal from the peripheral to the central nervous system. The function of the smaller and less numerous type II fibers is still largely unknown.

The neural cells in the cochlear nuclei have several complex firing patterns and wider dynamic ranges than the neurons in the auditory nerve. In response to simple tonal stimuli, several response patterns have been recorded in the various cells of the cochlear nuclei. These response patterns include a "primary" pattern that is similar to that of the auditory nerve ("primary-like" neurons); a "chopper" pattern that consists of repeated bursts of firing followed by short pauses ("chopper" neurons) (however, this periodicity does not match the periodicity of the stimulus; an "on" pattern, in which the cell fires only when a stimulus begins ["on" neurons]; and a "pauser" pattern in which the cell fires only at the onset of the stimulus, pauses, and then continues until the stimulus is turned off ("pauser" neurons) (Pfeiffer, 1966). Examples of peristimulus (PST) histograms, i.e., the histograms of the times at which neurons fire, as a function of latency following tonal stimuli, are shown in Figure 9-12.

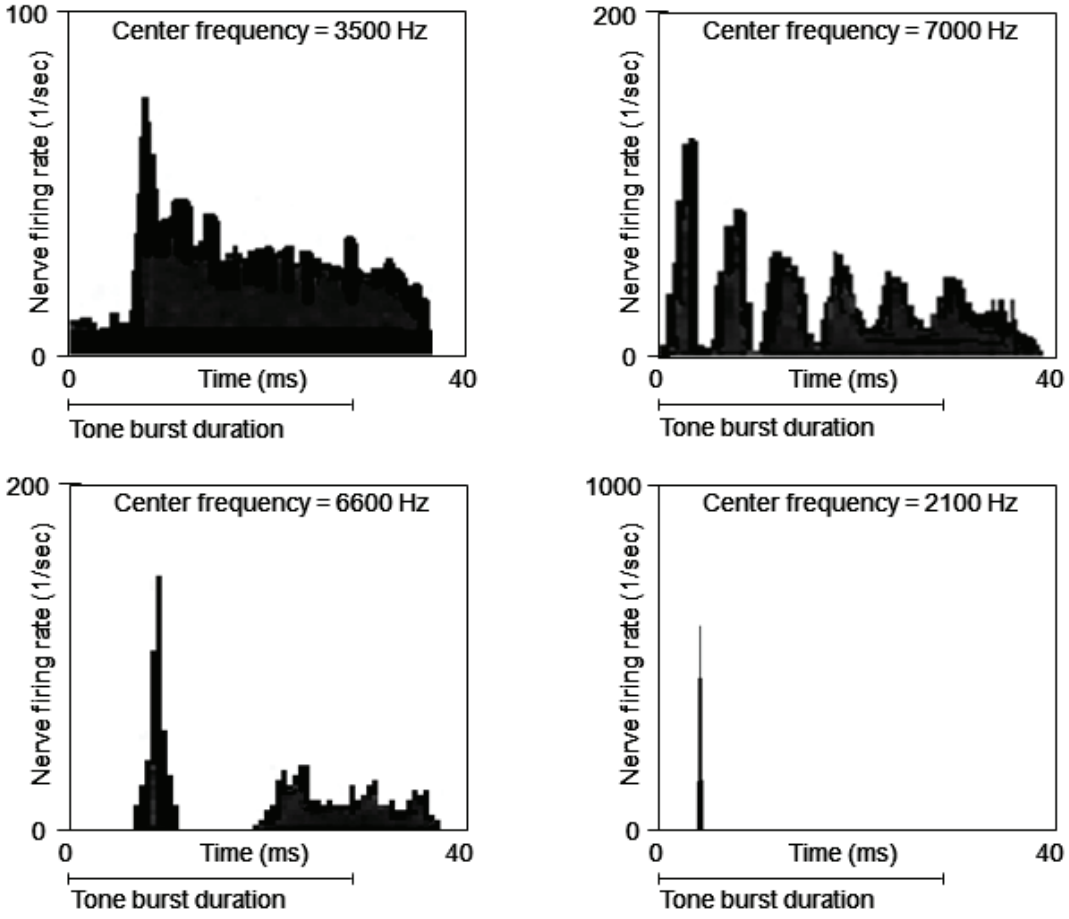

Figure 9-12. PST histograms illustrating different types of neuron firing patterns observed in the cochlear nucleus including: (a) primary like, (b) chopper, (c) pauser and (d) on (from Pfeiffer, 1966).

These firing patterns shown in Figure 9-12 are the most commonly reported firing patterns. However, there also are other firing patterns observed in response to simple stimuli. Further, there are reports of many different sub-

categories of firing pattern under each of these main categories, and the response of the cochlear nucleus to complex stimuli varies from the response to simple stimuli.

The type of response recorded from the neurons in the cochlear nucleus depends on a number of physical features associated with the cells (e.g. characteristics of the membrane, type of cell), the connection between the auditory nerve and the cochlear nucleus cells (e.g. many or few axon endings contacting many or few dendrites), and the presence of inhibitory input from other cells (Cant, 1982; Ostapoff, Feng and Morest, 1994; Rhode and Smith, 1986). For example, in the anterior ventral cochlear nucleus (AVCN), the most common cell types are the global and spherical bushy cell. These cells receive very few axonal connections from auditory nerve fibers from a localized frequency area of the cochlea and are the most likely contributors to the primary response pattern seen in the cochlear nucleus. Their frequency specificity may also be enhanced by their function as coincidence detectors, which reduce the random noise level from spontaneous activity of the auditory nerve (Joris et al., 1994; Joris, Smith and Yin, 1994; Louage, van der Heijden and Joris, 2005). Other cells may specialize in transmitting intensity of sound (multipolar cells) or temporal order of sound events (octopus cell).

In the posterior ventral cochlear nucleus (PVCN), the octopus cell is a common cell type, so-called because these cells resemble an octopus with long tentacle-like dendrites. These dendrites receive many more connections, across a broader frequency range of the cochlea, compared with the AVCN bushy cells, and they are thus more broadly tuned. These cells have been reported to respond well to amplitude modulated tones (Oertel et al., 2000) and clicks, but have a reduced activity in response to steady state noise (Levy and Kipke, 1998). In some species, the dorsal cochlear nucleus (DCN) has been recorded to respond to spectral differences that may indicate they provide some coding in response to monaural localization cues in a vertical plane (Spirou et al., 1999).

Superior olivary complex

The superior olivary complex (SOC) receives inputs from both cochlear nuclei (contralateral and ipsilateral) and has an important role in sound localization. The two largest nuclei in the SOC are the lateral SOC (LSOC) and the medial SOC (MSOC). The MSOC and LSOC are binaural integration sites where information from each ear comes together and the input from the right and left ears are compared against each other. Therefore, these two centers play a primary role in the creation of coding cues for the sound localization in the horizontal plane. The differences between the ears are used to create codes that represent ITDs, primarily in the MSOC (Masterton, Jane and Diamond, 1967; Masterton et., 1975) and IIDs, primarily in the LSOC. The MSOC, similar to the cochlear nucleus, appears to use coincidence detection as a mechanism for coding the different arrival times between cells. The actions performed by the MSOC and LSOC neurons are shown schematically in Figure 9-13.

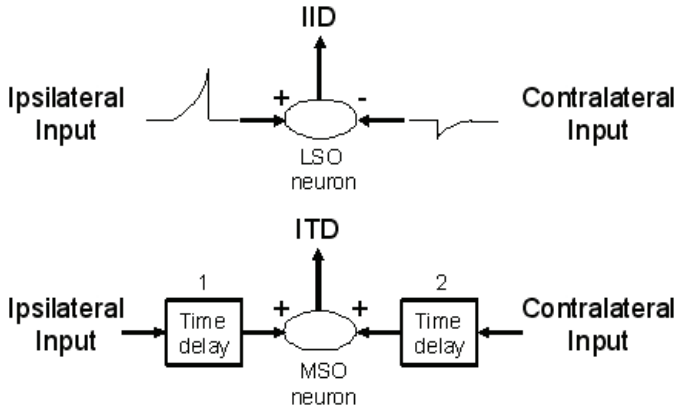

Figure 9-13. Coding of IID and ITD cues in the superior olivary complex.

All of the ascending fibers from the cochlear nucleus and superior olivary complex travel in a large fiber tract called the lateral lemniscus and synapse in the inferior colliculus. The inferior colliculus appears to be another site that processes intensity and timing differences between ears important for spatial sound perception (Masterton, Jane and Diamond, 1967; Møller, 2002). It also processes sound onset and duration and is generally believed to be the first site at which complex sounds are encoded.

Auditory fibers projecting from the inferior colliculus ascend to the medial geniculate body of the thalamus and from here to the neocortical structures of the primary and secondary auditory cortex located in the transverse temporal gyrus (Heschl's gyrus) of the brain. The medial geniculate body acts primarily as a relay station for all ascending auditory signals passing them bilaterally onto primary auditory cortex. However, because of the decussating of a majority of fibers prior to the lateral lemniscus, the primary signal from the right ear arrives in the left hemisphere of the brain and the primary signal from the left ear arrives in the right hemisphere. The primary auditory cortex has tonotopic organization with characteristic frequencies (CFs) of the neurons increasing from caudal to rostral locations. Somewhere in the center of the area there are also patches of more specialized neurons (Ehret, 1997).

Auditory signals are subsequently sent from Heschl's gyrus to other parts of the brain for feature extraction and further analysis by auditory, multisensory, and cognitive areas. One of such prominent other areas is Wernicke's area, located at and adjacent to the posterior end of the superior temporal gyrus. Wernicke's area is responsible for decoding a speech signal into a semantically recognizable message. The *planum temporale* of Wernicke's area is also involved in music and timbral changes perception. In most individuals, Wernicke's area is more developed on the left side, thus a speech signal directed to the right ear arrives directly to Wernicke's area and the signal from the left ear must travel from the left temporal lobe to the right temporal lobe via the large corpus callosum fiber track. Thus, most individuals demonstrate a right ear advantage, in which speech perception is superior when the signal is directed to the right ear. This effect is seen most clearly in children and diminishes with age. Another important area connected with primary auditory cortex is Broca's area that control speech production.

Descending (efferent) nerve fibers run from the auditory cortex back to the cochlea forming ipsilateral and contralateral synaptic junctions in reverse order to the ascending fibers (Figure 8-20). The corticothalamic projections descend from the auditory cortex to the thalamus and medial geniculate body whereas another group of projections, called the cortiocollicular projections, descends directly to the inferior and superior colliculi. Most of the descending pathways from the colliculi terminate at the lateral and medial periolivary nuclei of the SOC but some others project directly to cochlear nuclei. The descending olivo-cochlear projections connect the periolivary nuclei to the outer hair cells and the radial fibers in the cochlea modifying their responses (Oliver, 1997).

Although the complete understanding of the efferent system is still elusive, its main function seems to be to provide a gain control mechanism for the auditory system. The descending fibers conduct neural impulses that control the neuromuscular feedback system of the middle ear, the amplification function of the outer hair cells, and the sensitivity of the inner hair cells. Stimulation of the outer hair cells by signals descending from the medial periolivary nuclei through MOCB (see Chapter 8, *Basic Anatomy of the Hearing System*) decreases motility of the outer hair cells affecting sensitivity of the cochlear amplifier and improves speech recognition in noise (Giraud et al., 1997; Kumar and Vanaja, 2004). It has also been reported that efferent system affects detection of tones of unexpected frequencies (Scharf, Magnan and Chays, 1997) and controls an attentive state of a person (Froehlich et al., 1990; Yost, 2000).

It has to be stressed that the properties of the individual neurons and their synaptic connections do not represent a fixed structure in the central nervous system. They are affected by immediate surroundings, experience, selective attention, learning, and emotional states of a person and they are continuously modified throughout the lifetime.

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