

PRE AND POST-OPERATIVE AUDIOLOGICAL FINDINGS IN
PERIPHERAL HEARING DISORDERS - A REVIEW

Reg No. - M. 8911

An Independent Project Submitted as part Fulfilment
for First year Master of Science (Speech and Hearing)
to the University of Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING

MANASAGANGOTTHRI

MYSORE - 6

MAY. 1990

In
The
Profound Memory of
My Respectable Father
.... whose echoing speech
made me to achieve this goal

AND

My Beloved Harendra Bhaiya
... who has left a vacuum in my
heart which can never be filled.

CERTIFICATE

This is to certify that the Independent Project entitled: Pre and post-operative Radiological Findings in Peripheral Hearing Disorders - A Review is the bonafide work done in part fulfilment for First Year M.sc. (Speech and Hearing) of the student with Register No.M8911.

Mysore

May 1990


DIRECTOR

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CERTIFICATE

This is to certify that this Independent Project entitled: Pre and Post Operative Aadiological Findings in Peripheral Hearing Disorders - A Review has been prepared under my supervision and guidance.

Mysore

May 1990


GUIDE

DECLARATION

I hereby declare that this Independent Project entitled: PRE AND POST OPERATIVE AUDIOLOGICAL FINDINGS IN PERIPHERAL HEARING DISORDERS: A REVIEW is the result of my own study undertaken under the guidance of Dr.(Miss S. Nikam, Prof, and Head, Department of Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier at any University for any other diploma or degree.

Mysore

May 1990

Reg. No.M8911.

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INTRODUCTION

Man has come a long way from his animal skin and stone axe days. His life now consists of electronics, chemicals, as well as a variety of art and social function. No longer are meagre food, simple shelters or bare clothing enough for him. The human being is always striving towards sophistication in his lifestyle these days.

He has achieved a great deal in areas of science and technology along with those of the fine arts and economy. There are so many improvements in our life these days, in fact, that no one person alone can actually keep count of all of them.

With sophistication, the need for accuracy and precision has increased tremendously. The advance in all the different disciplines is simply too much now for a single person to improve his skills. Thus came the need for specialization in particular areas.

In the course of time, those specialising in particular areas of study have not been able to give enough attention to other areas. These areas are neglected as a result, even if they happen to be important. The area of audition and the process of hearing is one field which has been ignored because of increased specialization.

Not many people spend way time on their ears nor do many give much of a thought for hard of hearing individuals. This may have occurred because hearing has for a long time been accepted as a matter of fact. The sense of hearing is not something that is noticed easily, as is vision. Individuals with hearing impairments are often able to escape notice by using their vision and not committing themselves to anything. It is at this point that, lives may be affected by lack of knowledge. The lack of hearing affects our communication and therefore our efficiency in living our lives well.

It is at this stage, the growing need of a specialized field of Audiology finds its place, and an audiologist through his professional activities helps the growth of awareness among general public regarding Bar and Hearing disease and the kind of help they can seek for. Diagnosis, successful rehabilitation and an effective education for the public is the three major aspects with which an audiologist works. He with his sophisticated instrumentation and depth of knowledge of hearing problems, would be able to select the best for an individual.

Radiologists over the years have one common goal and one common problem and that is to assessthe hearing sensitivity of a large population which comes to them and then label them with less amount of doubt so that the proper

treatment would be available to them. This doubt is only because an audiologist has no standardized patterns and findings based on which he can specify the disease as such. And this problem is more especially in dealing with the peripheral auditory disorders. Sometimes the puretone audiological findings or impedance audiological findings or speech audiological findings of one disorder shows that though the lesions are different and the disorder is different but the findings of these tests are same and in that condition it is very doubtful for an audiologist to label a disorder or disease based on these limited findings. In that condition a clinical audiologist requires the findings of either audiological tests or a battery of test so that he can reach a particular type of an accurate diagnosis which will correlate with the diagnosis of medical professionals and a better and more appropriate treatment would be provided to those patients.

Both the clinical experience and rich research data enable us to group the audiological findings and associate them with specific problem in the ear or a specific condition like hearing loss due to old age or presbycusis. Such consistent findings allows the audiologist to select suitable test batteries for a patient with specific complaint and it also makes the diagnostic procedure more smoother, easier and with

least amount of doubt. It's help in saving the time and energy is also very important. Now in order to cross check the findings a primary need of an audiologist is the results of all the different audiological tests in different disorder which is not available.

Here is an attempt to fulfil this need, i.e. to put such consistent pre and post operative audiological findings in those peripheral hearing disorders in which surgical treatment is involved. Thus this attempt has got two purposes. The first purpose obviously is to help a clinical audiologist in his day-to-day clinical practice; and second purpose is to compare the pre-operative audiological findings with that of the post operative one, which in turn will help to evaluate the hearing improvement quantitatively after surgery, In other words to appreciate how effective the surgical treatment was.

In every chapter a brief description of the disease and summary of different audiological test findings is presented in a tabular form under two columns of pre-operative and post-operative audiological findings. This gives a quick and easy access to information without having to spend valuable time in retrieving details previously examined. Though in many diseases the findings show a progressive change over time, the value of the findings are nevertheless appreciate,

Here it is very important to be kept in mind that though this hand book deals with the peripheral hearing disorders, it is limited to only those peripheral hearing disorders in which surgical treatment is involved and needed.

REVIEW OF LITERATURE

Various structures and parts of our auditory system play different roles in the transformation, transduction, coding, transmission and decoding of the auditory signal. When we do audiological evaluations of different patients with complaint of hearing loss due to variety of reasons; we get a variety of audiometric patterns and findings in our clinical practices. These findings and patterns differ one from the other because of differences in the structure of the auditory system involved.

The first investigator who encountered this differential symptomatology perhaps was Ernst Weber in 1834. He put a vibrating tuning fork on the mastoid portion and noticed the hearing sensitivity through bone conduction in different pathological conditions. He noticed that in conductive hearing loss cases, the bone conduction signals lateralized and were referred to the poorer ear. After a 21 years of gap, in 1855 Heinrich Rinne described another method of tuning fork testing to quantify the air-bone gap by comparing the duration of perception by air conduction to the duration of perception by bone-conduction. Both of these tuning fork tests, so called the Weber test and the Rinne test were used as a cornerstone of diagnosis.

Though these two tests provided fundamental test results for differentiating conductive from sensorineural hearing loss, these tests could not meet the following needs i.e. these tests have certain limitations. They

are :

- 1) The tests could not differentiate bilateral mixed hearing loss from conductive and sensorineural hearing loss.

- 2) They could not differentiate among very mild losses and also in profound loss cases it could not differentiate whether the loss was mixed or sensorineural in nature.

- 3) They could not explain the mechanism or the part of the auditory system involved in various types of pathological conditions of the ear.

So, there was a need for developing other methods of testing which could detect and determine the site of disorder and specify the mechanism involved in different types of auditory disorders.

The first half of the twentieth century saw the invention of the vacuum tube, the development of the electric audiometer and an explosive increase in studies of auditory sensitivity. There was a little more progress, however, in diagnostic audiometry. The year 1948 was of major significance in the field of audiology because the interest now turned towards

the diagnostic audiology and developing more and more methods of testing which are specific to certain disorders e.g. cochlear versus retrocochlear pathology. This new interest and orientation in a very new field of audiology was initiated by the work of Dix, Hallpike and Hood who developed a test in England. The test came to be known as Alternate Binaural Loudness Balance (ABLB) test which is still used by the clinical audiologist for the measurement of loudness recruitment. The phenomenon of hypersensitivity to loud sounds often accompanied by hearing loss was studied in the middle of the nineteenth century by Fowler (in the period between 1928 to 1937). But Dix, Hallpike and Hood were the first to assign correct significance to the symptoms by showing its unique correspondence with the cochlear site of lesion.

The next decade came up with different types of procedures designed to measure loudness recruitment indirectly (i.e. objectively). Different attempts were made to exploit the differential diagnostic potential suggested by the work of Dix, Hallpike and Hood. In such attempts intensity and frequency discrimination, masking of tones by noise, adaptation, fatigue were all investigated exhaustively. The ABLB test result was regarded as validation yardstick against which

the efficacy of all the new procedures was evaluated. Then it was felt that the loudness recruitment as such was not the parent phenomenon which differentiates cochlear lesion from the eighth nerve lesion, but only one of the many possible manifestations of cochlear disorder, i.e. if recruitment is absent it does not mean that the cochlear pathology is not present. It is possible that the transduction and biological amplification system in the auditory system will be affected. As a consequence of this, it was realized that there was a lack of relations among various psychoacoustic measures and loudness recruitment; and finally it was realized that there was a need for developing such a valid test which should be aimed at predicting the site of disorder rather than prediction of recruitment. More precisely it was felt that our procedure should be so developed that it would show the relationship between the symptomatology and function.

Though abnormal adaptation like loudness recruitment, was well known to the nineteenth century otologist, realization of its full potential as differentiating symptom however was not possible because of the unavailability of sophisticated electronic instruments. In 1947, Bekesy introduced a new and exciting avenue of investigation

and provided an unique tool for a precise study of temporal changes in auditory sensitivity to sustained stimulation.

It was in the 1960s when new activities were started in the field of audiology, more specifically in the area of diagnostic audiometry. These new activities included :-

1. The development of new speech audiometric measures which played a very important role in differentiating peripheral auditory disorders from that of the central auditory disorders.
2. The development of instrumentation for impedance audiometry which is of great importance in the differential diagnosis of sensori-neural loss and conductive loss which in turn help determine the lesion in peripheral as well as in central auditory disorders.
3. The development of tests to determine the eighth nerve site especially in the case of retrocochlear pathology.

Although a number of nineteenth and twentieth century otologists and audiologists especially in Germany, experimented with the diagnostic potential of speech audiometry, the important break through came in 1955 when Bocca and his colleagues, in Italy showed how speech signal could be used to identify temporal lobe disorder. This pioneering work of Bocca and his colleagues stimulated an important series of research throughout the world in 1960s which was aimed at

the development of such material and techniques which exploited the diagnostic potential of speech audiometry. As a result of this work, speech audiometry has become one of our most important techniques for identifying and evaluating central auditory disorder and for differentiating peripheral from central site.

Impedance audiometry had its roots in the Scandinavian countries. Following the new classic 1946 monograph of Metz, a number of investigators in Denmark and Sweden developed and refined tympanometry and acoustic reflex measurement. It was not until the late 1960s that the clinical impedance audiometry developed in the United States. During the 1970s, however, its use became widespread to the point that it is now considered an integral part of the basic audiometric evaluation. It is probably fair to say that no new technique has had such a profound impact on clinical audiological practice since the development of electric audiometer itself. But still there were difficulties in testing the hearing sensitivity of babies and young children.

finally, the 1970s saw the development of another powerful new technique known as auditory brainstem response (ABR) audiometry. Sohmer and Feinmesser in Israel, and Hecox and Galambos, in the United States, were

among the first to demonstrate the potential value of the Auditory Brain Stem Response in audiological evaluation, especially in testing babies and young children. At present, this technique has established its great importance in the evaluation of virtually every type of auditory disorders from the middle ear to the brainstem. At present with our present knowledge by using impedance and ABR audiometry we can now differentiate, relatively, successfully, among five potential sites of disorder; the middle ear, the cochlea, the eighth nerve, the brainstem auditory pathway, and the auditory cortex in the temporal lobe.

CONGENITAL ANOMALIES OF THE EAR

Description of the disease:

Congenital malformations of the external ear results from the developmental failure of the first and second branchial arches. These malformations of external ear are often associated with malformations of the Middle ear and inner ear (Mowson, 1979). There are various types of congenital abnormalities of the ear. The common abnormalities are -

Macrotia - size of the auricle will be bigger than normal size.

Microtia - size of the auricle will be smaller than normal size

Anotia - Absence of auricle

Accessory auricles

Atresia of the external auditory meatus

Atresia of the external auditory meatus with microtia and abnormal middle ear.

Atresia of the external auditory meatus with abnormal inner ear etc.

Among these various types of abnormalities the atresia of external auditory meatus associated with microtia and

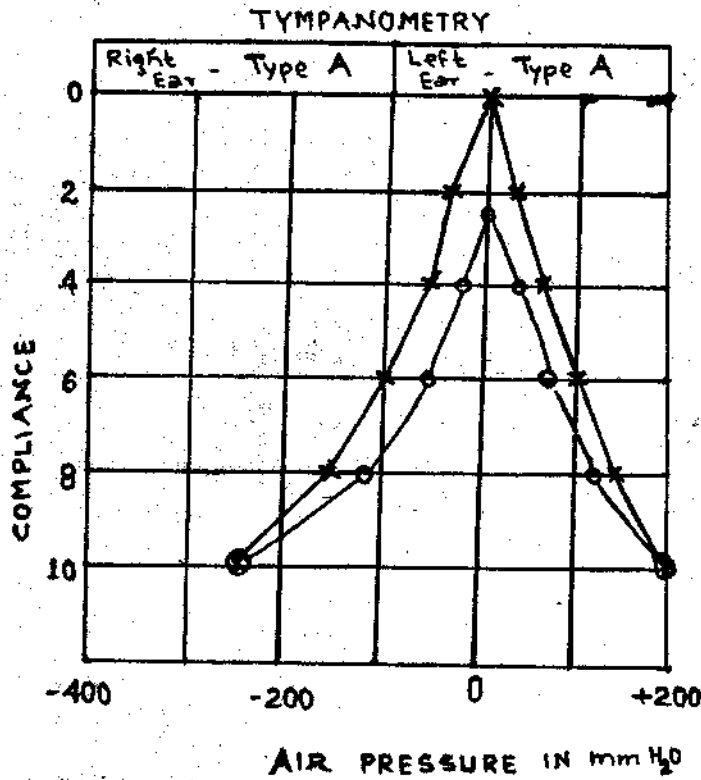
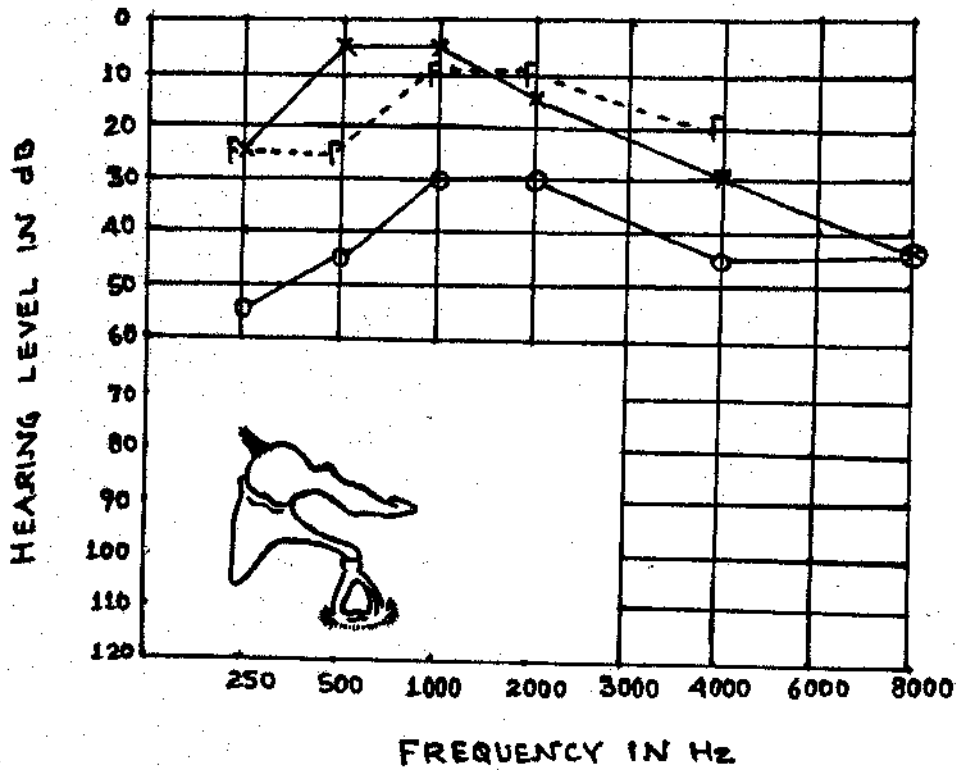
middle ear abnormalities is more common. Absence of auricle is often associated with atresia of the external auditory meatus. Congenital anomalies of the external ear and the middle ear are most commonly seen with the anomalies of other structures which are developed from the first two branchial arches. such as Treacher Collins's syndrome, whose features are Bilateral atresia of the ear, hypoplasia of the mandible, hypoplasia of the molar and maxillary bones, and antimongoloid palpebral fissures. At the same time several other congenital abnormalities can also occur in the same individual. These abnormalities produce cosmetic deformities, infections and hearing loss (Dayal, 1981).

Atresia of the ear canal results in a conductive type of hearing loss. Hearing loss associated with atresia of the external auditory meatus and middle ear defect can be unilateral or bilateral. In unilateral cases with normal hearing on one side causes no immediate concern, and if surgery is required it should be advised when the patient reaches 16 or 17 years of age. Infants with bilateral hearing loss due to such malformations need a good hearing for their proper development of speech and language. Hence in that case an appropriate hearing aid should be given as early as possible.

So far as the question of surgical correction in these infants is concerned, some surgeons advocate early surgical correction, while others recommend that surgery on one side should be done between 5 years to 7 years of age. But such surgery carries a risk of facial nerve injury because the nerve often runs an anomalous course in these cases. This surgery includes repair or reconstruction. Apart from this an audiological evaluation, and amplification is must for rehabilitation of these type of cases (Bergstrom, et al. 1974).

Pre operative and post operative audiological findings in congenital anomalies like atresia of the external auditory meatus.

Sl. NO.	Name of the test	Pre operative audiological findings	Post operative audiological findings
1.	Puretone audiometry.	<p>Puretone audiogram shows an unilateral or bilateral conductive hearing loss with PTA of less than 60 dB. sometimes mixed loss is also present (Finitzo, et al. 1979).</p> <p>Audiogram shows to flat curve with AvB gap of around 50 dB. In bilateral Cases there is a bilateral symmetrical mixed hearing loss of around 65 dB. (Bergstrom, et al. 1974).</p> <p>Bone conduction is generally within normal limits but in some cases it may be affected. In Around 10 percent of the cases have also been reported who have sensori-neural hearing loss in which inner ear was involved (Finitzo-Hieber, et al 1979)</p>	<p>61 percent to 73 percent of cases show improvement in hearing by 35 dB (Bellucci, 1981; Cremars et al. 1984 and Lesinski, 1984).</p>
2.	speech audiometry.	<p>Unaided SRT is reported to be around 48 dB. and aided SRT is reported to be around 38 dB which indicates an improvement of 10 dB in hearing sensitivity when the patient is fitted with a hearing aid (Grundfast, and Camilon. 1986).</p>	<p>Hearing improved within 25 dB SRT or better in 68 percent of cases (McDonald 1986).</p>
3.	Impedance audiometry	<p>Tympanograms seen in these cases is of flat type.</p> <p>Acoustic reflex depends upon the involvement of the inner ear.</p>	



	500 Hz	1000 Hz	2000 Hz	4000 Hz
Right Ear	Absent	Absent	Absent	Absent
Left Ear	Absent	Absent	Absent	Absent

ACOUSTIC REFLEX

Fig:1 Congenital malleus fixation (Moon and Hohn 1978)

DISCONTINUITY OF THE OSSICULAR CHAIN

Description of the disease:

Discontinuity of the ossicular chain refers to a disruption of normal articulation between the ossicles (malleus, incus and stapes) (Jerger and Jerger, 1981).

A history of deafness, usually unilateral, following head injury or previous cortical mastoidectomy, should indicate the possibility of ossicular dislocation or discontinuity of ossicular chain (Ballantyne, 1979). It can occur as a consequence of congenital defects, skull trauma, and middle ear disease. A variety of lesions can result but dislocation of the incus is the most frequent. (Ballantyne, 1979). Hough and Stuart, (1968) report that the most common site of the ossicular chain disruption is the incus and/or the incudostapedial joint. If the stapes has been involved there may be perilymph leak and progressive sensori-neural hearing loss superimposed on the conductive hearing loss. This condition is rare and an urgent surgery is done. (Jerger and Jerger, 1981).

Sometimes in the patients with ossicular chain discontinuity, the ossiclea may become rejoined by fibrous tissue or they may get fixed by a mass of new bony growth.

This condition is also known as ankylosis between the ossicles. This discontinuity in the ossicular chain or ankylosis between ossicles and adjacent bony walla of the middle ear, causes a maximum conductive hearing loss of 50 to 60 dB. This amount of hearing loss is nearly equal at all frequencies (Engush, 1976).

This ossicular chain discontinuity may be congenital. If it is congenital it may be associated with branchial arch and facial anomalies i.e. Pierre Robin and Treacher Collins syndrome and atresia or microtia. Generally it is unilateral (Jerger and Jerger, 1981). If the loss is bilateral, a child may manifest speech and language retardation. This speech and language delay will be associated with articulation errors and there will be significant disparity between verbal and nonverbal sections of intelligence tests. Absence of the oval window or round window, either in isolation or combined with ossicular chain anomalies, gives a similar picture. However, absence of both oval and round windows produces total deafness in the affected ear. This total deafness occurs because the movement of cochlear fluids which is essential for perception of sound cannot occur (Engush, 1976).

In the case of congenital ossicular absence there are also a variety of lesions. Absence of stapes superstructure

with or without the long process of the incus is the eommonest one. Polytomography may demonstrate osscicular absence or dislocation or reveal fracture lines. A small location of incus by 1-2 mm is enough to cause a marked deafness. (Jerger and Jerger,1981).

If this disease is associated with a longitudinal skull fracture, it may be accompanied by external ear canal lacerations or collapse, rupture of tympanic membrane, hemorrhage of mucous membrane and seventh nerve disorder.

Pre and post operative audiological findings in ossicular chain discontinuity cases.

Sl. NO.	Name of the tests	Pre operative audiological findings.	Post operative audiological findings
1.	Pure tone audiometry	Pure tone audiogram shows a conductive hearing loss of 40 to 60 dB. A-B gap will be 30 to 50 dB and the audiometric configuration is generally flat (Jerger and Jerger, 1981)	<p>According to Ogale, et al. (1986) report 93% of the cases improve their hearing with a closure of A-B gap to within 15-20 dB.</p> <p>According to Shea and Emmett, (1978), average AC gain is around 20 dB and according to Chang, and Cho, (1977), the closure of A-B gap is within 20 dB.</p> <p>There remains a presence of A-B gap of 20 dB in 84% of cases as reported by Lesinski (1984).</p>
2.	Speech audiometry PIP function	<p>SRT is within normal limit and SDS is also within normal limit.</p> <p>PIP function shows no roll over phenomenon (Jerger and Jerger, 1981)</p>	
3.	Impedance audiometry	<p>Tympanogram in later stage shows an abnormal deep type A i.e. Ad type, where as in early stage it may be type A.</p> <p>Static compliance is well above the normal range of 0.3 to 1.6 cm³.</p> <p>Acoustic reflex is absent or elevated and the reflex pattern i.e. Jerger box pattern is inverted L-shaped in configuration (Jerger and Jerger, 1981).</p>	

4. Weber Test

Weber is lateralized to the affected ear side (Prescod, 1978).

occlusion effect

It does not bring any change in the occluded ear results.

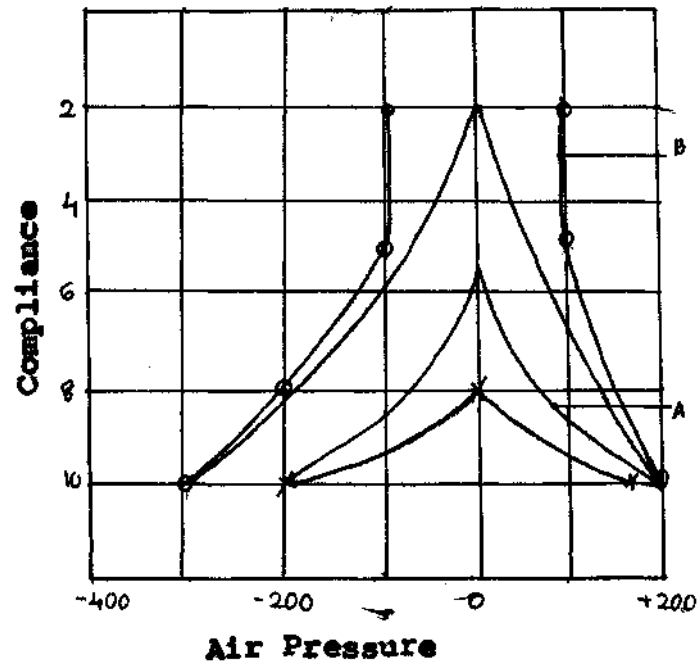
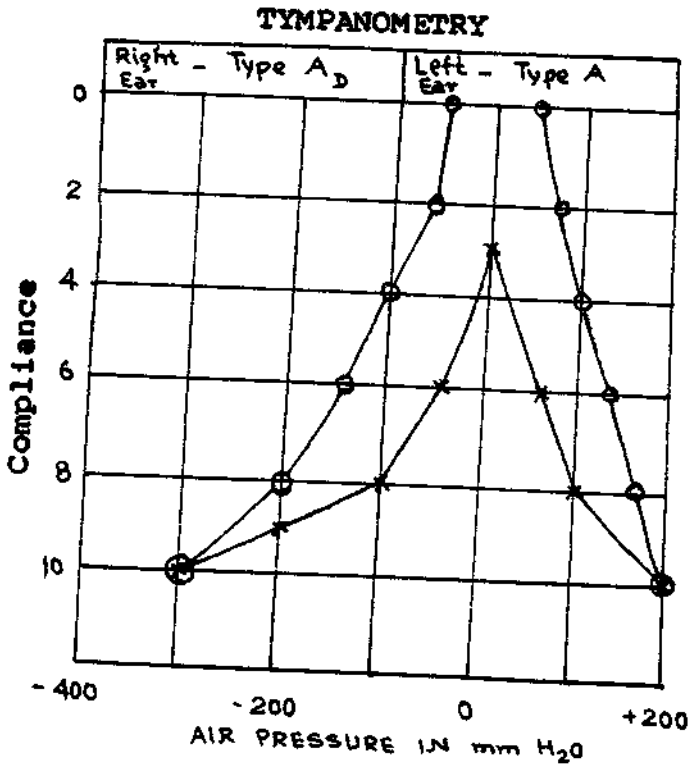
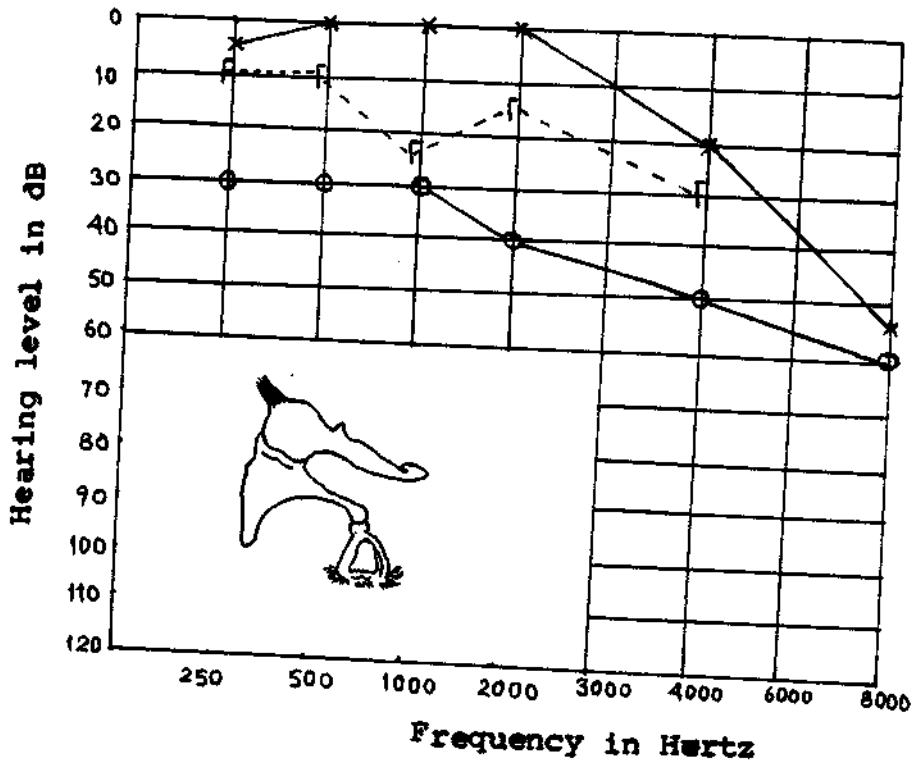


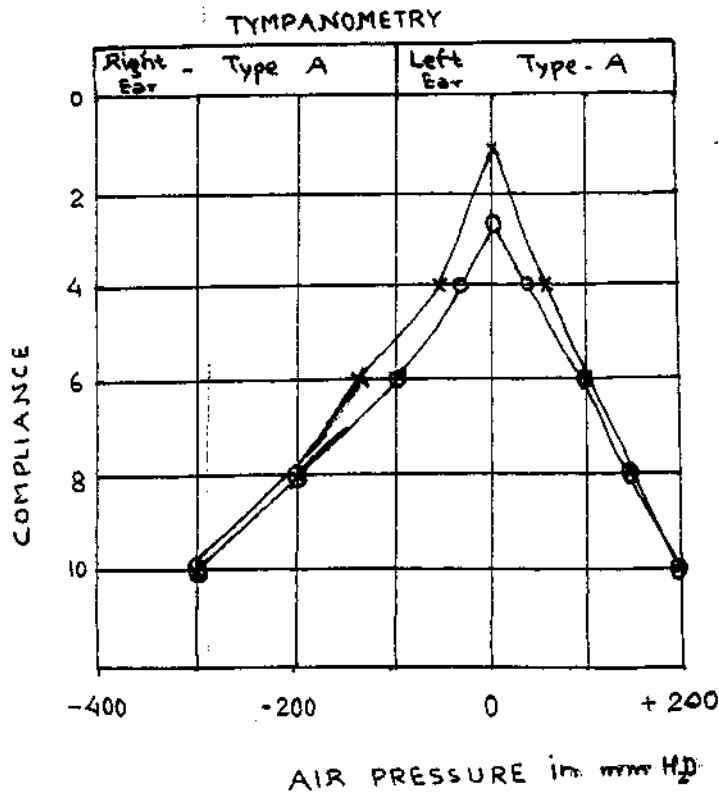
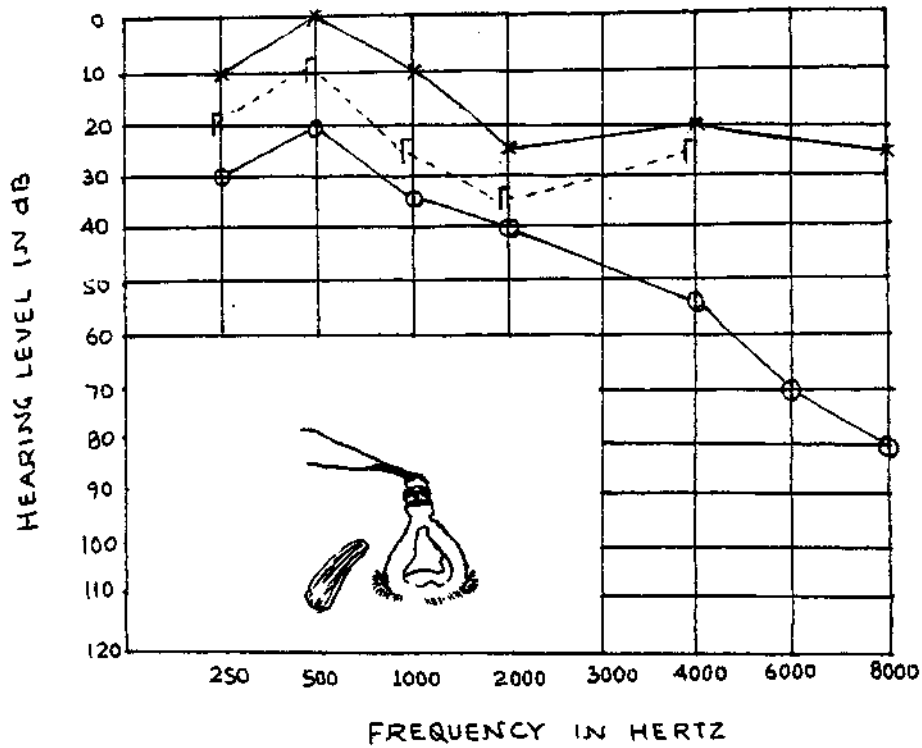
Fig-2 Madsen Acoustic bridge demonstrating tympanogram findings in -
A - Ossicular fixation and
B - Ossicular chain discontinuity



	500 Hz	1000 Hz	2000 Hz	4000 Hz
Right Ear	Absent	Absent	Absent	Absent
Left Ear	Absent	Absent	Absent	Absent

ACOUSTIC REFLEX

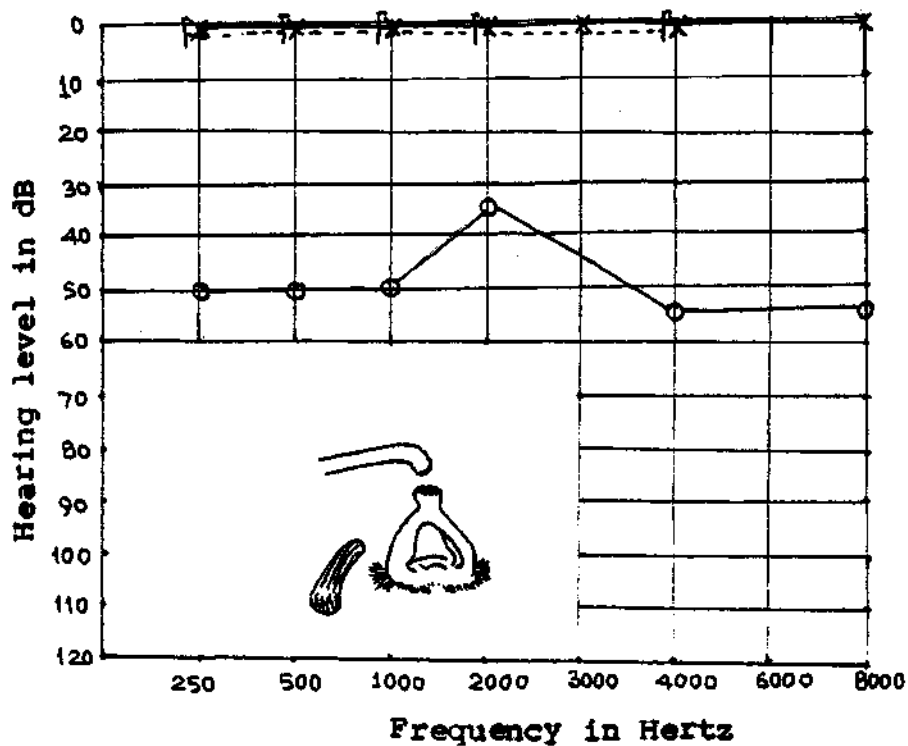
Fig.3 Malleus-Incus fixation with atrophic tympanic membrane (Moon and Hohn 1978)



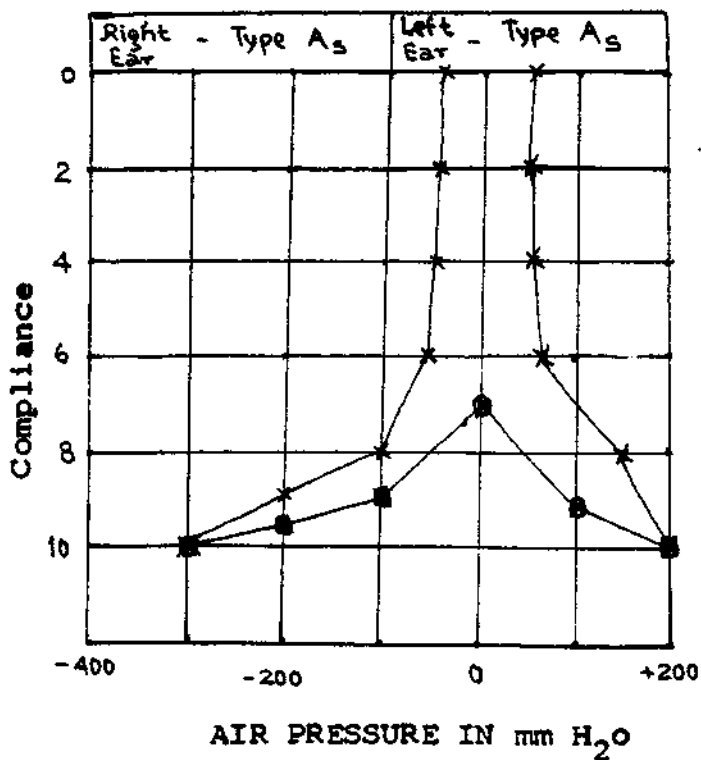
	500 Hz	1000 Hz	2000 Hz	4000 Hz
Right Ear	Absent	Absent	Absent	Absent
Left Ear	Absent	Absent	Absent	Absent

ACOUSTIC REFLEX

Fig:4- Ossicular chain discontinuity



TYMPANOMETRY



	500Hz	1000Hz	2000Hz	4000Hz
Right Ear	Absent	Absent	Absent	Absent
Left Ear	Absent	Absent	Absent	Absent

ACOUSTIC REFLEX

Fig:5, Ossicular chain discontinuity Incus stapes separation after skull fracture

(Moon and Hahn, 1918)

OTITIS MEDIA

Description of the disease:

Otitis media, literally, means an inflammation of the mucoperiosteal (mucosis + periosteum /membrane covering the bone/) lining of the tympanum or middle ear cleft. This inflammation can include eustachian tube, tympanic cavity, attic, aditus, mastoid antrum and mastoid air cells (when present) (Reichert, Specter, 1979).

Otitis media is a very common infectious disease of early childhood. It affects children of all ages, races and social groups. The mucosal lining of the middle ear cleft is continuous with that of the upper air passages at the level of the nasopharynx. Normal aeration of the middle ear cleft is maintained by activity of the eastachian tube whose opening equalizes the pressure (of the middle ear to that of the outer ear) (Coulthard, 1979).

Ventilation and normal aeration of the tympanic cavity is a must. If an adequate amount of aeration is not provided to the middle ear due to dysfunction or obstruction of the eustachian tube, the middle ear ventilation is impaired and a static air in the middle ear space may be absorbed by blood vessels in the mucosal lining. As a result of this, a negative

air pressure is created in the tympany cavity. The tympanic Membrane may become retracted. This reduced middle ear pressure may result ia edema of the mucosa and secretion of fluid from mueoperiosteum. (Friedmann, 1974).

The eustachian tube is the commonest route by which Inflammation travels from the nose and nasopharynx to the middle ear. In infants and children the middle ear infection is very common because the eustachian tube is shorter and straight. Following are the sources of infection:

1. Recurrent upper respiratory tract infection.
2. Common cold and influenza with secondary infection later.
3. Enlarged adenoid
4. Tumours of the nose and nasopharynx
5. Nasopharyngeal or nasal packing
6. Rhinitis and sinusitis
7. Childhood diseases such as measles and scarlet fever
8. Carelessness on the part of mother for keeping the baby in flat position while feeding, when milk regurgitates into the nasopharynx, then to middle ear cavity through euatachian tube.

Swimming and diving can cause forceful enterance of infected water through the eustachian tube (Macqbool, 1988).

Various stages of inflammation are -

1. Stage of hyperemia

2. Stage of exudation
3. Stage of suppuration
4. Stage of resolution and
5. Stage of complication

(shyamal Kumar, 1982).

This disease may be acute or chronic, and it can be suppurative or non-suppurative in nature. A common mechanism involved in acute infectious otitis media is a spread of upper respiratory tract infection through the eustachian tube. In suppurative otitis media a pus-like fluid may collect in the middle ear space (Gerwin and Read, 1974). In contrast to acute course, suppurative otitis media may be chronic in some individuals CSOM (chronic suppurative otitis media) may result in pathologic changes or destruction of the middle ear mucosa, ossicles, and mastoid bone. Together with this perforation of the tympanic membrane and a discharge of pus from the ear may occur (Jerger and Jerger, 1981).

In some individuals the disease may progress and it may lead to complications of otitis media which include tympanosclerosis, perforation of the tympanic membrane, and cholesteatoma formation. If the inflammation spreads to adjacent structures in the temporal bone or inside the cranial cavity, complications may include labyrinthitis, mastoiditis, facial paralysis, brain abscess, and meningitis. Some individuals may develop a permanent sensori-neural hearing loss.

It may occur at any age. However, according to the second National Conference on Otitis Media held in March, 1978, the incidence of acute otitis media is highest among infants of 6 to 24 months of age. Approximately 80% of the patients are children less than 5 years old. Chronic suppurative otitis media most often has its onset between 5 and 10 years of age (Jerger and Jerger, 1981). In Boston study by Teele, Klein (1978) and other studies by McEldowney and Ressler (1972) reported high prevalence of acute middle ear infections in males than in females. But other studies report that the sex ratio varies with age (Bjuggren and Tanevall, 1952).

The onset of otitis media may be acute or insidious. There will be a complaint of fluctuating conductive hearing loss. Otitis media of acute onset may be accompanied by earache, hearing loss and rupture of tympanic membrane. Symptomatology may include hearing loss, a feeling of fullness in the ear, a low pitched, pulsating or continuous tinnitus, cracking sounds within the ear, chills and fever. (Jerger and Jerger, 1981. In some patients otitis media may recur over a period of years. Severe recurrent otitis media in the first 3 years of life may be associated and cause delayed speech and language development in some children (Lewis, 1976; Zinkus, et al. 1978).

The primary and best treatment of otitis media is by drug therapy and surgery.

Pre and post operative audiological findings in Otitis Media

Sl. No.	Name of the test.	Pre operative Radiological findings	Post operative audiological findings
1.	Pure tone audiometry	<p>Puretone audiogram shows unilateral or bilateral conductive hearing loss, In acute stage the loss is typically between 20 dB to 40 dB HTL. But the degree of loss varies depending upon the stage and the type of otitis media. In an initial stage of otitis media the hearing may be within normal limits but in more advanced stage it may be associated with a loss of greater than 40 dB.</p> <p>In the case of Cholesteatoma the hearing loss may be unilateral or bilateral and the degree of loss varies from mild to moderate depending upon the site and size of the cholesteatoma.</p> <p>Audiometric contour may vary with the progression of the disease. In initial stage when there is a reduction in the middle ear pressure, and stiffness of the ossicular chain, the audiometric contour will be rising with greatest loss in the low frequency region than high frequency region. When the fluid accumulates, high frequency may be affected and the audiometric contour will be flat.</p>	<p>Hearing improvement is significant in all groups except the ears undergoing reconstructions from the footplate. There is a significant hearing gain with a PTA of 33 dB and a reduction in A-B gap which is less than 20 dB in 94% of ears undergoing myringoplasty and 69% of the ears undergoing tympanoplasty (Palva, 1987).</p> <p>Anderson, et al (1983) report of a significant difference in A-B gap at 500 Hz, 1 KHZ and 2 KHz.</p> <p>In majority of the cases the hearing gain brings up to 20 dB and more. (Sushko, 1978)</p>

	<p>. Bone conduction threshold is within normal limits. But it may be affected depending upon the duration and severity of the disease.</p> <p>If there is sensori-neural hearing loss, the audiogram shows a flat or downward sloping curve (Jerger, and Jerger, 1981).</p>	
2. Speech audiometry.	SRT and SDS; both are found to be within normal limits (Jerger and Jerger, 1981)	Normal
3. Impedance audiometry	<p>Results of impedance audiometry varies depending on the nature and extend of ossicular chain involvement. Impedance audiometry shows an abnormal tympanogram. In the case of cholesteatoma tympanogram may be shallow type A in shape.</p> <p>Static compliance is reduced and found to be below the normal limit.</p> <p>Acoustic reflex will be absent if there is fluid in the middle ear. In the case of choleatoma acoustic reflex threshold (ART) may be elevated and acoustic reflex may be absent.</p> <p>In initial stage of otitis media the peak of the tympanogram may show slight positive pressure. As air in the middle ear space is exhausted the peak of the tympanogram may occur at an increasing degree of negative pressure. In this initial stage the tympanogram obtained will be of type 'C' configuration. At this time reflex may be seen if air pressure is successfully maintained at the</p>	Post operative impedance audiometrical findings shows varying patterns of tympanograms (Anderson, et al. 1983).

peak of the tympanogram. Gradually the amplitude of the negative peak may decrease. Then the peak may become rounded or blunted. Subsequently the tympanogram is of a flattened shape. At this stage the type of tympanogram we get is of type B configuration.

NOTE: In patients with cholesteatomas that have eroded the ossicular chain, the tumour itself forms a functional connection between the ossicles. In this case also we get rounded shape tympanograms, and surprisingly a little air-bone gaps may be observed. (Jerger and Jerger, 1981).

4. PI - SSS
function
SSIT scores

It is normal in both the ears.

SSIT scores agree with PTA sensitivity (Jerger and Jerger, 1981).

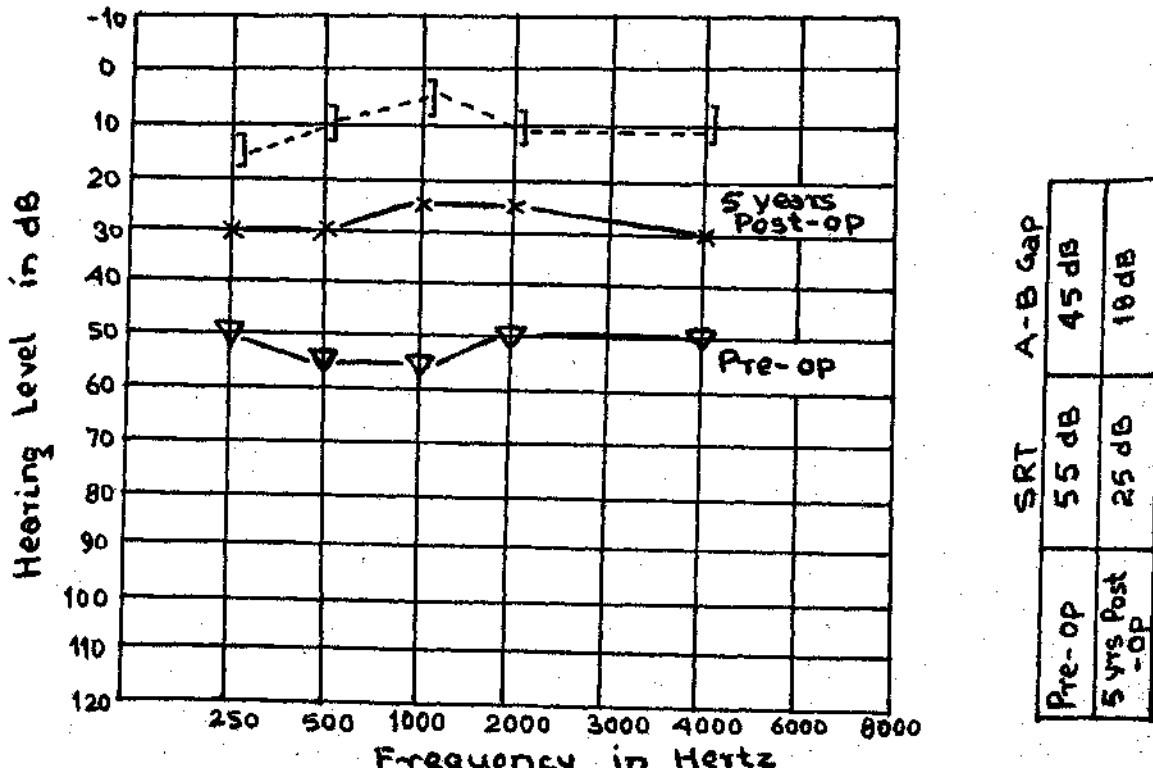


Fig:6, 5 Years Post-operative audiogram showing 25 dB. SRT with 18dB of A-B gap (Lesinski,1983)

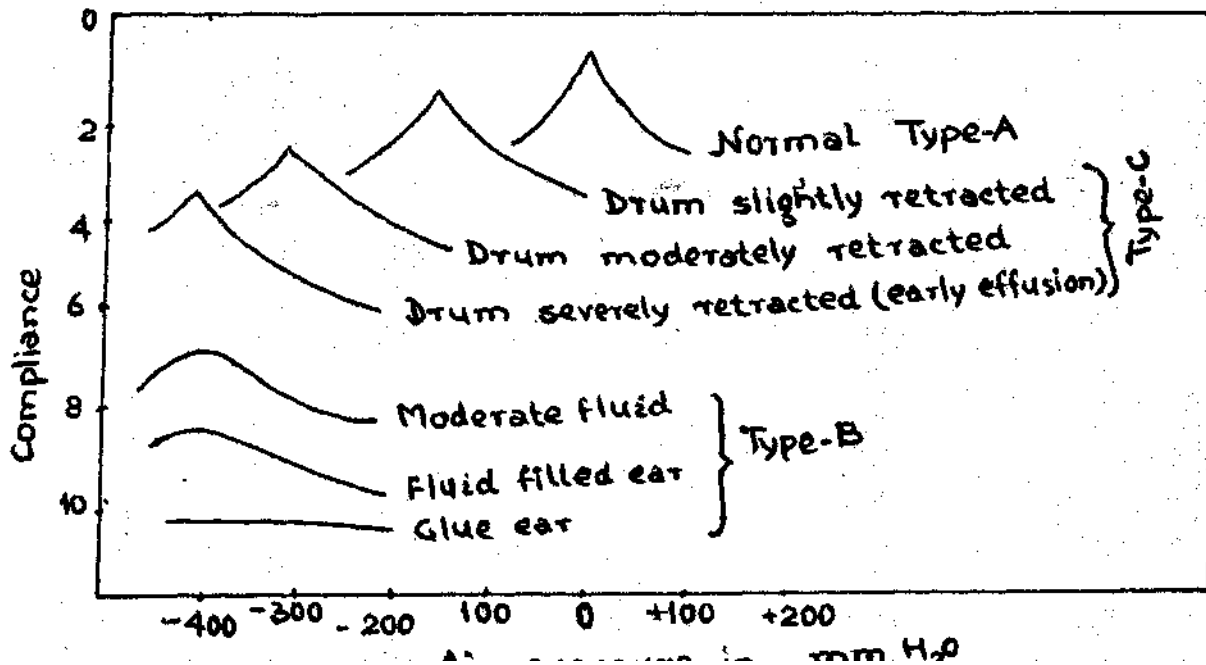


Fig:7, A Series of tympanograms illustrating the various Stages through which the tympanogram progresses during the development of serous otitis media. (Jerger and Northern, 1980)

OTOSCLEROSIS

Description of the disease:

The term "oto" refers to the ear, and "sclerosis" refers to a new spongy bone formation and remodelling. When both the terms are combined together it becomes otosclerosis which refers to a pathological condition of the ear. This pathological condition called otosclerosis refers to a new spongy bone formation which affects primarily to the labyrinth of the inner ear and it causes progressive deafness.

Otosclerosis is a common cause of hearing impairment. It is a disease or disorder of the bony wall (capsule) of the labyrinth. It is a localized osteodystrophy which has some similarity to Osteitis fibrosa cystica (Recklinghausen's disease) and Osteitis deformans (Paget's disease). It can occur anywhere within the petrous bone. It becomes manifested when the bony disorder has involved the oval window and interferes with the normal movement of the stapes. This produces a conductive hearing loss (Boies, 1961). This disease is characterised by spongy bone formation around the oval window and the promontory. This type of bone formation occurs in the middle layer of the bony labyrinth and it causes ankylosis or the fixation of stapes which causes interference with sound conduction to the inner ear (Nagar, 1969).

The exact cause of this disease is not known. The otosclerotic process generally develops in an area of the labyrinthine capsule, in which embryonic cartilage often persists. The first indication of the disease comes as an enlargement of the perivascular spaces. It has been reported that there is an increased vascularity of the tissues (and the cells) in the mucous membrane around the promontory and the atapes, This increased vascularity causes edema which results in vascular otospongiosis. This vascular otospongiosis after resorption of bone by new bone formation results in otosclerosis (Nagar, 1969). Hereditary and pregnancy are some factors which influence the development of otosclerosis. In a great majority of cases i.e. in 70 to 90 percent of cases the otosclerosis is located in the so-called area of predilection, which is anterior to the oval window (Bickert, 1965).

Generally this disease is bilateral but in 20 percent to 30 percent of cases it is unilateral (Guild, 1944, Fleischer, 1958; Lindsay, 1959). In 90 percent of the cases, the onset of otosclerosis is between 15 years and 45 years of age (Booth, 1978). It rarely starts before ten years and after 40 years of age (Prescod, 1978). Occurrence of this disease is more in females than in males and fair complexioned than dark. The incidence of this disease is fairly high in India and lowest among the American Negroes (Nagar, 1969).

As the disease progresses the patient complains of -

- (i) Hearing loss - in 80 percent of the cases there is a bilateral symmetrical conductive hearing loss. Onset of hearing loss is reported to be gradual (Booth, 1978).
- (ii) Paracusis is present, i.e. the patient may hear better in noisy than in quiet surroundings.
- (iii) Tinnitus is often present.
- (iv) Vertigo - generally it is not reported and even if it is there, it is minimal in degree.

There is no standard medical treatment for otosclerosis, but a restorative surgical procedure called stapedectomy can be done which improves the hearing sensitivity in most of the cases. If surgical treatment is not possible in that condition, a suitable hearing aid will be the choice of treatment.

Pre-operative and post-operative audiological findings in Otosclerosis.

Sl. No.	Name of the tests	Pre-operative audiological findings	Post-operative audiological findings
1.	Puretone audiometry	<p>Average PTA is around 45 dB. (Birch, 1986).</p> <p>Jerger and Jerger (1981) report of the following puretone findings:</p> <ul style="list-style-type: none"> - Puretone audiogram shows a bilateral symmetrical conductive hearing loss with presence of Carhart's notch at 2000 Hz for bone conduction. This Carhart's notch is very pronounced in early stage of otosclerosis. - The degree of loss is generally mild to moderate. - Greater loss is seen in low frequencies. - In early stapes fixation the air conduction curve is flat or gently rising <p>Bone conduction threshold is best at 250 Hz and poorest at 4 KHz. as reported by Ginsberg, et al.1978.</p> <ul style="list-style-type: none"> - A-B gap is around 15 to 20 dB. (Smith & Hop, 1986). 	<p>Average PTA becomes 35.38 dB HTL and air conduction threshold following stapedectomy has shown to reduce in increasing frequencies from 250 Hz to 8 KHz (Ginsberg, et al 1978 and Mair and Laukcl, 1986).</p> <p>PTA which was 45 dB changes to 20 dB i.e. there is a 25 dB improvement But it also depends upon the age at which the surgery was done.</p> <p>Bone conduction gain after surgery is in the same order as air conduction gain (Birch, 1986).</p> <p>Air conduction shows an average gain of 16 dB (Shaj and Farrior, 1987)</p> <p>Best bone conduction threshold at 250 Hz and poorest at 4 KHz. A slight negative change is evident at 250 Hz. But all other frequencies in a series of 2405 cases revealed a positive improvement in bone conduction threshold. In most of the cases the bone conduction threshold does not improve at 250 Hz, 500 Hz and 4 KHz (Ginsberg, et al. 1978).</p>

	<p>In stapedial otosclerosis the SDS is oftea excellent from 88 percent to 100 percent. But if inner ear is involved i.e. in cochlear otosclerosis the SDS varies from 50 percent to 80 percent depending upon the severity of the problem (Prescod, 1978).</p> <p>Average SRT is found to be ground 62.7 dB HTL and average SDS around 81.30 percent (Ginsberg, et al 1978)</p> <p>Tympanogram shows type A or shallow type A (As) (Preacod, 1978).</p> <p>The static compliance ia usually within the range of from 0.20 to 0.40 cc. If it is below normal limit it indicates that the mobility of tympanic membranc is reduced. (Jerger and Jerger, 1981).</p> <p>Acoustic reflex is usually absent in stapedial otosclerosis. But this acoustic reflex is present in the case of cochlear otosclerosis (Ballantyne, 1979).</p>	<p>Audiogram shows a sloping curve that drops in the high frequencies at the rate of approximately 5 dB per octave. (Ginsberg, 1978).</p> <p><u>Vestibular disturbance is reported to be reduced (Pappa, et al, 1984).</u></p> <p>An average SRT in a series of 2405 patients with otosclerosis was found to be 29.4 dB HTL and there was an agreement between the average improvement in SRT and PTA. Average improvement in SRT is found to be 34.84 dB HTL and SDS is 84.29 percent (Ginsberg et al, 1978).</p> <p>SDS becomes better than 90 percent as reported by Shea and Farrrior, 1987.</p>
<p>3. Impedance audiometry</p>		

There is a good deal of variation in depth of the tympanogram. Many patients with otosclerosis will show typanograms with no obvious abnormality. Similarly the static compliance is often well within the normal range. This very "normality" however, is diagnostically significant. It is the "Impedance signature" of ossicular fixation (Jerger, 1980).

In the case of stapedial otosclerosis, loudness discomfort level cannot be obtained; whereas in pure cochlear otosclerosis the loudness discomfort level may be present at 100 to 110 dB with affected frequencies. (Ballantyne, 1979).

In stapedial otosclerosis there is no recruitment. But in the case of unilateral cochlear otosclerosis full recruitment is present. (Ballantyne, 1979)

SISI score is low i.e. around 0 to 10 percent in the case of stapedial otosclerosis. But in the case of pure cochlear otosclerosis the SISI score is high i.e. around 50 to 80 percent as reported by Ballaatyne, 1979.

In atapedial otosclerosis the Jerger type Bekesy tracing is of type I and in cochlear otosclerosis it is of type II (Ballantyne, 1979).

4. Loudness discomfort level test.

Alternate
Dinaural Loudness Balance (ABLB) Test

5. Short Increment Sensitivity Index (SISI) Test

7. Bekesy Audiometry.

8.	<p>Tuning Fork Tests</p> <p>a) Rinne Test</p> <p>Rinne is negative i.e. BC is greater than AC which indicates conductive hearing loss. If Rinne is positive i.e. AC greater than BC then it is indicative of sensorineural hearing loss which is indicative of cochlear involvement.</p> <p>b) Weber Test</p> <p>In conductive loss cases Weber is lateralized towards the poorer ear side. But in sensorineural hearing loss cases it is lateralized towards the better ear side.</p> <p>c) Absolute Bone Conduction (A-B-C) Test</p> <p>A-B-C is good in early stage of otosclerosis and becomes poorer in later stage as reported by Prescod, 1978).</p>
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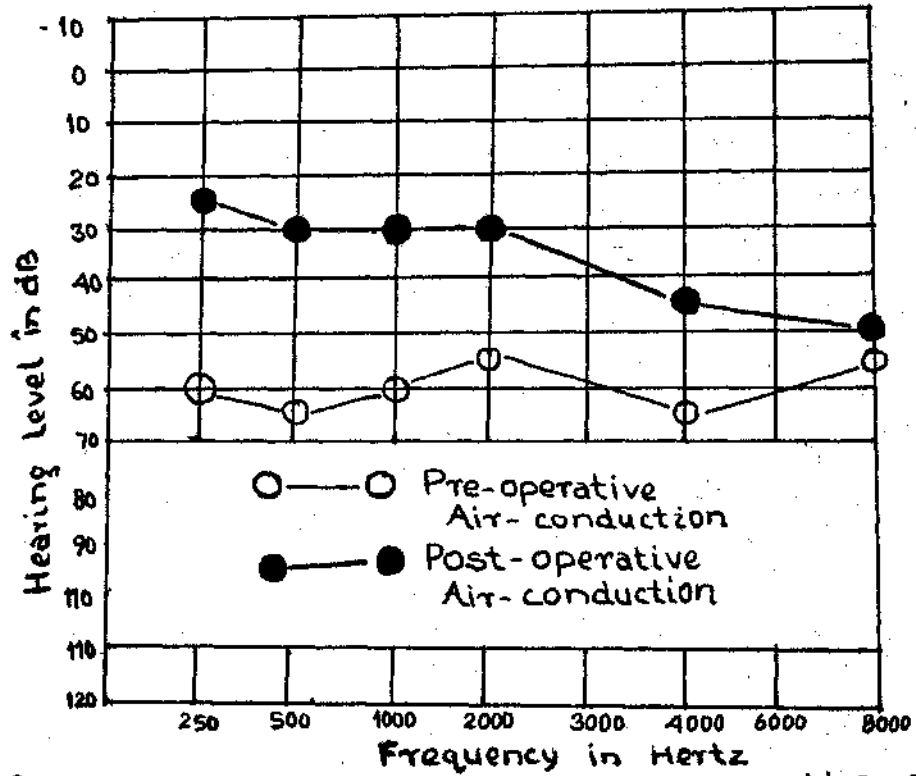


Fig:8; Mean pre-operative and post-operative air conduction thresholds obtained in otosclerotic patients

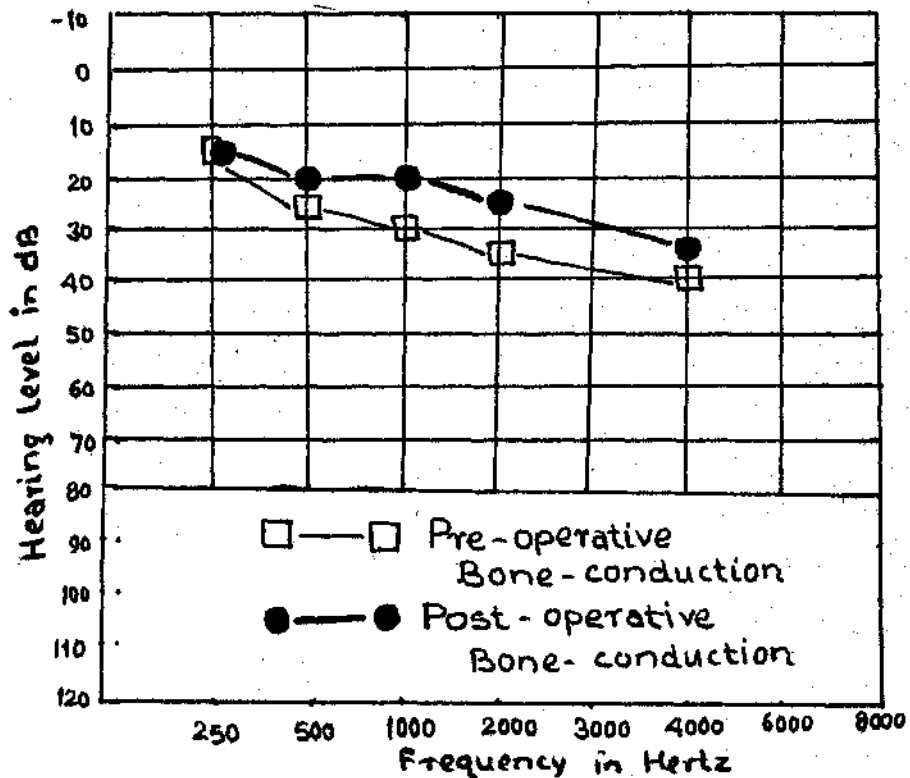


Fig:9; Mean pre-operative and post-operative bone conduction thresholds obtained in otosclerotic patients. [Ginsberg et al, 1978]

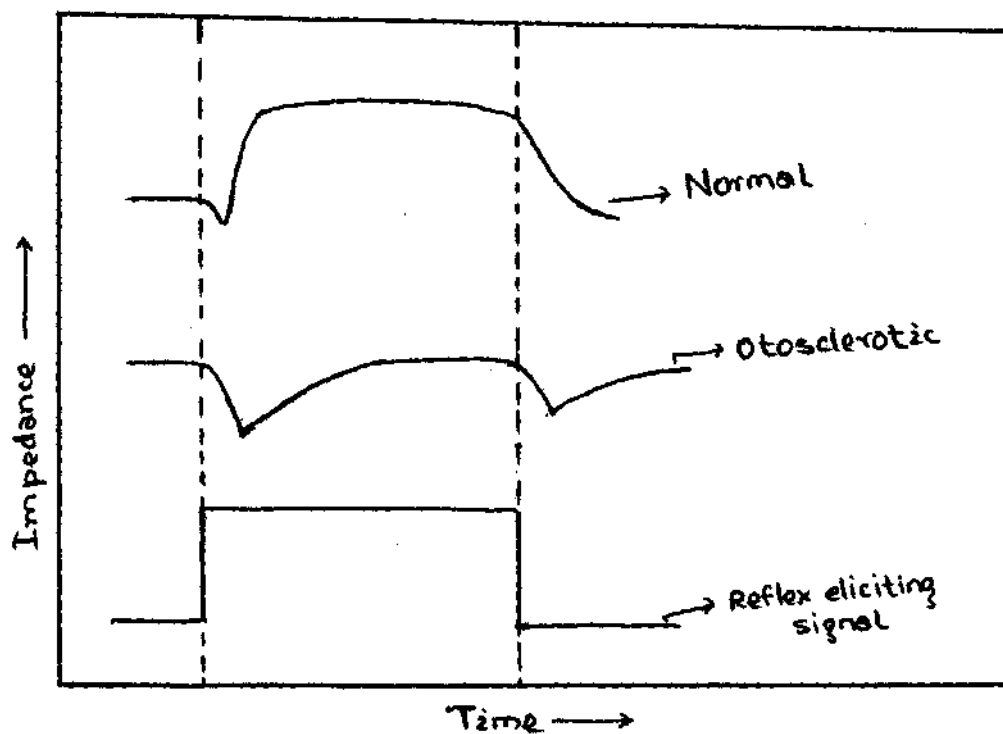


Fig:10: The course of the acoustic reflex in a normal and in an otosclerotic ear. Note that both show a negative deflection at the onset, but that only the otosclerotic reflex shows a negative deflection at the offset (Djupesland, 1963, 1969, Terkildsen, et al.1973).

MENIERE'S DISEASE

Description of the disease:

Description of this disease was first given by Prosper Meniere himself in 1861. In 1972 the American Academy of Ophthalmology and Otolaryngology Committee on Equilibrium defined Meniere's disease as follows: - "Meniere's disease is a disease of the Membranous inner ear characterized by deafness, vertigo, and usually tinnitus, which has its pathologic correlate hydroptic distention of endolymphatic system" (Alford, 1972). This definition is similar to that which was given by Prosper Meniere, the only thing is that this definition included the concept of endolymphatic hydrops as an integral part of the Meniere's disease which was not included in Prosper Meniere's description.

In brief the combination of vertigo, hearing loss and tinnitus characterized by episodic attack of fluctuating severity in hearing loss is called Meniere's disease. This is the most important disease of the inner ear which affects the fluid physiology of the endolymphatic system (Gibb and Smith, 1982).

The incidence and prevalence of Meniere's disease is not known. It varies greatly because of inconsistency in establishing the diagnosis by primary physicians.

The onset of this disease is usually between 49 and 60 years of age. It is rare in children (Stable et al. 1978). It is usually unilateral in 80 - 90 % of cases.

The exact cause of this disease is not known. Histopathological observations have shown that there is an increase in endolymphatic fluid which causes dilation of the scala media of the cochlea and saccule. This increased (abnormal) endolymphatic fluid pressure causes ballooning and rupture or stretching of Reissner's membrane (which is the thinnest membrane of the inner ear, having no connective tissue layer). It has been claimed that the episode of vertigo, hearing loss and tinnitus are caused by this increased accumulation of endolymphatic fluid (Dayal, 1981). Others are of the opinion that the episodes coincide with rupture of Reissner's membrane. When the Reissner's membrane ruptures, it causes mixing of perilymph and endolymph fluid. Because of these pathological findings, this condition is also called as "endolymphatic hydrops" (Dayal, 1981). Due to this condition of mixing, the cochlear microphonics and action potentials of cochlear nerves are upset.

When this disease is in progress, there is degeneration of organ of Corti with loss of hair cell population (Kimura, et al. 1976). According to Shea (1975) there are different types of Meniere's diseases.

1. Lermoyez Syndrome - This is a type of Meniere's disease in which hearing is improved during and immediately after a vertiginous attack.
2. Vestibular Meniere's disease - In this condition vertigo is present without hearing loss. It is also known as vestibular hydrops. and
3. Cochlear Meniere's disease or cochlear oydrops - In this condition auditory symptoms are present without vertiginous episodes.

Signs and symptoms of this disease includes -

- Feeling of intra-aural fullness before attack.
- Sudden onset of vertigo, tinnitus* nausea and vomiting.
- Fluctuation in the hearing ability. Hearing loss and tinnitus may become progressively worse (Haye and Quisthanaen, 1976).
- Severe headache
- Psychological (and emotional) instability.
- Repetition of these acute phases for few months.
- Diplacusis is present i.e. the patient perceives different pitches in the involved ear.
- Intolerance to loud noise i.e. recruitment is present.
- There will not be any history of pain in the ear or discharge from the ear.

This disease is treated medically. If medical treatment is not effective than surgical treatment like endolymphatic shunt operation is performed.

Pre and post operative audiological findings in Meniere's disease.

Sl. No.	Name of the test	Pre-operative audiological findings.	Post-operative audiological findings.
1.	Pure tone audiometry	<p>Radiogram shows an unilateral sensori-neural hearing loss. On frequent reevaluation hearing loss may be fluctuating.</p> <p>In initial atage the audiogram shows a rising curve with loss in low frequency region. In later stage the audiogram shows downward sloping curve with greater loss in high frequency. (Jerger and Jerger, 1981; and Musiek, and Geurkind, 1976).</p> <p>PTA is found to be ground 63 dB as reported by Avenberg, 1978.</p> <p>These findings also dependa upon the stage of disease (Pulec, 1972).</p>	<p>Post operative audiogram shows an improvement in hearing. Pure tone audiometry shows a 25 dB improvement and there is a significant improvement of 10 to 30 dB in HTL at the middle frequencies. (Ballad, Clemis, 1987)</p> <p>62% of the patientas who underwent ahunt operation, there was a stabilized improvement of hearing (Pulec, et al. 1972)</p> <p>50% of the patieata remain the same as far as pure tone hearing as concerned (Pulee, 1973).</p> <p>But the audiological findinga vary depending upon the atage of the diaease (Pulee, 1973).</p>
2.	Speech Auditor metry	<p>Speech discrimination score is disproportionately poor as compared to SRT and PTA. The mean value of SRT is around 62 dB and SDS is 52% (Stahle, 1976 a)</p> <p>But Arenberg (1978) reports of SRT around 79 dB and SDS around 27%.</p>	<p>There is a aignificant hearing gain with good apeech diacrimination score in 64.28% of the cases. (Agarwal.1984)</p> <p>Arenberg, et al (1978) reports of 30.5 dB improvement in SRT and 48% improvement in SDS (Ballad, and Clemis 1987; also reports of improvement in SDS</p>

	<p>Pulec (1973) also reports of improvement in SDS from 54-88%.</p>
<p>PB-PI test</p>	<p>It shows a "roll-over" phenomenon (Jerger, Jerger, 1981, Musiek, and Geurkink, 1976).</p>
<p>3. Impedance audiometry</p>	<p>Tympanogram shows normal type A. Static compliance is normal. Acoustic reflex is present at normal hearing level (Jerger and Jerger, 1981)</p> <p>There is no reflex decay at 500 Hz and 1000 Hz (Jerger, and Jerger, 1981).</p> <p>Acoustic reflex may be present in pathological ears at a reduced sensation level, thus indicating recruitment (olsen, et al. 1975; Musiek, and Geurkink, 1976)</p>
<p>4. ABLB Teat</p>	<p>ABLB test shows partial or complete recruitment for the diseased ear. (Ballantyne, 1979; Jerger, and Jerger, 1974) and Musiek, and Geurkink, 1976).</p>
<p>5. SISI Test</p>	<p>SISI is positive and SISI score ranges from 80% to 100% (Ballantyne, 1979) In Pulec, 1972 reports 80% of the cases have the SISI score of 100%.</p>

6.	<p>Tone decay test</p>	<p>Tone decay is usually negative. But this TDT may show abnormal tone decay with severe cochlear damage or beginning of VIII nerve lesion. (Prescod, 1978, Jerger and Jerger, 1981).</p>	<p>Horowitz, et al (1989) reports of improvement in tinnitus. But Agarwal, (1984) reports that the tinnitus disappears in affected ear in 60% of the cases.</p> <p>Apart from this tinnitus and vertigo is also controlled in 57% of cases. (Kerr, et al 1989)</p> <p>Savary, and Charissous, (1984) in his study on 157 cases of Meniere's disease, found that vertigo was eliminated after surgery.</p>
7.	<p>Bekeesy Audiometry</p>	<p>Bekeesy tracing shows type II (Jerger classification) tracing which is indicative of cochlear or hair cell damage (Ballantyne, 1979)</p> <p>Most commonly type II Bekeesy tracing is observed but type I and type IV may also be seen. (Hedgcock, 1968; Musiek, and Geurkink, 1976)</p>	<p>Bekeesy tracing shows type II i.e. it remains the same after surgery as reported by Pulec, 1973.</p>
8.	<p>Loudness discomfort level (IDL) test</p>	<p>Loudness discomfort level occurs below the normal hearing level which shows the presence of recruitment which is indicative of cochlear pathology (Musiek, and Geurkink, 1976; Ballantyne, 1979).</p>	
9.	<p>Difference limen test</p>	<p>DL test shows lowered DL for intensity (Ballantyne, 1979)</p>	

10. Weber test

Weber is lateralized towards the better ear indicating sensori-neural hearing loss. (Preacod, 1978).

11. Electro-cochleography.

Shows an alteration in the action potential scan and delayed conductivity depending on the type of lesion present (Ballantyne, 1979).

TYMPANOGRAM

Impedance audiometry for the patient with Meniere's disease.

(Please see fig 14 on Page 51

COMPLAINTS

Air pressure in mm H₂ O

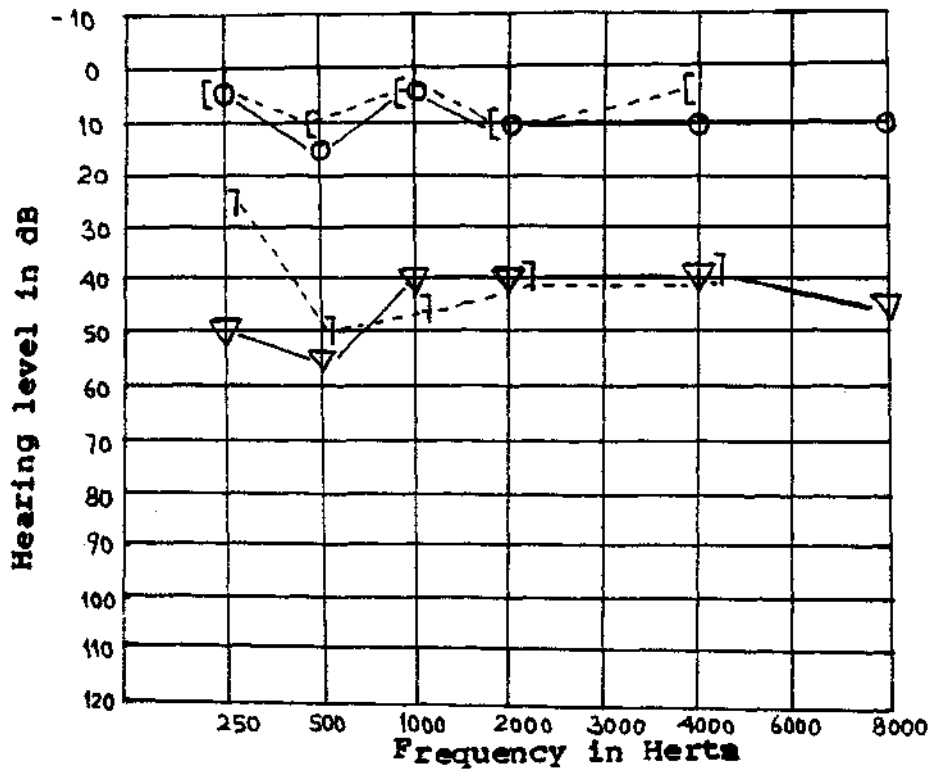


Fig.11 Pre-operative puretone audiogram in Meniere's disease

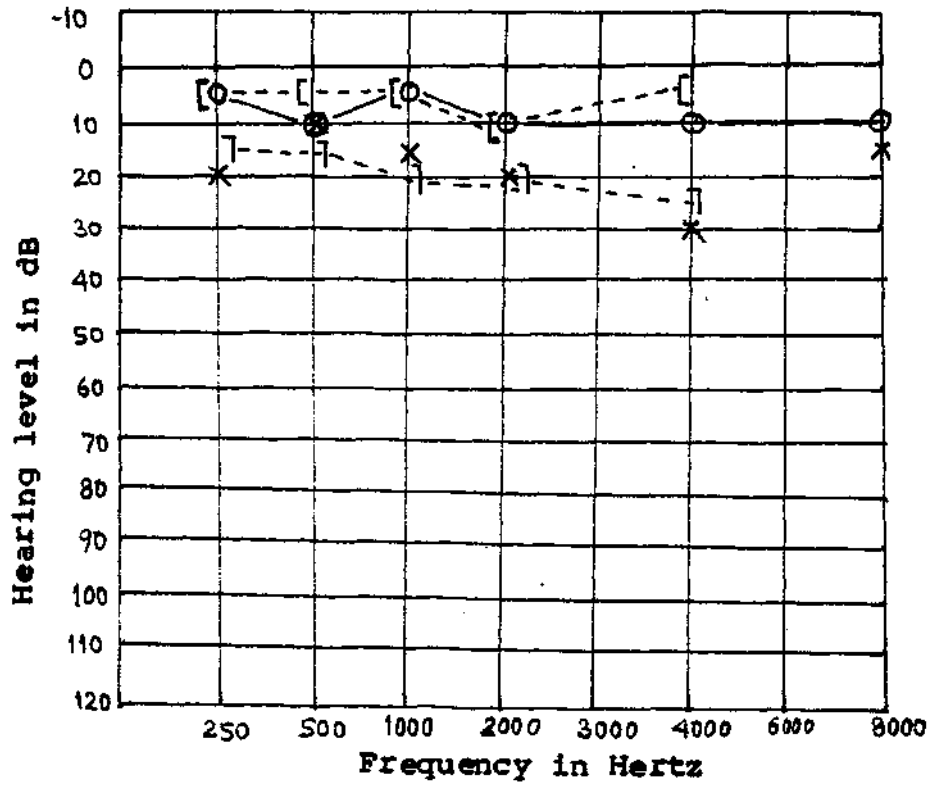


Fig: 12 Post-operative puretone audiogram in Meniere's disease (Meyerhoff et al, 1978)

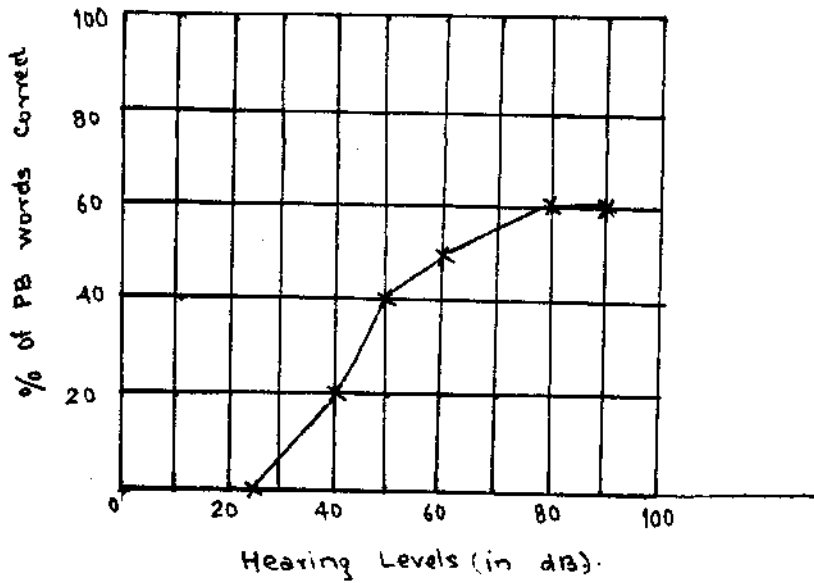


Fig: 13: - Typical PI-PB Function results from an ear with Meniere's disease. (Musiek and Geurkink, 1976)

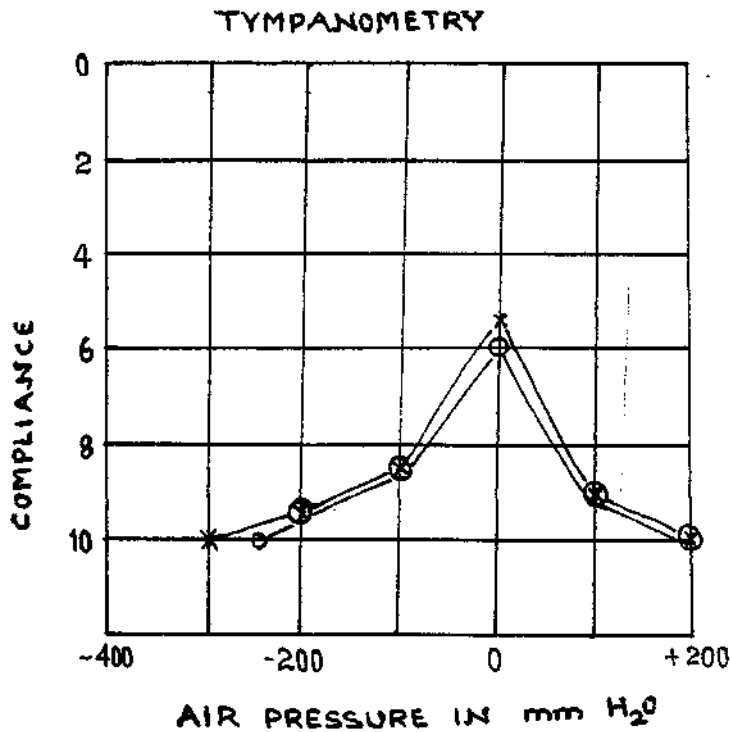


Fig-14, Shows tympanometry obtained in a patient with Menieres disease (Martin, 1975)

ACOUSTIC NEUROMA

Description: Acoustic neuromas or acoustic schwannomas are benign lesions that arise at the glial-schwann cell junction which always lies within the internal auditory meatus (Mafee, 1982 and Cartin, 1984). In other words we can say that the acoustic neuromas are the tumours that arise from the neurilemmal sheath (the sheath of schwann) of the eighth cranial nerve and also known as neurilemmomas which account for 10 percent of all intra-cranial tumours, 80 to 90 percent of all extra-axial cerebellopontine angle masses and more than 50 percent of posterior cranial fossa tumours (Valvassori, 1969; Kieffer, 1974; Clemis, 1984).

Characteristically, this tumor arises within the internal auditory canal from the vestibular portion of the eighth nerve, so it can also be called as vestibular schwannoma. As this tumor increases in size, it may extend medially from the internal auditory meatus into the cerebellopontine angle. This increase in size of the tumor causes compression and it invades the auditory and vestibular nerve trunk. This compression and invasion causes interference with the blood supply to the cochlea, biochemical disturbances of cochlear fluids (Endolymph and perilymph), pressure on adjacent cranial nerves, and/or compression or displacement of adjacent brain stem structure. (Jerger and Jerger, 1981).

These tumours occurs in about 8.7 persons per 1000 and can be classified in two groups:

- (a) Intracanalicular acoustic neuromas which occurs within the internal auditory canal
- (b) Extracanalicular acoustic neuromas which is extending or arising outside the internal auditory canal.

The onset of the symptoms is usually between 30 and 50 years of age. It is more in females than males. The tumour is unilateral in 95 percent of patients. Clinical course and signs and symptoms of this disease vary depending on the site of origin and size of the tumour. It includes -

- a progressive, unilateral sensorineural hearing loss.
- the onset of hearing loss is characteristically insidious in general
- the loss is preceded or accompanied by unilateral tinnitus in more than 75 percent of the cases
- dizziness or unsteadiness may be observed in 65 percent of persons
- many patients report of sensation of fullness within the ear, and
- compression of vestibular nerves may cause vertigo (Eagash, 1978)

The symptoms generally occur in a predictable sequence. With rare exceptions, the first sign of an acoustic schwannoma is hearing loss and tinnitus..

Hearing loss is accompanied by the other symptoms in the following order - " Labyrinthine symptoms, suboccipital discomfort, incoordination and staggering gait, involvement of other cranial nerves, headaches, vomiting, endolymphatic hydrops, and dysarthria, dysphagia, and respiratory difficulties (Bebin, 1979). The three typical symptoms of acoustic neuroma are -

- (i) Unilateral progressive sensori-neural hearing loss
- (ii) Unilateral tinnitus and
- (iii) Intermittent vertigo and/or unsteadiness.

The eighth nerve is the primary site of the disorder. The secondary sites may be the cochlea or the brainstem. (Jerger and Jerger, 1981). The treatment of acoustic schwannoma generally involves surgery. But in some patients the tumour may recur after surgical intervention.

Pre and post operative Audiological Test finding in Acoustic Neuroma cases.

Sl. No.	Name of the tests	Preoperative audiological findings	Post operative audiological findings
1.	Puretone audiometry	<p>Generally unilateral or a symmetrical bilateral sensori-neural hearing loss. (Jerger and Jerger, 1981).</p> <p>Puretone audiogram shows a downward sloping curve showing sensori-neural hearing loss (Glasscock, et al. 1989).</p> <p>Wiegaad and Fickely (1989) report that this unilateral hearing loss is followed by unsteadiness, tinnitus and headache.</p>	<p>Hearing is preserved in about 50 percent of the cases (Gantz, et al. 1986).</p> <p>Bat Glaascock, et al. (1989) reported that the hearing preservation is based on the hearing level in the ipsilateral and contralateral ears, tumour size, location of tumour in the relation to the internal auditory canal, ENG, ABB and the overall prognosis of the patient. He reported that an attempt to conserve hearing is generally not recommended in patients with tumours larger than 1.5 cm.</p>
2.	Speech audiometry	<p>SRT and PTA correlate closely.</p> <ul style="list-style-type: none"> - SDS may often be inappropriately poorer and it is out of proportion to the degree of hearing loss. (Glasscock, et al. 1989). - But according to Jerger, and Jerger (1981), it also depends upon the size and site of the tumour. 	<p>In Glasscock, et al (1987) study post-operative PTA was found to be around 43 dB.</p> <p>Speech discrimination is improved to 48 percent (Glasscock, et al. 1989).</p> <p>In 32 percent of cases PB score of 30 percent or less was found. Around 50 percent of the cases achieved PB scores of 90 percent or better. (Johnson, 1970).</p>

<p>3. Impedance audiometry.</p>	<p>Jerger and Jerger (1981) report the following impedance audiometric findings.</p> <p>Tympanogram shows type-A. Static compliance is normal. Acoustic reflex is reported to be absent or elevated (Glasscock, et al. 1980).</p> <p>Bekesy type Jerger box pattern shows a diagonal pattern.</p> <p>Reflex decay test shows the presence of reflex decay at frequencies below 2KHz which is a strong indicator of VIIIth nerve site.</p>	<p>But in some of the cases SDS changed from good SDS to zero percent. On the other hand in some of the cases the SDS in drastically improved. (Johnson, 1970).</p>
<p>4. Brainstem Evoked Response Audiometry (BSERA)</p>	<p>ABR Shows abnormal findings in 98 percent of the cases. The most common abnormality is an increase in the I and V inter-peak latency in the affected ear. (Telian, et al. 1989).</p> <p>According to Josey (1987) ABR shows changes in latency or morphology or both in the affected side. It is also reported that the peaks I and V are prolonged in affected side.</p>	<p>According to Josey, (1987); ABR findings after surgery are improved.</p> <p>Glasscock, et al (1989) finding shows waves I, II and V to be intact.</p>

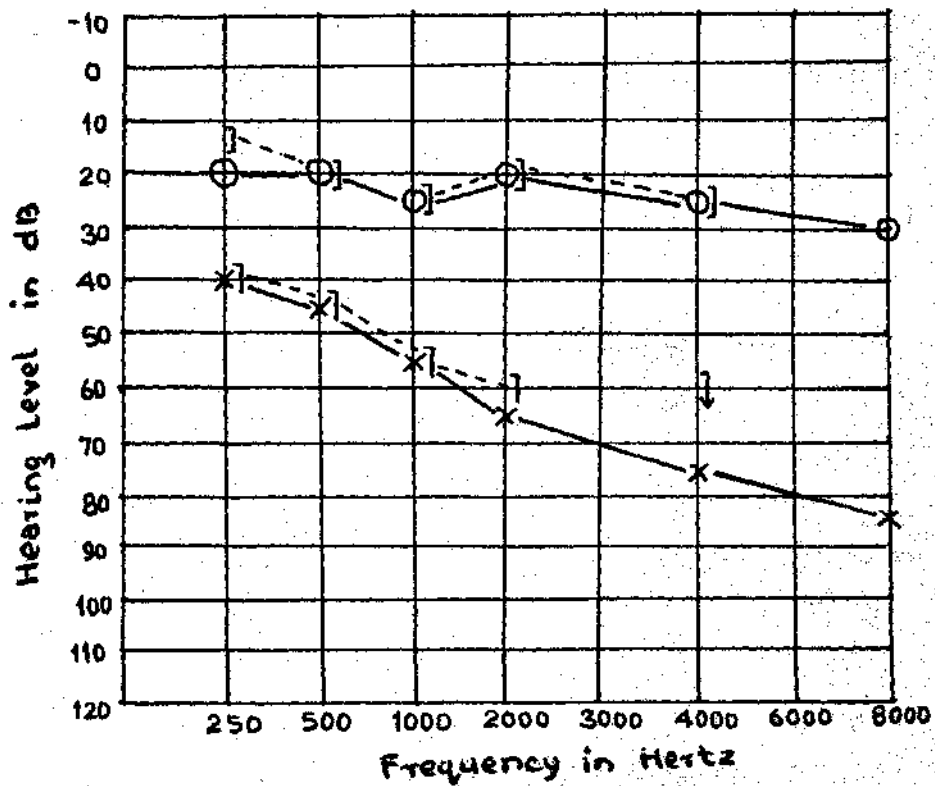
<p>SISI (Short Increment Sensitivity Index) test</p>	<p>SISI shows positive result in cochlear pathology and negative in retrocochlear pathology (Josey, 1967).</p>	
<p>6. Tone Decay Test (TDT)</p>	<p>In most of the cases TDT is positive which is indicative of retrocochlear pathology (Dayal, 1981; and Josey, 1987).</p>	
<p>7. ABLB (Alternate Binaural Loudness Balance) Test</p>	<p>It shows that the recruitment is absent in VIIIth nerve tumours (Josey, 1987).</p>	
<p>8. Bekesy Audiometry</p>	<p>Bekey tracing generally shows Jerger type IIIrd or type IVth which is indicative of retrocochlear pathology (Sander, et al, 1974; Johnson, 1977; and Dayal, 1981)</p>	<p>Bekesy tracing shows Jerger type IVth before surgery is changed to type IIIrd (Johnson, 1970)</p>

Some other findings which are important and confirm the presence of the tumour are as follows:-

1. **Caloric test** - In almost all the cases of cerebellopontine angle tumours, the caloric response is reduced or absent on the affected side (Dayal, 1981).
2. **Temporal bone x-rays** - A stenver's view may show assymetry of internal auditory meatus in the ease of acoustic neuroma. (Engush, 1976).
3. **Petrous pyramid polymography** - This radiographic examination in capable of demonstrating small changes in the size of the internal auditory meatus which is highly suggestive of a tumor in the VIIIth cranial nerve (Engush, 1976, Dayal, 1981).
4. **Pneumocephalography** - The contrast medium in this case is air in the subarachnoid space. This examination is useful in identifying larger tumours and in demonstrating possible displacement of the brainstem due to pressure from a large tumour (Engush, 1976).
5. **Brain scan** - A brain scan of the posterior cranial fossa will demonstrate a lesion of 2 cm. or greater but is of little benefit in identifying smaller tumors (Engush, 1976).
6. Any patient with a progressive sensori-neural hearing loss and persisting unilateral tinnitus should be suspected of having nerve VIIIth tumour. In addition to the presence

of unilateral sensori-neural hearing loss, if the calorie response is reduced or absent on that side, it is likely that the patient has an acoustic neuroma, in that case definitive investigations should be performed (Dayal, 1981).

7. **Computerised Axial Tomographic Scanning (CAT-Scan):** A CAT-Scan is very useful and it will detect the presence of small size of tumours.



Speech Audiometry

	PTA	SRT	SDS
Right Ear	21.66 dB	20 dB	100% at 60 dB
Left Ear	55 dB	55 dB	44% at 95 dB

Fig: 15. Audiogram of a patient with Acoustic neuroma which shows unilateral sensorineural Hearing Loss with poor discrimination of speech (in the Left ear). Hearing in the right ear is within normal limit.

(Dayal, V.S, 1981)

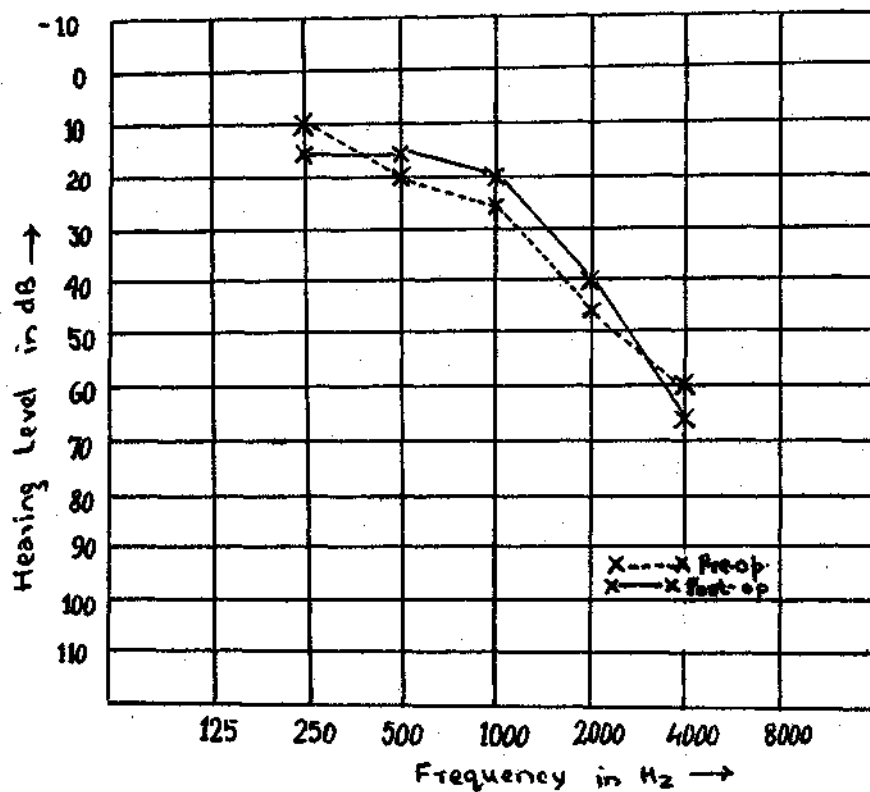


Fig-14 Preoperative and postoperative audiogram for 145 cases with acoustic tumor

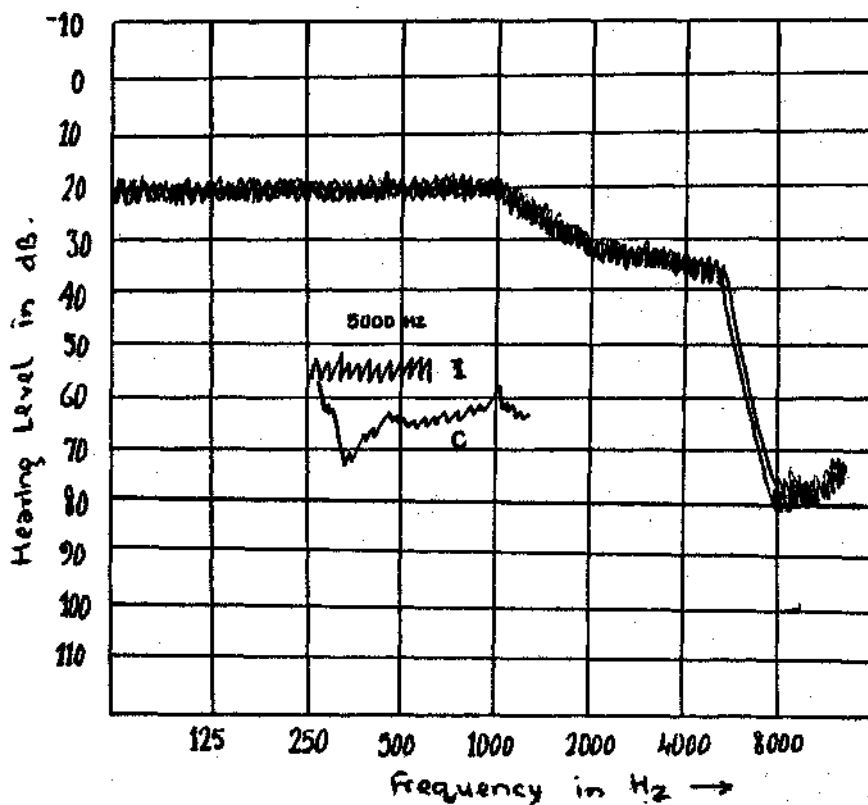


Fig:17 Post-operative Bekesy audiometry for cases with acoustic tumor.

(House, et al 1968)

Speech discrimination in the right ear at 65 dB is 62 percent

Speech discrimination in the left ear at 40 dB is 85 percent

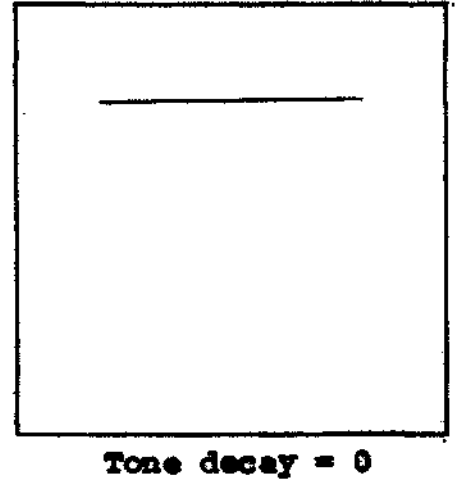
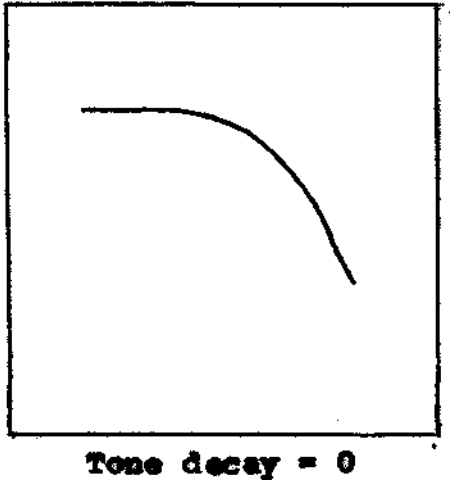


Fig.18 Shows an audiogram with reduced SDS in right ear (i.e. in affected aide).

Calorie test

Fowler's ABLB Test at 4 KHz

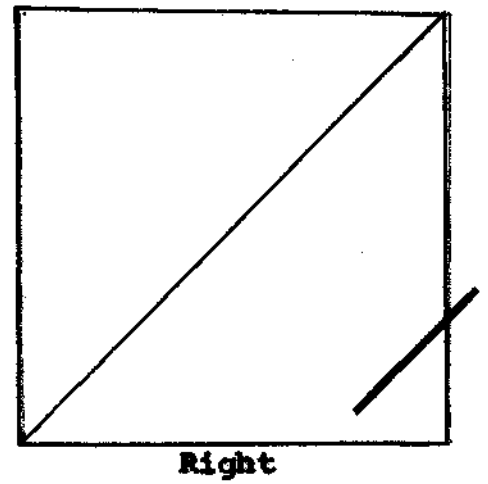
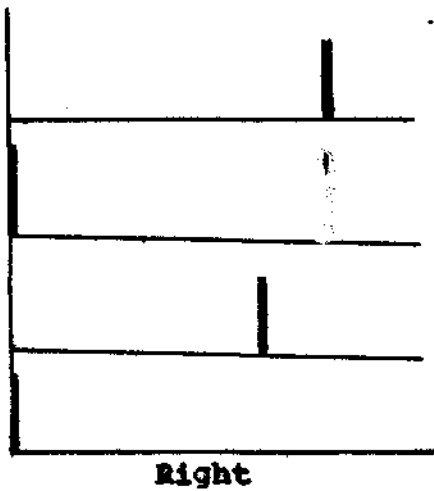


Fig.19 Shows absence of calorie response and absence of recruitment in the right ear

(Ballantyne, J. 1979)

SUMMARY AND CONCLUSION

Though this handbook is entitled: "Pre and Post operative audiological findings in peripheral hearing disorders", it is restricted only to those peripheral hearing disorders which involves surgical treatment, so that we can compare the pre-operative audiological findings with that of post operative with it then gives a clear cut objective information regarding the gain in hearing after surgery. In other words it helps us in better appreciation of surgical output. If we want to see, evaluate and realize the amount of change that has taken place, we have to keep one eye on the past events or past findings and another eye on the present one. And then by looking at these two we evaluate and realize the amount of change both in terms of quality and quantity. So is the ease here in our treatment of hearing disorders.

If we want to know how effective is our surgical treatment program, we have to have a preoperative audiological findings of different audiological tests. Then, after the surgery is performed, a post operative audiological findings of all those tests (performed earlier) at a regular interval should be done. Based on these two findings we can evaluate and realize the amount of improvement which is an objective evaluation.

If the same tests are performed at regular intervals it will give a clear cut information about the long term effect of surgery, It is because in some of the cases the long term effect of surgery is not very good.

It is also significant to note that we have enough informations regarding the preoperative audiological findings but comparatively few publications are available on postoperative audiological evaluations. This could be due to high cost of audiological evaluations which includes all the basic and some special tests. In addition, professionals fully engaged in clinical activities may not find it important worth their while to publish their findings. Another reason for this may be because after surgery we are more concerned about the patients speech discrimination ability and also to monitor his middle ear condition for any possible recurrence or complications. For that purpose at least a routine examination and audiological evaluations of all the different test should repeatedly be done. So, there is a need for the same so that we can have a better and specified identification of the disease, its treatment and research purposes.

Though there are several studies published in which output of post-surgical findings is also reported to be good and appreciable but reports of different audiological tests are not available. So there is a need of reports on a large number of cases

similar to oae by Ginsberg, et al (1978) in which 2045 cases of stapedectomy, and studies by Johnson (1977) in which results of surgery in 500 cases of acoustic neuromas, is reported; so that we can generalize these findings to overall population of the same disease.

BIBLIOGRAPHY

- Adkins, W.Y. and Osguthorpe, J.D. (1981): Management of canal stenosis with a transposition flap. *Laryngoscope*, 91, 1267-1269.
- Agarwal, P.N. (1984): Experience with ESR (endolymphatic sac revascularization) in cases of sensorineural hearing loss. *J.Laryngology and Otology*, 98(2), 139-151.
- Alford, B. et al (1977): Current status of surgical decompressions and drainage procedure upon the endolymphatic system. *Annals of Otology, rhinology, and laryngology*, 86, 683-688.
- Altenau, M.M. et al (1978): Tympanoplasty: Cartilage prostheses - a report of 564 cases. *The Laryngoscope*, 88(6), 895-904.
- Anderson, L. et al (1983): Treatment with sulfur hexafluoride in childrea with serous otitis media. *Archives of Otolaryngology*, 109(6), 358-359.
- Aram, G. and Gerwin, K.s. (1972): Otitis media-proceedings of the national conference callier hearing and speech centers, Dallas. Charcels C Thomas Publisher, U.S.A.
- Arenberg, I.K. et al (1978): Undirectional inner ear valve impact for endolymphatic sac surgery in Meniere's Disease. *Archives of Otolaryngology*, 104(2), 694-704.
- Ballad, W.J. and Clemis, J.D. (1987): Monitoring of the glycerol test with sinusoidal harmonic acceleration in Meniere's disease patients. *Annals of Otorhinolaryngology*, 96, 286-290.
- Ballantyne, J. and Groves, J. (1979): *Scott Brown's Diseases of the Ear, Nose and Throat*. 4th Edn. 2, Ear. Butterworth's and Co.(publishers) Ltd.,
- Ballenger, I.J. (1977): *Diseases of the nose, throat and ear*. 12th Eda. Lea and Febiger, Henry Kimpton Publishers, London.
- Bellucci, R.J. (1981): Congenital aural malformations: diagnosis and treatment. *Otolaryngologic clinic of North America*, 95-124.

- Bergstrom, L., Stewart, J.M., Kenyon, B: (1974): External auditory atresia and the deleted chromosome. *Laryngoscope*, 84, 1905-1917.
- Bicknell, M. and Morgan, N. (1968): A clinical evaluation of the Zwislocki acoustic bridge. *Journal of Laryngology*, 82, 673-691.
- Birch, L. et al. (1986): Hearing improvement after spapedectomy: upto 19 years follow-up period. *Journal of Laryngology and Otology*, 100, 1-7.
- Boies, L.R. (1961): *Fundamentals of otolaryngology: A book of ENT diseases*. W.B.Saunders Company, London.
- Boles, R. et al (1975): Conservative management of Meniere's Disease. *Annals of Otology, Rhinology and Laryngology*, 84, 513-517.
- Coppel, M. and Clani, S. (1979): Audiometric results following endolymphatic sac surgery. *Journal of the American Auditory Society*, 4(5), 165-169.
- Dayal, V.S. (1981) *Clinical otolaryngology*. J.B.Lippincott c Company, Philadelphia, Toronto.
- Djupesland, G. (1963): Middle ear muscle reflexes elicited by acoustic and non-acoustic stimulation. *Acta Otolaryngology, Supplement*. 188, 287-292.
- Djupesland, G. (1969): Use of impedance indicator in diagnosis of middle ear pathology. *Audiology*, 8, 570-578.
- Dela Cruz, A. et al (1985): Congenital atresia of the external auditory canal. *Laryngoscope*, 95, 421-427.
- Dix, M.R. (1968): Loudness recruitment and its measurement with special references to the loudness discomfort level test and its value in diagnosis. *Annals of Otology, Rhinology and Laryngology*, 77, 1131-1151.
- Engush, G.M. (1976): *Otolaryngology - A text book*. Herper and Row Publishers, INC London.
- Gibb, A.G. and Smith, M.W. (1982): *Otology*. Butterworth Scientific Co. Boston.

- Glasscock, et al (1987): Acoustic neuroma surgery. *Laryngoscope*. 97(7), 785-789.
- Glasscock, M.E. et al (1989): Management of bilateral acoustic tumour. *The Laryngoscope*, 99(5), 475-484.
- Goodhill, V. (1979): *Ear diseases, deafness and dizziness*, Herper and Row publishers. Inc. New York.
- Grundfast, K.M. et al (1986): EAC stenosis and partial atresia Without associated anomalies. *Annals of otology, Rhinology and Laryngology*, 95, 505-509.
- Hannley, M. and Jerger, J. (1981): PB Rollover and the acoustic reflex. *Audiology*, 20(3), 251-258.
- Haye, R. and Quisthansses, S. (1976): The natural course of Meniere's disease. *Acta Otolaryngology*, 102, 847-
- Hedgcock, L.D. (1968): Audiometric findings in Meniere's disease, edited by Pulec, J. *The otolaryngologic Clinics of North America*, 489-497.
- Hicks, G.W. et al (1978) III use of plastipore for ossiculat chain reconstruction: An evaluation. *Laryngoscope*, 88 (6), 1024-1033.
- Horowitz, M. et al (1989): Cryosurgical treatment of endolymphatic hydrops. *The Journal of Laryngology and Otology*, 193, 481-484.
- Hough, J. and Stuart, W. (1968): Middle ear injuries in skull trauma. *Laryngoscope*, 78, 899-937.
- House, et al (1968): "Middle cranial Fossa approach to Acoustic tumor surgery" *Archives of Otolaryngology*, 88, 630-638
- Jerger, J. (1970): Clinical experience with impedance audiometry. *Archives of Otolaryngology*, 92, 311-324.
- Jerger, J.F. and Jerger, S. (1974): Radiological comparislon of cochlear and eighth nerve disorders. *Annals of O t o l o g y , Rhinology and Laryngology*, 83, 275.
- Jerger, J.F. and Jerger, S. (1971): Diagnostic significance of PB word functions. *Archives Of Otolaryngology*, 93, 573.

- Jerger, S. and Jerger, J (1981): Auditory disorders - A manual for clinical evaluation. Brown and Company, Boston.
- Jerger, J. and Northern, J.L. (1980): Clinical impedance audiometry (edn.2) American electromedics Corporation, Massachusetts.
- Jerger, J. et al (1974b): Studies in impedance audiometry. III. Middle ear disorders, Archives of Otolaryngology, 99, 165-171.
- Johnson, E.W. (1977): Auditory test results in 500 cases of acoustic neuroma. Archives of Otolaryngology, 103, 152-158.
- Josey, A.F. (1987): Audlological manifestations of tumours of the VIII nerve. Ear and Hearing, 8(4), Supplement, 19S-21S.
- Katz, J: A handbook of clinical audiology. Third edn. William and Wlkins, Baltimore.
- Kenneth, H.Brokler,: Early diagnosis of acoustic neuromas: Maico Audiological Library series, VO1.XIV. Report 3.
- Kenneth, M. Grundfast, Camilon, F. (1986): External auditory canal atresia without associated anomalies. Annals of Otology, Rhinology and Laryngology, 95, 505-509.
- Kerr, A.G. et al (1989): Role and results of cortical/and endo-lymphatic sac surgery in Meniere's disease. Journal of Laryngology, and Otology. 103, 1161-1166. mastoidectomy
- Klein, J. (1979): The changing challenge of otitis media. Hearing aid Journal, 32, 11.
- Lesinski, S.G. (1983): Homograft tympanoplasty in perspective. A long-term clinical histologic study of formalin-fixed tympanic membranes used for the reconstruction of 125 severely damaged middle ears. Laryngoscope, 93, 1-37.
- Lesinski, S.G. (1984): Reconstruction of hearing when malleus is absent: TORP vs hemograft TMMI. Laryngoscope. 94 (II pt 1), 1443-1446.

- Lewis, N. (1976); Otitis media and linguistic incompetence. Archives of Otolaryngology, 102, 387-390.
- Liden, G. (1969a): Tests for stapes fixation. Archives of Otolaryngology, 89, 399-403.
- Liden, G. et al (1970): Tympanometry. Archives of Otolaryngology, 92, 248-257.
- Liden, G. et al (1974): Tympanometry for the diagnosis of Ossicular disruption. Archives of Otolaryngology, 99, 23-29.
- Lippy, W.H. et al (1978): Stapedectomy for otosclerosis with malleus fixation. Archives of Otolaryngology. 104(7), 388-389.
- Hair, I.W.S. and Laukli, E. (1986): Air conduction thresholds after myringoplasty and stapes surgery: A conventional and high frequency audiometric comparison. Annals of Otology, Rhinology, and Laryngology, 95, 327-330.
- McDonald, T.J. (1986): surgical treatment of stenosis of the EAC. Laryngoscope, 830-833.
- Meyerhoff, et al. (1978): Meniere's disease in children. Laryngoscope, 88, 1504-1517
- Moon, C.N. and Hahn, M. (1978): Pneumatic otoscopy and impedance studies in middle ear diagnosis. Laryngoscope, 88, 1439-1448.
- Musiek, and Geurkink, N.A. (1976): Meniere's disease: Symptomological audiological, and remedial aspects. Maico series. Vol.XV, report one,
- Nagar, G.T. (1969): Acoustic neuromas-Pathology and differential diagnosis. Archives of Otolaryngology, 89, 252-279.
- Nagar, G.T. (1969): Histopathology of Otosclerosis. Archives of Otolaryngology, 89, 341-363.
- Naunton, R.F. et al (1970): Acoustic neurinomas with normal internal auditory meatus. Archives of otolaryngology, 91, 437-443.
- Hewby, H.A. and Popelka, G.R. (1985): Audiology (5th Edn). Prentice-Hall, ISC, Englewood cliffs, N.J.

- Ogale, S.B. et al (1986): Styloid process - a new ossicular prosthesis - A pilot study. Indian Journal of Otolaryngology, 38(3), 108.
- Palva, T. (1987): Surgical treatment of chronic middle ear disease. Acta Otolaryngology, 104,279-284.
- Palva, T. (1987): Surgical treatment of chronic middle ear disease, Acta Otolaryngology, 104, 487-494.
- Prescod, S.V. (1978): Audiological handbook of hearing disorders. Van Nostrand Reinhold Company, London.
- Pulec, J.L. (1973): Meniere's disease: Etiology, natural history and results of treatment. Symposium on vertigo, otolaryngologic clinics of North America, 6(1), 1973.
- Pulec, J.B. (1972): Symposium on Meniere's disease I - Meniere's disease: Results of a two and one half years study of etiology, natural history and results of treatment. Reprint from the Laryngoscope, Vol.LXXXII, No.9, 1703-1715.
- Reichert, T.J. and Spector, G.J. (1979): Acute otitis media - A self instructional package from the committee on continuing education in otolaryngology. American Academy of Otolaryngology.
- Savary, P. and Charissoux, G. (1984): Surgical opening of the endolymphatic sac. in Meniere's disease - our experience from 1962-1980. Journal of Otolaryngology, 13(12), 73-75.
- Schechter, G.A. (1969): A review of cholesteatoma pathology. Laryngoscope, 79, 1907-1920.
- Shea J.J. and Emmitt, J.R. (1978): Biocompatible ossicular Implants. Archives of Otolaryngology, 104(4) 191-196.
- Shea, J.J. and Farrior, J.B. (1987): Stapedectomy and round window closure. Laryngoscope, 97 (1), 10-12.
- Smith, M.F.W. and Hop, M.L. (1986): 1984 santa barbara state-of-the-art symposium on otosclerosis. Result, conclusions, and consensus. Annals of Otology, Rhinology and Laryngology, 95, 1-4.
- Saow, J.B. et al. (1979):, Assessment of surgical procedures for Meniere a disease. Laryngoscope, 89 (5 point L) 737-747.

- Statch, B.A. (1987): Acoustic reflex in diagnostic audiology. *Ear and Hearing*, Vol.8,4) Supplement, 395.
- Telian, s.A. et al (1989): Normal auditory brainstem response in patients with acoustic neuroma. *Laryngoscope*, 99(1),10-14.
- Terkilden, K. et al (1973): Acoustic middle ear muscle reflexes in patients with otosclerosis. *Archives Otolaryngol.* 98, 152-155.
- Tos, M. et al (1989): The contralateral ear after translabyrinthine removal of acoustic neuroma: Is there a drill noise-generated hearing loss. *Journal of Laryngology, and Otology*, 103, 845-849
- Wieg, and Fickel, V.D. (1989): Acoustic neuroma - The patients perspective: Subjective assessment of symptoms diagnosis, therapy and outcome in 541 patients. *Laryngoscope*, 99(2), 179-187.
- Zaman, K. (1986): Oosciculoplasty in post myringoplasty cases. *Indian Journal of Otolaryngology*, 38, Conference Issue, 3-4.
- Zemlin, W.R. (1988): *Speech and hearing science, anatomy and Physiology*. Prentice Hall, Englewoodcliffs, New Jersey.1

APPENDIXKEY TO SYMBOLS

	<u>RIGHT</u>	<u>LEFT</u>
AIR CONDUCTION		
UNMASKED	○	×
MASKED	△	▽
NO RESPONSE	⊙	⊗
BONE CONDUCTION		
UNMASKED	[]
MASKED	┌	└
NO RESPONSE	└	┘