GERIATIC AUDIOLOGY

C.S. VENKATESH

Reg. No: 11

An Independent Project Work submitted as part fulfilment for First Year M.Sc (Speech and Hearing) to the University of Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING

MYSORE - 570 006.

DEDICATED

TO MY

GRAND FATHER (appa)

Who gave a

good start

to my

Education

C E R T I F I C A T E

This is to certify that the Independent project entitled "GERIATRIC AUDIOLOGY" is the bonafide work, done in part fulfilment for First Year M.Sc (Speech and Hearing) of the student with Register Number 11

Director,
All India Institute of Speech
and Hearing,
MYSORE - 570 006.

C E R T I F I C A T E

This is to Certify that the Independent Project entitled "GERIATRIC AUDIOLOGY" has been prepared under my Supervision and guidance.

(GUIDE)
P.J. KUMAR
Lecturer in Audiology
A.I.I.SH.
MYSORE-570 006

D E C L A R A T I O N

This Independent Project entitled "GERIATRIC AUDIOLOGY" is the result of my own study undertaken under the guidance of Shri. P.J. KUMAR, Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore; and has not been submitted earlier at any University for any other Diploma or Degree.

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CONTENT

Chapter-1

INTRODUCTION

Geriatric Audiology, is a branch of audiology, which deals with the process, diseases, disabilities, disorders, and rehabilitation of hearing,, in aged people.

Until recently the entire area of geriatrics has been neglected not only by medical professionals but also by speech and hearing professionals.

The aging process has strong effects on man's physiological, physical, psychological and social well-being. The number of aged people(i.e., over 60 years) is growing rapidly in India. In 1931 this geriatic population was only 2% ; in 1961 it increased to 4.8% and; in 1971 this was 6.3% The recent census in 1981 indicates geriatric population in India as 11% and it also calculates that there will be 18.5% of geriatric population by the end of this century. Another survey conducted in India indicates that 87% of geriatric population are suffering from different types of chronic illness, and of which only half could afford medical treatment.

We do not have the incidence of speech and hearing problems in aged population in India. In U.S.A., MILLER and ORT(1965), and SCHOW and NERBONNE(1976) have indicated that the incidence of hearing loss in elderly ranges from 20% to nearly 97%; depending upon the nature of the group evaluated.

Lot of literature has been available with regard to the hearing of the aged. They are interms of hearing process, hearing disorders, and hearing assessment. Primary aim of the present study, is to compile and review all the literature regarding the aged primarily with reference to the hearing and secondarily with reference to psychological, physiological and physical changes those occur with increasing age.

Present compiler, assumes that this will help in future research in India. This project is the first one that has been compiled, in India, hence, it is expected that will throw some light on the aspects of the hearing of the aged. These aspects can well be utilized by us and also by others while doing hearing research with the aged population. This is the primary implication of the present project also.

On the whole, present project is an effort to collect, compile and review the needs of the geriatric population, which has not attracted much attention from us.

Chapter-II

Normal Aging Process

" Aging is defined as a certain kind of changes in living systems due to passage of time '* . Hence aging of an individual starts from the womb of mother. But in this chapter we are talking about changes during senescence.

With increasing age comes a decline, a regression, or return to an earlier pattern of behaviour, and a simple level of functioning. However as PARKER(1961) said " Aging is not a disease or a disintegrative force, nor is senescence a state of pitiable decrepitude of mind and body to which we must all succumb- if we live long enough ". "Both aging and senescence are inherent part of life, that we must acknowledge, accept, and seek to understand and thereby enjoy".

MEDAMAR(1951) defines senescence, as change of body faculties sensibilities and energies, which accompanies aging, and which renders the individual progressively more likely to die from accidental causes of random incidence.

Origins of Age changes: Three tables from Time cells and Aging.

All people, as they grow old, experience physical, psychological and interpersonal changes of one sort or the other. Some times communicative handicaps cause many problems and sometimes they are the result of these changes. One common physiologic change associated with aging results in a communicative handicap. This chapter will provide an overview of several aspects of aging and changes associated with aging and will serve as a meaningful background to understand hearing problems in aged.

Biological changes: Associated with Advancing Age:

With advancing age all organisms, tend to be more susceptible to disease and deterioration and are less able to sustain and initiate the process of self repair. The physical deterioration associated with aging is either genetically determined or determined by enviornmental influences. JONES(1959) & BIRREN(1964) in case of man consider that hereditory factors as of lesser importance than enviommental influences.

Aging of Cells: At the most fundamental level hereditory and enviommental factors influence cellular function in body tissues. Two general problems associated with aging may be observed at a cellular level. Firstly, it is well documented that several cells die with increasing age in critical organs of the body and that the replacement of these cells in either reduced or absent among the elderly. Secondly, the remaining cells in the organs may not operate at their optimum. Efficiency in older organismsXwolff,1959) These two condition: result in reduced functional capacity of many organs of the body.

Aging may selectively influence highly differentiated cells that can not under go further mitosis. Hence the cellular organizations such as of the nervous, vascular, and muscular systems should be particularly susceptible to the effects of Senescence(BIRREN 1964).

One commonly accepted explanation for cell deteioration is that over time there is damage to DNA. Components of the chromosomes, with damage to DNA defective messenger RNA may be generated which are unable to synthesize the necessary enzymes for maintaining cell operation. As a result cells may die or they may not undergo proper division.

Aging of General Body Systems:

The most common characteristic problem of Aging persons is circulatory impairment caused by cardioc insufficiency, which is attributable to any of the following,

a) Vascular heart disease

b) Hyper tension

c) Pulmonary, metabolic or infectious diseases and

d) localized ischaemia resulting from arteriosderosis (SIMONSON 1965)

Cardio Vascular disturbances have a profound impact on other body systems, particularly the Nervous system. Many of the deficits in sensory motor performances associated with aging may be attributed to Cardio Vascular disease.

Atrophy of Skeletal muscles is another obvious change associated with advancing age. W0LFF(1959) stated that it is not entirely clear whether the decrease in muscle fibre mass is attributable to loss of fibres or a decrease in muscle fibre diameter, or both. A reduction in skeletal muscle mass is a characteristic of advancing age. Age explains much of the decreased muscle strength observed among the older. BIRREN(1959) noted that some muscles do not evidence a dramatic decline in strength probably because of continued high level of use.

The Endocrine system is considered to be relatively less susceptible to the aging process. Normal or near normal functioning of the pituitary adrenal mechanism has been observed even in very old people (Freeman 1959; BIRREN 1964) But certain hormonal productions like that of insulin is reported to decrease with advancing age. Gonadal failure is also reported to be common in old people.

WOLFF(1959) stated that, in aging body, there is a persistence of the integrity of the pituitary- adrenal mechanism and there is also a maintenance of the functional capacity of the catabolism- mediatering glands in contrast to a progressive loss of capacity in the glands with anabolic activities.

Aging of Nervous System: The nervous system is highly susceptible for aging. At least three reasons may be offered to account for this-

(a) neurons in the central nervous system are not replaced by the division of the remaining cells;

(b) the brain is highly sensitive to lack of oxygen, and therefore temporary interruptions in the blood supply is likely to result in the death of neurons and

(c) there is a proclivity for pigment deposits (lip ofusion) in cells of aging brain. It has been commonly thought that the accumulation of lipofusion may have an effect on the physiological capabilities of neurons and may even result in the death of cells (B0NDAREFF 1959)

One of the major changes of the nervous system associated with aging is decrease in its weight. APFEL and APPEL (1942) reported a decrease of 11 to 15% in the weight of nervous system.

BONDAREFF (1959) has described a decrease in brain volume relative to skull capacity as a function of advancing age. This gives the brain a shrunken appearance with an exaggerated pattern of cortical convolutions characterized by narrowed gyri and widened Sulci. In addition to these changes in cortical surface structure, there may be thickening of the meninges, atrophy of the Corpus Callosut, shrinking of the basal gahlia, distended ventricles an increase in the cerebrospinal fluid and cerebral arteriosclerosis.

Number of studies have shown that there is a definite decrease in central nervous systems' neurons. With advancing age, BRODY (1955) noted the greatest cell loss in the superior temporal gyrus with lesser reductions in the precentral gyrus, area striata, inferior temporal gyrus, and postcentral gyrus. Neuron cell loss has also been recorded in the peripheral nervous systems of the older people. Specifically there appears to be a reduction in the number of larger fibres (MAGLADERY, 1959).

BIRREN (1964) has argued that aging will have a progressively more serious influence on the less primitive aspects of the nervous system. The later evolved structures, such as, the cortex is reported to be more susceptible to the effects of senescence than the more primitive structures such as the vegatitive centres of brain stem.

Sensory-perceptual changes Associated with Advanced change:

There is a vast literature about sensory and perceptual changes in the old population. Here, initial attention concerning this topic focuses on the special senses of vision gustation, olfaction, pain, touch and vibration. We have considered, sensation as awareness of stimuli through the exteroceptive systems and perception as interpretation of sensations.

Vision: The principle test of visual adequecy is acuity, which is measured with respect to smallest object that can be perceived at a given distance. It is reported that there is generally a gradual little change or no change in the acuity from age 15 to 50 years, then onwards the change is reported to be rapid. BOTWINICK(1973) states that by the age of 70, without correction, poor vision is a rule rather than an exception. This visual deterioration is attributed to pupil size, which diminishes with age, there by reducing the amount of light reaching the retina. Accomodation ability of pupil decreases with advancing age.

WEISS(1959) states that maximum accomodation is roughly 20 dioptres at age 5 and decreases at the rate of 0.3 dioptres/ year until minimum accomodation of 0.5dioptres is reached at the age of 60. There is a definite deterioration of illumination level, contrast discrimination, colour vision, and critical flicker fusion, with advancing age (WEISS 1959, BOTWINICK 1973).

The reasons for deterioration in visual performance among the elderly are many and include retinal decay, reduction in pupil diameter, opacities and bubbles in the lens and loss of lens elasticity. Some of these eye problems result from degenerative and which may be of vascular, metabolic or endocrine origin.

Taste: Many studies suggest that sensitivity for the major taste qualities of salty, sour, bitter, and sweet; and discrimination ability between the tastes decrease in older subjects (COOPER & BILASH 1959) BALOGH and LELKES(1961) state that sensitivity for bitterness actually increase with age. Aging appears to have a differential impact on taste buds in certain areas of tongue (SCHIFFMAN 1977).

Olfaction: BIRREN(1964) reported a decrease in the number of olfactory nerve fibres with advancing age, implying a reduction in the sense of smell. Many research studies supported this finding. However, factors such as history of smoking, presence of nasal obstructions and occupational conditions may contaminate many efforts to assess the effects of aging on olfaction.

Touch and Pain: Touch and pain sensitivity decreases in older people. Specifically, Stereogenosis, Sensitivity to VonFrey hairs, vibratory sensitivity, and radiant heat pain sensitivity have all been shown to decrease with advancing age. However, as BOTWINICK (1973) has pointed out some of the diminished sensitivity reported in the literature may be a function of caution. That is older subjects tend to exhibit more c nservative thresholds for many sensory tests because of the need for certainty regarding the presence of stimulus. It should be noted that diminished touch sensitivity may have some impact on speech.

Perception: It must be appreciated that sensation and perception can not be easily separated in most exceptional tasks because an impairment of one usually influences the adequacy of the other,. RAJALAKSHMI and JEEVES(1963) and

MALEPEAI and HUTCHINSON(1977) have established that older subjects require much greater exposure time to identify designs, words, and pictures. The age effects are wore profound when interfering visual stimuli and reduced contracts are introduced. BOTWINICK (etal 1959) say that older people have difficulty in understanding complex and ambiguous visual stimuli.

There are four important explanations to reduced perceptional ability observed in older people. They are-

(a)due to anatomic changes in the sensory systems, the CNS, receives loss information upon which processing decisions are made;

(b) the threshold responses may be elevated in part because of certain caution in responding,

(c) CNS alterations due to aging may deteriorate the ability to integrate information from several senses or within a simple sense, and

(d) Elders may display a rigidity in responding and they may have a reduced ability to alter their original percepts

Psycho-Motor changes and Advancing Age:

Older people exhibit slower responses to envioRNmental stimuli, as a result of deteriorations in sensory and perceptual function. Gerentologists consider reductions in psychomotor speed to be a principal indicator of nervous system senescence (BIRREN,1964) Many experiments using reaction time and movement time have been carried out to explain psycho motor changes associated with advancing age. KOGA and MORANT(1923) found that reaction time rapidly decreases with increasing age from birth to about 20 years and then onwards there is a steady increase in reaction time as age increases. Many researchers have confirmed this finding.

It has also bean reported that the reaction time is determined more by the life style of a subject than his aging.(SPIRDUSO-1975)

SMITH & GREEN (1962) demonstrated that tasks frequently performed were less affected by aging than the tasks performed infrequently . The increase in the reaction time, may also be due to slowness in the muscular movement. There is a large dispute between researcher about the principal locus of reaction time increment as to whether it is due to changes in the peripheral functions or due to delay in the central mechanisms. The perceptual speed is reported to be slower among older subjects and it is also reported that it requires higher intensity of stimulation. BOTWINICK(1972) suggests that factors other than those associated with the input may be primary contributors to decreased reaction time with increasing stimulus intensity. In studies using measures of peripheral nerve conduction velocity, BIRREN and WALL(1956); HUGEN, NORRIS and SHOCK (1960) and LAFRATTA and CENESTRARI (1966) reported a small decrease in the nerve conduction velocity in the elderly when compared with younger population. This decreased nerve conduction rate in elders who attributed to the trivial component of elevated reaction time.

The role played by the peripheral mechanisms to bring about psychomotor changes in the elderly requires more attention to draw any definite conclusion. Owing to the unsignificant role of peripheral factors in accounting the direct relationship between reaction time and age, it appears that central deficits may give some account to explain psychomotor slowness in the elderly. WELFORD (1965) accounts four changes in the CNS which may have an influence an psychomotor performance in elderly. They are:

(a) reduction in the signal strength and processing capacity owing to reduction in number of functional neuron cells;

(b) a noise in the older brain, which might be due to an increased random neural activity that might be acting while processing certain stimulus response events.

(c) the ability of the brain in processing the more recent activities may reduce due to longer " after effects " of neural activity which interfere or blur new signals coming to it, in the aged; and

(d) the optimum activity levels of central neurons or neuron sets might be diminished due to diminished aroused level of CNS in older people.

However there is no satisfactory explanation available to account the reduced psychomotor ability in the aged at present either through peripheral factors or through the central areas. This area still requires a thorough investigation.

Intelligence and Memory changes in the Aged:

Many investigators agree that the geriotric population performs poorly an traditional intelligence tests when compared to younger population.

BOTWINICK (1973) says that there is a definite reduction in intelligence as age increases. Aged performed better on verbal subjects and showed poor performance on performance subjects, when Wechsler Adult Intelligence Scale test was administered. These experimental data need not be true of all individual cases of aging because of number of important variables affecting the interpretation of these findings. Education is one such important factor affecting the interpretation and standardization of the intelligence tests.

BIRREN and MORRISON (1961) re-evaluated WAIS scores along two dimensions of age and education to discover that education level played more important role than the age in determining mental capacity. Failure to account the factor of education level greatly inflates the age related decrement in intelligence test scores.

The health of the subjects appear to have a significant impact on intelligence test scores. BOTWINICK and BIRREN (1963) observed healthier subjects performing better than others as WAIS though not their results did not statistically differ.

Most of the experiments to study the changes in intelligence in the aged involve cross sectional designs for obvious reasons. However, inspite of the findings those are observed as these studies, longitudinal studies may provide a better thought an intellectual decline in the aged. MlLLER(l977) observed that cross sectional experiments tend to overestimate the rate of intellectual decline as a function of age.

MILLER (1977) stated that " fluid " intelligence (the ability to adapt to new situations, adduce new relationships, acquire new ideas) in more susceptible to aging than - " Crystallized " abilities (learned intellectual skills) using the results of the experiment by cunningham clayton and Overton (1975) They (Cunningham etal) observed older subjects performing relatively poorer as fluid intelligence test such as Raven's progressive matrices. Any index of intellectual functioning involving considerable fluid skills, such as problem solving, creative thinking and developing new ideas; tend to decline with increasing age (BOTWINICK 1967, 1973) "The unequivocal presentations, by different researchers on the aspect of intelligence changes in the aged, warm us very much as the factors affecting them, to draw any single conclusion Memory, one aspect of intelligence is reported to be affected in the aged (GILBAR 1941; DAVIS & OBRIST 1966) GILBER & LEVEE1971 MILLER 1977) SCHOW, CHRISTENSEN HUTCHINSON & NERBONNEL 1978) assume that memory involves

(i) " a temporary short term storage of limited capacity and rapid decay " and

(2) a long term storage with considerable capacity and relatively more permanent fixation of traces. They consider there as two fundamental levels of Memory.

GIlBERT (1941), DAVIS & OBRIST (1966), GILBERT and LEVEE(1971) observed that older people performing poorer when compared to younger people on various tests involving memory BOTWINICK(1973) observed elderly people exhibiting more serious decay in the long term storage process than the younger ones after reaching a criterion level of learning involving memory

While interpreting any data involving memory, one has to be very careful with regard to the factors such as stimulus intensity, speed of stimulus presentation, attention and sensory modality used etc.

Psychological changes and Advancing age:

Many psychological changes have been reported to occur with increasing age. However it is difficult to draw a general conclusion about these changes because of unequivocal reports by researchers and because of the influences of critical factors such as family size, family attitude, health, site of residence, ethnic and religious background, education, socio-economic status, cultural background etc., which determine the psychological profile of an older individual. Personality changes such as disengagement, cautiousness and rigidity have been reported to appear with increasing age.

BOTWINICK (1973) observed that older people tended to withdraw and to become relatively more introverted because of dis-engagement. GUMMING and HENRY (196l) earlier had argued that disengagement was a natural and pre-programmed phenomenon. An individual starts becoming more pre occupied with himself and starts having less concern about his interpersonal contact. In addition to this the societal forces may play a crucial role of forcing withdrawal an aging people. Since, they (CUMMING & HENRY 1961) consider disengagement as natural event, they consider that elderly will usually be satisfied, happy and well adjusted inspite of disengagement, BOTWTNICK(1973) considers this happiness- disengagement relationship to be dependent upon the age of the person, his physical and mental status his personality, the social role he has the t pe of activity he has to engage himself and whether he has confidence or not, cautiousness and rigidity have been considered the other characteristic dimensions of personality changes in the senescence. It has been reported that older people make more cautious life decisions than the younger one.

Even when the potential for marked financial gain is present, the older tend to be more cautious in taking the decisions involving the finance (WALLACH and KOGAN 1961)

Rigidity or rigid behaviours implies stereotyped response patterns and resistance to change. Older subjects tend to offer such inflexibility. Lifelong personality characteristics, educational history intellectual skills cultural factors etc, seem to influence rigidity(BOTWINICK-(1973)

Depression is another personality characteristic that is attributed to the aged. This may be due to depressive episodes that occur in life. BOTWINICK(1973) feels this can be well combated if health professionals can help the elderly to restore the feelings of self worth, social contact and restructuring their life goals.

However there are reports with regard to personality being remarkably stable over the adult years (FOZARD and THOMAS 1975) Many factors are necessary for a stable personality. They are good health, vitality, family relationships, family attitudes, emotional support from family members, interpersonal contacts, reactions to the end of life, life review etc.

One should be very careful, while drawing a definite conclusion about psychological changes in the aged. The changes those may occur might be just reactions. The reactions might be similar to the reactions of the younger population to the same kind of situation. Segregation of all these factors and studying the young and old generations may throw certain light on the personality changes those occuring with age.

Theories of Aging:

There are several theories of aging. They can be broadly classified into two groups They are:

(a) Social Theories and

(b) Biologic Theories

(a) Social Theories: Social theories can be further classified into:

(i) Theory of Disengagement;

(ii)Developmental theory;

(iii)-Multiple cause- deficit repair and

(iv) Active Theory.

(i) Theory of Disengagement: This theory has been putforth by CUMMING (1960) and CUMMING & HENRY (1961) The theory is suggestive of the gradual withdrawl from society a mutual dis involvement of the elderly individual and the social forces, sorrounding him with growing age. This has been considered a natural phenomenon. This is attributed to increasing biological limitation, the perceived inevitability of death and the escalating loss of social relationships.

The theory considers the disengagement as a self-perpetuating process. In that, the search for the ties, by the aged becomes less rewards and the need for the new existence becomes more. The theory views two stages In the first stage of aging, there is a severance of bonds established by employment and family leadership roles. In the second stage, the femilial dependency plays a crucial role, because the aged individual will not be able to maintain behaviour necessary for health, cleanliness or propriety.

(ii) Developmental Theory of KASTENBAUK(1961, 1964): This theory considers old age, as culmination of personal growth and as an end of development. The Theory accounts for individual differences. It also provides for four alternatives: They are-

(a) development may stop at certain point but the life may continue;

(b) an individual may continue to develop never reaching an old age;

(c) an individual may continue to develop inspite of growing age but without reaching a plateau to characterize the old age.

(iii) Multiple Cause- deficit- repair- Theory of LINDSLEY1964:

The Theory implies the following:

(a) A longterm accumulation of deteriorating influences may increasingly manifest with fading youth;

(b) A life time physical and emotional abuses may culminate in specific behavioural deficiencies which can impede performance,

(c) A diversity of disabling physiologic and psychologic agents, acting upon the systems during earlier years may manifest themselves as multiple deficits both physiologically and mentally individual depending upon the person's ability to cope with and compensate for his unique arrays of disabilities

(iv) Active Theory: This theory stresses that there is one to one relation between the older person's decreasing level of participation in social activities and personal satisfaction of life.

HAVIGHURST and ALBRECHT(1953) reported that the older persons may be forced to reduce their social participation due to physical and enviornmental changes owing to loss of occupation or due to altered living conditions. As the activities in the enviornment reduce, the older individuals may feel segregated from the enviornment(or society) and their self image starts deteriorating (MADDOX and EISDORF3R, 1962)

The theory asserts that an aging individual can still life actively in the society, if he is able to substitute more realistic activities for the ones which he is made to or relinguished and if he is able to replace the old lost human associations with new human associations (HAVIGHURST,l963)

The active theory also assumes that the levels of activity patterns and value systems of middle age continue to be preserved in old age.

(b) Biologic Theories of Ageing: Biological theories of ageing can be further classified into:

(i) Genetic theory

(ii) Somatic mutation theory

(iii)Free Radical theory

(iv) Immunologic theory

(v) Hayflick's theory.

(i) Genetic theory: This theory suggests that Biologic ageing is strongly associated with heredity. It considers that the biologic process of ageing as most inevitable and immutable process. As the human body ages, its composite systems deteriorate but not in a uniform rate. In addition, there will be an accumulation of foreign substances within neural and muscle cells and some extracellular deposits that interfere with normal activity and the growth The Theory also presumes that physically, there is an outward similarly in aging characteristics within the Species, and also a consistancy among the above factors. It also argues strongly in favour of a genetic determination of aging. It postulates that the life span is programmed to extinction by specific genes coded for such terminal changes and that the information carrying molecules in these genes accumulates errors over time which can eventually interrupt cellular functions, causing degeneration and health.

Somatic Mutation Theory: This theory postulates that increasing longevity of bodily cells is accompanied by a progressively higher frequency of cell mutations. CURTIS & MILLER (1971) consider that this increase in the number of unseparable mutations interfere with the primary function of the cells and that the duration of life-span and the rate of ageing are directly related to chromosonal dangers occuring due to mutation process.

Free Radical Theory: A free radical is a type of molecule which produced by the reactions within the mitochondria and that possesses a free, unbound electron. Radicals that escape from their systems may react with other molecules, threatening the integrity and function of cellular activity. Free radicals can react with RNA, the messenger agent of DNA, causing errors in protein synthesis and this in the " script " that the cell is programmed to perform The degeneration of molecules and changes in body chemistry may account for the loss of tissue elasticity, arteriosclerosis and calcification commonly- observed among the elderly and longevity may directly proportional to the rate of metabolic degradation (HARKAN & PIETTE 1966)

Immunologic Theory: According to this theory cellular ageing represents a gradual decline in immunity to various disease states. As cells grow old, they undergo an immunogenetic diversification and they become less resistant to infections processes to which they were once naturally immuned (WOLFORD 1969, PETER 1973)

HAYFLICK'S THEORY: Microbiologist HAYFLICK'S(1973) investigations of human factors and adult cell strains, have attached considerable credence to the concert that ageing is an inherent property of normal cells, Cells possess an inherant biologic " time piece " that programs them for extinction. Arresting cell division for several years and then restoring there limits of the doubling process, nor did transplanting aging cells in to more youthful systems alter their proliferation The genetic program for growing old, apparently has a " memory ".

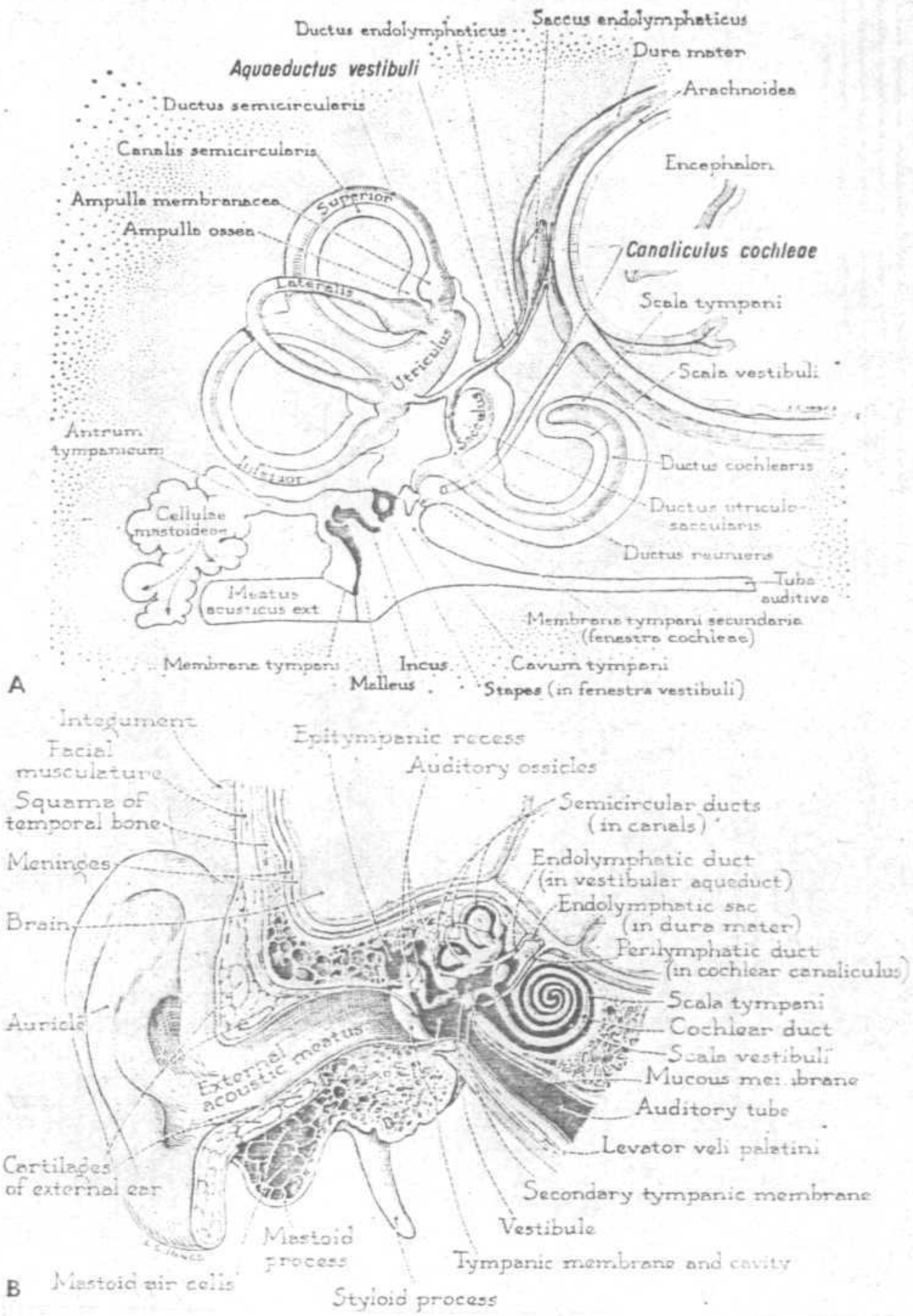


Fig: A- Schematic diagram of human ear.

B- Sectional diagram of human ear.

Chapter-III

NORMAL HEARING PROCESS

The knowledge about anatomic and physiologic bases of hearing will improve the clinician's ability in interpreting various audlological tests, in diagnosis of the - problem and in the rehabilitation of the patient. If one knows what is normal anatomy and physiology, then he can easily describe an abnormal anatomy and disturbed function

Auditory system can be divided into two parts . They are:

(i) Peripheral auditory system and

(ii)Central auditory system.

The peripheral auditory system can be sub-divided into:

(a) External Ear, consisting of 'Pinna ' or 'auricle' and 'external auditory meatus '. The pinna collects and directs the acoustic energy towards the middle ear via the external auditory meatus.

(b) Middle Ear consisting of tympanic membrane, ossicular chain and muscles. This (i) converts the acoustic energy into mechanical energy, (ii) matches the impedence between outer and inner ear, and (iii) protects the inner ear from loud sounds.

(c) Inner Ear consists of the organ of sense of hearing. It converts the mechanical energy that is fed by the middle ear, into electro-chemical energy and analyzes the energy that is reaching. That is to say that the sound energy that reaches the ear is transformed into electrical energy, through the electro-chemical reactions in the inner ear. This happens in the peripheral auditory system.

Central Auditory System: The electrical energy, the final outcome of the peripheral auditory system is further directed towards the central auditory system, via the auditory branch of VIII cranial nerve, in the form of neural impulses. These impulses pass through initial structures of the brain stem, lateral lemniscus, inferior colliculus, medial geniculate body, to reach the temperal cortex, via the auditory radiations. The information received by the peripheral structures are decoded at many stages, all along the central auditory system. Final decoding of the information is done at the level of cortex, that is temporal cortex for the rurpose of perception.

The major anatomic and physiologic features of the auditory system are briefly discussed here.

Peripheral Auditory Mechanism:

External Ear: It is outwardly visible. It's chief function is to collect and to direct the sound waves towards the middle ear. It has two major structures, (i) the auricle and (ii) the external auditory meatus.

Auricle: (Pinna) It is a prominent structure of the hearing mechanism. It is vaguely shaped in the form of a cup or funnel and located on both the sides of the head. It consists of several pieces of cartilages that are held together by ligaments and is covered by skin, (It has a number of land marks on its contoured surface. The outermost rim of the pinna is referred to as 'helix'. Another ride parallel to this is called ' antilelix ' The extreme lower portion is called

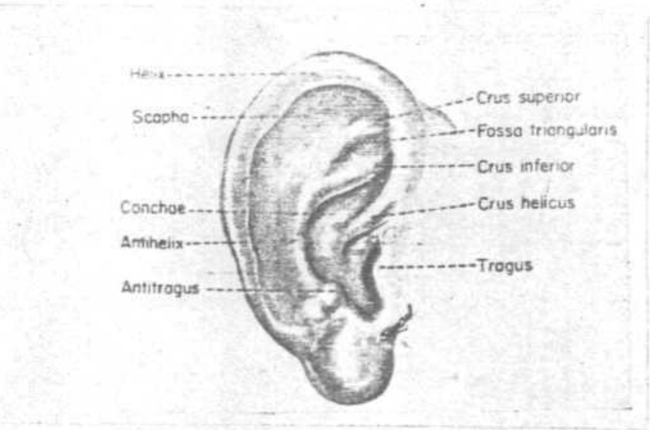


Fig: Human Pinna (Auricle)

'lobule '. Immediately above the lobule is the 'antitragus' A tiny protruding structure which is anterior to the opening of the ear canal, is the 'tragus' The depression in the pinna is 'concha ', which serves as the opening into the external auditory meatus.

The human auricle functions most efficiently as a sound collector for frequencies above 5000HZ The configuration of the ridges and depression of the auricle, function as a complex resonator for high frequencies. The vertical portion of the sound source to the auricle will provide cues for localization of sounds.

<u>External Auditory Meatus</u>:(Ear canal) This tube like structure is about 25 to 30 mm in length and (has a diameter about 7 to 8mm The outer two-thirds of the tube is cartiliginous and medial one- third is formed by the hard bone of the temporal part of the skull. The cartilaginous portion of the ear canal contains tiny cilia, and the subaceous glands. The subaceious giands have a secretory function. They secrete a waxy substance known as 'cerumen '. The presence of hairs and wax prevent the entrance of foreign bodies and insects. The ear canal is tilted at a slight upward angle as it traverses in to the skull' External auditory meatus obtains innervation from the V and X cranial nerves. The external auditory meatus directs the Sound waves towards the tympanic membrane and also behaves as a resonator (Rose 1978). The resonance provides l0-15dB ampli- ' fication in the region of 3000 to 4000 Hz.

Middle Ear: This is a cavity having a volume of about two cubic centimeters. It is enclosed within the temporal bone of the human skull. This cavity is situated between the external auditory meatus and the inner ear. The tympanum is lined by mucous membrane. Middle ear is a cubide six walls. It contains three auditory ossicles, air, muscles, ligaments and blood vessels. It also has nerve supply.

Lateral wall: It is formed by the tympanic membrane and a small portion of bone. It separates the ear canal from the middle ear cavity.

Tympanic membrane is made up of three layers of tissue. The outer layer, which is towards the external auditory meatus is made up of cutaneous tissue which is continuous with the tissue lining of the ear canal. The inner layer is composed of mucosal tissue which lines the entire middle ear cavity. The middle layer is composed of fibrous connective tissue, has medial and circular arrangements, having these three layers of tissue is termed as pars tensa. A small triangular portion of the drum, the "pars Flaccida " is located in the upper part of the membrane and it does not contain a fibrous layer. This makes pars flaccida more flexible than the parstensa, this allows the tympanic membrane to be highly responsive and mobile as sound waves impinge on it. The ear drum is cone-shaped, with its centre " umbo " attached to the handle of malleus. Tympanic membrane slightly protrude into the middle ear cavity. It is served by the branches of V cranial nerve.

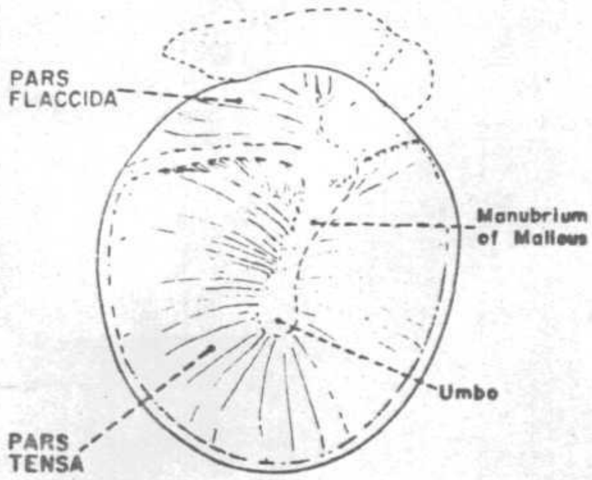


Fig: Lateral view of right Tympanic Membrane.

Medical Wall: It is a bony wall which is convex laterally. This c nvexity is due to the basal turn of cochlea. Due to the convexity there is a bulging portion, which is known as ' promontary '. This looks rough due to the tympanic plexus of glosso-pharyngeal nerve (ix). Just above the promontary there is an oval shaped opening, known as oval window/ " Fenestra Vestibuli ", which leads to "Scala Vestibuli " of cochlea. This oval window is covered by foot plate of the stapes. Just below the promontary there is a round opening known as ' Round Wincow/Fenestra Cochlea ' which opens "Scala tympany" of cochlea into middle ear. This round window is covered by a membrane known as secondary tympanic membrane. Above the promontary there are two bony elevations which are horizontal portions of the facial nerve canal, and the lateral semicircular canal.

Anterior Wall: It is very narrow and has four openings. The lower opening is the bony opening of the eustachian tube. Just above this there is an opening from which 'tensor tympany' muscle emerges.

Posterior Wall: Here there is a small projection known as 'Pyramid' which has an opening at its apex. Through this opening, the tendon of stapedius muscle emerges into the middle ear. Above this a small opening is present which is known as 'auditus' to ' mastoid antrum'. Through which the tympanum communicates with the tympanic antrum. Medial to auditus to mastoid antrum there is descending part of the facial nerve in the bony posterior wall.

Roof: It is formed by a plate of petrous part of the temporal bone, which is known as " tegmen tympani" . It separates the cranial fossa and tympanic cavity.

Floor: It is narrow and consists of a thin complex plate of bone which separates the tympanic cavity, from superior part of the internal jugular vein. In the floor there is a small aperture for tympanic branch of the 'Glosso-pharyngeal" nerve.

Ossicles: It is a chain like net work made up of three tiny bones, which connects the tympanic membrane and the inner ear. The first of these, the malleus, is attached to the middle connective tissue layer of ear drum. The next one is incus, is attached to malleus and also joins with the last ossicle, the stapes to form the entire ossicular chain. Stapes fits into the oval window, an opening of the bony covering of the inner ear.

Muscles and Ligaments: The entire ossicular chain is suspended by a series of ligaments which attach to numerous locations on the the three bones. These generally function to hold the ossicles in place during their vibration and to assist in the termination of ossicular movement once a sound has ceased minimizing the amount of distortion created in auditory system. There are two muscles the stapedius and the tensor tympani. The stapedius muscle originates on the pyramid of the posterior wall of the middle ear, and attaches to the neck of stapes. It obtains the nerve supply from facial nerve (VII cranial nerve) When this muscle contracts the stapes is pulled from the oval window, altering its normal movement.

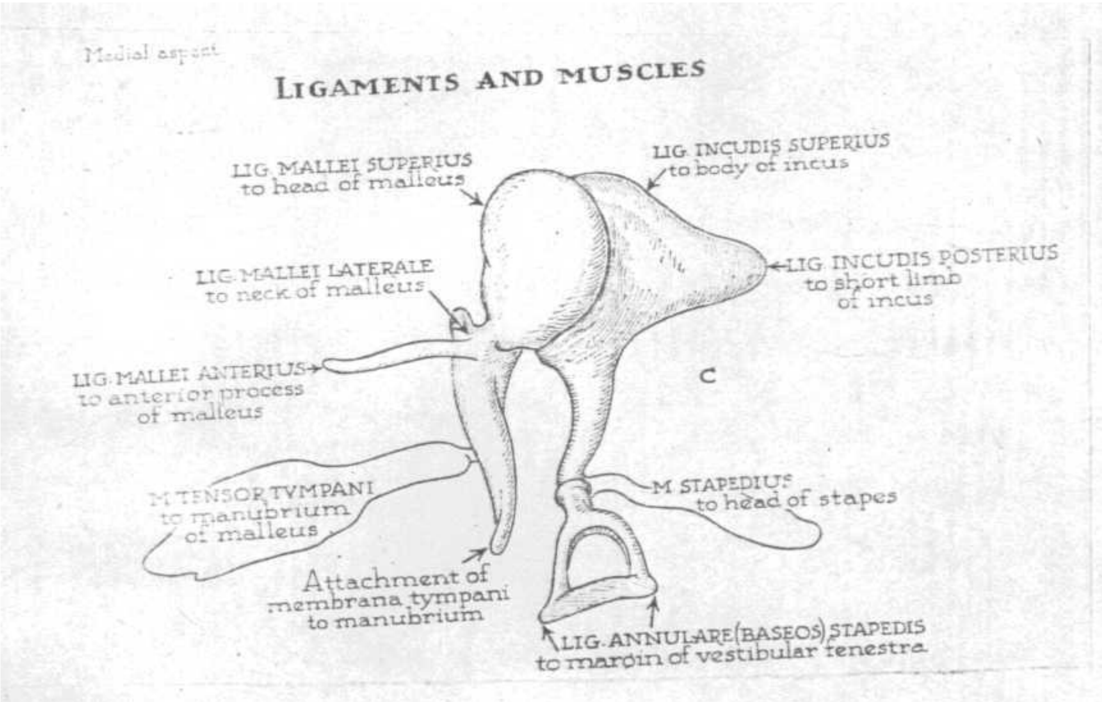


fig: Ossicals with Muscles and Ligaments.

The tensor tympani originates near the eustachian tube and attaches to the malleus. It's contraction exerts a strong pull on the malleus medially and anteriorly. This is supplied by V cranial nerve (Trigeminal).

The stapedius and the tensor tympani have been shown to contract automatically when an individual is exposed to intense auditory stimulation. The Contractions of these two muscles reduce the mobility of ossiclar chain and thus protect inner ear from damage resulting from exposure to intense sounds. Eustachian tube is a canal which connects the middle ear cavity with the nasopharynx. This tube is normally closed and can be opened through the muscular action associated with activities such as swallowing or yawning. It allows for an exchange of air in and out of middle ear cavity, thus equalizes the middle ear pressure, with reference to atmospheric pressure and it also drains the middle ear effusions to nasopharynx.

Middle ear primarily acts as the sound conduction mechanism from external ear to inner ear. Secondarily, it acts as an impedance matching transformer in matching the impedence of inner ear fluids with external ear. It also protects inner ear from being damaged by intense sounds by the contractile actionsof stapedius and tensor tympani muscle.

<u>inner Ear</u>: It contains the end organ of hearing as well as the end organ for balance. Both are en cased in the same bony capsule which is part of the temporal bone of the skull. The vestibular system,(the system covering balance) is composed of several critical structures, which include the saccule, utricle

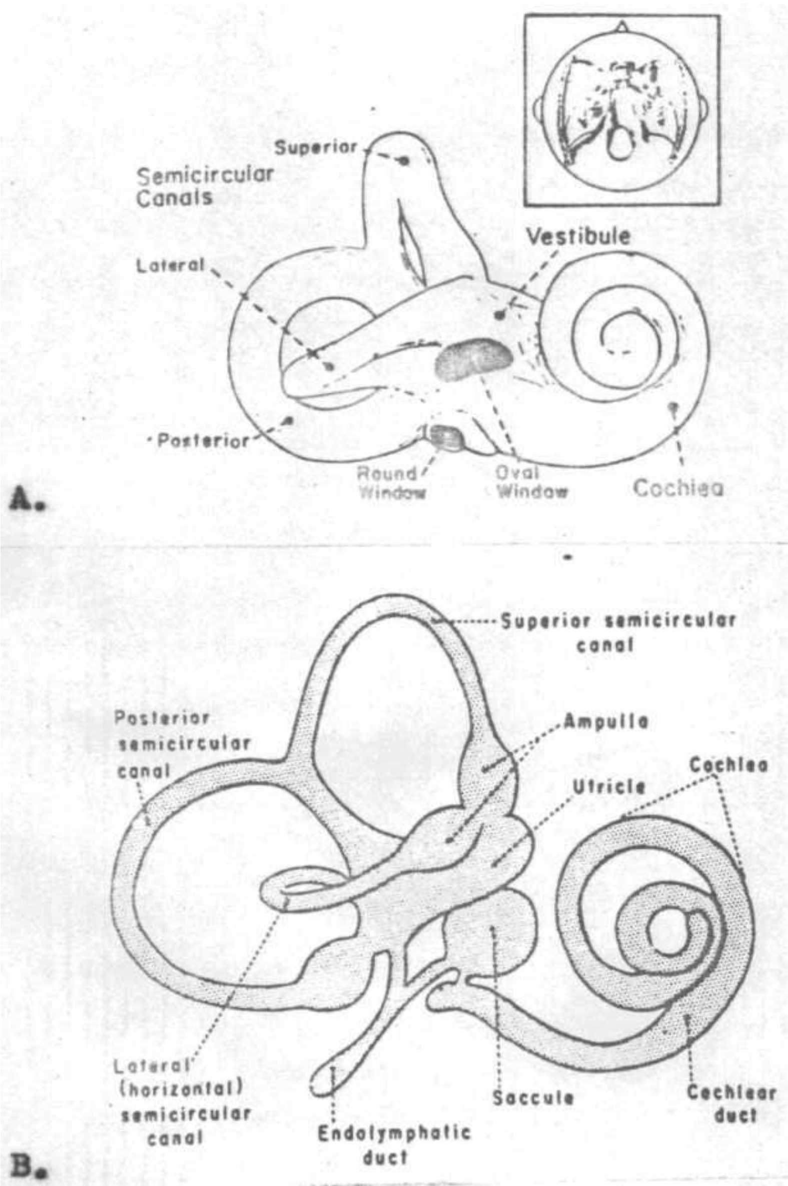


Fig: A- Right bony Labyrinth.
B- Right Membranous Labyrinth.

and three fluid filled semi-circular canals. These components work together in a complex fashion to provide us with much needed information pertaining to equilibrium.

In this paper our concern is with the end organ for hearing This portion of inner ear is termed as Cochlea It contains a highly complex net work of structures which are vital to the process of hearing. The inner ear contains both a bony/osseous labyrinth and a membranous labyrinth. The osseous labyrinth is a series of cavities- enclosed in the temporal bone while the membranous labyrinth is a network of fluids and tissues found within the bony labyrinth.

The cochlea, is a coil shaped, some what like that of a small snail shell. The foot plate of the stapes fits into the " oval window" which serves as an opening through the bony capsule into a space within the inner ear referred to as the vestibule. The vestibule is a common area within the inner ear for both the cochlear and vestibular systems and is connected directly with he basal portion of the cochlea.

The cochlea consists of three canals called as the "scala vestibuli " Scala media " and " Scala tympani" because of the presence of two membrane called "Reissners and Basilar membrances Scala vestibuli is a canal which runs from the vestibule till the apex, where as the scala tympani is a canal that runs from apex till the secondary tympanic membrane. The Scala media is the space in between the Reissner's membrane and the Basilar membrane.

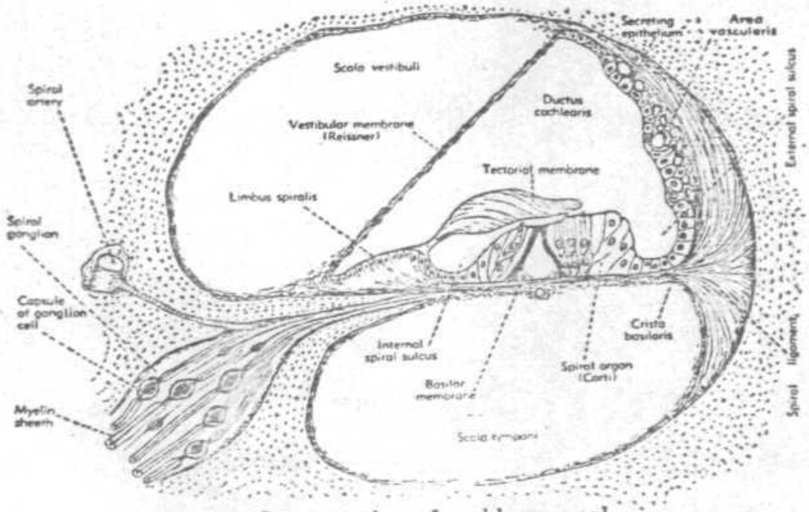
The scala vestibuli, and Scala tympani are filled with a fluid called "perilymph " as is the vestibule.

The Scala media is located between Scala Vestibuli and Scala tympani and is filled with a fluid called "endolymph ". " Reissner's membrane separates "Scala media " from the "Scala vestibuli ", while the " Basilar membrane " separates it from Scala tympani. In the Scala media, and placed on the Basilar membrane, is the organ of corti, the end organ of hearing The organ of Corti is made up of three rows of outer hair cells and a single row of inner hair cells, which have finy cilia on their superior surface that are in contact with the tectorial membrane, a structure which hangs over the hair cells

Nerve fibres associated with the auditory nerve are embedded in the basilar membrane and innervatie each of the more than 20,000 inner and outer hair cells in this very complex sensory structure

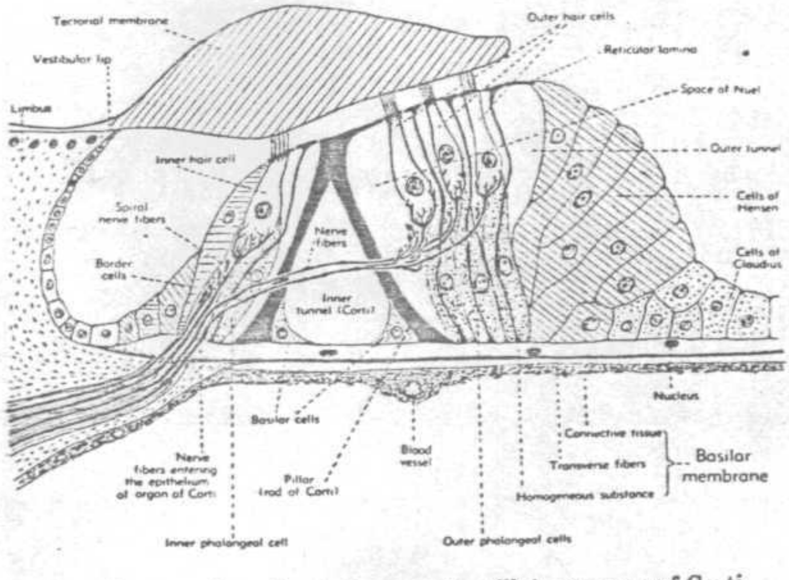
Movement of the stapes in and out of the oval window creates displacement within the perilymph , causing the round window to bulge in and out in response to the action of stapes. This fluid movement is also transmitted to the scala media and the sensory structures of the organ of corti.

Displacement of the endlymph and the basilar membrane occurs Movement of basilar membrane triggers the movement of the hair cell cilia through a shearing action which occurs between the basilar membrane and the fectorial membrane. This action indicates the electro chemical changes within the hair cells, which make the nearly nerve endings trigger neural impulses.



Cross section of cochlear canal

A



Cross section of spiral organ (papilla) or organ of Corti

B

These impulses are carried through the auditory nerve eventually to the next, major devision of the auditory syste-, the central auditory mechanism.

Central Auditory Mechanism:

Although basic intensity and frequency coding of auditory information occurs within the Cochlea, portions of the Central auditory tract provide further coding. All of this information is then processed by the important structures of the cen ral auditory system for the purpose of perception. We can divide the central auditory net work into two major divisions:

(i) The brain stem and

(ii) the cerebral auditory cortex.

Each division contains several important centers of neural activity along the afferent path-way of the auditory network which are briefly discussed here.

Eighth nerve and Brain Stem: Neural fibres from the hair cells within the cochlea collect within the modiolus to form spiral ganglia and then through the internal auditory meatus. This is the first of the auditory neural junctions and termed as ' first order neurons '. The cochlear portion of the auditory nerve (VIII C.N.) enters the brain stem at the junction of the medulla and the pons, and here the fibres split into two branches with one group proceeding to the ventral cochlear nuclei and other branch of fibers going to dorsal cochlear nuclei

The second order neurons begin at the cochlear nuclei. Some of them, then begin the processof decussation in which

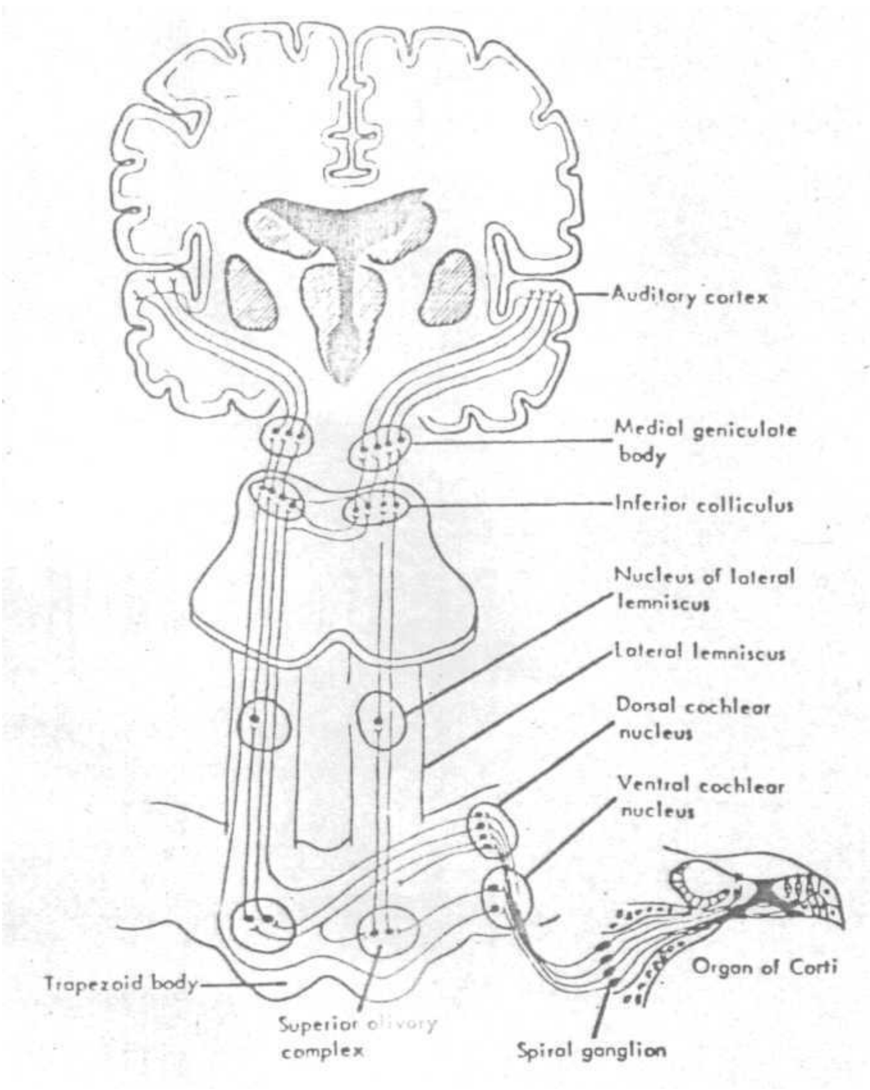


Fig: Ascending pathways of Cochlear branch of the 8th cranial nerve.

neurons cross to the contralateral side of the brain sterr via the trapezoid body and terminate within the superior olivary complex. The fact that nerve fibers from each ear do decussate here and at higher levels of the neural tract makes it less likely that neural destruction on one side of the central pathway will result in a total loss of hearing in that ear. Other second order neurons emerge from the cochlear nuclei and remain on as ipsilateral ascending tract. Sorne of these fibers proceed to a second neural junction, the superior olivary complex, before ascending as third order neurons through the lateral lemnisgus after leaving the cochlear neclei. All fibers reach the next neural junction, the inferior colliculus A portion of the neurons may pass through the inferior celli-culus, going directly to next point the 'medial geniculate body Further decussation of neurons occurs at the level of the inferior colliculus, which is felt to be the center for reflexive response to sound.

Cerebral Auditory Cortex: Apparently all ascending neurons synapse with neurons in the neucleus of the medial geniculate body The fourth or fifth order neurons, then travel through the area referred to as the auditory radiations, where the fibres are fanned out. These fibers then terminate in the auditory cortex. This particular portion of the cerebral cortex found in temporal lobe, below the sylvian fissure and bound posteriorly by the parietal and occipital lobes. Audition stands as the temporal lobes primary function. According to Brodman's numbering system, this particular region of the cortex includes areas 41 and 42 considered to be the primary acoustic reception areas.

The entire process of audition, particularly for complex stimuli like speech, requires the active participation of other portions of the cortex in addition to those already identified Working in conjunction with the anterior auditory reception areas are more posterior and superior auditory areas of the cortex. It is here that functions such as auditory recognition, association, and recall are th0ought to be accomplished. This is felt to be particularly true of the superior portion of the temporal lobe, where Wernicke's area is located.

The temporal lobes of both hemispheres of the brain appear to play very important roles in the process of audition Although decussation of ascending neural fibers does occur to link each of the two peripheral auditory systems, the two hemispheres are believed not to duplicate all functions in the processing of auditory stimuli. Rather it is felt that there is some degree of difference in between the hemispheres and specificity in audition. In most individuals the right ear appears to be able to retrieve more information than the left. The perception of speech in particular seems to be controlled for the most part by the left hemisphere, while the processing of nonverbal auditory stimuli is carried out predominantly by the right hemisphere. Yet even though a certain degree of specialization exists between the two auditory areas of the brain, a high degree of redundancy in functions of the two lobes is present.

Chapter-IV

The Aging Auditory Process

The human hearing system is a marvelous complex system. It is operationally mature even within the unborn fetus. The normal hearing mechanism provides the brain with a continuing nourishment of acoustic stimulation throughout the life. But this sensory system also changes in its structure, and in its sensitivity as the age increases. There has been an abundant literature which has reported changes in the auditory sensitivity and auditory system with increasing age.

The effects of aging on man's auditory system have been noted in the literature since the late 1800's(ZWAARDEMAKER-1894) Early investigations of this process focused on the structural changes; found to occur within the cochlea, for an extended period of tire. It was generally believed during those years, that the effects, of aging on audition were confined to this particular portion of hearing mechanism.

More recent investigations, however, have disclosed that any portion of auditory system is not immune to senescent alterations.

The decrease in auditory sensitivity with increasing age is referred to as presbycusis, that is, hearing loss securing with increasing age. The pathology of presbycusis has Shown to involve all the major divisions of peripheral and central auditory mechanisms.

SCHUKNECHT has contributed much towards what is currently known as presbycusis. This is especially true of the structural changes that can be associated with aging within the inner ear and the neural structures of the auditory system. The influence of genetic and enviornmental factors on presbycusis also have been studied. What ever the etiology may be, the fact remains that substantial alterations do occur in the entire auditory system of the elderly.

Changes have been reported to occur in the following divisions of the auditory system:

(a) the External Ear,

(b) the Middle Ear;

(c) the Inunerear and

(d) the Central Auditory System.

(a) Changes occuring in the External Ear with increasing Age:

The physical structure of the outer ear undergoes a number of changes FOWLER(1944) noted that changes begin to appear in individuals between 40 to 50 years of age. The skin becomes less resistant with aging, and there is shrinkage in its tissue. In addition, there might be a hair growth which proliferates along the periphery of the helix, antihelix, and tragus of the pinna. WILLEFORD (1971) observed that, these large, terminal hairs tend to have a wirelike texture, similar to brush bristles.

According to TASI et al(1958) there is an increased length and width of the pinna during the process of aging. The gradual loss of skin elasticity, muscle tenicity and the longitudinal forces of gravity are reported to contribute toward the physical enlargement of pinna.

Are, these age-related changes in external ear going to affect sound transmission to the middle ear? is the question that has to be answered. .

It has been reasonably documented that convolution or 'surface ridges ' of the pinna alter the frequency response of incoming complex signals. Angular resonance created by these convolutions provide an acoustic gain for high frequency components, which are essential for speech intelligibility. Normal ear canal resonance also selectively amplifies certain high-tone energies (above 2500 Hz) These characteristics of the normal pinna permit an input filtering process; that enhances speech discrimination. The outer ear plays a role in localizing both the direction and elevation of sound, it is angular shape enables a comparison between reflected and incidental sound waves, thus providing a peripheral model for gross sound localization, (BRTTEAU 1968; GATEHOUSE & OESTERRECH 1972) This structural capability, when enhanced by head movement and by the contribution of additional information received by the second ear, supports the ability to hear meaning ful signals in adverse noise conditions. Hence the changes in pinna with increasing age may contribute its part towards the hearing loss due to aging. The classical characteristics of hearing loss due to aging are (a)high tone hearing loss,; (b) reduced speech discrimination and (c) difficulty to hear in noisy enviornment.

PERRY (1957), revealed that there will be a decrease in the number of wax producing ceruminous glands, sweat glands and the tendency for ear wax to be some-what drier among older people.

Following are the certain problems of the external ear those interact clinically with the assessment of elderly -

(a) Hardened and sometime long standing wax deposits;

(b) Terminal hair growth;

(c) Prolapsed ear canal;

(d) collapsing ear canal and;

(e) decreased tactile sensation.

It seems doubtful that alterations in the external ear that are solely due to longevity have any pronounced effect on audition. Although functional changes in the pinna may alter, to some extent, the frequency response of the system. There is no research held has thoroughly investigated this aspect, and it's consequences on peripheral hearing. The incidence of prolapsed and collapsing canals among the aging is unknown but is likely to be quite rare.

The Middle Ear:

A number of age-related changes have been observed within the delicate structures of the middle ear. These alterations may result in some degree of auditory dysfunction.

Many investigators have reported sore alterations in the tympanic membrane, but the exact nature of these alterations are still not known.

COVELL(1952) pointed out that there is thinning and loss of rigidity of the tympanic membrane. ROSENWASSER (1964) noted the tympanic membrane of the elderly to be often thin and translucent. KLOTZ(1963) reported a sclerotic thickening or a nonreflective tympanic membrane to be present in older patients having chronic rheumatism and arthritis. MAURER and RUPP(1978) reveals that the drum is more translucent and rigid than former years, with adjacent ossicular land marks more visible; in the aged.

Substantial evidence is available regarding the development of increased rigidity of three bones of ossicular chain. ROSENWASSER (1964) discussed the ossification of the malleo-incudal joint with calcification in the articular cartilage. He also noted the presence of ossicular atrophy, particularly in the crura of the stapes, in the elderly. BELAL(1974) reported the incidence and severity of involvement of the incudomalleal and incudostapedial joints to increase significantly with progressing age. These alterations occured in both the ears and in both the sexes. The earliest signs consisted of fraying of the articular cartilage and the appearance of fibrils and vacuoles in the ground substance of the joints. Later changes included thinning and calcification of the cartilage atrophy and hyalinization of the elastic capsule surrounding the articular border of the cortilage and atrophy of the articular disc. More severe arthritic changes have been reported to occur among individuals of over 70 years of age. They include complete fusion of the space between the long bone joints.

Owing to above changes, the middle ear compliance may decrease with increasing age.

KLOTZ(1963) and CREBSE (1963) have reported the sclerosis of the joints of the ossicles to occur with increasing age. This results in increased rigidity of these bone. However, ETHOLM and BELEL(1974) have concluded that the above mentioned alterations may not affect the hearing.

A general degeneration and atrophy take of the tensor tympani and the stapedius muscles of middle ear and their ligaments have reported to occur. SCHUKNECTH(1955), KOT KLOTZ(1962) and GOODHILL(1969) have reported a reduced elasticity and atrophy of muscle tissue and calcification of the ligments. COVELL(1952) and ROSENW ASSER(1964) have stated that the deterioration in the functions of the two middle ear muscles may lessen the amount of protection provided by these structures of the ear, during the contraction of these muscles, in the presence of intense noise. DAVIS(1970) reported that degenarative changes in ligaments and muscles of middle ear may result in the inefficient operation of the ossicles, thus causing a minor decrease in the hearing acuity and producing some degree of distortion within the conductive mechanism.

DAVIS also noted the potential for the atrophy of the ligaments and muscles of middle ear to result in the production of less tension on the tympanic membrane, waking the membrane somewhat flabby and making it less able to respond to incoming sound waves.

The muscles responsible for the opening of the Eustachian tube has also been reported to under-go an atrophy Thus they do not function efficiently. Due to this the ability

of the Eustanchian tube to open properly, may be impaired. This inability may create a negative pressure in the middle ear cavity. MAURER and RUPP(1979) stated that increased patency of the Eustachian tube may cause the older person to hear his own voice more loudly. This condition is referred to as "autophony". Changes in elasticity and the displacement of the tissue in the nasopharynx may cause the mouth of the Eustachian tube to resist opening, resulting in an increase in the negative pressure in the middle ear.

Although the notion that conductive Presbycusis is a characteristic of the auditory aging process, it has achieved little consensus. Histologic reports indicate that these alterations range from subtle to profound, within this highly divergent population. Unless the changes are pronounced, they may not be detected by conventional audiometric measures or by visual otologic inspections. Hence, it is most important that any involvement compounding an already present sensorineural loss should be investigated. Obviously, there is a rather high premium on any sensitivity that can be restored through medical intervention. Consequently, audiologic assessment strategies, aimed at isolating possible transmissive lesions, should not be ignored simply because a person is old.

Inner Ear: Many researchers believe that the inner ear is the primary location for presbycusis. The degeneration of certain structures in the inner ear during old age has a substantial effect on hearing in elderly people. The aging process is going to affect the critical components of cochlea and other parts of Central auditory system. A review of the changes which occur with aging process are also discussed here.

SAXEN AND FIENDT (1937) noted the severe atrophy of organ of cortj is aged people. JORGENSEN(1961) found organ of Corti in a collapsed state and it is reduced to a small bulge on the basilar membrane and stated that at this stage it is virtually impossible to discern any anatomic details. SCHUKNECHT(1955) observed the atrophy of organ of corti in very specific area of the basal end of the cochlea and stated that, atrophy to be more at the basal end of the cochlea that at its apex.

A reduction in number and size of hair cells and supporting cells has been reported by many investigators SAXEN 1952; SCHUXNECHT 1955, 1964; JORGENSEN 1961; HANSEN and RESKENIELSEN 1965) SAXEN (1953) showed the flattening of the whole Organs of Corti and also showed Reissners membrane to be adherent to the hair cells. C0VELL and ROGERS (1957) study suggested that the epithelial lesions to be secondary to degeneration in the spiral gangliors.

SAXEN(1952) and SCHUNKNECHT(1955) have also supported this view. A comparison of available audiometeric data, with the histopathologic findings, gives a poor support to the view that these epithelial lesions in the cochlea have an anatomical base for presbycusis. JORGENSEN(1961) described these changes in all age groups, even down to the age of 7 years. SAXEN (1952) often found minor changes in anatomy in patients with major hearing defects and major lesions in patients where a minor hearing deficiency existed. Contrary to what one could except, the major pathology is not always found in the basal end of the cochlea(CROWE, et al 1934; SAXEN 1952; FLEISHER 1956; COVELL and ROGERS 1957). It is possible that these epithelial alterations are partly or wholly due to the agony of the patient or due to post mortem destructions or due to artifacts in the fixation(LANGE 1937; COVELL 1952; SAXEN 1952; JORGENSEN 1961) . HANSEN and RESKE-NIELSEN(1963-1965) reported no epithelial changes in the cochlea, in two patients. They studied the cochlea ten minutes immediately after the patients' death.

Calcareous deposits in the thickened basilar membrane and alterations in the stiffness of basilar membrane associated with aging were observed by MAYER(1920) and CROW et al (1934). CROWE et al (1934) reported some calcification and hyalinization at the very basal end of the basilar membrane. SCHUKNECHT(1967) observed the rupture and atrophy of basilar membrane, with the thinning of the membrane occuring at the site of the rupture.

CROWE et al (1934), SAXEN and VON FIENDT(1937),and SCHUKNECT(1964) reported the structural atrophy of stria-vascularis resulting in substantial interruption of the transducer activity within the cochlea. The degeneration of stria vascularis is a major factor in explaining the depression in hearing acuity observed in presbycusis; because the stria vascularis is felt to be the source or production of the endo-lymph (ZEMLIN 1968) and it is also felt to be the source of the D C potentials in the cochlea. The chemical composition of endolymph can alter concurrently with aging of auditory system (GOODHILL and GUGGENHEIM 1971).

CROWE et al (1934); SAXEN (1952); SCHUKNECHT (1955 & 1964): FLEISHER(1956) JORGE3EN(1961) constantly found the reduction in the number of ganglion cells in the spiral ganglian in the cochlea of the old patients and animals. Here again a comparison of the available audiometric data with the pathologic findings gives poor support to the view that these alterations in the spiral ganglion can bring about major changes and vice versa. SAXEN(1934) and HANSEN & RESKE- NIELSEN(1965) found a loss of less than 50% of the ganglion cells of the basal coil in presbycusis patients. FABINYI(1931) and CROWE et al (1934) found a correlation between frequency loss and the ganglion cell destruction found in the corresponding coil of the cochlea But unfortunately, the number of ganglian cells which can be damaged with out affecting the threshold for pure tones is given as high as 50% (CROWE et al 1934) and 80% (CITRON etal 1962

Central Auditory Mechanism:

The loss of neurons throughout the entire CNS system has been shown to begin early and to continue throughout the life. This loss of neurons becomes vigorous at old age (BRODY 1955). The degeneration of ganglion cells of the auditory nerve initially termed as 'Neural atrophy " has been changed to 'Neural Presb cusis' (SCHUKNECHT 1955, 1964). NEFF(1947)reported that the partial section of the auditory nerve in man results in a high-frequency hearing loss. However there is no parallelism between nerve fibre population and pure tone threshold. SCHUKNECHT and WOELLNER (1953) and CITRON et al (1963) found a normal threshold for puretones despite of severe degeneration of the cochlear nerve. The threshold loss in presbycusis is due to deficits in the population of the 1st, 2nd, 3rd and 4th order neurons. (SCHUKNECHT 1955). JORGENSEN(1961) found no loss of acoustic nerve fibers in patients with presbycusis.

CROWE et al (1934) and HANSEN and RESKE NIELSEN(1965) reported only slight degeneration in the peripheral part of the auditory nerve, but severe vascular alterations in the internal auditory meatus. COVELL and ROGERS(1957) and SERCER and KRMPOTIC(1958) report a narrowing of the openings of the tractus spiralis foraminosus by hyperostotic deposits. They would exert a continuous pressure and thus cause an atrophy of the acoustic nerve fibers, resulting in 'pure idiopathic presbycusis'

Asone cochlea is represented bilaterally, extirpation of one hemisphere does not cause threshold loss for pure tones in man (DANDY 1933) Bilateral temporal lesions, result in severe to total loss of hearing. KIRKAE, SATO and SHITARA (1964) found atrophy of neural structures in the ventral cochlear nuclei, the superior olivary complex, the lateral lemniscus, the inferior collicular and medial geniculate body and in the auditory cortex itself. SCHEIDEGGER(1963) mentions how in old patients the central pathways and nuclei are affected by general diffuse atrophy. HANSEN and R3SKE-NELSEN(1965) found severe degeneration in the glial part of the acoustic nerve as well as in the white matter of the brain stem and in the hearing centers. Alterations were most pronounced in the white matter of the hemispheres, next in the brain-stem and finally in the nuclei and the cochlea.

Bioelectric Potentials: PESTAL0ZZA (1956) and PESTALOZZA et al (1957) measured the Action potential, the cochlear microphonic and the summating potential of senile guinea pigs. They found that:

(a) The cochlear microphonic was smaller than in young animals. This was reported to be partly due to conductive hearing loss.

(b) Even with normal cochlear microphonic the Action potentials were less than in young animals. This was attributed to the loss of neurons in the spiral ganglion.

(c) The summating potentials were also reduced in old animals.

Cardiovascular Disease and Presbycusis:

Extensive Vascular lesions in the ear of the aged have been observed by many investigators (CROWE et al 1934; FIEANDT and SAXEN 1937; SAXEN 1952; SCHUKNECHT, 1955 & 1964; JORGENSEN 1962; HANSEN & RESKE-NIELSEN 1965). These local changes are not necessarily part of a general vascular disease. SAXEN(1952) states that the Vascular changes are. responsible for the epithelial lesions in the organ of corti. CROWE et al (1934) JORGENSEN(1961) and SCHUKNECHT(1964)describe extensive changes in the striavascularis, without any evidence of damage to the organ of Corti. BUNCH(1929-1931); PESTALOZZA & SHORE (1955); KLOTZ & KILBANE (1962), MILLER & ORT (1965) found no relation between presbycusis and cardio-vascular diseases . In contrast with these findings and opening, a positive relation between cardiovascular disease and presbycusis is suggested by SCHUKNECHT (1964); WESTON(1964) and ROSEN (1962, 1964, 1965).

TYPES OF PRESBYCUSIS: The histopathological and the patho-physiological (audiological) findings in the aged auditory system are not constant. For this reason a number of authors distinguished different types of presbycusis.

In 1955 SCHUKKECHT distinguished presbycusis due to epithelial atrophy and due to neural atrophy.

Presbycusis due to Epithelial Atrophy:

This is characterised by atrophic degenerative changes in the membranous cochlear labyrinth, including afferent and efferent nerve fibers. The loss of hair cells and spiral ganglion cells run parallel. The most severe changes are located in the basal coil. The process begins in the middle age and progresses slowly. Even at advanced age the lesion is reported to be limited to the very Basal end of the cochlea, and it does not or barely affects the speech frequencies.

Presbycusis due to neural atrophy: This is characteristed by a decrease in number of neurons of the auditory nervous pathway. It's onset is usually in older age and is superimposed upon the Epithelial atrophy.

Again in 1964, SCHUKNECHT described four different kinds of presbycusis:

(a) Sensory Presbycusis: This type is similar to the presbycusis due to epithelial atrophy as explained before.

(b) Neural presbycusis: This is the type described before as presbycusis due to neural atrophy. The hearing disability is characterised by a discrepancy between speech discrimination score and pure tone threshold, known as 'Phonemic regression' (GAETH 1946).

(c) Metabolic Presbycusis: The stria Vasculoris is an important structure in metabolic processes, simulating a battery providing a D.C. potential (DAVIS et al). An impairment of this structure results in an impairment of the bio-electrical process of the cochlea. Hence hearing loss. In this cases we can see a flat audiometeric curve.

(d) Mechanical Presbycusis: This isdue to a stiffening of the basilar membrane. The hair cells, the spiral ganglion and the stria vascularis show only minor changes. The functional correlate is a descending audiometric curve.

SAXEN (1962) classified presbycusis in man into three groups:

(a) Senile atrophy of the spiral ganglion: In this group a reduction in the number of ganglion cells in the spiral ganglion is found; mainly in the area of the basal coil. Normal ganglion cells can be found in the corresponding part of the organ of corti. This group is more or less identical to the Neural presbycusis of SCHUKNECHT.

(b)Angiosclerotic degeneration of the inner ear: The organ of Corti is flattened. Hair cells and supporting cells are reduced in number and size. There is widespread angiosclerosis of the blood vessels of the inner ear. In most of these cases a typical nephrosis is found. Angiosclerotic degeneration is seldom met as an independent disease. Usually a senile atrophy is also found in these patients.

(c) The central nervous system: There was no histopathologic changes found in cochlea and acoustic nerva. Blood vessels of the brain were reported to be intensely sclerotic.

Audiological Manifestations of Ageing:

It has been known for many years that the hearing acuity is diminishes in the old age, partic larly for high frequencies. The ageing process produces systematic changes in each of the two critical dimensions of hearing impairment- loss in the threshold sensitivity and loss in the ability to understand suprathreshold speech.

Pure Tone Threshold: ZWAARDEMAKER (1899) was the first to investigate this hearing acuity quantitatively in the elderly and he noticed that high frequency thresholds diminished with the increasing age of the patient and this process starts as early as the second or third decade of human life. BENZOLD (1894) STRUYKEN(1913), CICOCCO(1932) and SCHOBER(1952) have confirmed above findings. In 1929, BONCH used the electronic audiometer first time to determine the threshold for pure-tones in aged people.

Quantitative data for the pure tone threshold in different age groups have been collected by many investigators (KELLEY 1939; FOWLER & FOWLER 1936; BEASLY 1940; STEINBERG, MONTGOMERY, & GARDNER 1940; J0HAN3EN 1943; LEISTI 1949; GLORIG, SUMMERFIEID AND NIXON 1950; WEBSTER, HIMES & LICHTENSTEIN 1950; SATALOFF & MENTDUKE 1957; PIALOUX 1958; CORGO 1960; GLORIG & NIXON 1960; HINCHEUFFE 1959; JATHO & HECK 1959; GLORIG I960; GOETZINGER, PROUD, DIRKS & EMERY 1961; SKLAR & EDWARDS 1962; KLOTZ & KTLBANE 1962; CROSO 1963, MLLER & ORT,1965; SATALOFF,VASALLO & MENDUKE 1965). But it is necessary to realize that this is certainly incorrect. Presbycusis is a theoretical entity which could be defined as the hearing loss that results from the aging process. Unfortunately the aging of the individual is accompanied by a number of factors, such as exposures to noise, ear diseases, durgs taken etc. that in their turn can contribute to the hearing loss of aging patient, in addition to aging alone.

Later research served to provide further support concerning the influence of age on hearing; and also to provide documentation regarding the effect of sex on the progressive loss of acuity in the aged . The pure tone threshold results obtained in the National Health Survey (1962) in USA, demonstrated;

(a) an initial deterioration in hearing sensitivity to occur relatively early in life, with measurable changes observed in early adulthood;

(b) the males to experience sensitivity decrements at an earlier age than the ferales, with relatively large differences in thresholds occuring between the sexes by the third decade of life and ;

(c) the hearing loss to be more pronounced in higher frequencies .

RAIFORD(1931), CIOCCO(1932) JOHANSEN(1943)m, SATALOFF & MENDUKE (1957) HINCHCLIFF(1959), JATHO & HECK(1959) CORSO (1959 & 1963) MILLER & ORT (1965) reported a definitely poorer hearing in men than in women, especially at higher frequencies for the age groups of 40 years and above. But FOWLER & FOWLSR(1936); KlOTZ & KILBANE (1962); LEISTI(1949) PESTALOZZA & SHORE (1955) G0ETZINGER et al(1961) found no significant difference between hearing . threshold of men and women at old age.

It is generally accepted that preshycusis is sensory neural hearing loss; but recent studies (PESTALOZZA. & SHORE 1957; JATHO & HECK 1859; GLORIG & DAVIS 1961; ROSEN et al (1964, 1965) NIXON et al (1962) have shown ahair-bone gap in aged people and attributed that to conductive component i.e., middle ear changes due to ageing.

Speech Discrimination: It is a well known clinical fact that in presbycusis the intelligibility of speech is seriously affected and speech discrimination in the aged is frequently much poorer than is suggested either by pure tone audiometery or by speedh reception threshold (CAWTHORNE 1951, KONIG 1957; SATALOFF & MEADUKE 1957, KOLTZ & KILBANE 1962).

According to BOCCA (1958) discrimination difficulties of the aged are mainly of a cortical nature. CALVI & FINZI (1957) explain the poor discrimination as a diminished integrative capacity of the aged. The reduced ability to hear and to repeat common words at all supra-threshold levelsa as found in the aged was named " Phonemic Regression " by GAETH(1946).

PESTALOZZA & SHORE(1955) described how the discrimination becomes poorer as the hearing loss becomes greater. The discrimination was found independent of the shape of the audiogram. However, for spondees the hearing loss was smaller when the audiogram showed a steep slope instead of a flat sloping curve.

HUIZING & REYNTJES(1952) and HIRSCH, PALVA & GOODMAN (1954) found a low discrimination score usually together with recruitment. PESTALOZZA & SHORE (1955) and VAN DER WAAL(1962) however found in presbyjcusis poor discrimination combined with the absence of recruitment.

Time Factor & Speech Discrimination: According to FOURNIER(1954) the reduced speech perception in aged persons is partly due to a lengthening of the time required by the higher hearing centers including the cortex, to identify the message.

BORDLY & HASKI S(1955); FINZI(1956) CALEARO & LAZZARONI-(1957) DEQUIRAS(1964); STICHT & GRAY(l969); SCHOK (1970) and JERGER(1973 KONKLE, DEASLEY & BESS (1977) have demonstrated that the aged find difficulty in the discrimination of time distorted speech. These studies have found that time-compressed speech will create more errors in aged when compared with the performance of young listeners with similar hearing acuity.

Studies by WARREN (1961) WARREN & WARREN (1966) and OBUSEK & WARREN (1973) have shown a gradual decline in the number of verbal transformotions which occur in older subjects. WARREN(1976) has suggested that the reduction may be attributable to a reduced capacity for short-term storage of verbal information.

Special Tests:

In presbycusis the SISI score is quite unpredictable. At frequencies below 1000 Hz it is invariably low. Above 1000 Hz , however, the score can go to 0 to 100 % .

Usually in Presbycusis, there will be no recruitment or partial recruitment and in very rare conditions we can see complete recruitment. (DE BRUINEALTES 1946, 1949; JERGER 1952; MEURMAN 1953; SCHUKNECHT 1955- GOETZINGSR et al 1961)

There is no appreciable tone decay in presbycusis patient (JERGER 1960; GOETZIKGER et al 1961; HINCHCLIFFE 1962 ROSEN et al 1964).

KONIG (1957) and ROSS et al (1965) have shown that pitch discrimination abilities also deteriorate in the aged person. At the age of 70 the difference limen for frequencies is 2 to 3 times the value found at the age of 25. But KDNIG(1957) has reported that the sensitivity to frequency differences increases with the sensation level up till about 40 dB above threshold This means that the loss in pitch discrimination with increasing age may be made due to the increased threshold of audibility and not to the aging process.

MATZKER & SPRINGBORK(1958, 1959) reported that the ability to localize a phantom source decreased steadily after at age of 30.

The " binaural test " is based on binaural fusion of filtered speech. By using this test, the binaural integration of filtered speech was found to deteriorate with increasing age (MATZKER 1957, 1959).

Impedence Audiometery:

There has been some controversy as to whether different normative impedence values should be used with elderly (JERGER et al 1972; ALBERTI & KRISTEN3EN 1972; BEATTIE & LEAMY 1975; THMPSON, SILLS & BUI 1976; BLOOD & GREENBERG 1977 NERBONNE, BLISS & SCHOW 1977).

Static compliance changes as a function of age in adults (JERGER et al 1972; HALL 1979). Acoustic reflex thresholds also affected by aging (HANDLER & MARGOLIS 1977, JERGER et al 1978). Age differentially affects thresholds for noise Vs tone signals. As a result of the effect of age on the NTD, the accuracy of hearing prediction methods based on the NTD, such as the SPAR technique, tends to diminish as a function of age. The effect of age seems to be greatest for pure tone reflex thresholds. Here ART decreases as the increasing age. HANDLER & MARGOLIS (1977) found no evidence of decreased reflex threshold to ruretone signal in aged. According to him, noise reflex thresholds are relatively constant with age. THMPSON, SILLS & BUI 1976 found no significant difference from younger subjects.

CHAPTER 5

AUDIOLOGIC ASSESSMENT OF AGED

In order to diagnose and treat hearing disorders properly in the aged, it is advisable to seek the services of an otologist and an audiologist. An otologist needs much information besides that obtained from a physical examination. The results of audiologic assessment are invaluable in diagnosis. This chapter describes methods and tests used by audiologists in assessing hearing disorders in aged people.

Factors Essential for Better Assessment:-

The gathering of valid diagnostic data by an audiologist is of paramount importance. The accumulation of objective data, as with tympanometry, reflexometry, or with evoked response audiometry requires essentially technical competence with the equipment, with the data processing and with the interpretation of findings. The gathering of subjective information, a high interaction, and a co-operation between the patient and the audiologist requires artistry and empathy in addition to technical skill on the part of the audiologist. The ultimate effectiveness of skilled diagnostic audiologists result from their abilities to obtain reliable volitional information from problem populations as well as ideal-type clients.

The actual measures employed with the aged person may not differ greatly from those used with younger persons, but certain modifications may be necessary. Aging persons often present special problems in testing. Difficulties often can be circumvented, however, by providing the elderly client with clear, brief, carefully worded instructions at the outset. The goal is to minimize confusion and apprehension.

The general responsiveness of the older person should be continually monitored during the assessment. Fatigue can contaminate test results but must be counter balanced against reticence to travel to and from the clinical facility. Certain short outs may be necessary for some aspect of testing. The philosophy in such cases may be best described as one of the calculated expediency, where some measures are deliberately shortended in order to allow greater time for tests with higher validity. Impedence and acoustic reflex measures may replace bone conduction testing or masking. Traditional procedures for determining the SRT may be abbreviated. Such changes in conventional operating procedure must be calculated ones, reflecting a minimal loss in either the reliability or validity of the assessment battery-which is highly important, since, "the audiologic test qualifies and quantifies the handicap. It is an essential intervention" (MAURER & RUPP 1976).

SIMONTON (1965) proposes further a set of specific guides to the audiologist when assessing older clients:

1. Tonal presentations need to be lengthened (5 sec)
2. Use of frequency - warbled tones so that the listener can seperate the signal from the head noise.
3. Time between tonal presentations may be increased
4. Live voice presentations of speech meterial is preferred.
5. Familiarization of spondee words is essential, prior to determining the SRT.
6. If the patient can not handled standard phonetically balanced words lists, alternative lists should be available.
7. Half-list presentations of phonetically balanced listed may be acceptable. Try to avoid carrier phrases, which often confuse older persons and;
8. Possible collapse of the external ear canal under head phone pressure should be considered with each elderly client.

REGER (1965) gave classical admonitions for clinical diagnosic effectiveness: viz.,

1. Maintain well calibrated equipment,
2. Test in a quite environment with low ambient noise levels,
3. Employ appropriate masking procedures,
4. Ensure that head phones, oscillators, and sound-field placements are optimal,

5. Maintain a physical environment most conductive to patient's comforts,
6. Consider age intelligence and reaction time of the patient and modify the tasks accordingly,
7. Consider further the physiologic condition and mental attitude of patient,
8. Be aware of the relative complexity of the assigned listening task for the client,
9. Be careful to give instructions for patients response clearly and briefly and;
10. Be aware that complex recording methods will require careful interpretation.

Case History:- This is preliminary step in audiologic assessment. It is necessary to gather relevant information. The following information can be obtained through the case history interview

a) Reasons for having a hearing assessment,
b) Symptoms or compliants associated with hearing disorder, and
c) previous medical, occupational, rehabilitative and family history related to hearing.

Some-times older patients will discuss extraneous aspects of their medical history at length. It is often a challenge to use the write blend of patience and tact to move the interview forward. HARDICK (1977) stressed the need to express a genuine interest in the elderly person, about their hearing problem and it's effects. When an old person can not give the information what an audiologist needs or the given information is not reliable; then he should try to collect the information from family members or from any other source which is relevant to the person.

Self-Evaluation of Hearing Handicap:- A formal quantification of the handicap or difficulty imposed by the hearing impairment can be obtained by using this method. Eventhough a variety of handicap scales have been deviced for this purpose, the most popular one and the one used for elders is the hearing handicap scale (HHS), which was developed by High, Fairbanks, and Glorig (1964). This method may be used informally to alert persons to a need for clinical auditory testing. This method can serve to inform the presbycusis patient that his problem need not be ignored and that help is available to him beginning with a full audiologic assessment. Use of this method with a relative or friend of the aged person will provide additional information concerning the patient's hearing handicap.

Audiometeric Testing of the Aged Pure Tone Audiometery:-

Pure Tone Audiometery:- Here the auditory stimuli is produced by an electronic instrument called "Audiometer". The auditory stimuli may be delivered to ear through a set of ear phones. This type of examination is referred to as an "air conduction pure tone test", since the signal passes by air vibration through the ear canal and in to the middle ear. Sounds may also delivered via a bone conduction (BC) vibrator, which is generally placed on the mastoid process of temporal bone.

Through the audiometer, individual test tones of frequencies (250, 500, 1000, 2000, 4000, 6000, 8000) are presented at various intensities (0-110 db) to determine the "absolute threshold" (Lowest intensity at which the patient is able to hear the test tone). An audiogram has two important parameters (a) intensity in dB on Y-axis and (b) frequency of test tone in Hertz or CPS on X-axis, using pure tone average (average of 500, 1000 and 2000 Hz) audiologist can find out the degree of hearing loss. The American Academy of Ophthalmology and otolaryngology (1965) has given the scale of hearing impairment.

Degree of Hearing loss	-	**Hearing level**
Normal		26 dBHL
Slight		27-40 dBHL
mild		41-55 dBHL
moderate		56-70 dBHL
severe		71-90 dBHL
Profound		91 dBHL

The thresholds from airconduction and bone conduction pure tone testing may be compared to obtained information about the site of difficulty with-in the auditory system. (Conductive; mixed, or sensory neural hearing loss).

Pure tone testing by air aid bone conduction can be performed on aged patient without special modifications. HULL and TRAYNOR (1975) ZUCKER and WILLIAMS (1977) suggested that collapsed ear canals may occur in the aged due to earphone pressure on the soft tissue of the pinna. If B.C. thresholds are better than A.C. thresholds, this possibility should be considered.

REES & BOTWINICK (1971) and BOTWINICK (1973) noted that, given actual hearing function of equal levels, the older patient tend to show poorer thresholds than younger subjects becuase of a conservative response criterium. This is thought to be true for all sensory thresholds because of caution by the older patients in responding at, levels softer than these wh^re the stimulus is clearly present. Despite the need for caution in the interpretation of pure-tone findings from the elderly, this tendency does make older patients very consistant, reliable responders. Hence they are often relatively easy to test. However, some older subjects have unusualy long reaction times which slow down the test procedure (WILLEFORD 1971). Testing can be a very difficult process with patients having neurologic disorders, or extreme senility or with many who are emotionally depre-ssed. Abbreviated testing is appropriate in most of these cases. A.C. thresholds at 500, 1000, 2000, and 4000 Hz, along with impedance results will usually answer the same questions as complete air conduction and bone conduction find-ings, and, in a nonsound-treated environment, will provide more accurate results (TRAYMOR and HULL 1976). With these patients it may be necessary to divide the testing time, obtaining threshold in two to three sessions.

Speech Audiometery:- The processing of speech information is the most important task of auditory system. The audiologist employs testing referred to as speech audiometery to assess the effect of hearing loss on a persons ability to hear and understand speech. Speech audiometery is made up of several types the tests which are administered at controlled inten-sity levels through a clinical audiometer. Recorded speech meterial or 'live Voice' presentation may be used.

The first type of test is the "Speech Reception Thresh-old". Two syllable (Spondee) words are presented, and lis-tener must repeat back what he hears. The audiologist gra-dually decreases the sound intensity and the softest level where the listener can repeat back approximately 50% of words is considered the SRT. The results of this test often agree with the pure tone average. The SRT scale is similar to that used for pure tone thresholds.

The second test used in speech audiometery is "Speech Discrimination". Here a list of single syllable words is presented at a comfortable level (SRI + 40 dB). After presentation of the list the percentage of words correctly repeated by the listener is calculated. Scores of 90% and above are generally found in cases of normal hearing and conductive loss. In sensory neural hearing loss, discrimination scores will tend to be lower.

Routine procedures in S.R.T. and speech discrimination testing may be used with most elderly patients, but recorded speech meterials may lead to difficulty to the aged because of slow reaction times. Live voice presentation allows enough time for a slow response and therefore it is preferable in some cases. Several investigators have studied the speech discrimination performance of aged persons and have found reduced discrimination scores in groups of older persons. This decrease in scores may be a function of age as well as sensory-neural hearing loss. Performance of aged people on filtered or distorted speech tests are not only poorer than that seen in younger subjects, but also show a decrement of greater magnitude when compared to scores on undistorted tests.

Impedence Audiometery:- Here, the audiologist place a small probe tule at the entrance to the ear canal. A constant tone (220 Hz at 85 dB SPL) is sent into the ear canal through probe and the amount of reflected energy is measured. When a great deal of sound is reflected back, this indicates a high impedence of middle ear, which is possibly associated with the presence of fluid or oto-sclerosis. When a great amount of sound is absorbed and little reflected, this indicates unusually low impedance, which is possibly due to dislocation of ossicular chain or the tympanic membrane perforation.

Tympanometery is one of the impedance procedures. In this test, pressure changes (-200 mm H_2O to 200 mm H_20) are created within the sealed ear canal, to determine effect of pressure changes on impedance as measured at the ear drum. Usually impedance is measured in compliance units (C.C'S). The tympano meteric curves may be classified as type A, B, C, As and Ad and they may be described interms of pressure, compliance and shape. This information provides valuable diagnostic data, especially in determining the status of the middle ear. Type 'A' tympanogram, show a high peak in compliance at or near 0 mm H_2O of pressure. These tracings can be seen in normals and sensory neural hearing loss patients. Type 'B' curves show a relatively flat pattern across the pressure range, and is frequently seen in cases of middle ear fluid patients, perforation, hard ear wax obstruction, or other conditions that make the ear drum immobile with changes in ear canal pressure. Type 'C patterns show a high compliance peak at a negative pressure and seen in cases of Eustachian tube malfunction and in beginning and resolving otitis media. Type 'As' curves show a very low compliance peak at or near '0' mm. of pressure and it is seen in cases of otosclerosis and ossicular chain fixation. Type 'Ad' tracings are discontinued curves, which never make a peak and seen in cases of ossicular chain discontinuity. The difference between the highest and lowest point on a tympanogram is termed the "absolute compliance" The normal range for this value is from 0.39 c.c. to 1.3 c.c. (JERGER, JERGER and MAULDIN 1972)

Another important impedance procedure is the measurement of the acoustic reflex. Here the amount of compliance changes due to stepedius muscle reflex in response to a loud sound presented either ipsilaterally or contrulaterally. The lowest intensity which is enough to elicit Stepedial reflex is termed as "Acoustic Reflex Threshold". "Acoustic Reflex Decay" is another variation of impedance procedure in which the strength of the muscle response is measured over a 10 second period. Rapid decay (50%) is a diagnostic sign of a tumor of the VIII cranial nerve. The adequacy of Eustachian tube function can also be tested through impedence audiometery.

The use of impedance testing is valuable with the elderly, not only as an additional test but also as an alternative to routine bone conduction testing. An informal otoscopy is an important preliminary to put the probe in the ear canal. If ear was is extensive and near the entrance to the ear canal it canclog the probe. It is difficult to get air tight seal, in aged people with the unusually large pinnae. The use of glycerin or Vaseline or silicone putly as a coating on the outside of the proble tips has generally produced the desired seal (PURVIS 1974; HULL & TRAYNOR 1975}.

Special Test Battery:-

Usually,it is possible to determine whether the hearing loss is conductive or sensorineural from the results of pure tone audiometery. Speech audiometery and tympanometery can be used to confirm these results. In certain cases, it is desirable to know whether the hearing loss is due to cochlear lesion, neural lesion, brainstem lesions, cortical lesions or of non-organic origin.

Conventional Four-Test Battery:- Audiologist may use SISI, Bekesy, loudness balance and tone decay tests in a diagnostic test battery to isolate sensory and neural disorders.

The SISI test requires the patient to detect a small increment in the loudness of a steady tone. An increment of 1 dB is presented 20 times at 20 dBSL and the subject receives a percentage score depending on the number of increments he detects.

In tone decay testing the patient is required to listen to a continuous tone at his threshold to determine if he can hear it for a total of 60 seconds. If the tone fades, the level is increased until he is able to perform the task. The decay is the difference between the threshold and the intensity level at which the patient could hear it for full 60 seconds. There are several methods of administering tone decay test, given by several investigators.

With Bekesy audiometery, the patient is asked to use an automatic audiometer to trace his hearing threshold for continuous and interrupted tones. The relationship between these two tracings is used to classify the findings in-to one of five types depending upon normaly and lesion in the auditory system.

If the patient has unilateral hearing less (one normal ear), alternate binaural loudness balance (ABLB) may be administered. A supra threshold tone is presented to the good ear. Another tone is delivered to the impaired ear, and the patient is asked to adjust it's intensity until the signal is equal in loudness to the tone in the good ear. The loudness perception of the tone in the nonimpaired ear is assumed to be normal. If the loudness match occurs at about the same intensity levels rather than the same level above the threshold, then there is an abnormally rapid increase in loudness for the impaired ear. This indicates that "recruitment" is present. A variation of this procedure, "Monaural loudness balance" (MLB) involves a loudness match between two frequencies in the same ear. Recruitment can be measured or suspected using acoustic reflex test.

Results from this battery of four tests are often used to pinpoint the lesion. In conductive loss cases, there will be low SISI scores (0-20%); no or little tone decay (< 10 dB), type I Bekesy tracings and no recruitment.

If the damage is located in cochlea high SISI scores (60-100%); moderate (< 30 dB) tone decay, or no tone decay, type II Bekesy tracings; and complete recruitment will be seen an ABLB or MLB. In VIII cranial nerve lesions we can observe, low SISI scores (0-20%) [But some time there may be high SISI scores due to rapid tone decay. This is an artifact, not real score] rapid high tone decay (> 30 dB); type III or IV Bekesy tracings and no recruitment. The expected results do not always emerge on each of these tests, but a trend is usually evident among the majority of the test findings. Along with puretone, speech and impedance tests, these tests will contribute to differential diagnosis.

Functional Test Bettery:- If the audiologist suspects that the patient does not have an organic hearing loss, there are several tests to identify functional hearing loss cases.

Using case history, source of refferral; purpose of audiologic check up; and behaviour during case history like exaggerated attempt to lip read, will make us to suspect the case. Half word responses during speech audiometer and discrepency between pure tone average and speech reception threshold (more then +, 7 dB), make an audiologist to suspect functional hearing loss.

Lombord test, which is also known as voice reflex test can be used to detect functional hearing loss. Here the noise is introduced into normal ear while reading a passage. If that is normal ear, then the patient increases the loudness of his voice, if he has real hearing loss, then this voice change can not be observed.

In case of a symmetrical hearing loss (20 dB), audiologist can use Stenger Test; to determine the accuracy of patient's responses. With this test a tone or speech signal is presented to both ears, but patient will only be able to perceive it in the ear in which it is loudest. If the patient is not honestly reporting what he hears, this will produce in consistences in the test results.

In the delayed auditory feedback (DAF) test, the patient is asked to tap a pattern or read a paragraph. While he is doing this a slight delay is introduced in what he hears. This monitoring of the signal presanted via earphones is at a level below his admitted threshold. If he can hear the delayed sound, it will interfere with his performance and either the pattern will be disrupted or the reading rate will be reduced.

In the galvanic skin response (GSR) test, an equipment is used, which allows a measurement of skin resistance and simultaneously presents a slight shock to some location on the body. Patients are conditioned to expect a shock when they hear certain sounds and then these sounds are presented at low level and increased until an anticipatory, emotional response is observed through a change ir skin resistance. It can be assumed that the patient heard the sound when such responses occurs.

A type V Bekesy tracing or certain types of impedance (reflex) results also pinpoint a functional loss.

Central Test Battery: This test battery is used to test central auditory disorders. The tests in this test battery are as follows:

a) performance - Intensity function for phonetically balanced words (PI-PB function) - JERGER and JERGER (1971).

b) Filtered speech Test - Bocca et al (1954)

c) Staggered Spondic Word Test - KATZ (1962).

d) Synthetic sentence Identification Test with contralateral competing massage and/or Ipsilateral competing message - JERGER and Speaks (1965) & JERGER (1973).

e)Temporally Distorted Speech Test.

f) Masking level difference (M.L.D)

g) Evoked Response Audiometery

h) Brain stem evoked response audiometery.

i) Auditory skills test Battery (ASTB)

This is a 15 part comprehensive assessment protocb that evaluates auditory processing abilities in four major areas viz;

1. Selective attention for speech in three kinds of noise
2. Auditory discrimination with a plotting of a "sound confusion inventory".
3. Auditory memory with special emphasis on recognition, content, and sequencing; and
4. Sound-symbol relation-ships with assessments of sound mimicry, sound recognition, sound analysis, sound blending, association reading and spelling.

This ASTB has^deve loped by GOLDMAN , FRISTOE, and WOODCOCK (1974) and they also developed norms for populations who are aged between 3 and 80 years.

An audiologist need several kinds of information to assess hearing loss in the aged people and to make meaningful recommendations. This will include non-audiometeric and audiometeric procedures as well as medical and neurological examinati on. These results are used to determine if the hearing loss can be medically or audiologically remediated. If the loss does not involve activwg pathology and is simply a consequence of aging, very little can be done medically. In considering the aural rehabilitation, it must be remembered that there is a complex relation between the results of audiometeric findings and the effect of these hearing problems on a persons communicative ability. This must be considered if good management is occur.

The need for rehabilitation and various communication m needs of a hearing impaired old people are assessed by using the case history, and self evaluation tools.

Pure tone audiometeric findings will indicate the frequencies where there is hearing loss. Aging tends to cause a loss in high frequencies at first, but later extends to low frequencies. An audiogram will show the degree of hearing loss and type of hearing loss (conductive mixed, sensory neural). With age there will be a slow steady progression in hearing impairment. The greater the degree of loss the greater will be the need for remediation.

Speech audiometeric measures can be used along with the pure tone findings to analyze the effect of hearing loss on the reception of coversation. Useful information in this regard is the S.R.T. and pure tone average. It may be assumed that as the S.R.T. approaches 45 dB, there will be corresponding difficulty with normal conversation.

The aged may have even 40 to 45 dB of loss and may not admit handicap, if compensation is made by persons talking louder or if the subjects have minimal social interaction.

When considering the every day influence of hearing loss, speech discrimina tion scores must be carefully interpreted since in the routine examination these scores represent the clarity of a persons hearing when he is listening at a comfortable listening level rather than at a normal conversational level. Another important factor in analyzing speech discrimination findings for the elderly patient is the relative importance of vowels and consonants in understanding speech stimuli. The vowels in speech are general ly louder than consonants and thus easier to hear. Also the vowels tend to be low pitched sounds, while many; consonants are higher in frequency. For aged people hearing is better in the low frequencies than in high frequencies. Thus they hear the vowels better than consonants.

CHAPTER 6

Aural Rehabilitation of Aged

Medical Treatment: If it is true that the presbycusis is caused, by the loss of neural elements and possibly by the destruction of epithelial elements in the organ of Corti, then the medical treatment is highly questionable.

SCHUKNECHT (1955), VON SCHULTHESS (1961) doubt the possibility of a regeneration or replacement of such highly differentiated cells. On the other hand the amazing improvement in bone conduction, that can be found in Menier's disease, is just unexplained as a dramatic therapeutic success of any drug would be in the treatment of presbycusis.

Four main groups of drugs have been widely used for the treatment of presbycusis.

1. Hormones, 2. Vasodilative drugs, 3. Lipoproteinolytic drugs, and 4. Vitamins.

Hormones:- ALFOLDY (1936) described hearing improvement in old patients after intravenous administration of hyprophysis hormones. KOCH (1937,1947); HOFER (1956); KROATH (1958); BOGNER (1954) and WESTON (1964) used sex hormones on presbycusis patients and found a favourable effect on the hearing and a disappearance of the tinnitus. SCHURMANN (1944) administered female hormones to women between 40 and 61 years old and round no hearing improvement. ZAJICEK (1938), MENZEL (1942) and HOFER (1956) applied a special hormonal ointment and found hearing improvement in all kinds of deafness. There was 0 to 30 dB improvement and definite reduction or disappearance of tinnitis, after hormonal therapy.

However, this area requires further investigations, as the results have been very much unequivocal.

Vasodilative Drugs:- Nlcotinic acid and its derivatives cause peripheral vasodilatation and reduce the concentration of blood lipids. In presbycusis cases it dilates cochlear and cerebral blood vessels. FERRARI (1955), WEDER AND MISSUKA (1959) found a temporary improvement in hearing of presbycusis patients after the administration of nicotinic acid. The effect of hyderyin, as a sympathicolytic drug on hearing and tinnitis in old people (TANNER, 1955), is ascribed, to the vasodilative action of this drug.

Lipoproteinolytic Drugs: Tnese drugs have something to do with arteriosclerosis and are also used for the treatment of presbycusis. MAYOUX et al. (1955) used 'heparin' with presbycusis patients and found an audiometric improvement in 25% of cases. In 45% of cases tinnitus and vertigo disappeared. But BOUCHE (1956) says this drug is more useful to treat tinnits and vertigo than for hearing. GALLI DELIA LOGGIA (1956); CERRI & MARCUCCI (1957); and BREU (1956) support BOUCHE'S statement. DEL GUIDICE & AMORELLI (1960) believed that these drugs helps in delaying the onset of presbycusis.

Vitamins:- COVELL (1940) studied the role of vitamins in deafness. Vitamin 'A' deficient animals showed a degeneration of cells in the stria vascularis and parts of the organ of Corti with slight to moderate demyelinisation of the cochlear nerve. Vitamin 'B' deficient animals showed degenerative changes in the external hair cells and of the cochlear nerve. The data available indicated that the cochlear nerve is the cranial nerve which is most vulnerable to lack of vitamin 'B'. Vitamin 'C deficiency was found associated with swollen and partly degenerated cells in organ of Corti. Vitamin 'D' deficiency was associated with demyelinisation of the cochlear nerve. An increased incidence of middle ear infections was found in all vitamin deficient animals.

LOBEL (1949 & 1951); ANDERSON, ZOLLER & ALEXANDER (1950); BAU & SAVITT (1951); BARON (1951); NAGER (1952); RUEDI (1954); ESCHER & RUPP (1953); STOPPLER (1956); FLEISHER & KNOBLOCH (1957); and WEDER & MISbURA (1959) found vitamin 'A to be useful in prebycusis treatment. They found improvement from 12 to 46% of the patients. PERLMAN (1949); WILLEMSENS & HULK (1960) and WARD & GLORIG (1960) found no hearing improvement in presbycusis patients treated with high doses of vitamins.

Audiological Management

Traditionally, aural rehabilitation has been considered to be consisting of hearing aid evaluation and orientation, counselling for client and family, auditory training, speech reading and speech conservation (NEWBY, 1972). It is any effort made to rehabilitate the hearing impaired through non-medical procedures. RASSI & HARFORD (1968); NORTHERN & SANDERS (1972); OYER, FREEMAN, HARDIC, DIXON, DONNELLY, GOLDSTEIN, LLOYD and MUSSEN (1976) observe that traditional aural rehabilitation has not been popularly received by the adult hearing impaired. ALPINER (1973) felt that aged hearing clients do not have motivation to get rehabilitated and most of the time it is not successful. But there is some evidence to indicate that aural rehabilitation can be successful (NORTHERN, CILIKK, ROTH & JOHNSON, 1969; ESERSTEN, 1974; BINNIE, 1976).

The importance of aural rehabilitation is underscored by the fact that there are so many hearing-impaired persons needing rehabilitation. When compared to persons with other difficulties, the hard-of-hearing person is grossly neglected. Failure to recognize and rehabilitate people with hearing impairment may have serious consequences besides the demoralizing effects (YARINGTON, 1976). KAY, BEAMISON & ROTH (1964) found a substantial proportion of elderly psychiatric patients diagnosed as Schizophrenics of late onset had a severe auditory deficit.

It is apparent, that hearing loss may have devastating effects, in the elderly. If medical management is not indicated for correcting the disability, then aural rehabilitation is needed. Several forms of a total adult aural rehabilitation program havebeen suggested recently (SANDERS, 1971; NORTHERN, 1972; ROSS, 1972; ARA, 1973; O'NEILL & OYER, 1973; A.S.H.A. 1974; OYER & FRANKMANN, 1975;

BINNIE, 1976; and OYER & HODGSON, 1977). All of these programs suggest a broadly based approach to aural rehabilitation, and most include the following areas after preliminary hearing assessment aspects are completed:

1. Hearing aid evaluation
2. Hearing aid Orientation
3. Counseling of client and family
4. Communication rehabilitation including auditory training and speech reading.
5. Comprehensive planning for emotional, social, and vocational adjustment.

Hearing Aid Evaluation:- It is a most important step in aural rehabilitation, to determine, if a hearing aid can be used by the patient. The most appropriate aid is selected if amplification is indicated. The need for a hearing aid evaluation can be determined by a thorough audiologic assessment.

HODGSON (1979) has suggested a cursory guideline which outlines when individuals should be referred for hearing aid evaluation. It is based on the pure tone average and speech reception threshold, in better ear. There are exceptions to this guide, as in the case of persons with unilateral loss or for those with unusual audiograms, who may need hearing aids. The self-evaluation of hearing through a scale provides a helpful guideline regarding the need for rehabilitative help (HIGH, FAIRBANKS, & GLORIG, 1964).

When a hearing aid evaluation is undertaken, a number of approaches may be used. The most popular method is the

Carhart's method (1946). In this procedure, the subject is clinically tested in several conditions, both with and without amplification. Other procedures are (a) the use of a master hearing aid (WASSON, 1963); (b) Prescription procedure (BERGER, 1976); and referral to a hearing aid dealer for selection after audiometric assessment and counselling by an audiologist (RESNICK & BECKER, 1963). R.P.A.G. (1973); H.E.W. (1975) and ASHA (1977) cited that hearing aids are sometimes dispensed to persons who do not benefit from using these devices. This is particularly serious in case of elderly persons where numerous problems may emerge in obtaining a good fit and in achieving an adequate adjustment to the aid. In case of elderly persons audiologists can provide very helpful services before and after an aid is provided. Through aural rehabilitation programs much can be done in helping the new user in the adjustment process. In addition to these potential problems, there are many who have not obtained an aid but who should. One advantage of having an audiologist perform a hearing aid evaluation is that if a hearing aid is not indicated* then the person having hearing problem may obtain help through other forms of aural rehabilitation. These rehabilitation services are seldom provided by hearing aid dealers (LIBBY, 1974). It may be noted that hearing aids are not recommended for approximately 10-20% of hearing loss population (HOOPLE, 1960; and HARDICK, 1977).

Through audiologic procedures decisions can be made as to whether or not a person can benefit from an aid; what type of aid (ear level or body) should be used; the ear(s) for which the aid should be used and specific characteristics (such as frequency response, gain, M.P.O., ear mold coupling, distortion) which the aid should have. Once a person obtains an aid, periodic hearing aid re-evaluations, are recommended, to ascertain that optimal help is being achieved from amplification (ALPINER, 1975; HODGSON, 1977).

While hearing aid evaluation is conducted with all age groups in most clinics, but noteexclusively for elderly (ALPINER, 1973; BOWMAN, 1974; BLOOD & DANHAUER, 1976). The elderly require a special consideration which may not be required with younger patients (CANFIELD, 1973). RUPP, HIGGINS & MAURER (1979) have developed a scale for predicting the feasibility of hearing aid use with older individuals. This scale incorporates the following factors: magnitude of hearing loss, self assessment scores; motivation to hear better, adaptability, attitude towards hearing aids, age, manual exterity, visual ability, financial resources and family support. All these things play some part in predicting the prospects of success.

Another procedure designed for the use with the elderly calls for the utilization of the sentence portion of "Utley Lip Reading Test". DODDS and HARFORD (1968) use this test to allow for visual input along with auditory input which is heard under two conditions, both with and without amplification. In this way, some elderly clients who do very poorly on both aided and unaided auditory discrimination tests can be shown to benefit from an aid. This can serve as an encouragement to patients who might otherwise reject amplification. Sometimes with elderly, the opposite problem is encountered. Most experts agree that the person with presbycusis should get a hearing aid before the time of intense need is upon him (RUPP, McLAUCHLIN HARLESS & MIKULAS, 1971). The old person will probably be able to make the adjustment to amplification better before he has an extreme loss and before he reaches an advanced age. Thus, careful monitoring of the loss from year to year is indicated and in some , cases an aid will be recommended for an elderly person sooner than it would be tor a younger adult client.

The problem with the traditional hearing aid evaluatio is that the procedure is too lengthy for some elderly subjects. OYER et al. (1976) have suggested that one reason rehabilitation has not been successful with the elderly is that they have become discouraged after enduring the tedium of the usual hearing aid evaluation. HARDICK (1977) uses a procedure in which a hearing aid is selected in a series of shorter sessions over an extended period of time.

Hearing aid Orientation and Counselling:-

After selection of an appropriate hearing aid, it should be obtained for use in a trial period. The new user sometimes able to adjust quickly to amplification, but quite often there is some difficulty with a longer coping process. HARDICK (1977) estimated that of all new hearing aid users about 75% need only a brief introduction to the benefits, limitations, and care of hearing aids. He claimed, among the aged people, only a smaller percentage make quick adjustment and that many need extensive orientation .

The important components of a hearing aid orientation and counselling session shave been described by SANDERS (1975 and HASTEN & WARREN (1977). They suggest inclusion of the following information:-

1. Introducing the hearing aid, including it's components and controls.
2. Putting the aid on, and listening to amplification.
3. caring for earmolds and batteries,
4. using the telephone, T.V., alarm clocks, and other communication devices and,
5. checking the aid and getting it repaired.

In counselling HASTEN & WARREN (1977) suggest:-

1. Describing the levels of hearing such as primitive, warning and symbolic;
2. Explaining the audiologic findings,

3. Discu-ssing the handicapping aspects of hearing loss
4. Describing the benefits and limitations of aids.
5. Analyzing total communication needs and;
6. Finding solutions for communication problems.

TANNAHILL (1973) developed a hearing aid adjustment program, which incorporated.

1. The gradual increasing in the wearing time;
2. using appropriate volume settings and
3. increasing speech discrimination performance. A

spirit of comradery developed in the groups tried to achieve their goals in this program than individuals. Through such a process, clients were helped to make the initial adjustment to amplification while learning and sharing experiences in a group setting. This program has also been applied with as few as four sessions through a combination of group and individual therapy.

The most important factors in predicting success were health, mental status, and language function. Age, visual impairment motor disturbances and personality were of limited influence. According SMITH & FAY (1977) the extent of auditory impairment was also a secondary factor in predicting the success. They stated "Poor speech discrimination predicted nothing, except the need for much training".

Communication Rehabilitation:-

Auditory trainin g and speech reading are usually the , procedures that first come to mind, when aural rehabilitation is mentioned. Actually, both terms are used to refer to a wide variety of techniques. To some professionals, auditory training includes the various activities discussed in the above paragraphs. To others it means a series of listening activities, and to others it simply means learning about the dynamics of conversational situations.

Speech reading is a process of being visually alert to all lip, facial, gestural and environmental clues.

FLEMING et al (1973) reported a varie ty of activities of auditory training and/or speech reading to be included in communication rehabilitation. These consists of teaching and encouraging the patient to:

1. physically position himself to maximize lighting, reverberation and signal to noise factors.
2. Develop expertise in interacting with conversational patterns.
3. Maximize acoustic and lighting conditions in his usual conversational locations.
4. Become a better listener by being more interested and attentive and;
5. Deal with the impatient normal hearing conversational partner.

LUNDBORG, LINZANDER, ROSENHAMER LINDSTRCM, SVARD and FRANSSON (1973) recommended a selectivex use of such communication training. They also suggest that more extensive therapy is required in cases with more extreme loss, and th at these procedures become more difficult to apply with the aged. While communication aspects may thus be applied on a selective basis, they can also be merged in a total therapy program.

Several total rehabilitation programs have been discribed for the elderly. HARDICK (1977) described an aural rehabi litation program for the aged. He feels that a progra for the elderly should be:-

1. Client centered to meet individual needs. This is can be done through a combination of individual and group therapy.
2. revolving around amplification and /or modifying the communication environment.
3. requiring the attendance of a relativelor friend to gain admittance to the group.
4. short-term, involving a weekly meeting for 6-12 weeks, depending on how quickly the goals of the group is accomplished and;
5. able to disseminate as much informatiomas possible to th group.

The goals HARDICK describes for the program include the major aspects of aural rehabilitation. Clients are told about their hearing loss and their hearing aid. There is an extensive experimentation with hearing aid use until the right aid is selected. The electro acoustic aspects of the aid are then measured and recorded. Listening practice is given to awaken the person's interest in hearing again. They are informed about speech-reading, with it's difficulties and are encouraged to use their native skill. Attitudes and behaviours are discussed and some are discouraged, such as bluffing, self-depreciation and guilt. Finally, helpful suggestions are given about alarm clocks, T.Vs, telephones; and personal safety. HARDIC admittedly takes a conservative view of the benefits of speechreading and auditory training

The basic approach is to present to the client a variety of ways by which he may improve his overall communication, Thus, by pooling all information available to him (SANDERS, 1975) it is possible to compensate for the loss of audition. Alternative source of information include amplification, knowledge of language, awareness of situational constraints, the visual attentiveness to lip movements, facial expression and gestures. In addition attentive listening is discussed with suggestions for improving one's listening skills. In this therapy program speech reading is discussed interms of those factors which make it easier or more difficult. However, no extensive drill work is undertaken, and the emphasis is on visual alternatives rather than an visual phonemic reception.

The audiologist monitors progress and attainment of goals in these seesions with auditory, visual, and handicap measures. Auditory testing includes S.R.T. discrimination, and electro acoustic aid performance. Basic vision and pre-and-post speech reading scores are measured. Scoring is based not only on response accuracy but also on the attempts made. So that listening strategy of client can be assessed.

In rehabilitation of aged much depends upon the patients health, attitudes, and circumstances. These may influence on the acceptance of disability and the responses of therapy. Understanding and awareness of these factors is therefore important in the auditory rehabilitation efforts for the elderly. ALPINER (1963) advised that in working with the elderly one should remember that they are:-

1. Not as old as they used to be, physically or mentally:
2. No like children; they must be approached on a mature level
3. Individuals and are varied as in any age group;
4. subject to basic human needs; they need to love and be loved, to feel useful, to belong so mewhere, to lough to believe in something and;
5. Generally much more flexible and capable of learning new things than we. give them credit for.

CHAPTER 7

ROLE OF THE FAMILY

As a person gets older his family assumes an ever increasing amount of importance. The family is the older person's main source of emotional strength, companionship, and, more importently, a prime source of help in an emergency or in the case of illness. (RILEY & FONER). Aged persons will have fewer formal group re-lationship and closer ties with specific individuals. These lost social ties to which the elderly cling are often those of their immediate family. Aged people who are sick or whose activity is limited by chronic ailments are more likely to live with one of their children.

LEHMANN, DE LATEUR .. et al (1975) found that of a family is available and if it can get the needed direction and encouragement from a health care team, the communicatively handicaped patient can often live at home and receive better care as well. For this reason, delivery of health care services should not be directed solely to the aged person with a hearing disorder but should be a family-oriented activity. Quite frequently the family needs help as much as the patient does. Most families want to do something, to help in time of need, but in many cases, their efforts are unguided, non-productive, or even counter productive.

Unfortunately, the geriatiic patient with a communication problem is not always understood, even by family members, as to his limitations and reduced abilities. For these reasons, the re habilitative challange presented by the older hearing imparied patient will also require the family to seek and expect help from the health care professionals. When a family has some understanding of the na ture of an elderly patient's disorder, what can be reasonably expected, and how to affectively help, than adjustments are easier to make, mis understandings are avoided, and worthwhile patient care results.

The family of an aged handicaped person is often required to assume the role of nurse, occupational therapist, physical therapist, psychologist,Rehabilitation: audiologist, and speech therapist for months or years without any warning or training. In case of the aged person who has a hearing loss, both the family and the patient need to know how to communicate with each other effectively and to know the importance of monitor ing residual hearing. Similarly family members should seek information about the physical emotional problems of the hearing impaired. YOUMANS AND YARROW (1971) pointed out that an intact, supportive and intimate social environment is an important sociopsychological factor in resisting disorders in old age and is a significant factor in survival.

Sometimes it would seem that members of the family of someone with hearing impariment suffer as much and perhaps even more than the impaired person himself. The elderly person with hearing loss may appear serene and calm, basking in the peace and quiet which the impairment provides, while all around him everyone else tries in vain to communicate. Of course it is not generally pleasent to be cut off from daily communication, and having such a disability is worse than it may seem. Nevertheless, the family suffers as well.

Common complaints about the hearing impaired by the family include

(a) He hears what he wants to

(b) He blame everyone else for his hearing problem.

(c) He won't use his aid or, when he uses it, he still acts like he can't hear.

The family member should realise that it is possible to hear some parts of conversation or some voices more easily than others. Thus the hearing impaired are usually innocent of the thing they are so often accused of, namely, hearing only what they want to hear, When a person has a hearing loss, it often requires great concentration and use of visual and other clues to fill in parts of the message which are not heard. X^^^^^&^Q^fQ*tAA^KR9&APNtAbeapd. If full effort is not put forth, the conversational meaning can easily be missed. With normal hearing, by contrast, one can hear and understand with very little effort . When the hearing impaired are tired or not watchin g carefully, it may require more concentration than they can master to participate in communication.

It is not an easy thing sometime s to accept the fact that one has a disabili ty. Considering the gradual way in which pres by cubic losses develop, it is not surprising that many elderly persons who have a loss deny it and blame others. With gentel comments, the family members may be able to help the impaired person recognize that he possibly has a problem and encourage him to seek proper help. When a hard of hearing elderly person is seen by an audiologist, it is helpful for a member of the family to go w i th him. It is also important; to attend aural rehabilitation classes with the new hearing aid user.

BIBLIOGRAPHY

ALPINER.(J): Diagnostic and rehabilitative aspects of geriatric audiology. ASHA 7, 1965 (455-59).

ALPINER (Jerome G.) Aural rehabilitation and the aged client. MAIOO Aud.Lib.Ser. Vol.4. Rep. 4 (P.9-12)

ALPINER, J.G. (Ed.) (1978). Handbook of adult rehabitative audiology. The Williams & Wilkins Company, Baltimore.

AMERMAN, J.D. & PARNELL (M.M) The staggered spondiac word test: A normative investigation of older adults. Ear & Hear.. l(l), 1980, 42-45.

BABU PUNNAN, Audiological findings in oresbycusis: (Mysore University, Dissertation, 1976), 3304. Dissertation No. 34.

BAROCCI, (R) A hearing aid for the presbyacusic subject: A new technique end a new direction. Nuovo Arch Ital. ORL 7(1), 1979, 135-138.

BAROCCI,(R) et al. Speech audiometry in presbycusis: Revision of material and presentation of new techniques. Nuovo Arch. Ital. 0RL8(2). 1980, 269-273.

BASCHEK, (V) The influence of cerebral aging on directional hearing and the indication for application of a binaural hearing aid. Laryngol. Rhinol. Otol. 58(ll), 1979, 827-831.

BEASLEY,(W) Generalized age and sex trends in hearing loss. Hearing study series Bulletin. No.7, National Health Survey, Washington, D.C. US Public Health Service (1938).

BELOV. (I.M) et al. Measurement of duration of the residual auditory sensibility in various forms of auditory pathology (Russian text) Vestn. ORL, 41(5), 1979, 15-19.

BERGMAN (M) Hearing and aging: Implications of recent research findings. Audiol. Vol. 19, 1971 (P.164-17l).

BERGMAN (MOE) Effects of aging on hearing. MAICO. Aud. Lib. Ser.Vol.2, Rep. 6.

BERGHOLTZ,(L.M), HOPPER (R.E) & MEHTA,(D.6): Electro-cohleographr response patterns in a group of patients mainly with presbyacusis. Scand. Audiol., 6(l), 1977, 3-11.

BERKOWITZ (A.O) Audiological rehabilitation of the geriatric patient. Hear. Aid. J., 28(8), 1975, 8, 30, 32-34.

BERNATH (0): The results of hearing aid usage in a group of patients older than 65 years. HNO. 27(1), 1979, 35-37.

BERRUECOS (P): Binaural temporal integration in presbycusis: Aud. Vol. IX, 1970 (P. 309-313).

BIRREN (J): Handbook of aging and the individual. Chicago, University of Chicago Press (1959).

BIRREN (J): Relations of development and aging. Springfield III. Chos C.Thomas (1964).

BIRREN, (J), IMUS(H), & WINDLE (W): The process of aging in the nervous system. Springfield, 111. Cha C.Thomas (1959).

BLOOD (I) & GREENBERG (H.J): Acoustic admittance of the ear in the geriatric person. J.Amer.Audiol.Soc., 2(5), 1977, 185-187.

BLUMENFELD (Vera-Gruber) and others. Speech discrimination in an aging population. JSHR Vol.12, No.1, 1969, (P.210-217)

BOCHENEK (Z) AND JACHOWSKA (A): Atherosclerosis, Accelerated presbycussis and Acoustic Trauma: Contributed papers on population studies and presbycusis. Aid. vol.7, 1968 (P.509-510).

BOCHENEK (Z) & JACHOWSKA (A): Atherisclerosis, accelerated presbycusis and acoustic trauma. Aud.Vol. VIII, 1969, (P.312-3l6).

BONDING,(P): Critical bandwidth in presbycusis. Scand. Audiol, 8(1), 1979, 43-50.

BOHDAGARAY (P) & MENDOZA (c): Presbycussis and noise. Rev. ORL, 36(2), 1976, 44-47.

BUNCH (C): Age variations in auditory acuity. Archives of Otolaryngology, 9:1929 (625-636).

BUNCH (C): Further observations on age variations in auditory acuity. Archives of Otolaryngology, 13:1931 (170-180).

Clarissa R.Smoth & Thomas M.Foy: A program of auditory Rehabilitation for aged persons in a chronic disease hospital.

CORSO (J): Age and sex differences in pure tone thresholds Archives of Otolaryngology, 77:1963 (385-405).

CORSO (J.F): Sensory process and age effects in normal adults. J.of Gerontology, 26, 1971 (90-105).

CORSO (J): Presbycusis as a complicating factor in evaluating noise induced hearing loss in effects of noise on hearing, D.HENDERSON et. al. (Edi) New York, Review Press, 1976.

CORSO (J.F): Presbyacusis, hearing aids and aging. Audiology 16 (2), 1977 (146-164).

CRABBE (F): Acta Otolaryngologica Supplement 183: 1963, (24-26).

CRAIG (W) NEWMAN & JACLYN (B) SPITZER: Eustachian tube efficiency of geriatric subjects. Ear & Hearing, 2(3), 1981 (103-107).

CRAINE (M): Your hearing - The second 50 years. Hear. Rebab. 3(1), 1978, 4-7.

CROWLEY (DAFID E) Communications. Conference on animal models of aging in the auditory system. Ann Otol Rhinol. Laryngol, Vol.84, 1975 (P.560-561).

DAVID F.BARR: Aural rehabilitation and the geriatric patient. Geriatrics, 25(6), 1970, (111-113).

DAYAL (VS) & NUSSBAUM (MA): Patterns of pure tone loss in presbycusis - A sequential study. Acta Oto-Laryngol. Vol.71, 1971 (P.382-384).

DAYAL (VS) & others: Patterns of pure tone hearing loss. A comparative study of presbycusis, Multiple Sclerosis, Meniceres and Acoustic Neuroma. Acta Oto-Laryngol. Vol.69, 1970 (P.329-332).

DEMAN (Deuise): A comparison of the presbycisis speech discrimination performance in a clinical and non-clinical testing environment. Hear Instrum. Vol.27, No.9, 1976 (P-12).

DEMI CHELIS (G) & others: Index d'intelligibilite et bandes passantes on presbycusis contributed papers on population studies and presbycusis. Aud. Vol.7, 1968 (P.512).

DEMICHELIS (G) and others: Intelligibilte de la parole et filtrage chezles presbyacousiques, Aud.vol. VIII, 1969 (325-327) (Article in French).

DUM (N), SCHMIDT (U) & VON WEDEL (H): Age related changes in the auditory evoked brain stem potentials of albino and pigmented Guinea pigs, Arch-Ohr. Nas. Kehlkopfheilk, 228 (4), 1980, 249-258.

ETHOLM (B) & BELAL (A) Jr. Senile changes in the middle ear joints, Ann. Otol.Rhinol Laryngol. Vol.83. 1974 (P.49-54).

FELDMAN (R.Michael) & NEGER (Scott N): Relations among hearing, reaction time and age, J.S.H.R. Vol.10, No.3, 1967 (P.479-495).

FIELDS (W.S) : Neurological and sensory disorders in the elderly. NY: Stratton International Medical Book Corp. 1975, pp. 244.

FINDLAY (Robert C) and DENEKBERG (Larry J): Effects of subtle mid-frequency auditory dysfunction upon speech discrimination in noise. Aud, Vol.16, 1977 (P.252-259).

FOWLER (E): The aging ear, Archives of Otolaryngology, l0:1940 (475-480).

FREDERIKSEN (E), BEEGVAD (B) & ROJSKJER (C): Binaural hearing aid treatment of presbycusis patients aged 70 to 80 years, Scand.Audiol.. 3(2), 1974, 83-86.

GACEK (Richard A): Pathology of presbycusis, Round Table Session on Presbycusis, Aud.Vol.7, 1968 (P.445-446).

GACEK (R.R) & SCHUKKEGHT (H.F): Pathology of Presbycusis, Aud.Vol. VIII, 1969, (P.199-209).

GAITZ (C) & WARSHAW (H)Obstacles encountered in correcting hearing loss in the elderly, Geriatrics, 19, 1964 (83-86).

GERSDORFF (M.C.H): Impedancementric study of the variations of the auditory reflex in man as a function of age, Audiol., 17(3), 1978, 260-270.

GILAD (0) & GLORIG (A): Presbycusis: The aging ear Part II. J.Amer.Aud.Soc.. 4(6), 1979, 207-217.

GLATTKE (T.J): Anatomy and physiology of the auditory system; in the book "Audiologicel Assessment" by ROSE (D.E) Prentice Hall, Inc. 2nd Edn. 1978.

GLORING (A) & ROBERTS (J): Age, noise and hearing loss. Annals of Otology, Rhinology and Laryngology, 70:1961 (556-571).

GOETZINGER (C), PROUD (G) & DIRKS (D): A study of hearing in advanced age. Archives of Octolaryngology, 73:1961, (662-674).

GONZALEZ (M) & OOBO (J): Bekesy's automatic audiometry in old people, An. ORL Ibero-Amer., 2 (4), 1975, 67-85.

GRANICK (S), KLEBAN (M.H) & WEISS (A.D): Relationships between loss and cognition in normally hearing aged persons, J.Gerontol., 31(4), 1976, 434-440.

HALL (J.W) Effects of age and sex on static compliance, Arch. Otolaryngol., 105(2), 1979, 153-156.

HANSEN (C.C) & NELSEN (Edith Reske): Pathological studies in Presbycusis, Arch Otolar, Vol. 82, 1965 (P.115-132).

HANBEN (C) & RESKE-NIELSEN (E): Pathological studies in presbycusis, Archives of Otolaryngology, 82:1965 (115-132).

HANSEN (C.C.) & RESKE-NIELSEN &Edith): Cochlear and Cerebral pathology in aged patients: Round Table Session on Presbycusis, Aud. Vol.7, 1968 (P.450-452).

HARBERT (E), YOUNG (T) & MENDUKE (H): Audiologic findings in presbycusis, Jr. of Audi&ory Research, 6:1966 (P.297-312)

HAYES (D) & JERGER (J): Low frequency hearing loss in presbycusis. A central interpretation, Arch. Otolaryngol., 105(1), 1979, 9-12.

HAYERS (D) & JERGER (J): Aging and the use of hearing aics, Scand. Audiol., 8(l), 1979, 33-40.

HENRY (K.R.) & CHOLE (R.A): Genotypic differences in hehavioral physiological and anatomical expressions of age - related hearing loss in the laboratory mouse, Addiol., 19(5), 1980, 369-383.

HINCHCLIFFE (R): The threshold of hg. as a function of age Acoustica,9, 1959 (303-308).

HINCHCLIFFE (Ronald): The anatomical locus of presbycusis: J.S.H.D. Vol. 27, No.4, 1962, P.301-310.

HINCHCLIFFE (R): Population studies concerned with Presbycusis. Ind.J.Otolaryngol. Vol. 20, 1968 (P.52-57).

HIPSKIND (N.M): Aural rehabilitation for adults, Otolaryngol. Clin.N.Amer. 11(3), 1978, 823-834.

HOLMGREN (L): Psychology of deafness in the aged, Audiol, Vol.8, 1969 (P.281-289).

HOLMGREN (Lennart): Revalidation of presbycisis: Psychology aspects, Round Table Session on Presbycusis, Aud.Vol.7, 1969 (P.460-462).

HOLMGREN (L): Psychology of deafness in the aged, Add. Vol. VIII, 1969 (P.281-289).

HOOPLE (G): Diagnosis presbycusis and susceptibility, Laryingoscope (Special issue), 68:1958 (477-486).

HUDSON (Atwood): Communication problems of the geriatic patient, JSHD, Vol. 25, No.3, 1960 (P.238-247).

HULL (H) RAYMOND : Hearing evaluation of elderly in the Book, Handbook of Clinical Audiology, Edn. by KATZ (J) WILLIAM & WILKINS COMPANY, 1978, 2nd Edition.

HULL (R.H) & TRAYNOR (R.M): Community-widd program in geriatric aural rehabilitation, ASHA 75, 1975 (33-48).

HURLOCK (E.B) 3rd Edi. Developmental psychology, McGraw-Hill Book Company, 1968.

JATHO (Kurt): Population survdys and norms, Round Table Session on Presbycusis, Aud. Vol.7, 1986 (P.453-455).

JEFFERS (Janet): The "Social Adequacy" of two selected samples of presbycusis, contributed papers on population studies and presbycusis, Aud. Vol.7, 1968 (P.510-512).

JEFFERS (J): The social adequacy of two selected samples of geriatric - presbycusis with significant hearing losses, Aud. Vol.VIII, 1969, (P.317-324).

JERGER (J): Audiological findings in aging, Adv.Oto-Rhino-Laryngology, 20:1973 (115-124).

JERGER (J) & HALL (J): Effects of age and sex on auditory brainstem response, Arch-Otolaryngol., 106(7), 1980, 387-391.

JERGER (J) & NORTHERN (J.L) (1980): Clinical impedance audiometry American electromedics Corporation, Massachusetts, 2nd Edn.

JERGER (J), SHEDD (J) & HARFORD (E): On the detection of extremely small changes in sound intensity, Archives of Otalnaryngology, 69, 1959 (200-211).

JOHNSSON (L): Degenerative changes and anomalies of the vestibular system in man, Laryngoscope, 81:1971 (1681-1969).

JOHNSSON (Lars-Goran) & HAWKINS (Joseph E) Jr. : Sensory and neural degeneration with aging, as seen in microdissecations of the human inner ear, Ann Otol.Rhinol. Laryngol, Vol.81, 1972, (P.179-193).

JOHNSSON (Lars-Goran) & HAWKINS (Joseph E) Jr. Vascular changes in the human inner ear associated with aging, Ann Otol. Rhinol Laryngol, Vol.81, 1972, (P.364-376).

JOKINEN (K): Presbycusis I. Comparison of mannual and automatic thresholds, Acta Otolaryngol, Vol.68, 1969, (P.327-335).

JOKINEN (K): Presbycusis II. The effect of pulse duration and of the rate of intensity change on pure tome thresholds, Acta Otolaryngol, Vol. 69, 1970,(P.155-159).

JOKINEN (K): Presbycusis III. Perstinulatory threshold adaption, Acta Oto Laryngol, Vol.69, 1970 (P.324-328).

JOKINEN (K): Presbycusis IV - Masking of speech, Acta Oto-Layyngol Vol.76, 1973 (P.426-430).

JOKINEN (K) & KARJA (J): Presbycusis IV. Forward Vs. Reverse prequency sweep audiometry, Acta-Oto-Laryngol. Vol. 70, 1970, (P.227-321).

KASTENBAUM (R) (Edi) Contributions to the psychobiology of aging, New York, Springer, 1965.

KATZJ (Ed): Handbook of clinical audiology. The Williams & Williams Company, Baltimore, 2nd Edition.

KEIM (R.J): How aging affects the ear, Geriatrics, 32(6), 1977, 97-99.

KIRIKAE (Ichiro): Aditory function in advanced age with reference to histological changes in the central auditory system Bound Table Session on Presbycusis, Aud.Vol.7, 1968 (P.448-450).

KIRIKAE (I): Auditory function in advanced age with reference to histological changes in the central auditory system. Audiol, Vol.8, 1969, (P.221-230).

KLEIN (H): A filtered speech test for an aging population, J.S.African Speech Hear. Assn., 23, 1976, 87-97.

KONIG (E) : Tests audiometriques dams be cadre de la presbyacousic, Round Table Session on Presbyacusis, Aud. Vol.7, 1968, (P.455-457) (Article in French).

KONIG (E): Audiological tests in presbyacusis, Aud. Vol. VIII, 1969, (P.240-259).

KONIG (E): Problems of hearing aid fitting in elderly adults, HNO, 26(1), 1978, 28-40.

KONIGSMARK (Bruce W): Aging of cells and structures, Round Table Session on Presbyacusis, Aud.Vol.7, 1968, (P. 443-444).

KONIGSMARK (B.W): Aging of cells and structures. Audiol. Vol. 8, 1969, (P.191-198).

KRMPOTIC-NEMANIC (J): Presbycusis and retrocochlean structures: Round Table Session on Presbyacusis, Aud. Vol. 7, 1968, (P.446-448).

KRMPOTIC-NEMANIC (3): Presbycusis, presbystasis and presbyosumia as consequences of the Analogous Biological Process, Acta. Oto-Laryngol. Vol.67, 1969 (P.217-223).

KRMPOTIC-NEMANIC (3): Presbyacus and Retrocochlear structures, Aud. Vol. VIII, 1969 (P.210-220).

KRMPOTIC-NEMANIC (Jelena): New concept of the pathogenesis of presbycusis, Arch. Otolar Vol. 93, 1971 (P.161-166).

KRMPOTIC-NEMANIC (J) & Others: Weiterer Beitrag Zur Atiologic Der presbyacusis, Acta Oto-Karyngol. Vol.75, 1973 (P.211-215).

LANSING (I) Edi. 1952: Cowdry's problems of ageing: Biological end medical aspects. The Williams and Wilkins Company, Baltimore, 3rd Edition.

LAYTON (Barry): Perceptual noise and aging, Psychol.Bull. Vol. 82, 1975 (P.875-883).

LEHNHARDT (D(: The question of the term "Presbycusis". HMO, 26(12), 1978, 406-413.

LENZI (P), COhTI (A) & ARPIKI (A): Late evoked responses to acoustic stimuli in relation to age, Audiol. 17(2) 1978, 152-158.

LEUTENEGGER (Ralph R) & STOVALL (Janice D): Pilot graduate Seminar concerning speech and heaiing problems of the Chronically III and the aged, ASHA. Vol.13, 1971, (P.61-66).

LOPOTKO (A.I): Some peculiarities of the age involution changes in the peripheral structures of the auditory analyzer (Russian text) Vestn. ORL, 37(3), 1975, 22-27

LOPOTKO (A.I): A comparative assessment of sound conduction of the cranium in young and old individuals, (Russian text) Vestn. ORL, 38(6), 1976, 38-39.

LOWELL (Seth H) & PAPARELLA (Michael M): Presbycusis: What is it? Laryngoscope, Vol.87, 1977, (P.1710-1717).

MAURER (F.J) & RUPP (R.R) (1979), Hearing and aging: Tactis for intervention, Grune & Stratten, Inc. New York.

MCCOY (C), BUTLER (M) & BROEKHOFF (J): Effects of age and sex on dichotic listening: The SSW test, J.Aud.Res.. 17(4), 1977, 263-268.

MENEGAUX (A) & PAILER (J.P): An approach to troubles of central integration of a message in the elderly, Rev.Laryngol Otol. Rhinol., 100 (9-10), 1979, 557-566.

MILLER (Lucille V): The adult and the elderly health care and hearing loss, Volta, Vol. 77, No.l, 1975, (P.57-63).

MILNE (J.S): Air-bone gap in older people, BJA, Vol.11, 1977 (P.1-6).

MILNE (J.S): A longitudinal study of hearing loss in older people, Brit. J.Audiol.. 11(1), 1977, 7-14.

MOLLICA (V): Traumatisme acoustique et presbyacoysie: Contributed papers on population studies and presbyacusis, Aud. Vol.7, 1968,(P.508)

MOLLICA (V): Acoustic trauma and presbuacusis, Aud. Vol. VIII, 1969, (P.305-311).

MORLEY (D.E): Training and exoerience of graduate students in communication problems of the aging, ASHA .Vol.5. 1963, (P.819-821).

NADOL (J.B): Electron microscopic findings in a presbycusic degeneration of the basal turn of the human cochlea, Otolaryngol. & Head & Necksurg, 87 (6), 1979, 818-836.

NAVARRP (Maroin R): Earmold for the Geriatric patient J.S.H.D.. Vol.42, 1977 (P.44-46).

NERBONNE (M.A), BLISS (A.TJ & SCHOW (R.L): Acoustic impedence values in the elderly, J.Amer. Aud.Soc. 4(2), 1978, 57-59.

NETTING (E) & SAMPSON (A), Hearing loss and an inservice programme formthe elderly, Soc.Work, 24(4), 1979, 333-335.

NOMURA (Y): Lipidosis of the basilar membrane, Acta Oto-Laryngol Vol. 69, 1970, (P.352-357).

NOMURA (Y) & KIRIKAE (I): Presbyacusis. A histological study of the human cochlea, Acta Oto-Laryngol. Vol.66, 1968, (P.17-24).

NOVOTNY, (Z): Aye factor in auditory fatigue in occupational deafness caused by noise, Leskoslov. Otolaryngol, 24 (l), 1975, 5-9.

OSTERHAMMEL (D) & OSTERHAMMEL (P): High frequency audio-metrety. Age and sex variations, ^cand. Audiol. 8(2), 1979, 73-81.

OSTERHAMMEL (D) & OSTERHAMMEL (P): Age and sex variations for the normal stapedial reflex thresholds and tympanometric compliance values, Scand. Audiol, 8(3), 1979, 153-158.

OUELLETTE (S.E) Ed. 1978: Biennial conference proceedings Part I. .7.Rehab. Deaf 12(4), 1979, 1-108 (i-xii).

OYER (H.J) & OYER (E.J): Social conseauences of heating loss for the elderly, Allied Health & Behav. Sci.. 2(2), 1979, 123-129.

PALVA (A) & JOKINSEN (K): Presbyacusis. V.Fiitered speech test, Ada Oto-Larvngol.. Vol.70, 1970, (p.232-241).

PARNELL (M.M) & AMERMANH (J.D): Aging and decoding of co-articulatory cues, J.Speech Hear. Res.. 22(3), 1979, 433-445.

PEDERSEN (C.Brache) & ELBERLIKG (C): Temporal integration of Acouetic energy in patients with presbyacusis, Acta Oto-Laryngol, Vol. 75, 1973, (P.32-37).

PEDERSEN (C.Brache) & POULSEN (T): Loudness of brief tones in hearing impaired ears - Temporal integration of acoustic energy at suprathreshold levels in patients with presbycusis, Acta-Oto-Laryngol, Vol.76, 1973, (P.402-409).

PHILON (Y) & LESCOUFLAIR (G): Acoustic reflexes in presbycusis and noise induced hearing loss, Audiol.. 16(l), 1977, 38-48.

PLOMP (R) & MIMPEN (A.M): Speech reception threshold for sentences as a function of age and noise level, J.Acoust. Soc. Amer., 66(5), 1979, 1333-1342.

POWERS (J.K.) & POWERS (E.A): Hearing problems of elderly persons: Social consequences and prevalanece, ASHA.20 (2), 1978, 79-83.

PRAZIC (M): Snoring and presbyacusis, Acta Oto-Laryngol. Vol. 75, 1973, (P. 216-219).

PRICE (G.Richard): Age as a factor in susceptibility to hearing loss: Young versus adult ears, JASA, Vol.60, 1976, (P.886-892).

Quaranta (A), Amorso (C) & Cervellera (G): Remote masking in presbycusis, J.Aud.Res. 18(2), 1978, 125-129.

QUARAKTA (A), CASSANO (P) & AMOROSO (C): Acoustic reflexometry in presbycusis, Audiol.. 19(4), 1980, 310-315.

REYNAND (J): Enquete sur la presbyacousis en mitieu rural et nomade Africai Contributed papers on population studies and presbyacusis, Aud.Vol. 7, 1968 (P. 507-508).

REYNAUD (E) & others: An investigation into presbyacusis in Africans from rural and nomadic enviroments, Aud. Vol. VIII, 1969, (P. 299-304).

RITCHIE (B.E): Aural rehabilitation for the elderly - A model program, Hear Aid J.. 32(2), 1978, 4, 34-36.

ROBINSON (D.W) & SUTTON (3.J): Age effect in hearing - A comparative analysis of published threshold data, Audiol.. 18(4), 1970, 320-334.

ROP (I), RABER (A) & FISCHER (G.A): Study of the hearing losses of industrial workers with occupational noise exposure, using statistical methods for the analysis of qualitative data, Audiol., 18(3), 1979, 181-196.

ROMAN ROJO (L.A) & INFANTE SANCHEZ (J.C): Values of auditory involution in subjects over 50 years of age, Acta ORL Expan.. 27(3), 1976, 67-80.

ROSENHAMER (H.J), LINDSTROM (B) & LUNDBORG (J): On The use of click-worked electric brsinstem responses in audiological diagnosis II. The influence of sex and age upon the normal response, Scand. Audiol, 9(2), 1980, 93-100.

ROSEN (Samuel) 3. others: Presbycusis study of a relatively noise-free population in the Sudan, Ann.Otol.Rhinol. Laryngol. Vol. 71, No.3, 1962 (Reprint).

ROSEN (Samuel) & others: High freqnency audiometry in pre-bycusis: Arch. Otolan. Vol. 79, 1964, P.18-32.

ROSEN (Samuel): Hearing loss and cardiovascular disease: Round Table Session on presbyacusis, Aud.Vol.7. 1968, P.457-459.

RUPP (R.R): Understanding the problems of presbycusis: An over view hearing loss associated with aging, Geriatrics. 25(l), 1970, P. 100-107.

SAGAZOVICH (B.M) & TSUKANOVA (V.N): Disturbance of the capacity of discerning the front of accretion of sound stimuli as a oathogenomonic sign of presbycusis, Russian text, Vestn. ORL. 40(3), 1978; 10.15.

SANDERSON (B.A.) AND others: An evaluation of vaso dilitation therapy for presbycusis, Aud. Vol.IX, 1970, P. 287-292.

SCHALLENKAMP (K.K) MAHANNA (S.K), LINGOR (G.J) FLIEHS (J.B) & KING (M.E): Incidence of hearing loss among the elderly, Hear. Aid.J., 33(3), 1980, 10, 48-49.

SCHMIDT (P.H): Presbyacusis and noise, Aud. Vol.VIII, 1969, (P.278-280).

SCHMIDT (P.H): Presbycisis and noise: Round Table Session on presbyacusis, Aud. Vol.7, 1938, (P.460)

SCHON (T.D): The effects on speech intelligibility of time-compression and expansion on normal hearing, hard of hearing and aged males, JAR, Vol.10, 1970, (P.263-268).

SCHOW (R.L) et al. (1978): Communication disorders of the aged. A guide for health prodessionals, University Park Press, Baltimore.

SCHOW (R.L) & GOLDBAUM (D.E): Collapsed ear canals in the elderly nursing home population, J.Speech Hear.Dis.. 45(2), 1980, 259-267.

SCHUKNECHT (Harold F): Further observations on the pathology of presbycusis, Arch. Otolan. Vol. 80, No.4, 1964, (P. 369-382).

SEIFERT (M.W), SEIDEMANN (M.F) & GIVENS (G.D): An examination of variables involved in lympanometric assessment of eustachian tube function in adults, J.Speech. Hear.Dis. 44(3), 1979, 388-396.

SELTZER (M): Growing older.... Whatever that means, Hear. Speech Action, 45 (4), 1977, 8-10.

SETH (S.M) & KACKER (S.K): Contours of normal hearing in Delhi as a factor of aging, Silent World. Vol.6, No.l, 1971, (P. 21-27).

SHEILA (M) CHOWN (Ed) 1972: Human ageing, Penguin Books Ltd., Harmondsworth, Middlesex, England.

SHOOP (C) & BINNIE (C.A): The effects of age upon the visual perception of speech, Scand. Audiol.. 8(l), 1979, 3-8.

SILMAN (S): The effects of aging on the stapedius reflex thresholds, J.Acoust.Soc. Amer.. 66(3), 1979, 735-738.

SMITH (Clarissa R) and FAY (Thomas H): Program of auditory rehabilitation for aged persons in a chronic disease hospital, ASH, Vol.1977, (P.417-419).

SODA (T) et.al. Physiological and bio-informative analyis of the aging of the auditory function, (Japanese text) Otol. Fukuoka. 23, (Suppl. 3), 1977, 738-747.

SPINK (U) JOHNNSEN (H.S.) & PIRSIY (W): Acousttically worked evoked potential dependence upon age, Scand Audiol. 8(l), 1979, 11-14.

SPOOR (A): Presbycusis values in relation to noise induced hearing loss, Aud. Vol. VI, 1967 (P. 48-57).

SPOOR (A) & PASSCHIER - VERMHER (W): Spread of presbycisis values contributed papers on general, Aud. Vol.7, 1968, (P.519-520).

STEVENSON (P.W): Responses to speech audiometry and phonemic discrimination patterns in the eledrly, Aud. Vol. 14, No.3, 1975, (P. 185-231).

STRECHLER (L.B) 1962: Time cells and aging, Academi Press, New York, & London.

SUGA (FLJMIRO) & LINDSAY (JOHN R): Histopathological observations of presbycusis, Ann. Otol.Rhibol. Larvnaol. 85, 1976, (P. 169-184).

THOMPSON (D.J) SILLS (J.A) RECKE (K.S) & BUI (D.M): Acoustic admittance and the aging ear, J.Speech Hear Res. 22(l), 1979, 29-36.

TYBERGHEIN (J) & FORREZ (G): Audory evoked potentials and presbyacusis: Contributed papers ondiagnostic neuro– physiological test, Aud. Vol.7, 1968 (P.424-425).

TYBERGHEIK (J) & FORREZ (G): Potentials evoques cerebrauz et presbyacousis, Aud.Vol. VIII, 1969, (P. 377-381). (in French)

WEDEL (H.V.) & OPITZ (H.J), The effect of aging on middle ear function, Laryngol. Rhinol.Otol.. 57(2), 1978, 152-158.

WEDEL (H.V.) & OPITZ (H.J): The influence of presbvcusis on evoked potentials, Laryngol Rhinol. Otol.. 58(7), 1979, 602-609.

WILLEFORD (J.A): The genatric patient; in the book "Audio- logical assessment" by ROSE (.DE) Prentice Hall, Inc. 1978, 2nd Edition.