Chapter 3

Human Hearing Mechanism

3.1 Introduction

Audition is the scientific name for the perception of sound. Sound is a form of energy that moves through air, water and other matter, in waves of pressure. Although the ear is vertebrate sense organ that recognises sound, it is the brain and central nervous system that *hears* sound waves perceived by the brain through the firing of nerve cells in the auditory portion of the central nervous system. The ear changes sound pressure waves from the outside world into a signal of nerve impulses sent to the brain.

Human hearing arises from airborne waves altering 20 to 20000 times a second about the mean atmospheric pressure. When a human being perceives sound, he is experiencing the effect of complicated sensor and amplification system. Sound wave causes vibrations of the tympanic membrane. These vibrations mechanically transfer to ossicle located in the middle ear which in turn acts upon the oval window located on the cochlea. The movement of the oval window translate into pressure waves with in the fluid filled cochlea which causes the movement of the basilar membrane. Sensory hair cells, which are located on the basilar membrane, bend in response to the movement. The cellular bending causes the generation of action potentials which are transmitted to brain. The action potentials travel from the left and right ears through the eighth cranial nerve to both sides of the brain stem and upto the portion of the cerebral cortex dedicated to sound, where sound is perceived.
3.2 Anatomy of Human Ear

The ear can be divided anatomically and clinically into three parts: the external ear, the middle ear, and the inner ear. The external ear and middle ear are concerned primarily with the transmission of sound. The internal ear functions as the organ of hearing and as part of the balance system of the body [89]. Figure 3.1 shows the anatomy of human ear.

![Anatomy of human ear](image)

Figure 3.1: Anatomy of human ear

3.2.1 The External Ear

The outer ear consists of visible portion, the auricle, and a tube leading from the auricle medially. The tube is the external auditory meatus (auditory canal). The auricle (pinna) consists of several pieces of cartilage that are held together by ligaments and covered with skin. The meatus is about 2.5 to 3 cm in length and has a diameter of about 0.75 cm. The function of pinna is to direct or funnel acoustic
waves from a wide area into the auditory canal. The auditory canal then conducts those sound waves to the middle ear. Sounds are filtered due to the geometry of the pinna and sound shadowing effects of the head.

3.2.2 The Middle Ear

The middle ear consists of the tympanic membrane, ossicles, tensor tympanic muscle and stapdexts muscle. The middle ear cavity having volume of about $2cm^3$. It is encased within the temporal bone of the human skull. The main purpose of the middle ear is to transfer the slight movement of the tympanic membrane, due to acoustic pressure on the outer side of the membrane, into movements of the ossicles which in turn act on the oval window, entry point into the inner ear.

There are three ossicles in the middle ear attached to the tympanic membrane is the malleus which connects to the incus, which in turn forms a flexible connection with the stapes. The stapes is the smallest bone in the human body. The flat portion of the stapes, footplate, moves in and out of the oval window much like a piston creating the pressure waves in the fluid filled cochlea. The middle ear cavity contains air provided by a tube connecting the nasophasynx and the cavity. This tube called Eustachian tube in human it is often closed, except during swallowing, and provides a means by which pressure is equalized across the tympanic membrane. The arrangement of the tympanic membrane and ossicles works effectively to couple the sound from the opening of the ear canal to the cochlea.

The middle ear is an impedance matching network that amplifies the speech frequencies and increases the efficiency of energy transmission so that the sound energy can get from the air filled external world to the fluid filled inner ear. Most of the impedance conversion results from the difference in area between the eardrum (receiving sound from air) and the oval window (transmitting sound into liquid, see
Figure 3.1). The ear drum has an area of about 60 mm$^2$, while the oval window has an area roughly 4 mm$^2$. Since pressure is equal to force divided by area, this difference in area increases the sound wave pressure by about 15 times.

### 3.2.3 The Inner Ear

The inner ear consists of the cochlea, which is a part of the auditory system, and the labyrinth, which is part of the vestibular system. The inner ear is encased in the hardest bone of the body. The cochlea itself is a spiral shaped organ with a length of 3 cm and an inner diameter of about 2 mm. Figure 3.2 shows cross section of the human cochlea.

![Cross section of the human cochlea](image)

**Figure 3.2: Cross section of the human cochlea**

The cochlea consists of two long fluid filled compartments, separated by a nar-
row, flexible membrane, called the basilar membrane, situated on this membrane is the organ of corti, which contains the sensory cells (hair cells) for hearing. There are two types of hair cells in the human cochlea, i.e. inner hair cells (IHC) and outer hair cells (OHC) [15]. The IHCs are primarily connected to the auditory nerve by afferent fibers, which deliver neural signals to the brain. The OHCs, on the other hand, are connected to efferent nerve fibers, which receive neural signals from the central auditory system. Figure 3.3 shows the cross section of the organ of Corti. The human cochlea contains approximately 4000 IHCs and 12000 OHCs. They are typically distributed as one row of IHCs and three rows of OHCs along the basilar membrane.

![Cross section of the organ of corti](image)

*Figure 3.3: Cross section of the organ of corti*

The tectorial membrane lies on the top of the organ of corti and is attached to the tips from the stereocilia of OHCs, but is not directly in contact with the stereocilia.
of IHCs as illustrated in Figure 3.3 [26]. Sound waves, which pass from the ear drum to the inner ear via the middle ear, causes the basilar membrane to vibrate, which results in angular displacement of the hair bundles. The shearing of the stereocilla opens ion channels in the cell membrane at their tips, resulting in a flow of ions and thus a change of voltage drop across the hair cell membrane in response to the stimulus is receptor potential [43, 83]. This voltage change leads to synaptic transmitter release from the basal end of the cell, triggering the neural impulse to the brain that forms the basis of our ability to perceive sound. The movement to voltage transduction by the inner ear is the fundamental component of the hearing process.

The basilar membrane is stiffest near the oval window, and becomes more flexible toward the opposite end, allowing it to act as a frequency spectrum analyzer [57]. When exposed to high frequency signal, the basilar membrane resonates where it is stiff, resulting in the excitation of nerve cells close to oval window. Likewise, low frequency sounds excite nerve cells at the far end of basilar membrane [122]. Figure 3.4 shows the tonotopic map of the cochlea. The numbers indicate the resonance frequency at different locations of the cochlea. These make specific fibers in the cochlear nerve respond to specific frequencies. This organization is called place principle, and is preserved throughout the pathway into the brain.

Another information encoding scheme is also used in human hearing, called the volley principle. Nerve cells transmit information by generating brief electrical pulses called action potentials. A nerve cell on the basilar membrane can encode audio information by producing an action potential in response to each cycle of the vibration. For example, a 200 HZ sound wave can be represented by neurons can produce 200 action potentials per second. However, this only works at frequencies below about 500 Hz, the maximum rate that neurons can produce action potentials.
The human ear overcomes this problem by allowing several nerve cells to take turns performing this task, for example, a 3000 Hz tone might be represented by ten nerve cells alternately firing at 300 times/second. This extends the range of volley principle to about 4 KHz, above which the place principle is exclusively used.

3.3 Hearing Problems

Anyone can have an ear problem that causes hearing loss. Some people are born with a hearing impairment. For others, hearing loss may results from an ear infection, genetic deficits, biochemical insult, exposure to intense sound, head injury, tumour growth or aging [4, 87]. Generally, the hearing loses fall into three major categories

1. Conductive hearing loss

2. Sensorineural hearing loss

3. Mixed hearing loss
3.3.1 Conductive Hearing Loss

Conductive hearing loss is due to attenuation in the outer ear or middle ear. Conductive hearing loss occurs because of some mechanical problem in the external or middle ear, build up of wax in the ear canal, a punctured ear drum, middle ear infection, malfunction of ossicles, or fluid in the middle ear. Conductive hearing loss can be alleviated by amplification provided by hearing aids and may be subject to surgical correction or with medication.

3.3.2 Sensorineural Hearing Loss

Sensorineural hearing loss (SHL) can be caused by anything that damages the delicate parts of the inner ear, the auditory nerve or the brain stem. Sensorineural hearing loss due to absence of internal hair cells results from genetic defects, biochemical insult, severe infection such as mumps or german measles, a head injury, an abnormal growth in the ear exposure to loud sound or aging. A hearing aid can often help a person who has SHL and some cases partial hearing functions can be restored with the cochlea implants. However, little can be done today to reverse this type of hearing loss.

3.3.3 Mixed Hearing Loss

Problems can occur simultaneously in both the conductive and sensorineural mechanism. In this hearing loss sound conduction will be attenuated by both middle ear and inner ear problems. This type of impairment is called mixed hearing loss.

Deafness could be either from birth or acquired. The Table 3.1 shows various causes based on these parameters.
Causes of Deafness

<table>
<thead>
<tr>
<th>Congenital</th>
<th>Acquired</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Maternal Rubella</td>
<td>1. Ototoxicity</td>
</tr>
<tr>
<td>2. Hereditary/Family Diseases</td>
<td>2. Severe birth asphyxia</td>
</tr>
<tr>
<td>3. Ototoxicity-drugs used during pregnancy</td>
<td>3. Cerebral palsy</td>
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<td></td>
<td>4. Febrile illnesses</td>
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<td>5. Age</td>
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<td>6. Infections</td>
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<td>7. Occupational</td>
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<td>8. Accidental</td>
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<tr>
<td></td>
<td>9. Tumour growth etc.</td>
</tr>
</tbody>
</table>

Table 3.1: Causes of deafness

3.3.4 Abnormalities of Temporal Bone

The temporal bone forms a part of the base of skull. They are among the hardest of all bones, enclosing the tiny organ of hearing and balance systems. Hence, the abnormality of temporal bone is a major cause for many hearing problems. The most important temporal bone abnormalities which can be detected from medical images are *Otosclerosis* and *Cholesteatoma* [40]. *Otosclerosis* is a hereditary disease resulting in an abnormal sponge like bone growth in the middle ear. This growth prevents the ear from vibrating response to sound waves. This lack of vibration lead to hearing loss that continues to get worse with time. *Otosclerosis* usually affects both ears. The hearing loss is primarily of conductive type but not uncommonly, ssesorineural hearing impairment also may be present. *Cholesteatoma* is a destructive and expanding sac in the middle ear and/or mastoid process. A *cholesteatoma* cyst consist of desquamating (peeling) layers of scaly or keratinized (horny) layers of epithelium, which may contain cholesterol crystals. If untreated, *cholesteatoma* can eat into the three small bones located in the middle ear (the malleus, incus and stapes), and can result in nerve deterioration, deafness, imbalance and vertigo.
Hearing has a key function for social life. The auditory sensory system allows us to communicate with each other and to realize around us. Therefore hearing impairment is a serious handicap. Several psychoacoustical tests are used to qualify and quantify a hearing loss in general pure tone audiogram, speech audiometry, electrocochleography, tympanometry, otoacoustic emission (OAE), auditory brain stem response (ABR-BERA) etc. OAE is of primary focus in this work.

3.4 Otoacoustic Emissions

Otoacoustic emissions are very low level sounds that are emitted by the ear either spontaneously or in response to sound simulation. Dr. David Kemp demonstrated that a healthy cochlea generated very low levels of acoustic emissions during the normal hearing process. Otoacoustic emissions are now thought to reflect the activity of active biological mechanisms within the cochlea responsible for the exquisite sensitivity, sharp frequency selectivity and wide dynamic range of normal auditory system. They are thought to be produced by outer hair cells of the cochlea as a by product of a frequency and threshold sensitivity increasing mechanism. One of the most attractive features of OAEs is their tight relation to the cochlear status: OAEs are universally present to various degrees in all healthy cochleas, where as they are greatly reduced in ears with mild hearing losses [32]. The OAE response acts like an on/off function around 30 dB of hearing loss. In general only normal and near to normal ears will produce OAE. The OAE signal as recorded in the ear canal is, however, not only a product of the generation mechanisms in the cochlea but is also affected by conductive properties of the middle ear, volume and acoustics of the outer ear canal. This aspect together with the extreme facility to perform the test and the high reproducibility had made the OAEs an increasingly widespread application in diagnosis of hearing impairments. OAEs have considerable potential
in clinical applications such as differential diagnosis of sensorineural hearing loss, and hearing screening.

3.4.1 Physiologic basis of Otoacoustic Emissions

OAEs are believed to be the byproducts of preneural mechanisms of cochlear amplifier and, in particular, to be linked to the normal functioning of OHCs. The cochlea consists of three fluid filled compartments which are separate by narrow flexible membrane, i.e. the basilar membrane. The organ of corti rests upon the basilar membrane. Direct measurements of basilar membrane motion support the theory that an active, vulnerable, biomechanical process within the organ corti uses metabolic energy to enhance the sensitivity to an input and frequency selectivity of the basilar membrane vibration. According to this theory, active mechanisms take place to sharpen the frequency selectivity and to amplify the vibromechnical response at low sound levels.

The organ of corti contains the sensory cells for hearing, the inner and outer hair cells. The inner hair cells are of sensoric type which detect the vibrations caused by sound and transform the vibration energy into nerve action potentials. The outer hair cells are, however, both sensoric and motoric containing several proteins with contractive properties which allow them to act as elementary movers into the organ of corti [10, 44, 81]. This motoric property explains the mechanism behind OAE where mechanical energy produced by the outer hair cells propagate backward along the basilar membrane towards the stapes. The active phenomenon is under the control of the efferent nerve system to hair cells where emission parameters can be altered by proper stimulation of the efferent pathway [10, 74, 103]. Emissions thus reflect leakage of energy from the nonlinear, mechanical process based on activity in the outer hair cells. Reduced electromotility in the
outer hair cells resulting in hearing loss has also been found to in reduce or absent OAEs. Thus OAEs provide an objective means to assess the status of an essential component of the inner ear functionality and to make conclusions on hearing loss [120].

A wide range of research findings supports this hypothesis. Among the most compelling is the fact that OAEs can be measured under circumstances in which neural responses are absent OAEs can be measured even when the eight nerve has been severed or when eighth nerve activity is blocked chemically [111, 114]. OAEs are unaffected by stimulus rate, unlike neural responses. In addition, OAEs are vulnerable to a variety of agents such as acoustic trauma and ototoxic medications that cause hearing loss, and in particular, damage to OHCs [18, 105].

OAEs are sounds found in the external auditory meatus that originate in physiologically vital and vulnerable activity inside the cochlea [64]. There is abundant experimental evidence that this activity is intimately associated with the hearing process. It happens that OAEs are generated only when the organ of corti is in normal conditions. It is well documented that when OHCs are absent or damaged (i) Auditory sensitivity is reduced by 40-60 dB as in Figure 3.5(a) (ii) The tips of tuning curves are elevated or absent (Figure 3.5(b)). (iii) Response to auditory stimuli as a function of stimulus level grown abnormally (Figure 3.5(c)). Also absence of OHCs is associated with a lack of otoacoustic emissions, supporting the hypothesis that OHCs are responsible for OAE generation.

In summary, it is believed that the motility of OHCs provides the mechanical source of OAE energy and that the efferent system allows for some regulation of this mechanism by the central auditory nervous system.
Figure 3.5: Schematic representation of the consequences of normal (solid lines) and abnormal (dotted lines) outer hair cell functioning.
3.4.2 Classifications of OAE

Figure 3.6 shows the various classifications of otoacoustic emissions. There are two basic phenomena (i) *Spontaneous Otoacoustic Emissions (SOAEs)* (ii) *Evoked Otoacoustic Emissions (EOAEs)*. SOAEs are emitted from the ear in the absence of stimulation. EOAEs can only be observed in response to stimulus applied to the ear. There are several different types of EOAEs, classified by stimulus used to evoke them, and two types have been extensively used. The first type is called *Transient Evoked Otoacoustic Emissions (TEOAEs)* and the second type is *Distortion Product Otoacoustic Emissions (DPOAEs)*.

![Diagram of OAE classifications]

*Figure 3.6: Types of otoacoustic emissions (OAEs)*

Figure 3.7 shows the typical SOAE waveforms recorded from normal hearing human ears. The spectrum of SOAE shows typically one or more pure tone like signals, at frequencies which are reported to be very stable over the time while their amplitudes may change. Multiple SOAEs from a single ear are common, and female subjects are more likely to have multiple SOAEs than male subjects. SOAEs are generally not observed in frequency regions with sensorineural hearing loss ex-
ceeding 30 dB. Majority of SOAEs from adult ears fall within the frequency region 1 KHz to 2 KHz with mean amplitude -3 to 5 dBSPL, which probably reflects the contribution of middle ear resonance characteristics [97]. Because SOAES are not measurable in all normal ears and because they appear at discrete and unpredictable frequencies, they are not the emission of choice for clinically assessing cochlear functioning.

Figure 3.7: Examples of spontaneous otoacoustic emissions recorded from normal hearing human ears

Transient evoked OAE responses are generated by movement of outer hair cells on the organ of corti, when the auditory periphery is stimulated by a click or tone burst. A click is presented to ear, and the response occurs after a brief time delay. The measurement of TEOAEs is accomplished using time synchronous averaging, although averaging will reduce the amount of noise in the trace, it will not remove the stimulus artefact at the start of the recording. The stimulus artefact under typical recording conditions is much larger than the recorded TEOAE. The energy from the stimulus may also persist in the ear canal long enough to obscure the onset of TEOAE response. Therefore, the first few milliseconds of trace are usually eliminated from the final averaged to remove any energy due to the stimulus. The Figure 3.8 shows the TEOAE waveforms detected from a normal full-term newborn.
Distortion product otoacoustic emissions (DPOAEs) are very low level stimulated acoustic response to two pure tones presented to the ear canal. DPOAE measurement provides an objective non-invasive measure of peripheral auditory function and used for hearing assessment. In this type of otoacoustic test, two pure tones with frequencies $f_1$ and $f_2$ are presented to the cochlea. For best results, $f_2$ is usually chosen at $1.22f_1$. Since the ear is a nonlinear structure, a number of very low level distortion products are generated due to the inter-modulation process within the cochlea. Out of which $2f_1 - f_2$ is usually the strongest and it is used for clinical purpose. DPOAEs occur at predictable frequencies that are mathematically related to primaries. DPOAE can be therefore being measured using narrow band filtering centered at frequencies of interest.

### 3.4.3 Clinical Applications

OAEs have great potential for use in three distinct clinical applications. First, because the mechanism that generates OAEs is specific to OHC system. OAEs complement the existing battery of audiological site-of-lesion tests, allowing, for
the first time, differential diagnosis of that cochlear dysfunction involving reduction of outer hair cell electro motility from other sensorineural or central losses. Second, because OAEs elicited from a normal ear extremely stable over long periods of time, allowing detection of very small induced changes, they are useful for longitudinal monitoring of subject at risk for hearing impairment. For patients receiving life saving but ototoxic drugs or undergoing surgery to remove auditory-nerve tumors, OAES may serve as a rapid, objective means in assess cochlear status. Similarly, monitoring of workers in noisy environments may allow identification of those most at risk for noise-induced hearing loss, so appropriate precautions can be taken. The third, and probably the most important application of OAEs, is for infant-hearing screening. Because the great majority of congenital hearing losses involve the outer hair cell system of inner ear and because OAEs can be measured quickly using relatively inexpensive equipment.

The clinical applications can be divided into following categories

**Infants:**

- Neonatal Screening (targeted or universal).

**Children:**

- Children’s hearing Screening (pre-school & school age).
- Ototoxicity monitoring.
- Tinnitus monitoring.

**Adults:**

- Detection of central auditory disorders.
• Ototoxicity monitoring (monitoring of the course of a potentially ototoxic drug).

• Early detection and monitoring of noise induced hearing loss.

• Tinnitus monitoring.

• Detection of acoustic neuroma tumors and other central auditory disorders.

3.5 Conclusions

This chapter has attempted to portray a general overview of human hearing mechanism and influence of external, middle and inner ear on hearing process. The common hearing problems, like hearing loss and temporal bone abnormalities, are discussed. The physiological basis of OAE generation, its relation to cochlea and applications of OAEs in hearing diagnosis are also presented. OAEs and diagnosis of temporal bone abnormalities are the major focuses of attention in this thesis for further investigations.