2. FUNDAMENTALS OF HEARING

2.1 INTRODUCTION:
The ear is a marvel of either creation, or evolution, depending on your point of view. What is not disputed however, is that it is an amazing device, whose performance has yet to be duplicated by man-made contrivances. The ear is an extremely sophisticated amplifier and frequency analyzer which transduces sound intensity over a $10^{13}$ range of amplitudes (from barely perceptible to the threshold of pain). It can reject the sounds of a loud cocktail party and single out a single voice. It can adapt to loud noises and protect itself from damage. It can hear the subtle tone differences between a 300 year old Stradivari violin and a modern copy. Even more amazing is that it does all this in a volume of approximately one cubic inch.

Objectives of this Lesson:
Upon completion of this lesson, you should have a basic understanding of the physical and physiological processes involved in hearing. The basic operation of the ear is described, as well as common disorders which result in hearing loss, including those resulting from noise exposure.

References:
The ear is the subject of on-going research and there are still many things we do not know about its functions (or at least that’s what the scientists keep telling the NIH to justify continuing research grants). If you want to know more than what is covered in this brief section, the following references are recommended:
An Introduction to the Physiology of Hearing, James O. Pickles, Academic Press, 1982
Hearing, Stanley Gelfand, Marcel Dekker, 1981
Experiments in Hearing, G. von Bekesy, Wiley, 1960

Some Interesting Web Sites:
http://www.boystown.org/ cel/ cochmech.html - Boystown National Research Hospital educational pages on cochlear mechanics
2.2 ANATOMY OF THE EAR

We typically divide the ear into three functional areas: outer, middle and inner.

**Outer ear**

The outer ear “gathers” sound and transmits it to the timpanic membrane. The outermost visible portion is called the Pinna. It aids somewhat in directionality detection (for high frequency). Some echolocating bats have very developed pinnae with high directional sensitivity. Dogs and cats can turn their pinna to locate sounds. The deep central portion of the pinna is called the concha (cave).

The auditory canal - ~7 mm dia, 27 mm long, acts like an organ pipe to amplify sounds reaching ear drum. It provides approximately a 10-15 dB gain from 2000-6000 Hz. The resonant frequency of the auditory canal is ~4000 Hz, while the resonance of the concha is around 5000 Hz.
The timpanic membrane (tympanus or eardrum) - separates outer and inner ears, and vibrates in response to incident sounds. It is a cone shaped, nearly transparent membrane, 55 - 90 mm² in area.

**Middle ear**

![Fig. 2. Middle Ear Ossicles (ref. On the Sensations of Tone, Helmholtz)](image)

The middle ear transfers sound energy (in the form of mechanical vibrations) from the tympanic membrane, on to the cochlea. Because of its transformer action, the middle ear matches the low impedance of air to the high impedance of the cochlear fluid, thereby causing a much more efficient transfer of energy into the inner ear. The middle ear cavity is about 2 cc in volume. It is connected by a 35-38mm long tube (eustachian tube) to the nose cavity. The eustachian tube provides for pressure equalization and prevents the eardrum from bursting due to static pressure differentials across it.

**Bones of the middle ear:** The moving parts of the middle ear are a mechanical linkage consisting of three tiny bones - the ossicular chain,
- hammer (malleus) - connects to eardrum
- anvil (incus)
- stirrup (stapes) - attaches to oval window

**Muscles of the middle ear:** Two small muscles are attached to the hammer and stirrup. They contract in response to: loud noises (>75 dB above threshold), vocalization, or general body movement. When contracted, they stiffen the chain and attenuate low frequency transmission (<2 kHz). This reflex action can occur in as little as 10 ms for high intensity sounds. This is too slow to greatly attenuate impulsive type sounds. The action of these muscles are thought to serve a number of useful functions, such as:
- a) protect the inner ear from damage
- b) provide an automatic gain control for loud, low frequency sounds
- c) because they contract due to vocalization, or body movement, they reduce the perception of self-generated sounds (such as your own voice, or noises due to movements)
- d) since low frequency sound can mask sounds of higher frequency (more about this later), selective attenuation of low frequencies can improve the perception of complex stimuli, such as speech.

**The middle ear as a transformer, or impedance matching device:** If the oval window were directly exposed to airborne sound (no outer or middle ear), very little sound
energy would be transmitted into the inner ear. When sound encounters a boundary between two different materials, some will enter the second material, some will be reflected. The amount that is transmitted is determined by the acoustic impedances of the two media (see Unit 5, Course 1 for more on impedance):

\[
\frac{E_2}{E_1} = 4 \frac{Z_1 Z_2}{(Z_1 + Z_2)^2}
\]

where \(E_2\) and \(Z_2\) are the energy transmitted into the second medium, and the impedance of the second medium respectively.

If both materials are the same, 100% of the energy is transmitted. In the case of the ear, the impedance of air is \(\sim 400 \text{ N sec/ m}^3\), while the impedance measured at the oval window of the cochlea is \(\sim 2 \times 10^5 \text{ N sec/ m}^3\) (for cats, at 1KHz by Khanna and Tonndorf, 1971, JASA vol 50, pp 1475-1483), a substantial mismatch. For this case, only 0.8% of the sound energy would enter the inner ear if there were no outer or middle ear.

The purpose for the middle ear then is to try to mend this impedance mismatch. The ossicular bones provide a small mechanical advantage of \(\sim 1.15:1\) and convert the displacement of the timpanus into smaller displacements (and higher forces) at the oval window. The ratio of the timpanus area (55 mm\(^2\)) to that of the oval window (3.2 mm\(^2\)) is about 17. This increases the pressure at the oval window. Together, these two effects result in an impedance matching (low force/ large motions @ eardrum are transferred into high force/ small motions @ oval window). The overall mechanical advantage from eardrum to oval window (including effect of the relative sizes of each) is \(\sim 1.15 \times 17 = 20\). (a 26 dB increase). In terms of impedance, this translates into an impedance change of 20\(^2\) or 400. This is in the range of what is required to properly match the impedance of the cochlea to that of the outer ear canal.

**Frequency response** The pressure gain of the ossicular chain shows a bandpass characteristic as shown in the accompanying figure. This is actually data for a cat, which is similar to a human. The pressure was measured just behind the oval window inside the cochlea for constant sound pressures at the tympanic membrane (Nedzelitsky, 1980 JASA vol. 68, pp 1676-1689). The irregularity around 4 KHz is due to the resonance of the middle ear cavity.
Inner ear

The inner ear is where “hearing” actually takes place. Here, the mechanical vibrations transmitted through the bones of the middle ear to the oval window are converted into pressure fluctuations in the cochlear fluid, which are in turn converted by the hair cells of the basilar membrane into nerve impulses sent to the brain. An alternative sound transmission path is directly through the skull bones to the inner ear. This is the mechanism by which we hear our own voices, and is the limiting factor for hearing protector effectiveness.

Much of what we know of the cochlea’s mechanics comes from the work of G. von Bekesy, who examined the movement of the cochlear partition in human and animal cadavers. He would rapidly dissect the ear soon after death, insert rubber windows for the round and oval windows and attach a mechanical vibrator to the oval window. Microscopic and stroboscopic techniques were then used to measure the displacement of Reissner’s membrane (which he assumed vibrated similarly to the Basilar membrane).
Structure: Structurally, the cochlea consists of a coiled, fluid filled organ divided lengthwise by the basilar membrane. When unrolled it is approximately 34 mm long from base to helicotrema. The basilar membrane tapers from base to apex and is wider at the helicotrema than at the basal end. The oval window is directly connected to the stapes. The lower chamber ends in the round window which acts as a pressure release mechanism (it probably prevents echoes back down the cochlea).

Action of the basilar membrane: Motions of the oval window induce a disturbance in the cochlear fluid and a traveling wave in the basilar membrane. The distance this disturbance travels depends on the frequency of the sound. As shown in Figure 7, low frequency sounds will propagate along the entire upper chamber, while high frequency (2kHz for instance) will result in motion in the first quarter of the cochlea nearest to the oval window.
All motions excite basal end, while only low frequencies get to the apex. This can explain why with exposure over time to excessive noise, high frequency sensitivity is lost more rapidly than the low. The base sees greater usage and the hair cells experience more cycles of oscillation and are more prone to wear out.

It is believed that the brain extracts the frequency information in the sound from the position of maximum excitation on the basilar membrane.

(see http://www.boystown.org/ cel/ waves.htm or http://www.neurophys.wisc.edu/ / animations/ for really cool animations of the traveling waves in the basilar membrane.)

**The Organ of Corti:** The organ of Corti contains a number of hair cells (outer and inner) as shown in the cross section below. There are 20,000-30,000 hair cells in the average human ear. Each of these hair cells has about 40 stereocilia (cilia or tiny hairs) attached to it. An electron micrograph of the top surface of the organ of Corti with the tectorial membrane removed, showing the stereocilia, is shown below.

Motions of the basilar membrane cause shear between the stereocilia and the tectorial membrane. Each hair cell is connected to a number of nerve fibers. The shearing action of the cilia causes the nerve cells to fire, sending signals to the brain.

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Figure 7. Traveling waves on the basilar membrane (ref. Yost fig 7.13)

Figure 8. Magnified cross section of the organ of Corti detailing the basilar membrane
Differences between inner and outer hair cells: The inner hair cells do not directly contact the tectorial membrane but are moved by viscous fluid forces. The output of the inner hair cells is proportional to the velocity of the basilar membrane. In contrast, the outer hair cells are connected to the tectorial membrane. They are either bent, or stretched by the relative motion (we don’t know which for sure), producing an output which is proportional to displacement. There is some thought that the outer hair cells actually may work not as sensors, but as small actuators, causing motions of the tectorial membrane in response to nerve impulses. This could allow the ear to selectively change the gain (turn the volume up or down) of selected areas of the basilar membrane.
Nerve cells: Two types of nerve cells are found in the ear - afferent and efferent. Afferent fibers are sensory nerves which carry information from the ear to the brain. Efferent fibers typically bring information from the brain to the ear. The inner hair cells are connected mainly by afferent fibers, so they appear to be primarily responsible for sending information to the brain. The outer hair cells have very few afferent cells and many efferent cells. The outer hair cells appear to function in some way to control the biomechanics of the ear. It is believed that the brain sends signals back to the outer hair cells in a form of active feedback loop. This is thought to help in filtering out unimportant sounds, and control directional interpretation. This can explain why you can concentrate (“focus”) on one conversation in a crowded, noisy room.

2.3 DISORDERS OF THE EAR

The basic types of hearing loss are:

Conductive Deafness - hearing loss due to an abnormality which prevents sound from reaching the inner ear

Nerve Deafness (sensorineural hearing loss)- hearing loss due to damage in the cochlea or auditory nerves

Conductive deafness -
Conductive deafness is caused by damage outside of the cochlea, such as in the outer or middle ear. People with conductive deafness tend to talk very quietly. They generally can hear their own voice quite well (via bone conduction) but cannot hear other voices as well. They instinctively try to speak so that they perceive their own voice at the same level as other voices or sounds. Conductive deafness can be helped by a hearing aid.

Causes of conductive deafness include:

outer ear
- blockage of ear canal by wax buildup, infection, swimmer's ear, dirt
• damaged or punctured eardrum, small damage will heal, whole eardrum can be repaired by grafting tissue middle ear
• infection (otitis media)
• otosclerosis - stirrup becomes fixed to oval window, can be replaced by a prosthesis
• aerotitis media - blockage of eustachian tube

Nerve deafness
Nerve deafness is caused by some defect in the cochlea or auditory nerve. People with nerve deafness tend to talk very loudly, in an attempt to hear themselves better. Hearing aids are of limited effectiveness in this case. Experiments are continuing in cochlear prosthesis, where the auditory nerve is directly stimulated by electrical means. Nerve deafness can occur suddenly, or gradually over a long period of time.

Sudden causes:
• disease - meningitis, infection, multiple sclerosis, mumps
• head injury
• acoustic trauma (i.e. explosions)
• drugs that effect the central nervous system

Gradual causes:
• neuritis - disease of nerves
• tumor - effects the nerve
• noise induced hearing loss
• presbycusis - normal effect of aging (see figure 11 below)

Figure 11  Threshold shift due to presbycusis:  M=men,  W=women, Speech sounds: male, normal voice at 1m.  Data due to Beranek  (from Bies and Hansen)
Noise induced hearing loss
Exposure to high noise levels can cause hearing loss as evidenced by an upward shift in the threshold of hearing – called noise induced hearing loss. If full hearing returns in a short period of time, this is called temporary threshold shift (TTS). This is also known as acoustic fatigue. Continued exposure to steady noise, or exposure to impulsive sounds can cause permanent loss or permanent threshold shift (PTS).

Damage to the hair cells is usually responsible for noise induced hearing loss. Prolonged exposure to high levels causes the hairs to exceed their elastic limit, analogous to mechanical fatigue. If hair cells are damaged, they will not regenerate or recover. Figure 13 shows scanning electron micrographs of normal and damaged hair cells in a chinchilla. The damage can be localized to a small area of the basilar membrane if the noise exposure occurred continuously at one frequency (common in factory workers who have worked next to the same machine for years).
**FIGURE 14.2** 14.2a Scanning electron micrograph of normal organ of Corti showing normal ultrastructure. 14.2b Scanning electron micrograph of organ of Corti that has been damaged by acoustic overstimulation. The effects of sound damage on the outer hair cells are clearly shown, as the cells are now missing (except for some remnants of OHC2). These photographs courtesy of Dr. Ivan Hunter-Duvar, Hospital for Sick Children, Toronto.
Aural reflex:

Luckily, the ear has several protective mechanisms called the aural reflex, to guard against high levels of steady noise. Two protective things happen:

- The bones of the middle ear behave non-linearly with amplitude. As previously mentioned, the muscles connecting the ossicular chain tense up when the sound is about 80 dB above threshold, and decrease the transmission into the inner ear.
- The ear drum increases curvature (and thereby its stiffness) when exposed to high levels. This takes ~10 ms to happen.

These effects are of little help for loud impulsive noises, because by the time the reflex happens, the damage has already occurred. The aural reflex can be activated by noise impulses or clicks. The reflex can last as long as 2-3 seconds before complete relaxation. The military uses this effect to try to protect gunners from the intense noise from cannon fire (such as in a tank or naval gun turret). A series of progressively louder clicks (but not as loud as the gun itself!) is presented just before the big boom, to give the ear a chance to tighten up and “get ready for the big one”. Continued activation of this aural reflex can cause acoustic fatigue and TTS.
**Tinnitus:**
Tinnitus, sometimes called “ringing in the ears”, is an indication that permanent hearing loss has been incurred. This is a disorder in which a person suffers from a ringing or other sound in the ear for which there is no obvious physical source. It is poorly controlled by drugs or surgery and the only real treatment is to mask it by external noise. There is considerable debate over the exact physiological causes of tinnitus, but it normally accompanies nerve deafness.

**4. HEARING EVALUATION**

Hearing acuity is measured with an audiometer. The most common type consists of an audio oscillator which generates single pure tones, an attenuator calibrated in decibels, and an earphone.

![Figure 14. The basic audiometer](image)

The usual test tones are spaced in octaves, from 125 to 8000 Hz. The reference level for each frequency is the normal threshold of hearing. In operation, a subject is presented with a tone of decreasing level in 5 dB steps. The subject is asked to identify when that tone is no longer audible and that level is recorded as the measured threshold of hearing. The difference in level between the reference level and the person’s measured threshold level is the hearing loss in decibels. A hearing loss level of –10 to +25 dB is considered within normal limits for adults. The reference sound levels which are used as the zero reference for a representative audiometer (Western Electric model 705-A) are shown in Table 1.

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