DO HEARING AIDS DAMAGE HEARING ? ' A REVIEW

BY

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An Independent Project Work
Submitted on part fulfullment of first year M.Sc, (Speech & Hearing)
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DEDICATED TO

My Parents

Sisters and Brother.

C E R T I F I C A T E

This is to certify that the Independent project entitled "DO HEARING AIDS DAMAGE HEARING? - A REVIEW" is the bonafide work done in part fulfilment of M.Sc., 1st Year (Speech & Hearing) of the student with Register No.

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C E R T I F I C A T E

This is to certify that the Independent project entitled, "DO HEARING AIDS DAMAGE HEARING? - A REVIEW" has been prepared under my supervision and guidance.

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D E C L A R A T I O N

This Independent Project is my own work done under the guidance of Mr. M.N.Vyasamurthy, Lecturer in Audiology, All India Institute of Speech & Hearing, Mysore - 6, and has not been submitted earlier at any University for any other Diploma or Degree.

MYSORE

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A C K N O W L E D G E M E N T

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C O N T E N T S

CHAPTER - I

INTRODUCTION

Hearing aid is an electroacoustic device used for the amplification of sounds. Hearidgaid is a boon to the individual with hearing loss who cannot be helped by either medicine or surgery and who experience difficulty in one or more of the daily activities either vocationally, educationally or socially. This amplification system merely increases the intensity of the sound reaching the ear, and the main purpose of amplification is to utilize the individuals residual hearing to the fullest extent possible.

Colebrander (1978) stated two ways of reducing the experiencing handicap, one by improving the ability of the individual and the other by reducing the listening demands of the environment, reflected by reduced listening effort. So, the amplification for the hearing handicapped is an important rehabilitative measure.

O'Neill and Oyer (1970) have given the basic goals for stimulating residual hearing as follows:

1. Greater understanding of speech by others.

2. More rapid development in the use of language by the child.
3. Better speech.
4. Higher attainment in school.
5. Better social and emotional adjustment through link with other people and world at large.
6. Over all improvement in the development of child.

Corso (1977) has given the purpose of electroacoustic treatment on hearing disorders as

1. To improve the threshold of hearing.
2. To increase the ability to discriminate speech sounds for more effective verbal communication.
3. To generate normal fields of hearing.
4. To provide possibility of directional hearing.

Among the population of hearing impaired, relatively a few are totally deaf, and because the ability to hear even a part of speech signal is an enormous help in speech reception and speech production, the hearing aids are most vital in rehabilitation.

Madell (1978) has mentioned the following goals of amplification for the hearing impaired:

1. To keep the child in contact with the environment.
2. To make the best use of the residual hearing.
3. To enable the child to develop the best possible auditory skills to enable to perceive speech.

Although hearing aid is a boon to the hearing handicapped, its continuous use may have harmful effects on the residual hearing. It is a well established fact that the continuous exposure to intense noise results in hearing impairment.

CHAPTER - II

NOISE AND HEARING

Ever since the age of Industrial revolution, there is increased amount of noise pollution and people are getting more and more annoyed with the noise that it is the best known universal enemy. With this constant persistance of noise, because of its harmful effects, noise is drawing increased attention of professionals. These effects of noise can be physiological or psychological. The physiological effects can be auditory or non auditory in nature. Former one is the one which is concerning the audiologists and those interested in hearing conservation. The most common well known hazard of noise exposure is the "Hearing loss", either temporary or permanent. There are many variables to be considered, the parameters of voice itself and individual factors such as susceptibility, previous exposure, etc. The gradual diminution of hearing acuity associated with noise exposure is referred to as noise induced hearing loss. If this loss is sudden onset after a brief exposure to intense sound, noise trauma is the result. Though, initially NIHL is reversible; the elevated threshold shift because of fatigue, with rest returns to pre-

exposure levels. This threshold shift is known as temporary threshold shift. But, as the permanent loss increases, the temporary threshold shift decreases. With repeated exposure TTS becomes permanent hearing loss.

This type of loss is often accompanied with tinnitus, recruitment and vertigo; at times with 4KHZ dip. Such losses are accompanied by or are as a result of temporary or permanent injuries to structures of the ear. It must be noted that permanent injury to inner ear is not always accompanied by permanent changes in auditory sensitivity i.e, auditory acuity of puretones is neither indicative of injury nor intact auditory system (Bredbery 1968).

Noise induced hearing loss can be conductive, sensorineural or mixed in nature. Conductive component may be as a result of rupture of tympanic membrane, dislocation of ossicles, epithelial cysts in the middle ear damage to squamous cells of middle ear, etc. Sensorineural loss is the result of inner ear changes. The area most vulnerable for high intensity sounds bambardment is the organ of corti. Though there are a lot of

individual variations seen regarding the site and extent of damage, the extent of damage is related to the intensity of sound, site is related to frequency of the sound and the severity is related to exposure times.

With the development of scanning electron microscopy, the structural damages within the cochlea are well known, but little is known about the modification within the organ of corti. It is difficult to state as to where earliest damage occurs but it is quite evident that rather early, sensory hairs on outer hair cells loose stiffness and they have a tendency to wilt and it is unresolved, whether it is reversible or not. If sufficient damage occurs, hairs may fuse and form conglomerations. Some parts of hairs become ingested into the surface of outer hair cell and can be found lying horizontally inside the plasma membrance above cuticular plate. In the inner hair cellsnumber of fused hairs can form a spatular plate protruding from the surface parallel with modification of surface, various grades of deterioration appear with hair cell cytoplasm. In cases of minor damage, structural changes can result in the formation of lysosomes appearing especially in the region below basal body. With repeated exposure, increase in lipofuscin granules occur.

If damage is lethal to the cell, both nucleus and cell cytoplasm begin to disintegrate. Vacuoles or cystic degenerations appear and the cells loose contact with the reticular membrane. The gaps are rapidly closed by the outgrowth of supporting cells, mainly from the Deiters cells, but even pillar cells participate. In less serious injury, outer hair cells are damaged first and regular pattern is broken. Scattered cells or groups ofsensory cells disappear and in more pronounced damage, all the outer hair cells degenerate in a more or less restricted area. Depending on type, or noise exposure, the damage can appear as small spotted regions of hair-cells or as a distinctly localized damage. The inner hair cells are more resistant, but, in severe exposure, large number of inner hair cells may also disappear. This is followed by neural degeneration in particular area.

In sections through organ of corti the fluid spaces are at first preserved. With the increased dis-integration, macrophages appear and fluid spaces fill with the outgrowth from supporting cells. These also

disappear after varying periods of time, nerve endings and nerve fibres and even the ganglion cells in spiral ganglion. The rest form a cuboidal layer on basilar membrane. During this process the tectorial membrane is lifted off from the organ of corti in its damaged area. It looks as though tunnel of corti can remain intact in undamaged regions but disappears in severely damaged regions.

The structural changes were seen in stria vascularis and permanent changes were seen in strial prominence.

These cochlear changes do not develop immediately after exposure but need varying time to develop, partly depending on extent of damage.

CHAPTER - III

NEED FOR THE STUDY

While considering all the advantages of hearing aid one must bear in mind that the person wearing hearing aid is exposed to high intensity sounds, and the above mentioned hazards of intense sounds are likely to be expected in a hearing aid user. There are a lot of controversies regarding this possibility of damage. Moreover the main interest in prescribing a hearing aid is to utilize the intact hearing present in the individual to the maximum extent and not to further deteriorate the individual's hearing level. This shows the necessity for the knowledge about the effects of hearing aid usage on person's hearing, before providing one with it. So, the present project is taken up to review the studies on the effects of hearing aid usage on the person's residual hearing.

With regard to the problem of whether or not a hearing aid usage results in deterioration of hearing, WHO, in 1967, reported that "formerly it was believed that the use of hearing aid might cause hearing to deteriorate,

but there is no evidence to support". Again in 1967, ASHA Conference on hearing Aid Selection Procedure pointed out the need for further research on the possible deleterious effects. Current food and Drug Administration regulations for aids recommend that aids should not provide more than 132dBSPLMPO, and instruments exceeding that should be labeled as providing a potential hazard to residual hearing. On the contrary, in 1973, the Consumer Guide Section of paying through ear answered the question, "can a hearing aid damage hearing?", by stating "yes, there is evidence that by hearing aids which are too powerful, dangerous damage to hearing can be seen."

There are many studies and experiments conducted on this particular topic, ofcourse with different results. The following discussion, includes item under two headings:

1. Studies indicating damage to residual hearing due to the use of hearing aids.
2. Studies indicating no damage to the residual hearing due to the use of hearing aids.

CHAPTER - IV

STUDIES THAT SHOW THAT

HEARING AIDS DC DAMAGE HEARING

Kinney (1953) reviewed records of 8800 children over 15 years period. 445 showed no change in hearing, 16 demonstrated progression of hearing loss. No explanation was given for deterioration, although he did state that hearing loss was more marked in the ear fitted with hearing aid. Acoustic characteristics of aids used or etiologies of hearing loss were not specified.

In 1955, Harlford and Markle presented a single case, an eight year old girl with congenital, symmetrical, sensorineural hearing loss, who had been wearing a hearing aid for three years before her hearing acuity was reevaluated. It was noted that her hearing thresholds were no longer bilaterally symmetrical but that the acuity in the used ear (left) was significantly poorer than in the non used (right) ear. The aid was fitted to the right ear, and after several months, it

became apparent that the left ear had recovered to its prehearing aid use level whereas the right ear demonstrated threshold shifts. The used ear was again reversed with the same phenomenon noted. When the aid was withdrawn from use for a week, the thresholds in both the ears recovered to prehearing aid use level. Harlford and Markle caution against interpreting their report "as evidence supporting the concept that hearing aids may cause damage to hearing".

Moller and Rojskjaer (1960) in their investigation of 390 cases of age range 10 - 78 years, with sensory neural hearing loss who were regular hearing aid users, reported that nine of these people showed a distinct deterioration of hearing in their used ear (MPO of 120dBSPL).

Kinney in 1961 again examined the files of hearing conservation program and selected the subjects according to following criteria:

1. All the subjects having used the hearing aid for atleast one year.
2. The subject of having a record of one ac testing prior to the aid use.

3. All the cases having sensory neural hearing loss.
4. The subjects who had normal or near normal speech.

178 subjects were selected between the age range of 6 to 16 years. Out of 178 subjects, 126 subjects were filed between 1938 and 1958, 52 between 1959 and 1960. The hearing aids prior to 1959 tended to be less powerful than the later. He observed absolute threshold shifts in used and non used ears. 13 subjects out of 126 subjects showed on average shift of 10dB or more in the used ear and not in the non used ear. 4 cases showed shift in the non used ear too, but in the used ear it was of greater magnitude. In the 52 subjects group, out of 39 subjects who were using monaural hearing aid, 19 subjects showed shift of 20dB. In the remaining 13 cases of binaural hearing aid, 9 subjects showed shift of 25dB in both ears. Kinney has recommended

"1. in sensory neural hearing loss, no aid of more than 40dB gain should be used. It is the MPO to be limited, not the gain, to protect the ear from traumatic effects ofintense sound.

2. Binaural amplification in children should be condemned. Again here MPO should be considered".

Kinney did not study the relationship between deterioration of hearing and etiology of deafness in his subjects. He was of the opinion that hearing losses due to hereditary factors were the most resistant to hearing aid trauma. He has concluded that there is a strong relationship between MPO of the aid and the amount of deterioration. He has also stated that in certain cases, the binaural amplification should be condemned. However, he has not specified in which cases the binaural amplification should be condemned.

Sataloff (1961) found a case with decrease in acuity of hearing after the use of hearing aid. This was a seven year old boy who had worn a hearing aid in one ear for four years. He complained his hearing was getting poorer in the used ear and audiometric results confirmed his impression. The hearing aid was removed, and after a short period his hearing acuity recovered to its former level. The sequence was repeated several times. Each time the hearing aid was worn, his loss increased

each time it was removed, his hearing recovered to its former level. The sequence confirmed the impression of trauma due to hearing aid amplification.

Ross and Treux (1965) have presented two cases in which an unexplained threshold shift occured in an ear using a powerful hearing aid and they also describe measures taken to protect the other ear to which use of an aid was switched Case 1. was 13 year old boy who started wearing moderately powered aid in his right ear at the age of five. After five years he was fitted with a powerful body worn instrument with MPO of 139dB. Several years later, he was enrolled for speech therapy and his audiogram at that time indicated bilateral sensory neural hearing loss with better acuity in the right ear than the left. The boy discountinued speech therapy and two years later, his mother called the clinic an3 reported that her son was having increased problems. The audiogram was taken and when comparison was made between the initial and final audiograms, it was observed that the threshold in right ear were 25 to 35dB poorer, whereas the threshold in left ear agreed within 10dB. Otologic findings were negative. He was recommended to wear the aid in the left ear, but the case was unable to comply with this because the case felt that it was painful

to wear the hearing aid in the left ear. This complaint had audiological justification; SRT was 62dB in left ear while, UCL was 82dB. But, without the aid, the boy would be severely limited in his functioning. So, an aid with AVC was recommended to left ear, which was the better ear at that time.

Case 2. 14 years old boy was under treatment from his fifty year for a severe bilateral hearing loss. His audiograms were consistent through the years. He had worn a hearing aid in his left ear since age four. At age 8, he had switched to an instrument of MPO of 139dB. When he was 13 years old, he appeared to have more difficulty; and audiogram was repeated. While previously the two ears were symmetrical, recent audiogram showed left ear thresholds poorer than the right ear. Otologic examination revealed no apparent reason for the threshold shift. As in the previous case, he was unable to function effectively without an aid, yet there was a possibility of his hearing acuity further decreasing with powerful hearing aid. So, aid with AVC was prescribed to the right ear.

Ross and Treux say that the above two cases are not meant to provide unequivocal evidence that hearing aid amplification can produce acoustic trauma and further decrease in a person's residual hearing. As yet there is inefficient evidence to assign this effect to the aid; nevertheless there is no theoretical reason why this cannot occur. So, they recommend that in the clinical practice one must often proceed as if a possible cause were a probably cause and take requisite remedial steps. The person should be able to receive the benefits of amplification with the possibility of further trauma due to hearing aid amplification minimized.

Best controlled and comprehensive investigation of this phenomenon was accomplished by Macrae and Farrant in Australia (1965). They compared changes in hearing acuity aided ear with that of unaided in 87 children with bilateral symmetrical sensory neural hearing loss. Subjects were 87 children whose ages ranged from 6 to 16 years. Ehologies of losses were described as endogenous and exogenous. 34 children wore hearing aids that provided 48 dB SPL average gain and an MPO of 124 dB SPL (moderate

power) 53 children wore hearing aids that provided 68 dB SPL average gain and an MPO of 130 dB SPL (high power). The length of the time the hearing aids were used ranged from 10 months to 10 years. Changes in hearing thresholds were recorded at four octave frequencies beginning with 250 Hz. The mean increase in thresholds in the high power aided ears were significantly greater than mean threshold increases in unaided ears at all the vested frequencies. Use of the moderate power hearing aid resulted in significant between ear threshold differences at 500 and 4000 Hz only. In children using high power hearing aid, it appeared that greater losses did not deteriorate as much as lesser losses. The amount of deterioration appeared to increase as the length of time, that the high powered bearing aids were worn, increased. This relationship was not specified for moderate powered aids. Macrae and Farrant concluded that

1. the use of powerful hearing aids does tend to produce deterioration of hearing in aided ears, and that the greater the power of hearing aid, greater the degree of deterioration.

2. Ears experiencing exogenous and endogenous losses appear to be equally susceptible to hearing deterioration as a result of hearing aid use.

3. hearing also appeared to deteriorate in the unaided ears, and the deterioration appeared less when the initial loss was greater.

So, their findings suggest that there is negative correlation between deterioration in aided ear and average hearing loss in aided ear at the time aid was fitted. There was positive correlation between deterioration in aided ear and total estimated number of hours the aid was used. There was a weak, though signif cant correlation between average deterioration and volume setting with group one users, significant increase in threshold at 500 and 4000 Hz was found. In second group negative correlation between average threshold deterioration and average threshold in the aided ear at the time hearing aids were fitted. No significant correlation fo first group. In any case, they suggest to take an account of possible damage before prescribing an aid.

Ross and Lerman in 1967 conducted a study on hearing aid use and residual hearing. The procedure for subject selection criteria and analysis were similar to

those utilized by Macrae and Farrant. Subjects were selected according to the following criterias.

1. Bilateral symmetrical sensorineural hearing loss.
2. Use of aid in only one par.
3. A reliable audiogram at the time the aid was introduced.

18 subjects met the criteria with age range of 7 to 19 years. The subjects had used their hearing aid from 1 to 5 years. Specific etiological information was unobtainable, records did indicate that each child's loss was of congenital origin. Each child was scheduled for an audiologic evaluation. All were accompanied to the clinic by one or both of the parents. The following information was obtained.

1. Make and model of hearing aid (receive type, internal and external adjustments) the manufacturer's specifications for an estimate of MPO.
2. Average hours per week the child used the aid.
3. The number of weeks he had used the aid.

The difference between initial and final audiograms, for both aided and unaided ears were noted. Then difference between these ditferences for the frequencies 250, 500, 1000, 2000, 4000 and 8000HZ were computed. For example, if at any given frequency the unaided ear showed a difference of 10dB between initial and final audiogram and the aided ear showed a difference of 15dB, then the relative threshold shift was 5dB. Justification for this procedure was explained by Macrae and Farrant. They state "It was hypothesized that the operation of factors (Other than aid use) which affected hearing was randomly distributed (tended to be equally distributed) between the aided and unaided ears of the group". On this hypothesis if the aid use had no effect on hearing, the change in average thresholds of the aided ears would not differ from the change in average thresholds of the unaided ears except within the limits of sampling error.

The results showed the relative shift between the two ears. Positive difference, i.e. the used ear shifting more than the nonused ear are found at 250, 500, 1000, and 2000HZ. Slight negative differences were found

at 4000 and 8000HZ. The average relative shift for frequencies 500, 1000 and 2000 HZ was evaluated by means of a t test for correlated means and found to be significant at 0.05% level of confidence. Perusal of raw data indicates, however, that only nine of these subjects contributed to this result. The remaining nine showed little or no shift or demonstrated a negative shift.

1. The average loss at the same frequencies at the time the hearing aid was first used was 0.64.
2. The hours per weeks the aid was used was 0.42
3. The MPO of the hearing aid was 0.21.
4. The number of weeks the aid was used was 0.02.

A multiple correlation of 0.80 was found when the average relative shift was correlated with the average hearing loss when the aid was first worn and the hours per week it was used. The results did not indicate significant correlation between MPO and the relative shift in hearing acuity. One child in this study was who fitted with a

hearing aid with an output greater than 130dB showed a substantial shift in the used ear relative to nonused ear. It is possible that this nonsignificant relationship between MPO and shift in the hearing acuity. The greatest correlation was between average relative shift and the average hearing loss at the time the aid was fitted. That it was a positive correlation and it indicates that those children with the greatest degree of hearing loss also showed the greatest amount of relative shift. This is an unexpected result as per Ross and Lerman. It would be more understandable if a high positive correlation were found between MPO and the average relative shift since children with greatest degree of loss are usually fitted with aids having higher MPOs. Another possible factor responsible for the relatively high correlation between degree of loss and average shift could be the fact that children with more severe losses would tend to wear their bearing aids a greater number of hours per week. However, correlation between number of hours and relative shift was very low (0.19), thus eliminating this factor as a possible explanation. This finding contradicts Macrae

and Farrant's study in which they found negative co-relation between degree of hearing loss and relative shift. The evidence suggests that shifts in hearing acuity due to hearing aid trauma are indeed a real possibility. These shifts are by no means inevitable, nor they are uniform when they do occur. The likelihood is that they are related to high MPO and number of hours of aid usage. Any shifts due to hearing aid trauma must be separated from effects reported by Barr and Wedenberg, the progressively associated with etiology and a moderate degree of loss. Ross and Lerman suggest that if trauma occurs, one should not conclude that hearing aid amplification perse is contraindicated for children who need amplification in order to improve their communicative functioning. Rather, aids with reduced MPOs should be recommended and children should be scheduled for audiological followups and be closely supervised in the use of their aids. Thus children can continue to receive the benefits of amplification, with assurance that if shifts do occur, they will be noted immediately and all possible remedial measures will be taken.

In his pilot study, Macrae (1968) has found substantial amounts of temporary threshold shift in aided ears of children with sensory neural deafness after the use of powerful hearing aids. 4 Children from school for the deaf were selected. All had sensory neural deafness not exceeding 85dB hearing loss at any frequency tested in the aided ear. The children's hearing was tested twice at about 3 pm on a Friday afternoon and they were then deprived of the use of their hearing aids for the weekend. At 9.30 am on Monday their hearing was tested and their aids were returned to them for normal use in classroom and to play. At about 11 am and 1 pm their hearing was tested again, then their aids were removed for the afternoon. Subsequently at 2 pm and 3 pm their hearing was tested again. Testing was done in the "reverse" direction from 4000 to 500 HZ and took 110 Sec. The testing was done in those with hearing aids worn, after 30 sec of removal of the aids. Results clearly showed that hearing aid use is causing substantial amounts of TTS ever a wide range of frequencies. Macrae has put forward two hypothesis based on the results:

> "1. In three of these children, recovery from TTS seems to be at a slower rate than that obtained for similar degrees

of TTS in normal hearing subjects. The rates obtained for these children appear to correspond more closely to the slow rates, reported by ward (1960) found in recovery from high values of TTS.

2. Although the effect of aid use is spread over all the frequencies investigated, there are possibly two main areas of effect: one centering around 2700HZ and another at about 800HZ. This is suggested more clearly by an early stage in development of TTS in one subject".

In his subsequent article Macrae (1968) has given the rate of recovery in these children. In three of the four children the rate of recovery from TTS appeared to be slower than that found for similar degrees of TTS in people with normal hearing. The criteria used were as in the earlier study and the hearing aids used were also the same ones as before. The children's hearing was tested twice at about 3 pm on Friday afternoon, and they were then deprived of the use of their hearing aids for the week end. At about 9 pm on Monday, their hearing was tested, aids were returned for normal use. At 1 pm aids were removed and 2 minutes after removal, their hearing was tested. Subsequent testing was carried

out at hourly intervals from the time of removal of hearing aid for four hours. All the tests were done with Bekesy audiometer in a "reverse" direction from 4000 to 500HZ, and the time required for testing was 110 sec-. Pulsed tones were used for testing. Once again improvement in the children's thresholds was found to have occured between 3 pm on Friday and 9 am on Monday, substantial amounts of TTS over a wide range of frequencies were found to have occured between 9 am and 1 pm. on Monday. For such child, the course of recovery from 1 pm to 5 pm was measured at the two frequencies most affected with TTS. Children were recovering from TTS at a rate of about 3 - 4 dB/(logt) during the range of time investigated. Again this is very close to the rate of 4-5dB/(logt) found for recovery from high values of TTS in normal hearing subjects. So, his conclusion were:

1. The use of powerful aids resulted in TTS.
2. Recovery of TTS was slow, after the aids were removed, rate of recovery followed that of normals experiencing high degree of TTS
3. Greatest shifts were between 800 - 2700HZ. The limitation of this study was that, the age of the children and the etiology of loss were not specified.

Macrae (1968) examined the amount of aided ear threshold shift in 134 children whose ages ranged from 5 to 18 years. The average hearing level from 500 to 4000HZ was examined for any change that might have resulted from the use of amplification. The MPO of aids that these children wore ranged from 115 to 130dBSPL. The degree of hearing deterioration was also examined relative to exogenous and endogenous hearing loss and specific etiology of the loss. Macrae concluded that observed changes in the average hearing level of aided ears were the same as those of unaided ears when the aids produced less than 119dBSPL MPO. Greater changes were noted in the aided ears tended to be less when the hearing losses were greater. This finding was inconsistent with Ross and Lerman's report, but consistent with Macrae an3 Farrants. The mean change in average hearing level attributed to hearing aid use appeared the same for exogenous losses. In unaided ears exogenous losses deteriorated more than endogenous. In most cases, hearing deterioration not attributable to hearing aid use appeared to be bilateral and of equal degree in both the ears.

Roberts (1970) has given a case report of permanent perceptive acoustic trauma following the provision

of hearing aid. He states that the study of possible adverse effects of amplification on hearing of children appears to be hampered by two main difficulties. Firstly, in children, the diagnosis of deafness is made and amplification is provided before an accurate audiogram can be obtained; secondly, in some cases there may well be a spontaneous deterioration of hearing. It therefore follows that this problem can only be studied in cases where reliable audiograms are available before amplification is begun and where amplification has been provided in one ear only, so that the unaided ear can be used as a control. Between 1965 to 1968, Roberts has examined 278 children with perceptive deafness at a regional audiology centre. All the children were reviewed approximately three times a year and allwore hearing aids. During this period a deterioration of hearing was recorded in 16, of whom 6 had siblings with perceptive deafness, and a further three gave a history of perceptive deafness in a close member of the family. The high prevalence of positive family history of deafness and absence of any potential audiological factors in obstetric, neonatal and past medical histories of these children strongly suggested that the observed deterioration of hearing was due to heriditary factors. These findings support Burr

and Wedenberg's (1965) view. The baseline preamplification puretone audiogram was not available for 13 of these children since the deafness had been diagnised before 3 - 4 years of age. Serial audiometry in the remaining three children showed evidence of deterioration of hearing in the aided ear in all the three cases. Roberts has reported one of these cases. John's language development during infancy and childhood was considered normal and at three years he spoke using 3 - 4 word sentences, but with defective pronounciation. No history of deafness in the family; and John's obstetric, neonatal and past medical history were normal, but his parents had begun to suspect he had difficulty in hearing. At five years of age, audiometric evaluation revealed a gently slooping high frequency perceptive deafness. Ear level amplification (right) was prescribed after confirming the diagonosis, in February. An audiogram in November showed severe deterioration of hearing in the right ear, most marked at 4000HZ, the classical dip of acoustic trauma. In December the aid was discontinued but follow up audiometry in January (1969) showed no improvement. The acoustic trauma appeared to be permanent since no recovery was seen in one year. There was only a 10dB

deterioration of hearing in the control ear (left) and this occured over the whole range of frequencies (unlike 50dB high frequency deterioration in the aided ear). Later it was found that John's sister (younger) also had perceptive deafness and over 44 months her hearing showed a 5dB deterioration throughout the frequency range in the absence of amplification. This finding confirms the diagnosis of progressive hereditary perceptive deafness. So, the evidence suggests that the use of amplification in children with progressive hereditary perceptive deafness may cause acoustic trauma and thus accelerate the hearing deterioration in the amplified ear. Fortunately this condition is uncommon (10% of all perceptive loss children) but, nevertheless it is essential to identify this type of loss before deciding to provide amplification. Roberts also says that presence of any or some of the following features may indicate that progressive hereditary perceptive deafness is present: a family history of perceptive deafness; the discovery in childhood, of a gently slooping 4O-5OdB perceptive hearing loss; a history of a reasonably normal speech and language development in early childhood (suggesting that hearing loss was either normal or minimally affected during this period) and finally a history of record of deterioration of hearing

over the previous 2 or 3 years.

Eastern and Braulin (1970) presented a case study in which they could create deterioration of hearing through the use of aid. Their patient was ten year old girl with bilateral moderate sensorineural hearing loss. She wore a body type of aid satisfactorily in one ear for 14 months and then began to complain that the aid was not helping as much as it did previously. Audiometric evaluation showed marked worsening of hearing in the aided ear and no change in the hearing of unaided ear. Subsequent hearing aid evaluation was done and she was advised not to use her present aid. Re-evaluation of her hearing seven weeks later showed 20 to 30dB improvement in the previously aided ear. They were able to show temporary deterioration in the aided ear, regardless of which ear wore the aid. The aid in question had an average gain of 34dB and average saturation sound pressure level of 120dB. They recommended a less powerful aid of 30dB gain and 100dB SPL. When the less powerful aid was fitted to the patient and worn alternatively between ears, evaluations verified that temporary deterioration of hearing was no longer present. Their

conclusion included that all children, and their fitted hearing aids be carefully reevaluated shortly after initiating hearing aid use. They also suggest that the clinicians should have some means of verifying the electroacoustic characteristics of each hearing aid.

Danaher and Picklt (1972) noted in subjects with profound loss that their most comfortable hearing level may actually be 125dB SPL with a loudness discomfort level of 128dB SPL. Many patients with profound hearing loss have no loudness discomfort at any level, while other patients with seemingly similar hearing loss, have loudness tolerance problems so severe that they cannot tolerate any type of amplification.

Laquillon reported 7.5% of 80 children with perceptive deafness showed an increase in deafness which could be attributed to aid usage.

Ishizawa and Nishiyama (1974) reported a six year old congenitally deaf child with bilateral and symmetrical perceptive deafness. The left ear was fitted with the aid. 40 days later following initial use, they found deterioration in hearing. Trauma was suspected

and aid was removed and changed to the right ear from the left ear. Eight months later, no recovery was seen in the left ear and no threshold shift in the right ear. It points out that several conditions such as susceptible age of hearing shift, endogenous deafness, sudden amplification increase and insufficient use of ARC gave countenance to diagnosis.

Biesalki and stange (1975) have specified the risk of providing aids too early in childhood. Age equipped therapy in the first year of age entails some difficulty in anatomical, physiological, central nervous and nursing aspects. There are additional therapeutic risks from maturity problems of the auditory system and the diagnostical impediments within first 6-8 months caution against providing aid before 9-12th month with-our thorough diagnosis. Measurement of central potential in 21 children and 11 adults and studies with progressive hearing disturbance confirmed the suspecion and proved risks of too early and too intensive amplification.

Jerger J.F. in 1975 reported a case in which apparently permanent damage in a child was related to the use of a powerful hearing. A nine year old girl with

severe bilateral sensorineural deafness that was caused by rubella had been wearing a powerful hearing aid in her left ear since the age of three years, three months. The MPO was 135dB. An audiogram made on February 7th, 1974, showed an essentially bilaterally symmetrical loss. A year later (January 30 1975) thresholds in the aided left ear had deteriorated substantially, whereas the unaided ear had remained unchanged Impedance audiometry indicated normal middle ear function on both sides. Because of the child's increasing communication difficulties, the parents expressed anxiety lest the reduced benefit from the hearing aid should influence scholastic achievement and long term habilitation goals. The child was advised immediate reduction of the aid's MPO from 135dB to 125dB without altering the gain curve. At the same time the parents were advised systematic alteration of the aid from ear to insure that neither ear accepted over-stimulation for long periods of time and to initiate auditory training in the previously unexploited right ear.

Itakura et al. (1978) observed some children wearing aids showing aggravation of hearing loss. Factors reported by Itakura et al. for hearing deterioration

were:

1. Resulted from gain even less than 4OdB.
2. Happened soon after hearing aids are changed.
3. Occured not only in ears with the aid, but also in the opposite ears. When aggravation occured, discontinuation of hearing aids was not found to be of much help.

Heffernan and Simmons in 1979 have questioned the possible relationships between hearing aid use and temporary increase is sensory neural loss in children. They have given case illustrations of two cases with temporary increases in sensorineural hearing loss following hearing aid use and demonstrate the need for a specific appointment schedule with children following hearing aid recommendation, both to assist during the adjustment periods with new hearing aids, and also to monitor hearing threshold levels in order to assure that no decrease on hearing acuity occurs. In each case, initial hearing evaluations demonstrated bilaterally symmetrical sensory-neural hearing loss, probably congenital in origin. Only one ear was initially amplified but hearing thresholds were monitored in both ears to determine the stability of

threshold response in the unamplified ear. In addition, both children were examined by an otologist to rule out the presence of middle ear disorders.

Case 1 (HT) was evaluated at the age five years four months. Otologic and audiologic assessment was done to rule out hearing loss as a contributing factor to his language delay and articulation aquisition problems. No middle ear pathology was evidenced and results of initial hearing evaluation using play audiometry, revealed a symmetrical bilateral hearing loss, of moderate degree. It was sensoryneural hearing loss of congenital origin. Right ear was, based on subjective clinical impressions, selected for initial trial with amplification. Since hearing thresholds were essentially equal bilaterally, goal was to fit binaural amplification. The body type hearing aid had HAIC gain of 45dB and maximum power output of 125dB. Speech test suggested that it provided adequate gain for his communication needs, after a follow up i.e, at seventh month of hearing aid fitting, his air conduction and bone conduction threshold in the unaided ear was stable. In contrast, both air and bone conducted thresholds in the hearing aid ear decreased in acuity significantly. Air conduction thresholds dropped from

5 to 25dB and there was no response to bone conducted stimuli at any frequency. Actual MPO showed a peak of 135dB. There were no ENT problems associated to explain the threshold shift. To determine if the loss was temporary or permanent,

1. The case was asked not to wear hearing aid for 14 days.
2. No auditory training instruments were used in the classrooms.
3. Repeating puretone testing at the end of two week period.

The results indicated that hearing in the aided ear returned to initial level demonstrating a reversible, thus temporary hearing loss. So, he was asked to wear an aid with HAIC gain of 32dB and MPO of 115dB, which showed no threshold shift in followup studies.

Case 2. (MF) was initially evaluated at 4 year 4 months, otoscopy revealed no middle ear pathology. The first hearing test depicted a bilateral hearing loss, mild to moderate in degree. Diagnosis was sensory neural hearing loss of congenital origin. A body type aid with

HAIC gain 45dB and MPO of 125dB was fitted to his left ear. After one year, the hearing thresholds in the ear without amplification had remained stable. In contrast, aided ear had decreased significantly in the auditory acuity (shift ranging from 20dB to 35dB). It was necessary to determine if the threshold shift was temporary or permanent.

1. Not to wear hearing aid for 14 days.
2. NO auditory training instruments were to be used in the classroom or during speech therapy.
3. Repeating audiometry after two weeks.

The tnresholds obtained two weeks later showed that air conduction thresholds in the aided ear had improved significantly to within 10dB of original thresholds. A postaural instrument of MPO 108dB and HAIC gain of 33 dB was recommended to him, which showed no deterioration of hearing during followups.

So, the data showed relationship between hearing aid amplification use and increased SN hearing loss. This increased loss was temporary in nature and its reversibility depended upon the discontinuing of hearing

aid use. In this study, the actual causative factor for the increase in hearing loss could not be pinpointed since variables such as exposure duration and volume setting were not controlled. However when hearing aids of different MPO were used, hearing loss did not increase. Therefore the threshold shift appear to be related to MPO of the hearing aid, a finding consistent with others. Heffernan and Simmons suggested MPO to be limited to 132dB in severe losses and in mild and/or moderate cases, 120dB or less. The frequencies to show largest shifts were 1000 and 2000HZ. Therefore whatever factor or factors combine to cause threshold shift, the pattern does not resemble that of prolonged noise exposure with a 4000 HZ shift. They also recommend that since hearing aid use causes increase in the hearing loss, the initial introduction of amplification should be always monaural. If subsequent testing for three months reveals no change in hearing, then the recommendations can be made for binaural amplification. One cannot assume that because hearing loss is symmetrical, the ear responds in the same manner to the amplification. So, whenever possible and applicable, binaural amplification must be provided with caution. In accordance with other authors,

they suggest routine followups

1. Check of performance with new hearing aid within 30 days of purchase.
2. Electroacoustic analysis of new hearing aid within 30 days of purchase.
3. Monthly appointments thereafter to monitor hearing thresholds until hearing levels stabilized for atleast 3 months.
4. Reevaluation atleast three months for next year.
5. Annual otologic and audiologic reevaluations.

For further investigation of TTS in hearing aid user using larger population, and more controls the authors give the following procedure.

1. If a decrease in hearing threshold levels is observed following hearing aid use, auditory "rest" is indicated, with total cessation of hearing aid use in the affect ed ear.
2. Auditory stability to be monitored until stability is noted.
3. The same aid must be tried again in the same ear and auditory thresholds should be monitored.
4. If again decrease is observed, a substantial relationship can be inferred between hearing aid use, duration of daily exposure and intensity level of amplification should be specified and controlled.

Kittel and Axmann (1981) have quoted Biesalski's study (1977) in which he has mentioned hearing deterioration attributable to the use of hearing aid. He examined audiograms of hearing handicapped children during the period of 1967-1975 and found that roughly 35% of children and progressive hearing loss between 20dB and 50dB which fell almost exclusively within the frequency range from 1000 to 6000HZ. This alarming observation raised the following questions in Kittle and Axmann,

"1. Is there a similar progressive nature of hearing disturbances among the pupils of school for the deaf and hard of hearing who are under medical care?

2. Is such a progression most likely attributable to the use of hearing aids or is rather due to an inexorable fate, despite the use of aids?

3. If changes in the individual dynamic ranges are likely to be caused by high electronic amplification, which is definetely required in case of residual hearing to obtain auditory efficiency, is it then to refrain from using super power instruments to restrict the values temporarily or to employ them only when speech is to be transmitted or do the

sole auditory impressions required for development of speech rhythm justify the use of high sound levels?"

To answer these questions, they evaluated records of residual hearing in 6, 7, 8 and 9 classes of school for the deaf in Nuremberg. Evaluation covered 55 children, 30 girls and 25 boys, and they were selected because their hearing thresholds had been determined accurately and the results were repeatedly checked before they had been fitted with hearing aids. Moreover, these subjects were also using electroacoustic hearing instruments daily with relatively high outputs over several years. Since, children had limited speech at first when tested, speech audiograms were not available. Therefore, only the old purchase audiograms were compared with new audiograms and a measured value tolerance of upto 20dB was allowed. 11 of the children revealed deterioration at one frequency only. Since no deterioration was evident at all other frequencies and since in all instances no deterioration was found in other aided ear, these 11 cases were not analysed further. Changes in individual dynamic ranges with a hearing loss increased from 15 to 50dB in 7 children (13%). The etiology was acquired loss

in 5 of these cases and hereditary in two of them. Heriditary factors were assumed to exist if other members of family suffered from such disorders, with the exception of presbycusis. Neither ENT nor impedence tests which were carried out furnished in indication that any of these 7 children suffered from a middle ear disease at the time of examination. The deterioration of hearing threshold in children K, K3 and K5 was within the frequency dependent amplification range of aids. In K2 and KA there was only a partial overlap between these ranges and threshold shift. The deterioration of K6 was outside the amplification range. The child suffered from scarlet fever with otitis media at the time of his hearing threshold deteriorated. Further more, his brother was also hearing handicapped. The hearing of child K7 deteriorated abruptly in the course of an influenza infection. Consequently the hearing aid be looked upon as the exclusively cause of hearing deterioration is KA and K7. As far as K2 and K4 are concerned, aid could not be regarded as exclusively responsible either, since, also frequencies outside the range of influence of hearing aids used were involved. As the mother of the child K3 also suffered from a severe hearing impairement, a

hereditary progressive nature of the disease could not be excluded, irrespective of burden imposed on the user by hearing aid. So, only in K1 and K5 out of 7 children, hearing aid produced pressures could be primarily seen as the cause of threshold deterioration. In Kl, hearing deterioration was observed only in one ear even though the same aid was worn in both ears. Consequently, only in KS, hearing deterioration was most likely due to the use of aid. Even in 7 children, with clear threshold deterioration almost invariably more likely causes were found atleast contributory to progressive nature of deterioration of hearing. Although the children examined furnished no conclusive evidence of instrument caused threshold deterioration, one would nevertheless draw attention to the possibility of hearing induced damage in case of high or relatively excessive output levels, especially when deterioration factors, independent of aid, are present. Kittle and Armann suggest to consider the possibility when deterioration develops incombination with intercurrent otic disease or in case of increased vulnerability. So, in the light of this investigation, it can be assumed that under roughly normal starting conditions, an impaired ear is hardly likely to

be additionally damaged by a correctly fitted hearing aid and regular setting checks.

Jassal (1982) in his article stresses the urgency of immediate action in seeking medical intervention and demonstrate the role of audiologist and hearing aid dispenser in the management of sudden threshold shift in a hearing aid user. Sudden deafness was defined as deafness that is rapid in onset, occuring instantaneously, within few hours or days, possibly accompanied by Tinnitus and Vertigo. Sudden threshold shift was defined as sudden decrease in hearing sensitivity over a preexisting hearing impairment. There are a number of causes for sudden hearing loss, such as intravascular occlusions, ototoxicity, allergy, systemic diseases tumours, unknown causes, etc. The case reported by the Jassal was 72 year old female with history of hearing loss gradual over a period of 10 years. She began wearing hearing aid monaurally earlier and later on amplification was binaural. One day she had stuffy, funny feeling in left ear and upon wearing hearing aid,she was unable to hear clearly. There was clear cut threshold shift. Lateron it was found that the cause was high

blood sugar level and sugar free diet treatment improved her thresholds to preexisting level. In such a case, she was advised against the use of aid in the involved ear during the recovery process. Because of her reduced sensitivity stronger aid could not be recommended because of potentially detrimental effect that would occur in the event, the patients hearing sensitivity improved. Any delay in treatment would have caused permanent, irreversible damage. It is of utmost importance to determine type of hearing loss, employing audiologic site of lesion tests, X-rays etc.

Hawkins (1982) has presented a well documented case of overamplification in which the conductive, retrocochlear and other possible factors of cochlear problems were ruledout and in which the hearing aid was set below the user's loudness discomfort levels. The client had come with the history of numerous incidents of congenital familial hearing loss, with grand-mothers, brothers, sister and uncles. The client's loss was since childhood and she wore the hearing aid from 1969, only on her right ear, with a MPO of 131dBSPL. The hearing aid was worn from 1969 to 1977 only on the right ear and the left ear was not aided during that time. Later

on in 1977, due to her complaints about the ineffectiveness of hearing aid, a more powerful aid with MPO l35dB SPL was recommended to the same ear. She had never complained of uncomfortable loudness. Hawkins has made a comparison between thresholds overtime in the aided and unaided ear for the evidence of over amplification. He has assumed that with a bilateral sensorineural hearing loss, unilateral progression is. quite rare and if it did occur, it would be probably the result of a conductive or retrocochlear overlay. Examinations of her thresholds of hearing acuity from 1971 to 1981 in the right ear showed drop in the sensitivity, approximately 30-35dB at 250HZ, 40-45dB at 500HZ, 40dB at 1000HZ, 35dB at 2000HZ and 25dB+(to the limits of audiometer) at 4000HZ. In comparison, the only noticeable shifts in the left ear are approximately 15dB at 1000HZ and 10dB at 2000HZ. Though the client had expressed her concern regarding deterioration of hearing, she was assured that aids do not damage hearing. Again when she was bothered of the experiencing tinnitus and decreased sensitivity in the right ear only after listening to very loud dance, she was advised by the otologist to avoid the intake of alcohols, cigar6t.es and loud noise exposures. No con-

sideration for lowering MPO was given. Hawkins gives two reasons for having not considered overamplification as the possible cause of additional hearing loss.

1. In early 1970's hearing aids were less powerful.
2. The client was receiving services from two different sources and as a result, neither book full responsibility for her continued monitering and care.

In 1981, she had never to profound loss in right ear and mild to moderate loss in her left ear. She was recommended to wear a hearing aid of MPO 115dBSPL in her left ear and right ear was of little benefit, if aided, but, she wore an aid with MPO of 124dBSPL. This report serves as a continuing reminder that hearing aids can cause further damage to auditory mechanism. Hawkins emphasises the need for close monitoring of hearing thresholds in all hearing aid users. He also stresses the need for providers of audiological services to define clearly for the clients what followup procedures are necessary and why they are important.

CHAPTER - V

STUDIES WHICH SHOW THAT
HEARING AIDS DO NOT DAMAGE HEARING

Berry (1939) concluded that the use of a hearing aid tends to increase rather than decrease acoustic intelligence. Disuse of any function encourages its atroptry, stimulating the ears does not improve our threshold performance, but it does make our acoustic perception keener.

Holmgren (1940) strongly suggested that in his experience amplification through hearing aids never adversely affected the residual hearing of children and he went on to say that in many cases the use of a hearing aid had an improving effect on the hearing.

Murray (1951) selected a group of people whose deafness was due to maternal rubella and he followed them up for a period of five years. All were using monaural aids with MPO 130dB SPL. He found changes in hearing acuity in aided ear of these children were similar on average to those of their unaided ear.

Nauton (1957) reviewed the records of 1480 hearing impaired individuals who had been fitted with Medresco hearing aids, instruments issued by British Government at that time. A total of 120 patients returned to clinics for audiologic reevaluation and constituted a basis for the conclusion that apparently no significant changes in auditory sensitivity occured as a result of hearing aid use. His data included etiology of loss, age of the case, period of the hearing aid use, and hours/days of use. Non using ear was the control and he observed relative shifts in the aided ear. The acoustic characteristics of Medresco hearing aids were not specified? nor were the extent and etiologies of losses specified. It was noted that an unspecified portion of finally selected sample was described as experiencing conductive pathologies, a fact which had considerable influence on the overall results of the investigation.

Whetnall (1964) presented the case of two siblings, a girl of four, who after using a hearing aid for 1½ years showed some deterioration and her brother who showed more severe hearing deterioration than his

sister inspite of the fact that he never used a hearing aid. Whetnall stated that "the progressive deafness was clearly familial".

Whetnall states that there is no evidence of acoustic trauma due to a hearing aid, and indeed it could not happen. Acoustic trauma results from long exposure to continuous noise at a high level of intensity or from a sudden explosion. Speech sounds, even with the powerful hearing aid are never delivered at an intensity of 100dB in normal deaf child continuously. Speech sounds are not a continuous sound but are changing in intensity from time to time along with frequency. Intermittent louder bursts of sound such as gunfire will also cause acoustic trauma, but a child is rarely exposed to this risk. Hence, neither of the condition which causes acoustic trauma is present.

Barr and wedenberg (1965) investigated

1. The progressiveness of hearing loss in children with different etiological categories.
2. The effect of hearing aid usage on progressiveness.

They separated the children as

1. Exogenous deafness and
2. Endogenous deafness.

84 children with bilateral perceptive deafness were selected out of which 44 children were with exogenous deafness.

1. 23 children with hearing loss due to maternal rubella observed over a period of 5 to 11 years.
2. 15 children with perinatal accidents observed over a period of 5 to 10 years.
3. 6 children with meningitis observed over a period of 7 to 10 years.

Endogenous group consisted of 40 children, who had hearing loss because of heriditary factors and this group was observed over a period of 3 to 15 years. They observed spontaneous progression in 2 of their groups, i.e., all the children with meningitis and 22 children with heriditary factors. Exogenous hearing impairment group did not show much progressivity. Even if progression is seen in some, the number was very small. This was the finding mainly with maternal rubella and perinatal accident group. The meningitic group showed

progression with or without streptomycin treatment and the authors Barr and Wedenberg considered it as spontaneous rather than a result of amplification. Although 50% of the endogenous group demonstrated increased loss these increases were also considered spontaneous. The conditions of hearing aid use were not specified nor were the number of monaural vs. binaural fittings. The acoustic characteristics were stated as follows, "Most hearing aids had an MPO of less than 13OdB SPL, and no aid exceeded an MPO of 138dB SPL". They concluded that the progressive loss was probably due to the constant use of hearing aids. They also stated that for those children who showed progressive deterioration, progressivity was the same or sometimes even greater in the unaided ear than in the hearing aided ear.

In 1968, Bellefleur and Vandyke made an attempt to explain the deterioration of hearing

1. By etiology.
2. By aid usage.

They conducted the investigation because of the conflicting evidence presented by many authors.

75 subjects were selected for whom good audiometric data were available for 8 to 10 years period.(1953-1964). The selection process was done with children having instruments which were of same manufacturer and model type. Initially 75 were selected for whom good audiometric data was available for 8 to 10 years period (1953-1964). Recorded informations were

1. PTA in each ear for 125-500HZ and for 500-4KHZ.
2. Make, model and data of purchase of aid.
3. Type of fitting (ear, V/Y cord) (4) etiology of loss.

Method was to substract PTA of initial from final for each ear. Ear differences were determined by subtracting average for the unaided ear from average of aided ear. After this procedure, actual subjects were selected who met the following criteria:

1. Hearing aid were always used is the same ear
2. The paid worn at the time of final audiogram was of the same manufacture and power classification as on initial audiogram.
3. They could be studied 8-10 years.

25 children were selected and classified as known group. Criteria 2/3 could be met, but aided ear was not determinable in 33, classified as unknown group. Remaining 18 were rejected. For known group, unaided ear was used as control. It was hypothesized that if high gain amplification had adverse effect, it would be apparent from an analysis computed by subtracting total threshold shift in aided ear from threshold shift in unaided. Any significant difference would indicate amplification effect. Three way analysis was used to evaluate exogenous and endogenous factors. Whole data was analysed to determine whether as a group, there was significant ear to ear deviation in recorded puretone information.

Analysis of variance was not significant at 1% level of known group. Comparison between aided and unaided ear for frequencies 125-500HZ or 500-4000HZ showed no significant change. Comparison of exogenous and endogenous also showed no significant effect. Mean initial and final test indicated that threshold shift under all conditions was extremely small. It is noteworthy that whether comparison of means over all frequencies included in the study (125-4000HZ) or only

those of speech frequencies (500-2000HZ) the greatest difference between aided and unaided was less than 0.5 dB. It was impossible to predict an ear difference in unknown group indicating that no threshold shift occured or that both aided and unaided ears shifted equally. The findings of this study agree with Naunton's (1957) view that hearing aids with high gain have little effect atleast on the population of children with severe loss.

Markides (1971) comments "There is no doubt that exposure to high intensity sounds for a prolonged period causes damage of hearing mechanism, in the same way use of powerful aids can cause further damage to the hearing of people suffering from sensory neural hearing loss". Markides opinion is that many a times it is not the case, very young deaf children react most unfavourably to excessively loud sounds and they soon develop ingenious ways of dealing with such situations. He says that evidences so far presented consists of a series of statements based onpoorly designed experiments or inadequate studies. Markides points out that Kinney (1961) has not taken etiology into consideration and

and that the deterioration of hearing observed in his subjects could be due to spontaneous progressivity resulting from pathology of deafness and or from treatment rather than hearing aid usage. Further, Markidds feels that Kinneys assertion that some of his subjects, were using hearing aids with MPO of 146dB SPL may be an exggeration.

Markides (1971) has critically evaluated some studies in the following manner: Macrae's (1965, 1968) were carried out without any serious control over experimental procedures. Tester differences environmental variables are not taken into account. Moller and Rojskjaer have used a large number of elderly people. Hearing deterioration could have been due to presbycusis rather than hearing aids. Macrae's experiments regarding TTS were carried out with only 4 children and definitely it is a small sample to conclude anything. Ross and Treux (1965) and Ross and Lerman (1967) related hearing deterioration to MPO 130-139dBSPL. But, it is well known that manufacturers figure supplied can be notoriously unreliable. These authors failed to carryout their own measurements with the aids used. Again it is

difficult to accept the reported isolated cases as strong evidence. The hearing deterioration observed in these cases could have been brought about by a natural progressivity relating to pathology of deafness and/or infectious diseases suffered after the aid was issued. On the other hand, Markides discusses, those authors who failed to find deterioration are not altogether water proof. For example, 60% of Naunton's (1957) subjects were suffering from conductive or mixed deafness. These are the persons for whom there is less likelihood of acoustic trauma. Of the 22 children who showed progressive hearing loss in endogenous group of Barr and Wedenberg (1965) only two of them were regular aid users. Also the MPO of hearing aids used by the subjects included in the sides of Naunton (1957) Murray (1951) Barr and Wedenberg (1965), Bellefluer and Van Dyke (1968) and Roberts (1970) may not be great enough to cause trauma.

From the above evidence it is very difficult to decide whether the use of hearing aids does or does not cause further deterioration to an already impaired mechanism of hearing. Clinically one must presume that what is possible may be probable and take appropriate

remedial steps. This justifies the provision of informed guidance and specialised supervision to hearing aid users, especially children, and it also poses the question "desirable MPO" of hearing aids. Watson (1967) stated that "an aid must be provided for the deaf which can give a sufficiently high level of output to enable the pupils to hear speech at adequate levels above their thresholds. Such levels are considered to be not less than 20dB and preverably 30dB or more. This requirement is subject to maximum level which an ear can tolerate without physiological damage, usually of 130-135dB. It should be also stressed that this requirement must be met when the inpur to the aid is not more than 65dB". It is rather difficult to specify regarding clear cut MPO of wearable aids, indeed such a question can only be answered on an individual basis. Extra care is needed in persons where the loudness discomfort level of these persons is above the audiometric limits. According to Coles (1971) persons with severe sensory lesions develop secondary neural degeneration and these are people who are quite likely to tolerate excessive levels damaging to their hair cells.

Markides concludes that there does not yet appear to be any conclusive scientific evidence that powerful hearing aids do or do not have a deleterious effect on user's residual hearing. Enough evidence has been presented to justify a cautious attitude when recommending such aids. Markides opinion is that when powerful aids are needed, it is better to wear them rather than deny auditory experience to deaf, especially children. It is wise not to use such aids binaurally unless they are with AVCs. Wherever applicable alternative use of the ears is recommended, thus providing periodic rest for each ear, or atleast, reducing total energy impinging on each ear over a period of time and thus reducing the rate of any hearing deterioration. Markides recommends more research in variables, such as kind and pattern of hearing loss, cause of deafness, MPO of the aids, actual hearing aid setting for each person, length of time of use of hearing aid, guidance given, extent of noise exposure, calibration of the equipment used to test hearing, testing environment and tester differences, etc. This of course would be a long term study and the variables are too difficult to control and evaluate. Periodic cross over of hearing aid from one ear to other

as a means of studying persistent threshold shift and long term recovery may be employed as another method of researching this topic.

Hine and Furness (1975) examined the annual puretone audiograms of 21 children, 5-9 years, in school for partially hearing where communication means is oral and emphasis on use of aid. Audiograms repeated were analysed for variability by analysis of variance and for trend by sign test. No cases of satistically significant deterioration of threshold were found. They concluded that regular use of aid does not damage residual hearing in any children. One third of them showed statistically significant improvement was reflected on enhanced ability to attend to auditory signals.

Markides (1976) reported on a longitudinal study into the effects of hearing aid amplification on residual hearing of four groups of hearing impaired children. After testing the hearing levels of the children at the main audiometric frequencies of 500 to 4000HZ both at the beginning of the investigation and

at subsequent 6 monthly intervals over a period of 3 years, he reported that the hearing acuity of the hearing aided ears of children showed slight improvement during the investigation, while the hearing acuity of their unaided ears showed slight deterioration in all frequencies tested. These results were in contrary to the findings and beliefs of a considerable number of workers in this field but, they were basically of good agreement with findings and beliefs of a list of contributors (Markides 1971).

Darbyshire (1976) did a study on the use of high power hearing aids by children with marked degree of deafness and the possibility of deterioration in hearing. 100 children of 3½ to 12 years with medium age of 7.3 years when first tetsted, 6.7 years and 15.2 years when retested with median age of 10.11 years. All were students of the same school tested in the same acoustic environment. All of them had marked hearing loss. The subjects on whom data were presented were the first hundred hearing aid users on whom tests and retests could be carried out with an interval of 2 years between them. The hearing losses were in excess of 60dB when

average puretone threshold of better ear was calculated for middle/speech frequencies. The frequency range for testing was from 250 to 8000HZ with audiometers having maximum output of 130dB. The longest interval to elapse between was 3 years 2 months and the shortest was 2 years 9 months. Comparisons between thresholds were not made when no responses were obtained at any frequency at either test/retest stage. Most of them were wearing body type hearing aid and most of them also changed aids atleast once between recorded tests. None was fitted with HAIC gain less than 50dB and maximum HAIC gain was less than 100dB. At the time of first series of tests all the children had body worn aids, but when second series was carried out 20 of 28 were with skislope losses had been given behind the ear aids, these being fitted to a total of 30 ears. Though 45 had two aids at the time of second test, only 10 had them at the time of first test. No child having two aids for less than one year was classified as binaural fitting in the results. These clear trends were shown in the data

1. There was no evidence that the wearing of aids caused deterioration of hearing.

frequencies, greatest at 4000HZ was seen. Comparing the recorded deterioration and improvements did not yield satistically significant results with all frequencies or at any one frequency.

It was possible for him to pick up 3 major groups of audiograms in terms of contours.

a. Steeply sloping (skislope)

b. Left hand corner

c. Flat

(a) Criteria for skislope was that no puretone readings above 5OdB at 250 to 500HZ and no readings below 70dB at higher frequencies. Some improvements were relatively great, the biggest being at 1000HZ, 4.8dB. Change at 4000HZ was 0.4dB and at 8000HZ, 1.2dB.

(b) Children with most marked hearing loss were those with left hand corner audiograms. Thresholds for a total of 60 ears which had received amplification were recorded, none of them having readings better than 75dB at any lower frequencies and none having any high readings at higher frequencies. Improvements were very small, biggest being 2.3dB at 500HZ.

(c) The flat group people had thresholds which did not vary by more than 30dB at any frequency. 28 ears and an average improvement was recorded biggest being 2.3dB at 1000HZ.

Careful scruting was made of the audiometric data of hearing aid users who were tested on occasions between tests and retests recorded. Relatively few cases with dramatic improvements/deterioration were seen. 26 showed changes, better or worse of 15dB at one/more frequencies. In marked deterioration upper respiratory tract infection was often suspected and/or impedence revealed middle ear pathology. 11 showed changes of 15dB/more in both ears and the greatest improvement was 20dB at 250HZ. Biggest deteriorations recorded at 4000HZ were 15dB. They were unilateral with no deterioration, on the other side. 11 ear deteriorations of 10dB and 17 of 5dB. No evidence was found by him to suggest that skislope or left hand corner types of hearing losses were made in areas most vital for their perception of information about speech even when the binaural fitting were made. 30 with monaural aids and no aid in other ear tended to show a slight mean improvement in the ear with an aid and a slight deterioration in

the other. None of the recorded improvements exceeded a mean of 20dB over octave frequencies and none of the deteriorations more than 15dB. Mean improvement was 7.8dB and mean deterioration, 4.5dB. No evidence to suggest that any recorded deterioration were or were not linked to high distortion levels of aids.

So, data provides evidence that sustained amplification by means of good fitting individual aids causes young children to give better responses to pure-tones after 3 years use and not worse. To certain extent according to him this is the function of the improved listening skill and the maturation. One is tempted to conclude from cumulative evidence of ears tested that most of the children with marked losses of hearing benefit from using two aids. If this is valid it is most probably under-pinned by the fact that children were fitted with aids best suited. Darbyshire also cautions to pay more attention to possible acoustic trauma. None of them were clinically , significant in this case although there was less improve-ment at 4000 - 8000HZ, than at other frequencies in some. Again he suspects that this could be the fact that result of hearing at these points is less stimulated by the aids

than at lower frequencies. He has concluded that there is no evidence that amplification was harmful to children with marked losses. The author recommends further research on.

1. Would a study of similar nature over a longer period, have produced comparable results?
2. What is the effect of etiology on the capacity of children's ears to withstand prolonged hearing aid usage and benefit from amplification?
3. Would ears that were subjected to intensive mainly bilateral auditory training give similar results of those now reported?
4. Do high distortion levels cause temporary or permanent threshold shift?
5. Would evident improvement in hearing for puretones shown in this study also manifest itself in speech discrimination scores in cases in which children's language comprehension levels enabled these to be obtained in early years?

Titche et al. in 1977 were interested in finding whether the hearing aids really do damage hearing?

Over a period of 1965 to 1975, 261 patients were fitted with the hearing aids at reexamined at various intervals. Additional patients had been fitted, but for various reasons, did not return for reexamination and were excluded from this study. The patients were not selected upon any basis except for the fact that a hearing aid would be of benefit to them. No attempt was made to ascertain if there had been any noise exposure or other contyaminating variables. The mean age was 55.20 years and median age of 54.69 years. Monaural aids were furnished to 261 patients and binaural aids to 14. These aids varied from a gain of 36dB and 110dB MPO to a gain of 72dB and 142dB MPO. All the testing was performed by one person. The tests were conducted in a two room sound suite. Puretone and speech audiometry was carried on. Speech reception threshold and discrimination scores were obtained with live voice. Pure tone audiometry was for frequencies 250HZ to 8000HZ for air conduction and the same frequencies, except for 3000HZ and 8000HZ for bone conduction. It was presumed that any acoustic trauma that was produced by the hearing aids, would be shown particularly at 3000HZ and 4000HZ. On puretone audiogram and

by changes in the speech reception thresholds. The discrimination scores were not being used in this report because there are too many variables influencing them that it was believed that it would not indicate changes in hearing accurately.

The pre aid hearing in the non aided ear was substracted from the hearing in the ear, which was fitted with hearing aid. At the time of reexamination, these differences were obtained again if the loss of hearing in the aided ear and increased more than in the nonaided ear, a positive value and if the reverse occured, a negative value resulted.

1. For entire group tested by air conduction at 3000HZ there was a mean difference of +0.4338dB which was not significant; at 4000HZ there was a mean difference of -0.2797 which was not significant; and the SRT showed the mean difference of + 0.3142 which was not significant.

2. For the sensory neural group, at 3000HZ there was a mean difference of -1.39dB which was not significant; at 4000HZ the mean difference was -0.2457dB which was not significant; and SRT showed mean difference of -0.16dB, not significant.

3. For the conductive group, by air conduction showed a mean difference of + 4.4853B, which was significant; at 4000HZ mean difference of - 0.348dB which was not significant; SRT showed mean difference of + 1.279dB not significant. At 2000HZ there was a relative shift of +2.5dB. The bone conduction tests for this group showed a relative shift of - 1.3dB at the 2000HZ frequency and OdB shift at 4000HZ.

In general these results show that the relative shifts in the entire group and the sensory neural group were less than those reported by Ross and Lerman and would indicate that hearing aid use had no detrimental effect upon hearing. The patients who had a conductive component in their deafness showed an increase in the loss of hearing at both 2000HZ and 3000HZ by air conduction. Titche et al. opinion that the loss at 3000HZ would seem to indicate evidence of acoustic trauma, inspite of no increase in bone conduction at 2000HZ and no significant change at 4000 HZ by either air or boyne conduction. They do not give any explanation for this. They felt that there seamed to be little effect produced by length of use of a hearing aid as far as deterioration of hearing was concerned, except for those who used an aid from 8 to 9 years. However correlation

between years of use and relative shift at the frequencies studied was not significant. So, Titche et al. by stating the limitation that the group was small to draw any conclusions, state that the prolonged use of hearing aids, like those being worn by patients at the present time, does not increase loss of hearing in aided ear.

Markides and Aryee (1978) have given a followup study of effects of hearing aid amplification on the user's residual hearing. 4 groups of deaf children, 30 children in each of tne first three groups and 10 in the fourth group, were taken as subjects. The children in group A (average age at the beginning of study, 10, 3 years, range 8.5 - 12.6 yrs) were fitted with commercial hearing aids with MPO varying from 130- l36dB SPL and worn at volume settings giving acoustic outputs ranging from 116 - 127dB SPL and with effective frequency range from 300 to 4000 HZ. The children in group B (average age at the beginning of study, 9.8 yrs, range 7.9 - 13.2 years) were fitted with commercial hearing aids with MPO varying from 116 - 128dB SPL and worn at volume control setting giving acoustic outputs ranging

from 95-ll5dB SPL and with effective frequency amplification from 350 to 4000HZ. The children in group C (average age, 13.1 years, range 11.4 - 15.4 years) were not using hearing aids while children in group D (average age, 10.4 years range 8.7 - 12.9 years) were fitted with binaural hearing aids similar to those issued to the children in group B. But in the followup study, it was not possible for them to include all of them. Only 18 children from group A; 14 from group B; 15 from group C; 8 from group D were eventually followed up. The children from group A and B were still wearing the same hearing aids that were issued to them at the beginning of investigation. At the end of initial investigation 15 children from group C, who were not using hearing aids, were given with monaural bodyworn aids, the same type as was for group B, and the 8 children in group D who were previously using binaural hearing aids were restricted to one hearing aid during the follow up study, mainly because of economic reasons. All the children had bilateral sensory neural loss, they were either born deaf or acquired deafness during first few years of life, in nearly half of the cases cause was unknown and this is mainly due to poor medical care and unreliable case histories. The follow up study was in the same environment with same equipments and audiologists.

CHAPTER - VI

The results were computed in the following way. The average hearing levels of children at 500, 1000, 2000 and 4000HZ respectively, both at the beginning and at the end of initial investigation and at the end of subsequent follow up year were tabulated in terms of hearing "aided" ears and hearing "unaided" ears, irrespective of whether the hearing aid was fitted in the left or right ear. Mean hearing levels of the children in each group at each one frequency was tested at the beginning and end of the follow-up years, compared with one another by using technique of one way analysis of variance. Resulting values were small varying from 1.38 - 3.02. None of them reached significant values and in view of this no further statistical treatment of results was undertaken. It is of interest to note, however that the initial tendency to "improvement" of hearing in the aided ears and the tendency to "deterioration" of the unaided ears in all the frequencies, as noted in the earlier investigation were also in evidence during this followup study. It is of interest to note the results of group C and D. At the end of the follow up study, children showed slight "improvement" of hearing in their aided ears in the region of 1 - 3dB in all frequencies tested whilst their unaided ears remained

the same in group C. The hearing aided ears of children in group D remained the same in terms of hearing acuity, whilst their unaided ears showed on average a "deterioration" of l-4dB in all frequencies.

So, the results are basically in agreement with those obtained previously during initial investigation and they tend to lend additional support to Berry's (1939) and Holmgren's (1940) statements to the effect of that amplification through hearing aids "tends to increase rather, than decrease in acoustic intelligence". But Titche et al. list some of the limitations that

1. Children were followed up only for a limited period of 4 years. A longer period may show different effects.

2. The children were using their individual hearing aids at volume control settings giving acoustic outputs varying from 95 - 127dB SPL. In theoritical assumption, these levels may not be strictly relevant to the pathological hearing mechanism. If more powerful aids were used, results would have been different.

3. Children were not consistent aid users. In average each child was using the aid

3 - 4 hours daily during school hours. Very few were using during vacations. This was due to limited number of aids available., and limited technical services available at school. More intensive use of aids might have produced different results.

4. There is possibility that the use of powerful hearing aids can effect differentially the various pathologies of deafness. This proposition could not be studied in this investigation. However, the fact remains that children did not show deterioration of hearing assigned to hearing aid use in this longitudinal study.

Further, again Markides and Aryee (1980) continued their study on 15 children to study the effects of hearing aid use on the user's residual hearing. Here they tried continuing with their previous investigations. Only 15 children, 12 from group A, 3 from group B could be tested, because some had left the school and some did not show consistent hearing aid use to include in the present study. Average age of these 15 children (9 male

6 female) at the beginning of original study was 9.7 years. (7.9 to 11.2 years range). All were using for 6 years monaural commercial body worn hearing aids with MPO varying from 116-136dB SPL and worn at volume settings giving acoustic outputs ranging from 95 - 127dB SPL and with effective amplification of frequencies from 300 to 4000HZ. Children were using same type and model of hearing aid in some ear. All the children had bilateral sensory neural impairment and they were either born deaf or acquired hearing loss within first few years of life. In more than half of the cases the cause was unknown, this was mainly because of poor medical care and unreliable case histories cause of remaining 6 were; 2 heredity, 2 meningitis, 1 rubella and 1 convulsions due to malaria. The equipments used were in accordance with previous investigation. Hearing levels were tested both at the beginning of the study; at six monthly intervals thereafter over a period of 3 years, at the end of fourth year and at the end of sixth year.

The results indicated that the average hearing levels of children at 500, 1000, 2000 and 4000HZ respectively both at the beginning of the investigation and at

the subsequent testing intervals over a period of 6 years were tabulated in terms of hearing "aided" ears and hearing "unaided" ears irrespective of whether the aid was fitted to left or right ear. One way analysis of variance was used to compare the ear differences at each frequencies. Resulted values were small from 0.95 to 2.93. None reached a significant value. It is was found that the initial tendency to "improvement" of hearing aided ears and the initial tendency to "deterioration" of unaided ears in all frequencies were also evident in this study as was in previous study (Markides 1976); Markides and Aryee 1978). Again the hearing in the hearing aided ear of children on average showed an initial "improvement" of 2-3dB in first 12 months, on the whole was, maintained over the rest of the experimental period. The hearing in the unaided ear of children on average showed an initial "deterioration" of 2-3dB in hearing which was again maintained for rest of the period.

So, the result support Berry's (1939) study. But Markides and Aryee suggest to remember that

1. Children were of average intelligence. There may exist a group of children with subnormal intelligence who may show different

results, no overt action to loudest sounds, etc. They feel that the other group which may have deleterious effects from powerful aid use are the children suffering from progressive hearing loss and wherein a hearing aid may have accelerating effects on hearing deterioration. They also question, in this case, would it not be advisable to use the residual hearing of each children to maximum before gradual deterioration interferes with whatever residual hearing they may possess? But, there may be a possibility that use of powerful hearing aid can cause differential affects on various pathologies of deafness.

2. The children in the investigation used MPO 95-127dB SPL. If more powerful hearing aids were used, different results would have got.

3. The children were not consistent hearing aid users. On average each one used aid 3 - 4 hours daily. More intensive use may produce different effects.

Katherine (1981) holds a sample of 20 children monitored for progressive hearing loss combined with 25 progressive loss cases. The effect of hearing aid use

on progressive loss were examined in context of etiology and period of progression. The criterion adopted was decrement of 15dB or greater for atleast two frequencies in one ear. Progression was greater at 1K and 2KHZ than 4KHZ. Mean peaks gain used by 45 progressive subjects was 62.2dB and peak 5SPL was 130.2dB SPL. In nonprogressive it was 62dB and 129.5dB SPL. Results of the study showed a limited role played by aids on the progressive hearing loss. In 31 (69%) of the 45 cases use of hearing aid was not implicated questionable in 9 (11%) and implicated in 5 (11%). So, Katherine has concluded that in monitoring the progressive loss cases, it is unwise to conclude that hearing aid use is cause of deterioration without considering all plausible factors. However, she suggests incentive to look beyond hearing aid is provided by a large number of possible etiological factors associated with progressive loss. Eg: 10 had, in her study progressive bilateral sensory neural hearing loss even after wearing the aid to only one ear. So, irrespective of hearing aid usage, progressive hearing loss is not uncommon in children and communication between parents, audiologists and other professionals is crucial for management. When etiology is unknown, genetic counseling

and laboratory studies are done to determine cause of loss and possibility of future progression (proctor 1977) Katherine has also said about the difficulty in deciding upon safe SSPL that this can be appreciated in conjunction with differences in inherent susceptibilities, etiologies and other factors. Rintelmann and Bess (1977), Hefferman and Simons (1979) believe that SSPL of 120dB or less would be a reasonable limiting level with mild and moderate loss cases. For more severe loss cases, level less than 130dB is suggested by Ross and Lerman (1976). None of the authors have specified standards for SSPL measurements nor did they distinguish between average and peak SSPLs. Recently defined SSPL 90 (Kastern 1978) would be appropriate, barring extreme peaks. Although Rintelman and Ross (1977) cited evidence suggesting that children with profound hearing loss may be able to tolerate high levels of acoustic output without experiencing threshold shifts, they recommended extreme care with SSPLs approaching 130dB SPL. Other safegaurds such as monaural, alternative use and AVCs are appropriate. Close auditory monitoring is essential. In individual cases desirability to provide best amplification must be carefully balanced in context of possible damage from aid use.

CHAPTER - VI

DISCUSSION

There does not yet appear to be any conclusive evidence that powerful hearing aids do or do not have a detrimental effect on listener's residual hearing. Many variables such as

1. Hearing loss, its severity and type; the cause of hearing loss; individuals susceptibility and tolerance;
2. Factors within the aid such as maximum power output; the presence of noise within the hearing aid; performance of aid over time;
3. Number of hours of aid usage; type and extent of noise exposure, etc. are the important ones to be considered to draw any conclusive remarks.

Most of the studies have selected children as subjects on the grounds that adults are more likely to be exposed to contaminating variables such as, presbycusis, and industrial noise hazards are generally less likely to be available for regular testing. But, there are serious

pitfalls of using children as subjects. Many young children tend to suffer from a fluctuating hearing loss as a consequence of episodic respiratory infections. Children attending schools for the deaf, moreover, are likely to receive high level amplification from auditory training equipments, and group aids. These systems provide binaural stimulation. It would seem that lack of correspondence among various studies might be due in part to methodological difficulties inherent in the use of children as subjects. Children as a rule cannot give information about exposure durations, volume control settings and temporary threshold shift, all of which might be relevant to the problem.

Above all it is often necessary to fit a hearing aid on a young child before obtaining an accurate assessment of residual hearing. In such a case, if the hearing aid should have any detrimental effect, children may still be unaware of such additional permanent decrements in the auditory sensitivity.

Even with the knowledge of such harmful effects, hearing aids have to be fitted as early as possible,

STUDIES WHICH INDICATE THAT HEARING AID DAMAGES HEARING

AUTHORS OF THE STUDIES	YEAR	NO.OF PERSONS	NO. AFFE-CTED	MPO OF AIDS	DETERIORA-TION OF HEARING IN dBs
Kinney	1953	8800	16	?	?
Hardford & Markle	1955	1	1	?	?
Holler & Rajskjaer	1960	390	9	120	?
Kinney	1961	178	41	?	0.25
Sataloff	1961	1	1	?	20
Ross & Trevx	1965	2	2	139	25-35
Macrae & Farrant	1968	27	?	121-126	9.6-11.2
Ross & Lexman	1968	18	9	130	1.2-9.4
Macrae	1967, 1968	4	4	130	4.5
Macrae	1968	32	?	115-117	4.2
		40	?	117-119	3.9
		38	?	120-124	8.1
		24	?	125-130	8.6
Roberts	1970	278	16	?	?
Eastern & Bravlin	1970	1	1	120	?
Ischizawa and Nishiyama	1974	1	1	?	?
Jerger	1975	1	1	135	?
Heffernan & Simmons	1979	2	2	125	5-35
Kittle & Axmann	1981	55	7	?	?
Jassal	1981	1	1	7	?
Hawkins	1982	1	1	135	20-45

STUDIES WHICH INDICATE THAT HEARING AID DOESNOT DAMAGE HEARING

NAME OF THE AUTHOR	YEAR	NO.OF SUBJECTS	NO.AFF-ECTED.	MPO OF AIDS	DETERIO-RATION IN dBs.
Holmgren	1940	?			
Murray	1951	?		130	
Naunton	1957	120		126	
Whetnall	1964	9		?	
Barr & Wedenberg	1965	84		130	
Bellefleur & Vandyke	1968	58		?	
Hine & Furness	1975	21		?	
Markides	1976	100		116-136	
Darbyshire	1976	100		130	4.4
Titche et.al	1977	261		110-142	0.3
Markides & Aryee	1978	55		116-136	1.38 to 3.02
Markides & Aryee	1980	15		95-127	.95 to 2.93
Katherine	1981	25		62-130	

On the theoretical grounds, the amplification levels and exposure durations that many deaf children sustain in daily hearing aid usage appear to carry a risk similar to that of a very noisy industrial plant; yet research has not always shown that this type of exposure is dangerous for all deaf children. Presumably, children are most likely to derive communicative benefit from the use of aid and hence, most likely to use it persistently at a level that will compensate optimally for their hearing impairment. So, hearing aids are not intrinsically dangerous. They can damage hearing only when they deliver sounds high enough to have permanent threshold shifts. It is well known that many severely deafened children wear aids without benefit and presumably without harm. Moreover not all ears that sustain temporary threshold shift from overstimulation develop permanent threshold shift. TTS is necessary, though not a sufficient condition in the generation of PTs. Several reports have shown that TTs returns to preamplification level once the instrument is removed (Macrae 1968, Kastern and Braulin 1970, Sataloff 1961 etc.). This absence of information about the relative suceptibility of children to acoustic injury and noise induced hearing loss; as well as absence of definitive information about the effects of noise on diseased or previously injured cochleas has implifications to clinical methods and research needs.

CHAPTER VII

CONCLUSIONS AND RECOMMENDATIONS

The literature on possible destruction of residual hearing through the use of powerful aids does not permit from conclusions in either of the direction, but it does contain sufficient evidence to show that some sustain temporary or presumably permanent threshold diminution as a result of protected use of powerful instruments. Either of the events have serious consequence on' the individual. So, in this case, the presence of evidence does not mean evidence of presence. No individual should be deprived of the benefits he ought to receive because of the fear of risk of further handicap. Once the following guidelines are followed, the aid need not be denied.

1. Detailed understanding of individuals hearing: type and amount of loss, site of pathology, etiology of loss, onset of loss, progressivity of susceptibility to NIHL, tolerance level, and the time of consultation.
2. Analysis of the need for the aid and its usefulness.

3. Accurate electroacoustic measurement of hearing aid its gain, SSPL, MPO, distortion level and inherent noise (all must be within acceptable limit, with a note of make and model of the aid).

After establishing these baselines, care must be taken to recheck these factors periodically. Careful and regular audiometric checkups indicate the possible damage to certain extent, if any, by comparing the results of preaided ear to post aided ear.

Even if progressive loss is attributable to aid usage, it has to be monitored by adjustments in MPO, alternating use of ears or reducing the length of wearing times to reduce the exposure.

Since there is a possibility of loss of hearing with aid usage, at first, monaural fitting with frequent monitoring must be done and only later, if no harm detected, the binaural fitting to be recommended. Due caution and consideration has to be taken while recommending binaural amplification. One cannot assume that two ears behave in the same manner if they have symmetrical loss. As far

as possible those who use powerful aids must wear hearing aid at minimum setting of volume control.

1. Check the performance of the aid after fitting within 30 days.
2. Electroacoustic analysis of aid within 30 days.
3. Monthly appointments for at least 3 months.
4. Reevaluation for every 3 months in the following ear.
5. Annual otologic and audiologic check up.

Along with these careful considerations, the user must be made aware of the facts regarding the pain threshold. This is especially must with parents of small children. (Vyasamurthy.M.N. 1981).

In 1977 Food and Drug administration went into effect, regulating professional and labeling requirements and conditions for the sale of hearing aids. In accordance, the hearing aid dispenser should advice the user to consult a physician if any of the following conditions do exist:

1. Visible congenital traumatic deformity of the ear.
2. History of active drainage within previous 90 days.

3. History of sudden or rapidly progressing hearing loss within previous 90 days.
4. Acute or chronic dizziness.
5. Unilateral loss of sudden or recurrent, onset within previous 90 days.
6. Audiometric a - b gap of more than or equal to 15dB at 500, 100 and 2000HZ.
7. Visible evidence of cerumen accumulation or foreign body in the canal.
8. Pain or discomfort in the ear.

So, the frequent and regular auditory monitoring is of utmost importance in the rehabilitative tool of hearing impaired, as does the correct and best fitting aid, with the "individual" in consideration. Cur aid should be to provide the benefits of amplification, with the further trauma due to aid use minimized.

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