AUDITORY BRAIN-STEM RESPONSES IN GERIATRICS - A REVIEW OF LITERATURE

Reg. NO. M. 9109

An independent project submitted as part fulfilment for the first year M.Sc (Speech and Hearing) to the University of Mysore

v.

All India Institute of Speech and Hearing
MYSORE - 570 006

MAY 1992

DEDICATED TO:

BAPA,BA,PAPA & MUMMY

CERTIFICATE

This is to certify that the Independent Project entitled **AUDITORY BRAIN-STEM RESPONSES IN GERIATRICS -A REVIEW OF LITERATURE"** *is a bona fide work, done in part fulfilment for the First Year Degree of Master of Science (Speech and Hearing), of the student with* **Reg.No.M 9109.**

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CERTIFICATE

This is to certify that this Independent Project entitled "**AUDITORY BRAIN-STEM RESPONSES IN GERIATRICS - A REVIEW OF LITERATURE**" *has been prepared under my supervision and guidance.*

MYSORE
MAY 1992

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DECLARATION

/ hereby declare that this Independent Project entitled **-AUDITORY BRAIN-STEM RESPOHSES IN GERIATRICS - A REVIEW OF LITERATURE'** *is the result of my own study under the guidance of* **Dr.(MISS).S. NIKAM,** *Professor and Head of the Department of Audiology, All India Institute of Speech and Hearing. Mysore, has not hem submitted earlier at any University for any other Diploma or Degree.*

MYSORE **Reg.No.M.9109**
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C 0 N T S N T S

INTRODUCTION

Brainstem auditory evoked potential (BAEP) audiometry has rapidly become a very important tool in the field of clinical audiology, otology, neurology and neuro- otology and is one of the widely used electric response audiometry (ERA).

The ABB is recorded by attaching electrodes to the surface of the scalp and amplifying the electrical activity obtained immediately following an auditory stimulus. The response is low in amplitude and is buried in other ongoing activity in the nervous system therefore, special common mode rejection amplification response filtering, and computer averaging are needed to differentiate the response to the stimulus from the other activity.

Early reports on the ABR were provided by Jewett, Romano and Williston (1970), Jewett and Williston (1971) and Sohmer and Feinmesser (1967).

A normal ABR waveform is characterized by five to seven vertex positive peaks that occur within 1.4 to 8.0 milliseconds, the labelling convention for the waves was suggested by Jewett and Hilliston (1971) by using Roman numerals I through' VII to identify the peaks. The most prominent vertex positive peaks in the waveform are waves I, III, V.

Some of the early researches suggested that wave V cane from the inferior colliculus, wave IV from the lateral lemniscus, wave III from the olivary bundle, wave II from the cochlear nucleus and wave I from the VIIIth nerve.

Holler and Janneta (1981) reported that wave I and in part wave II can be attributed to VIIIth nerve activity. There are no simple generator sites for waves III-VI, and each is most likely a reflection of complex inter-relationships and 'overlapping activity of brainstem structures at several sites within the central auditory system (Achor and Starr, 1980a, 1980b; Kevanishvili, 1980; Mair, Elverland, and Laukli, 1978) and even wave II has at least two putative sites.

Of all the evoked potentials, BAEP shows the least variability as the quality and reproduclbility of the ABR is quite independent of the state of the subject and can be obtained in subjects who general anaesthesia or comatose. Because of this replicability, consistency among subjects, and sensitivity to disorders in these auditory pathways, the ABR has become an important tool in both clinical evaluation and intra-operative monitoring.

Nevertheless, in BAEP, the latencies and amplitude of the potentials are influenced by many factors like stimulus polarity, stimulus rate, stimulus intensity, stimulus duration and rise time, stimulus frequency, monaural Vs binaural stimulation, electrodes montages, filters, one Vs two channel recordings, age, gender,attention, medication and drugs etc.

Age is one of the important factors. ABR obtained in premature and term infants vary from these obtained in adults (Hecox and Galambos, 1975: Starr, Amlie,Martin, and Sanders, 1977; Jacobson,Morehouse and Johnson, 1982; Schulman Galambos and Galambos, 1975). Waves I, II & V are most visible and the normal wave V latency for a newborn approximates 7.0 milliseconds at 60dB nHL. Interwave latencies may be prolonged upto approximately 12 to 18 months of age (Hecox and Galambos 1974; Starr. Amlie, Martin and Sanders, 1977) and the amplitude of wave I may be greater than wave V.

Wave I *may* be prolonged in infants, but generally not as much as wave V, often generating a longer interwave latency interval than observed in adults. This say be related to cochlear Maturation (Rubel and Ryals, 1983; Ryan, Woolf and Sharp, 1982), neuronal maturation (Starr, Amlie, Martin and Sanders, 1977; Stockard and Stockard, 1981), reduced efficiency in external and/or middle ear sound transmission and occasionally collapsing ear canals.

In contrast to the interest in the development changes in the ABR, the potential influence of aging in adults has recevied remarkably, little attention.

EFFECTS OF AGE

Age is an important factor in behavioural audiometry. The age-related decrease in pure-tone sensitivity for higher frequencies and in some patients, lower frequency, is well-documented. Depressed performance in speech understanding for both single word and especially in sentences in competition is associated with aging. Age is also a factor in impedance audiometry static compliance decrease as a function of age. With increasing age, acoustic reflex thresholds usually improve slightly for pure-tone signals, and are elevated for noise signals, even in subjects with normal hearing. Consequently, the noise-tone difference is decreased as a function of age. Gersdorff (1978) reported decreased amplitude for crossed (contralateral) and uncrossed (ipsilateral) acoustic reflexes, in subjects with normal hearing sensitivity. The view of these age effects in other aspects of auditory function, it seems reasonable, to suspect an age factor in the ABR (Jerger and Hall, 1980).

Mortality rates for the aging population have declined, morbidity often has not. We may thus expect to encounter an ever increasing population with the diseases of aging.

The effect of aging process on the human auditory system has been reported in the literature as early as 1800 A.D. Jerger (1973) stated, the aging process produces systematic changes in each of the two critical dimensions of hearing impairment-loss in threshold sensitivity and loss in the ability to understand suprathreshold speech.

Vision and hearing are ranked first in importance for a healthy old age. The general adjustment problems. Facing the elderly are complicated by both general physical illness and loss of hearing.

The incidence of hearing loss in the elderly population is significantly high and the most common cause of hearing is presbycusis, or the loss of hearing due to aging process (Sataloff, 1966). Pusbycusis manifests changes in the entire auditory system (Schuknecht, 1955).
It must be the goal of those on whom the responsibility rests to insure that the best kind of assistance is given to the elderly individual who desperately may need assistance in resolving his problem.

An effective programme of aural rehabilitation for the geriatric patients should be as comprehensive in approach as the hearing problem itself. The first step for such programme is proper audiological testing which should involve a battery of tests including ABR. In order to assess, adequately, the severity of hearing impairment, the factors of prime importance is reference standard against which a person's hearing is evaluated. Various age related changes in ABR waveform morphology, latency and amplitude should be known to an audiologis't to arrive at an appropriate diagnosis.

The available data have been controversial about the role of ace. eg., Rowe (1978) demonstrated significant differences in ABR between old and young subjects, while Beagley and Sheldrake (1978) hardly found any significant differences.

Thus reviewing past efforts may allow us to project future directions for aural rehabilitation for older adults.

AGING AND IT'S CONSEQUENCES

The branch of medicine that concern with the prevention with aging is called "geriatrics", the term coined by an American Physician, Ignaz Leo Vaschers (1909).

Over the age of 65 constitute the group identified as "the aged". However, this group is not homogeneous. With regard to most factors, and gerontologist have divided the aged into the younger (age 65-75 years) and older aged (75yrs and older) (Busse and Pfeiffer, 1977). Shanas and Maddox (1976) have identified 3 subgroups of aged; the "young old" (45-64 years), "the old" (65-74 years) and the "old old" who are over 75 years.

Birren (1959) distinguishes 3 kinds of aging these are:

1) Biological, or the length of life in years, months etc.
2) Psychological, which indicates the adaptation capacity of the organism.
3) Social age, or the social output and performance of the person relative to his or her culture and social group.

The period of life designated as "old age" is unique in that it inevitably brings with it various declines in functioning. It demands that the person experience and adjust to some degree of debilitation and reduced capabilities.

Changes take place on almost *every* aspect of a person's functioning, at rate which varies from individual to individual and within each individual. How rapidly and in which areas physiologic and psychologic changes occur is largely dependent on a person's genetic endowment, the environments in which he/she was reared and presently lives and the total life history of the person prior to old age.

The normal biological and physiological changes concomitant with aging are not due to diseases are many. They
include;

1) Decline in heart output and blood supply, particularly to the brain.
2) Reduced stomach and intestine motility, which produces digestive problems and constipation.
3) Decreased vital capacity of the lungs.
4) Bone decalcification leading to osteoporosis.
5) Stiffening of joints and ligaments.
6) Loss of teeth.
7) Graying and loss of hair.
8) Loss of genitourinary system efficiency.
9) Reduction of subcutaneous fat.
10) Decrease in oxygen utilization.
11) Lowered hormonal output.
12) Changes in sensory processes and many more (Rosenwasser, (1964).

Psychological changes in cognitive-intellectual aspects and emotional-personality alterations are noted (Thompson 1973).

Memory deteriorates in old age. Learning ability is also influenced by central processing problems.

Particularly important for everyday activities are the changes in sensory processes which accompany old age.

They have increased threshold and touch, decreased pain sensitivity, and a lowered ability to perceive tastes. They have trouble identifying common substances by smell, correlated with a loss of taste buds and reduced taste sensations.

The visual problems of the aged include reduced peripheral vision, greater vulnerability to glare, increased visual threshold, yielding decreased perceptual abilities, and poor dark adaptation, visual acuity, contrast sensitivity, accommodation and colour matching. Cataracts, glaucoma, poor visual orientation and visual problems which reduce nobility and increase the isolation of the aged (Butler and Lewis, 1977).

Hearing impairment is one of the most widespread sensory deficits associated with the normal aging process.

The effect of aging on the human hearing system has been reported in the literature as early as 1800 A.D. Zwaardemaker {1891), observed progressive deterioration of hearing for high frequency sounds with advancing age.

Bezold (1894) using whispered voice test in elderly (50-70yrs) demonstrated that the hearing loss for whispered speech increases as a function of age. He also observed that males have poorer hearing than females. On the basis of Rinne'S test, he concluded that reduced hearing power of the aged resulted from an affected inner ear and not from disease of middle ear structure.

For all age groups (20-60yrs) pure-tone sensitivity is more viable in the high frequency than in the low frequency and primarily for high frequency above 1024 Hz there is a progressive deterioration in auditory sensitivity as a function of age (Bunch, 1929).

1935-1936 National Health Survey (NHS), indicated an exponential increase in hearing loss with advancing age.

Seth and Kacker (1971) assessed the level of hearing in a sample of 100, ranging in age from 5 to 77 years. The results revealed a gradual increase in the loss of hearing with increasing age especially after fourth decade of life. The hearing loss was more marked at high frequency while the speech frequencies were preserved.

Punnan (1976) evaluated 100 subjects of age ranging from 35 years to 74 years. The analysis of data revealed a gradual increase in hearing loss with age and frequency. The onset of hearing loss was reported to be earlier by about 5 year compared to Western reports.

Indrani R. (1981) studied the age and sex variations in hearing in 180 subjects (10.6 years to 87 years)of Indian Nationality. She indicates increase in hearing threshold as afunction of age.The dependency of hearing acuity on frequency was moat marked in the older population.

Rosen etal(1962)compared the median hearing levels the 10-70 years old Mabaans to hearing levels of corresponding age groups who were tested in the 1954 ITiaconain atate Fair Hearing Survey and found better hearing in Mabaana and showed that the Mabaans do not experience deteriorations of hearing as a function of age to the same degree as the subjects exanined in the Wisconsin study.

" Similar study was reported by Kapur (1967) who surveyed the hearing sensitivity in Todas, a tribal population living in the hilly regions of Nilgiris. This tribe has minimal exposure of noise. Total number of subjects included 50M and 43F of age ranging from 6 years - 70 years. The analysis of results revealed normal hearing in 74% and hearing loss in 26% of the test population. Of the latter, 25% had conductive loss and only 1% manifested SN hearing loss.

Thus data on hearing and aging gleaned from field studies reveal that:

1) Measurable changes in auditory sensitivity for high frequency puretones (4000 - 8OOOHz) occur by fourth decade of life of men and women.

2) The variability in puretone thresholds, especially in the higher test frequency, is greater for the older age groups than in the younger age groups.
3) Men lose auditory sensitivity in the higher frequencies more rapidly as a function of age than women do.
4) The noxious elements in our culture and the unique life-styles that people lead have a significant effect on the degree to which hearing sensitivity changes as a function of age.

The aging process produces systematic changes in each of the two critical dimensions of hearing impairment loss in threshold sensitivity and loss in the ability to understand suprathreshold speech (Jerger, 1973).

Pitch discrimination, auditory acuity and speech perception are worse among older persons (Fozard and Thomas, 1975) though the inability to understand speech may be a function of vocabulory as well as audiometric considerations (Farrimond, 1961) and is accentuated when listening takes place under environmentally stressful conditions (Corso, 1977).

Phonemic regression (Gaeth, 1948) is a condition in which the ability to discriminate speech (measured with phonetically balanced monosyllabic words) is diminished in elderly individuals with presycusis. The presence of an age related gradual decrease in the speech perception, out of proportion to hearing loss for pure tones, has been confirmed in different studies (Pestalozza, Shore, 1955; Goetzinger, Rowsey, 1959; Klotz, Kelban, 1962).

Moller (1981) however, found only a minor decrease in the discrimination score for monosyllabic words with age.

Further limitations in hearing concern a decrease in directional hearing in old person. Their ability to identify very small interaural time differences deteriorates.

In short, the loss of hearing in old age affects high-frequency sensitivity, resulting in deterioration of consonant intelligibility (phonemic regression), diminished ability to compensate for discrimination loss by interpolation and combination on the semantic sentence plane, decrease in cerebral processing of speech and other acoustic patterns, decrease in pitch and volume discrimination as well as speech discrimination ability in noisy situations, general decreased ability to compensate centrally for a peripheral hearing loss, and decreased visual acuity resulting in diminished ability to use visual cues from lip and facial movements and gestures.

The general adjustment problems facing the elderly are complicated by both general physical illness and loss of hearing or vision.

Anderson et al (1968) noted that more than 70% of the patients over 65 years of age who were admitted for psychotic or maladaptive behaviour exhibited either obvious hearing impairment or difficulty in comprehension of conversation.

Hearing problem places the elderly at risk of unnecessary communication difficulties and sensory deprivation. Hearing loss can lead to problems with social isolation either from the impatience or misunderstanding of those trying to communicate with them.

The anxiety, fear and anger due to hearing impairment generates greater emotional distress.

Hearing impairment hampers one's communicative efficiency and undermines an individual's ability to foster and maintain interpersonal relations. Self-reports of hearing handicap by hearing impaired individuals have revealed that hearing impairment cuts them off from the environment in which they were once actively involved. Hearing impairment in elderly individuals triggers a chain of psychological reactions (Hull, 1978, Maurer and Rupp, 1979). According to Hull, the cycle begins with a sense of frustration growing out of a decreased capacity for interaction and communicative effectiveness.

The measured loss of hearing sensitivity does not reflect completely the extent of handicap imposed by a hearing impairment (ASHA, 1981).

Reduced perceptual abilities in audition (and other senses as well) are more likely due to problems in central processing, than to sense organ changes (Forzard and Thomas, 1975).

The general consensus is that central factors predominate and are related to decreased cell count in the temporal lobes, an increased time for information processing in the brain and possibly an increase in synaptic delays on the central auditory pathways.

Presbycusis is broadly defined as an age-related decline in auditory function. This decline encompasses both the effects of true cellular aging on the auditory and central nervous system and the cumulative effects of other factors such as acoustic trauma, cardiovascular diseases and ototoxic medications.

In the process of aging, the ear and it's structures like other parts of the body undergo changes. Many types of auditory system deterioration occur. These include,

1) Loss of hearing sensitivity through chemical and mechanical changes in the inner ear.
2) Breakdown of inner ear structures and
3) Complex degenerative changes occurring along nerve pathways leading to the brain.

Schuknecht has suggested (1955, 1964) 4 categories of presbycusis based on histopathologic and clinical characteristics.

Sensory presbycusis: Presents with a bilateral abruptly sloping high frequency SN hearing loss. Their speech discrimination is related to the frequency involved and is generally good. Loudness recruitment is common and hearing aid prognosis is good.

Histology demonstrates progressive degeneration of the basal portion of the organ of corti. There may be degeneration and atrophy of sensory and supporting cells with primary damage beginning at the basal end of the cochlea and moving towards the apex. Secondary degeneration involves the acoustic nerve.

Neural presbycusis: Here patients usually notice a rapid hearing loss and have severe difficulty understanding speech. Their audiograms demonstrate a moderate, flat pure-tone loss with disproportionately poor speech discriminations. Hearing aid prognosis is not good for this type of pathology. This conditions is often seen in persons with severe ateriosclerosis.

This is an advanced stage of sensory presbycusis which involves a greater loss of neurons in the central nervous system. The cause is a greater (but diffuse) loss of neural cells in the spiral ganglion or nerve fibres. There also appears to be an angiosclerotic degeneration of epithelial tissues of the organ of corti and of the basilar membrane, resulting in a loss of elasticity.

Strial/Metabolic Presbycusis: Is characterized by a familial slowly progressive SN hearing loss, flat loss with good discrimination. Hearing aid prognosis is good.
Histology demonstrates patchy atrophy of the stria vascularis in the middle and apical turns of the cochlea.

Cochlear (conductive) presbycuais: demonstrate sloping high frequency loss with conductive components and preservation of speech discrimination are found in these patients. Hearing aid prognosis is good.
It is speculated that it is a disorder of motion of the mechanics of the cochlear duct caused by a stiffening or calcification of the basilar membrane which would be consistent with a loss of elasticity and increase of the internal frictions in the basilar membrane.

Soucek, Michaels and Frohlich (1987) studied pathological changes in the organ of corti in presbycusis by microslicing and staining. A marked loss of outer and a mild loss of inner haircells was present in the elderly subjects. Complete atrophy of variable lengths of the terminal basal coil cells was also noted in all elderly cochleas. Giant sterociliary degeneration takes place in hair cells (except basal coil outer hair cells) before they disappear.

The loss of neurons throughout the entire central nervous system has been shown to begin early and to continue throughout life. This loss of neurons become vigorous in old age. Brody (1955), Neff (1947) reported that the partial section of the auditory nerve in man results in a high frequency hearing loss. Whereas Schuknecht and Woelliner (1953) and Citron et al (1963) found a normal threshold for pure tones despite severe degeneration of the cochlear nerve.

Crowe et al (1934) and Hansen and Reske Neilsen (1965) reported only slight degeneration in the peripheral part of the auditory nerve, but severe vascular alterations in the internal auditory meatus (IAM). Covell and Rogus (1957) and Scrcer (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 fibres, resulting in pure "ideopathic presbycusis".

Kirkae, Sato and Shitara (1964) found atrophy of neural structure in the ventral cochlear nuclei, the superior olivary complex (SOC), the lateral leminicus, the inferior colliculus and medial geniculate body and in the auditory cortex itself. Hansen and Reske - Neilsen (1965) found severe degeneration in the glial part of the acoustic nerve as well as in the white matter of the brainstem and in the hearing centers.

Geriatric patients are also vulnerable to otosclerosis (Farrior, 1963).

Some conductive pathology is present in aged (Glorig and Davis, 1964).

Rosen Wasser (1964) studied structural changes in aged ears as ossicular atrophy particularly in the crura of the stapes, ossification of the incudomalleolar joint with calcification of the articular cartilage, degeneration and atrophy of the middle ear muscles, a thin and translucent tympanic membrane and atrophy and thinning of the skin that lines the external auditory meatus together with loss of elastic tissue elements.

Various findings are summarized in following table (adapted from Saucek et al 1986).

Investigator	Hg. Loss	Tech. used	No. Pt.	No. ears	Age yrs.	Findings in coch/ brainstem
Guild, Crowe et al	High tone	Ser- ial	3	5	60-64	Loss of s.g.c. in b.c.
Von Fieandt & Saxen 1937	-	Sec- tion	100	?	50-90	Loss of s.g.c. Arteriosclerosis
Schuknecht 1955	High tone	Ser- ial Sec- tion				Loss of hair and supporting ceils in b. c.
Schuknecht 1964	Flat curve	Ser-- ial Sec- tion	4	8	76	Partial atrophy of stria in apical 1/2
Fleischer 1956	-	-'	100	?		Loss of s.g.c.
Scrcer and Krmpotick 1958		Unde- cal Sec- tion				Hyperostosis in IAM
Bredberg 1965	High &low tone	Sur- face Prep	?5	?10	70-100	Loss of hair cells in b.c. m.C.& a.c.
Hansen & Reske Neilson 1965	High &low tone	Sec- tion	12	24	>80	Loss of s.g.c. in b.c.degenera= tion of VIIIth nerve & brain
Johnson & Hawkins 1972		Sur- face Prep	150	?	?	Halrcell loss Loss of vessel in spiral ligament
Suga & Lindsay 1976	SN	ser- ial sec- tion	17	34	>65	Loss of s.g.c. Loss of hair cells. Strial atrophy
Arnesen 1982	SN	Sec-" tion	6	12	76-89	50% loss of neurons in coch. nuclei

s.g.c. = Spiral ganglion cells
o.c. = Basal coil
m.c. = Middle coil; a.c. = Apical coil

Hoolpe (1960) reports that it is difficult to accept the aging factor alone as the full culprit in the hearing problem. There is a multiplicity of causes of hearing loss. Fowler's (1959) partial list of such factors includes severe strain, fright, grief, frustrations, poisonous drugs, antibiotics, overexertion, bacterial and viral infections, electric shock, metabolic disorders, pregnancy, vitamin deficiency, exposure to cold, allergies and thromboses along with acoustic, psychic and other traumas.

The aging person may have hearing loss from presbycusis, noise induced hearing loss (NIHL), and chronic middle ear disorder (Surjan, Devald and Palfal, I, 1973).

Presbycusic defect seen in many cases is gradually sloping, gradually progressive, high frequency sensorineural hearing loss. The loss increases gradually at first and then accelerates more rapidly with increasing ages, especially for the higher frequencies (Berger et al, 1977; Corso, 1963; Spoor, 1967) and is bilateral symmetrical (Dayal, Kane and Mendelsohn. 1971; Klotz and Kilbane, 1962; Sataloff and Menduke, 1957). But not all presbyscusic hearing losses follow the typical audiometric configuration. Dayal et al (1971) reported 31% incidence of flat audiometric configurations.

Carhart (1958) describe "Bernero Effect" and he suggested that reduced bone conduction response at 500Hz is more likely a reflection of central auditory dysfunction. Glorig and Davis (1961) described a high frequency air-bone gap that they ascribed to an age-related increase in stiffness of the cochlear partition. The air-bone gap was in evidence at 4000Hz and increased from l0dB at 50 years of age group to 40db by 80 years of age group. Nixon, Glorig and High (1962) found air-bone gap at 4000Hz only and it was not due to noise exposure and suggested that the conductive component was related to pathologic changes in the connective tissue of middle ear.

Several auditory manifestations are seen clearly in pure tone audiometry, speech audiometry, impedance audiometry, brainstem evoked response audiometry and so on. Several findings regarding all these tests are summarized in Table-I.

TABLE-1

A. PURE TONE AUDIOMETRY

Investigators	Procedure (methodology)	Findings
Miller & Ort, 1965	Evaluated subjects of wide age-range	Generally aged revealed bilateral SN hearing loss, sloping type, having more loss above 2KHz. He reported this slope in 88% of a group of individuals over the age of 65 years.
Dept of Health, Education & Welfare U.S.A.		Loss is greater for men in frequency above 4KHz, whereas women show a greater loss towards the lower frequencies.
Bunch, 1929, 1931		There is gradually progressive reduction in sensitivity beginning at age 30. The hearing loss primarily affects frequency above 1000Hz and tend to progress through beyond age 60yrs.
Sataloff & Menduke, 1957		Little increase in hearing loss (bilateral) in men or women from age 65 years through age 90 years.
Melrose, Walsh & Luterman, 1963	Tested aged population using pure tone audiometry	Gradually sloping, bilateral symmetrical SN hearing loss with greater loss in the high frequency.
Milne & Lauden, 1955	Examined pure tone sensitivity of 500 persons between 62 & 90yrs of age.	Women showed a greater hearing loss at 1000Hz & below, whereas men showed greater loss at 2000Hz and above.
Pederson, Rosenhall & Moller, 1989		Hearing loss was most pronounced at higher frequencies for both sexes and men had an average of 10dB greater hearing loss at 8Khz than women. The decrease in hearing threshold in men between the age of 70 and 81 years was more pronounced at 2KHz (27dB) than at 4 KHz (15dB) and 8KHz (20dB). The average hearing loss in women increased at a constant rate between the age of 70 and 79 years (15dB), while between the age of 79 and 81 years the change in pure tone threshold was minimal.

Investigators	Procedure (methodology)	Findings
Burn, 1968		Higher the frequency, greater was the hearing threshold and older the person, the greater was the deterioration.
Milne, 1977	Performed longitudinal study of persons, age 62-90years	Hearing losses were small after one year but greater after 5 years especially at higher sound frequencies.
Ronald et al 1980	Studied the specific freq. and degree of hearing loss in 202 elderly nursinghome resident (159F & 43M)	PTA (500, 1000 & 2000Hz) for better ear showed a substantial deterioration in each decade interval beginning from ages in the 60s and extending into the 90s.
Plom, 1978		24% of the population is handicapped at the age of 65 years, over 30% by age 70 years and 50% by age 75 years.
B. SPEECH DISCRIMINATION:		
Pestalozza & Shore, 1955	Studied speech discrimination in a group of subjects over 60 years of age.	The reduction in speech discrimination could not be related to degree of hearing loss or slope of the audiometric configuration. They suggested reduced speech discrimination was related to degenerative changes involving spiral ganglion cells and fibers of the VIIIth nerve.
Goetzinger, 1961	Studied age & ear effects for discrimination score obtained using the CID-W-22 word list.	Found significant age and ear effect. The speech discrimination problems in aging were the result of a composite of changes in the auditory system and were not related to degeneration at any single level.
Harbert & Mendulle, 1966, Kasden, 1970		Reduction in speech discrimination scores were related to the degree of SN hearing loss rather than age.

Investigators	Procedure (methodology)	Findings
Harbert et al 1966		Reduced speech discrimination scores for the W-22 word list for the individuals over 60yrs
Kasden, 1970		Failed to demonstrate an age related reduction on speech discrimination.
Surr, 1977		Did not find any difference in speech discrimination scores across age groups with mild high frequency hearing losses for NU-6 word list at 40 dBSL.
Rintlemann and Schmmaier, 1974		Demonstrated significant age related reduction in speech discrimination in subjects over 60yrs of age. matched in terms of degree of hearing loss with younger hearing impaired persons
Jerger, 1973	Examined mean PB max scores as a function of age for groups with varying degrees of hearing loss	Found decrease in PB Max .with aging is similar to the decrease in absolute sensitivity with aging.
Bess & Townsend, 1977	Found age effects in the speech discrimination abilities of 556 subjects with flat hearing loss (age 14-98yrs)	Found significant age effect for mild hearing losses, the speech discrimination ability at 40 dBSL. decreased very slightlywith age. For greater amounts of hearing loss, speech discrimination decreased dramatically with age.
Luterman, Welsh ft Melroae 1966		Found more errors for elderly than for young listeners on W-22 word list at 40dBSL.
Bergman, 1971	Examined the speech discrimination ability of normal adults, between the age of 20 & 79yrs using a variety of altered speech tasks.	Reported a significant reduction in discrimination ability in the absence of any peripheral sensitivity loss.

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Investigators	Procedure (methodology)	Findings
Konkle et al 1977		Reported that time compressed speech discriminations scores showed a consistent decline with increasing age. The grater the amount of time compression, the greater was the age effect.
Orchik & Burgess, 1977	Examined (SSI-ICM) as a function of age of listener in four age-graps with normal peripheral hearing.	The two oldest age groups showed a significant reduction in discrimination.
Korobic et al, 1972		Poorer performance for elderly listeners in comparison with young listeners, where the elderly listeners had high frequency SN hearing losses and poorer speech discrimination scores unaltered speech and the test words were presented at relatively low SLs.
Gang, 1976		Found a strong relationship (r=0.83) between the age of the listener and the amount of roll over suggesting that the likelihood of Vlllth nerve involvement increases with age.

C. IMPEDANCE AUDIOMETRY

Investigators	Procedure (methodology)	Findings
Blood & Greenberg, 1976	Examined dynamic compliance of the middle ear system among the aged.	A significant increase in impedance in population aged 50 years and above.
Alberti & Kristensen,1972		Middle ear system becomes increasingly compliant upto middle age and then stiffens with further aging.
Jerger, Jerger & Mouldin, 1972		A systematic decline in static compliance with advancing age.
Hall, 1979	Studied static compliance in men and women.	Sex differences in S.C. were more pronounced between 30&60 years of age. Beyond 30 yeas, both men and women showed a systematic decline in static compliance.
Jepsin, 1963; Jerger et al 1972		A systematic decline in acoustic reflex threshold with advancing age. No sex difference in acoustic reflex threshold in geriatrics.
Habenuar & Snyder, 1974	64 normal individuals from 3-68 years.	Found poorer reflex threshold in the younger age groups.
Seaverton & Lennor 1976	Studied 3 age groups. 20-30, 40-50 & 60-70yrs	Acoustic reflex in 3 age groups were at equal SL but the supracoustic reflex amplitudes were reduced in the oldest group (60-70yrs).
Jerger, 1978		There was decline in ART for pure tone stimuli but for broadband noise, the age effect was non existent.
Nerbonne, Bliss & Ronald, 1978	Mean S.C. as a function of age & sex in 3 groups. 20-29yrs, 60-69yrs, & 70-79yrs.	Slight but no significant tendency for the S.C value to decrease with age. The acoustic impedance value increases with age particularly above 70 yrs. This is due to increased stiffness of the conductive mechanism as the age advances.

Investigators	Procedure (methodology)	Findings
Baethi & Leany, 1975 cited in Marshall, 1982	Admittance value in Two groups, of elderly (60-78yrs) and of young (17-29yrs).	Admittance value was higher in the elderly group as compared to the young adults.
Silman, 1979a		No difference in ART for pure tone between young & elderly normal hearing adults, but found increased acoustic threshold for white noise in the elderly subjects.
Thompson, Sils, Recke & Bui, 1980.	Studied the growth of the acoustic reflex to filtered noise & tone of 500Hz, 1KHz, 2KHz in 30 subjects between 20-70yrs.	No change in ART for either pure tone or filtered white noise as a function of age for normal hearing adults but did find decreased growth in amplitude of the acoustic reflex with Increasing age.
Hall (1982)	92 subjects graped according to age as 20-30, 60-69&70-80. The crossed & uncrossed reflex amplitude were measured.	Uncrossed reflex amplitude decreases with increasing age. Decrease in the amplitude of reflex with age is apparent only at the maximum signal intensity level (HOdBSPL) over the range of 20-80yrs, max. reflex amplitude of the average decreased by *56%.*
Wilson, 1981	Evaluated two groups of subjects using Impedance test battery (below 30yrs and above 50 yrs).	Found no significant difference between the static immitance value for the two groups of subjects although the below 30 yrs group had slightly higher static admittance than the above 50yrs group, but difference was not significant. The ART for the two groups were the same in the low to mid frequency region (250 Ha - 2000Hz); the reflex threshold for the above 50yrs group were elevated significantly (8dB) for 4000Hz and 6000Hz. In all the condition, the magnitude of the AR was substantially smaller for the above 6Oyrs group as compared with the below 30yrs group. The variability of the reflex magnitude was large for the both groups of subjects. Saturation of the individual growth function which was frequency dependent occurred twice as often with the above 50 years groups as with the below 30 yrs of group.

D) BEKESY AUDIOMETRY

Investigators	Procedure (methodo1ogy)	Findings
Jokinen & Kaiya 1970	60 Presbycusic ears were evaluated with forward & backward sweep frequency.	The sweeps with the continuous tone gave slightly better threshold values in forward than the reverse direction at middle and high frequency and the situation being the opposite at low frequency.
B) LODDHESS FONCTION 1) SISI test:		
Jerger et al 1959	Administered SISI on a grp, of 34 elderly subjects.	SISI scroes were unpredictable. SISI scores at lOOQHz & 4000Hz ranged from 0 percent (in retrocochlear involvement) to 100 percent (in cochlear disorders).
Young & Harbert 1967	Evaluated presbycusis subject & other cochlear disorders & compared based on •SISI	Did not find any difference between presbycusis and various cochlear disorders for SISI scores across a range of SPLs or at high levels.
Punnan (1976)		Scores show unpredictability. The presbycusic ear behave like normal ears, no pathology is detectable in the chochlea.
2) Loudness balancing:		
Pestalozza & Shone (1955)	Roger's technique of monoaural bifrequencies loudness balancing was used to assess recruitment on 20 elderly individuals	20% of the patients had complete recruitment, 30% had partial recruitment and 50% had none.
Goetzinger 1961	He tested 80 males and 40 females in a population of the aged subjects.	Among the male population 34% showed complete recruitment 51% incomplete recruitment. Among females they found 25% with complete recruitment, 40 % incomplete recruitment & 30% no recruitment.

Investigators	Procedure (methodology)	Findings
Harbert et al 1966	Used two monaural loudness balance test to assess the incidence of recruitment in 50 subjects over the age of 6 years.	Recruitment was found in less than 30% of the subjects. The absence of recruitment was often associated with an elevated aural overload threshold, suggesting that the cochlear dysfunction in aging may be a mechanical disturbance such as increased stiffness of the B.M. (basilar membrane).
Traynor (1975)	In subjects ranging from 78-92yrs used the impedance audiometry to detect recruitment.	60% of the subjects didnot show acoustic reflexes that would be indicative of recruitment while *40%* demonstrated reflexes at normal or near normal intensities.
3) Audiotory adaptation:		
Goetzinger (1961)	Used a threshold tone decay procedure in evaluating the subjects between 60-90yrs.	Ho correlation between the presence of presbycusis and tone decay. None of them exhibit significant decay at 1KHz, 2KHz and 5QOHz.
Willeford (1971)		Abnormal tone decay for only a small number of elderly subjects.
Milen & Peterson (1972) (cited in Mainer and Rupp 1979)	Studied normal & sensorineural hearing loss ears among the elderly & compared with the normal young adults.	tone decay pattern for their older population were not different from these of their young population.

F) FREQUENCY DISCRIMINATION AUDIOMETRY

Investigators	Procedure (methodology)	Findings
Mermauram (1955) cited in Marshall 1982	Difference liroen for frequency (DLF) at 20dBSL across the frequency 125Hz-400DHz.	DLF at this frequency range larger than normal but lesser than pathological condition such as Meniere's disease.
Konig (1957)	DLF for pulsed sinusoidal at 40dBSL for 125Hz-4000Hz 10 listeners per decade through 20-89 years.	Poor discrimination as a function of age.
Ross (1965)		At the age of 70yrs, the DLF is 2-3 times the value at the age of 25 years.
Zwicker & Senon (1978) cited in Marshall 1982	72 hearing impaired subjects 10 of whom had presbycusis	Showed greatly reduced frequency selectivity results compared with normal young adults.

G) DIRECTIONAL HEARING AND LOCALIZATION VS. LATERALIZATION AMONG AGED

Investigators	Procedure (methodology)	Findings
Matyker (1958) & springbon (1959)	Localization of phantom source.	The ability decreased steadily after the age of 30 years.
Kirikae (1969)	Tests used were 1) directional hearing 2) Time Vs. intensity trade.Subjects were divided into 2 groups 50-75yrs and 20-30yrs.	Minimal interaural time differences necessary to lateralize the sound image (TDL) for the aged was 0.082 whereas for the young it was 0.05. The younger people perceive the lateralization events with a slight difference in time.
Basatra & Russolo (1982)	Used 3 central tonal lists. Age range of subjects 50-88yrs.	Results show a considerable reduction of the performance intensity function with age whereas tonal test remains within the value of younger subjects.

Hi

EVOKED RESPONSES

Compared *to* developmental studies done on infants, literature on stud: es done on adults is limited. .

It was only in late seventies when Beagley and Sheldrake (1978) measured brain stem evoked potentials in response to clicks at 60dB, 70dB a*nd* 80dB from a group of 70 normally hearing subjects, who were divided into second to eight decades having 10 subjects. (5M+5F) for each of the decades. Results indicated only a minimal increase in latency of wave V as a function age and females had shorter latencies than males in most cases.

On the other hand Rowe (1978), took 25 young (mean age 25.1 years and range 17-33 years) and 25 old (mean age 61.7 years, range 51-74 years) and recorded ABR to 60dB HL stimuli at rate 10/sec and 30/sec and at 30dB HL at 30/sec rate. Have II and IV were found to be absent from one or both sides. He found a difference between means of 0.2 msec for wave I, 0.44 msec for wave III, and 0.36 msec for wave V, old subjects showing longer latencies than young ones.

Haves III and V were the most constant and reproducible peaks were seen-in all subjects at 60dB. At 30dB wave III was absent in 16% and wave V in 3%.

Have II for 30dB, 30/sec rate showed relatively decreased latency compared to 60dB, 30/sec trials in the old subjects. but only suggestively changes in the young subjects.

They have given one tailed 95% and *99%* confidence intervals for conduction times for 60dB stimulation at both 10/sec and 30/sec with suitable correction factors to be added when used in older subjects.

Limitation of this study is that his procedure for selecting the subjects did not rule out the possibility, and participation of subjects with high frequency hearing loss. Subjects were not matched for gender and hearing loss, there is no information regarding ear difference. Presentation level was not at equal SL.

Rosenhamer et al (1980) found significant difference of all peak latencies between young and old females, but not between young and old males. He found no significant latency difference between males and females among the old subjects. They reported shorter latencies in young females (below 50 years) compared to young males. It was not significant above the age of 50.

Thomson et al (1978) studied the better ear in Meniere's patients and found that age has some influence, with latency (of wave V) increasing approximately 0.1msec/decade.

Thorasen et al (1978), Jerger and Ball (1980) have found significant latency difference for peak V, between young and old individuals whereas Johnson and Lehn found this difference for peaks III, IV, V and VII.

Prolongation of latency of wave I upto 0.1-0.2 msec and of wave V upto 0.2 msec was seen with increasing age. For wave III there was a slight but significant difference between the age groups (25-34 years) and (55-75years) (Rosenhall, Bjorkman, Pederson and Kall 1985).

Older patients have a slight high frequency hearing loss and deviate from the younger individuals which causes age related BAEP changes (Stockard and Stockard, 1983).

But is not supported by the observation (Rosenhall et al) in younger individuals slight high frequency loss doesnot have any influence on the peak latencies.

In old individuals increased stiffness in the middle ear can induce a slight conduction hearing loss (Gliad and Glorlg, 1979). A combination of high frequency SN hearing loss and conductive hearing loss, might explain the longer peak latencies observed in elderly individuals.

Rosenhall et al (1985) reported no difference in the latency between females and males.

Debruyne (1986) reported a greater latency prolongation when the stimulus rate was increased. He found no statistical correlation between the scores on the synthetic sentence identification task and prolonged latency of wave V in the ABR in old groups. So they seen to be the result of two independent processes.

Von Wedel (1979) studied difference in ABR with age and sex. The Percentage difference between young and old object showed a decreasing wave occurrence with growing age. At higher intensity levels the difference between the age groups show comparable results as obtained for 30dB HL. They found well established fourth wave (IV wave) complex as a dominant wave. The possibility of the absence of the IInd, IIIrd, and Vth wave complexes increased with age. For the Ist wave complex there was growing wave latency with age. This was true for IVth wave complex also.

Jerger and Hall (1980) examined amplitude and latencies of ABE waveform as function of chronological age (20-79years).

In the normal group, latency increased as a function of age for both sexes. The average latency in the oldest group was 0.2msec. Longer than the average latency in youngest group Combining all age groups, the average latency for male subjects was 0.14 msec greater than the latency for female subjects.

In the SN group, latency showed little change as a function of the age. For male subjects, the average latency in the older group was only 0.10msec longer than the average latency in the younger group. For female subjects, there was no consistent change in latency as a function of age.

The latencies of the waves I, III and V as well as /P/ (interpeak interval) I-V and III-V were significantly shorter in F than in M. A correlative linear dependence on age could be shown for the latencies of wave I, III and V in M and for the wave V latency in F (Stuzebecher and Herbs, 1987). They hypothesized that age and sex dependence is not caused by different mechanisms. The ABR latency difference between M and F are in the main the result of the ABR age-dependence being less pronounced in females than in males.

Presbycusis is likely to be one cause of the age dependence of ABR. It is the result of sensory and neural degeneration on the auditory pathway, which begins in early childhood (Johnsson and Hawkins, 1972). Presbycusis increases with age and in more pronounced in males (ISO 7029, 1984). The age and sex dependence of ABR latencies and IPI parallels the presbycusis and one can assume a close relation between the two phenomena (Stuzebecher and Werbs, 1987).

Jerger and Johnson (1988) studied the interactive effects of gender, age and degree of SN hearing loss in 412 subjects. Click test level was selected on the basis of PTA2 wave V latency increased systematically as high frequency hearing loss increased. Wave V latency showed little change with degree of hearing loss upto the 50 to 60dB region. Above this, it increased in linear fashion with further increase in hearing loss.

Females showed shorter latencies than males and little change in wave V latency with increasing hearing loss. In elderly group the gender difference in the absolute latency of wave V was not apparent at high click levels, however, as effective click level decreased, latency increased for males but not for females.

They suggested 3 factors as possible bases for the overall gender effect on ABR latencies, differences in head and brain size, difference in whole body temperature and difference in hormonal mileau.

Otto and Mc Candless (1982) evaluated 30 elderly subjects, 30 young subjects with comparable SN hearing losses and 30 normally hearing subjects.

The groups with high frequency hearing loss produced waveforms that differed from normal responses.

Elderly subjects had slightly longer latencies when compared to young hearing impaired subjects.

When advanced age and high frequency hearing loss interact, high frequency loss found to be greater factor in morphological and latency changes.

This study lacks external validity, as the sample size is limited. Details regarding statistical analysis are not mentioned.

Soucek and Mason (1987) studied hearing in the elderly using ECOG and ABR.

There was no significant delay in brainstem conduction times for the elderly, indicating normal transmissions through the brainstem.

ECOG showed lower levels of the activity in cochlea and probably the contributing factor they thought was the reduced myelinations of the nerve tracts which is a feature of advanced age.

Spivak and Malinoff (1990) demonstrated spectral differences in the ABRs of old and young subjects. 80 subjects were divided equally into 4 groups. Group A, 18-35; group B, 55-64; Group C, 65-74; and group D, 75-80.

On spectral analysis, 3 major energy peaks at approximately 200, 500, and SOOHz were seen. „ Composite spectral profile suggested increase in low frequency energy concentration as a function of increasing age (spectral profile measurement of the amplitude of spectral components at l00Hz intervals). The amplitude of the low frequency spectral component was significantly longer in the older age group than in the younger group. Significant differences were not found in between A Vs B or C Vs D but between A Vs C or D and B Vs D.

Underlying sources of the excessive amounts of noise and consequently the poor morphology of ABRs from old subjects may be related to general change in physiological processes related to aging (Spivak and Malinoff, 1990). They suggest modification of recording parameters when ABRs are recorded from older patients. This can be done through optimizing S/N ratio through signal averaging, to use greater number of sweeps. Second approach is analog or digital filtering during response acquisition.

The time from wave I to III and therefore from I to V, increased slightly with increased frequency of stimulation and increased age. The time from III to V and V to VII was unaffected by change in stimulus condition or age (Rowe, 1978).

Rosenhamer et al (1980) reported gender related I-V IPL differences in young individuals which diminished with increasing age and was not observable in the old age group.

There is a significant correlation between age and interpeak latency (IPL) I-V in males (Maurizi et al 1982). But, Rosenhall et al (1985) and Rosenhamer et al (1980) on the other hand could not show any age dependence of the IPI I-V The constancy of this parameter indicates that the neural function of the auditory pathway in the brainstem is not affected to a measurable degree in old individuals with normal hearing.

Chu (1985) showed a small increase with age in the IPI III-V and I-V, whereas Allison et al(1983) demonstrated a significant increase with age in the IPI I-III and I-V.

Kjaer (1980) found a pronounced increase of IPLs with increasing age for males, while the IPLs of older women were only slightly longer than those of younger women.

Mean interpeak latencies for elderly and young hearing impaired subjects were within 0.1msec, except for the difference between left ears between waves I and III. This suggests that age differences as the whole are quite small.

Although it is popular that amplitude of ABR is much more variable within subjects than latency, and clinical application is limited, many significant consistent age related changes have been reported.

Psatta and Matei (1988) studied age-dependent amplitude variations of BSAEPs. Results show components I-V undergo a significant variation in amplitude with age over a life time (l-70years). All the brain stem auditory evoked potential components (except-I) increased markedly in amplitude between 1 and 10 years. There after a progressive decrease was shown by the majority of components (I-V) between the ages of 10 and 50 years. Components VI and VII had smaller amplitude change with age. Wave VII being stable between 10 and 70 years at a stimulation of 80 dBHL. The amplitude difference between initial (I-II) and subsequent BAEP components (III-V) was greater in old age due to an unequal reduction (greater reduction of initial components). At 10 and 30 years, wave III is also normally larger in amplitude than the wave complex IV-V. Slowing of amplitude changes occurred in old age which may be related to physical factors influencing volume conduction during old age. Theoretically, the difficulties in audition of presbycusis, particularly in the high frequency domain (4-8KHz), to which the click stimuli belong, would suggest a greater diminution of amplitude at this age.

Amplitude ratios between components V and I at different ages show subunitary ratio at 1 year and it increases progressively upto 2.6 at the age of 70.

BAEP amplitude is also a controllable parameters. The use of standard amplitude values of the IV-V complex is superior to the amplitude ratio between component V and I as ratio depends strongly upon age (Psatta and Matei, 1988).

Beagley and Sheldrake (1978) gave two possible explanations for diminution of amplitude with age, until in the 8th decade.

1) Lack of synchrony between individual responses following individual click stimuli. The mean latency of wave V from trial to trial and subject to subject did not show very great changes, so it is necessary to postulate a greater scatter or individual response latency and thus poor synchrony with increasing age.

2) Increased tissue impedance may have played a part in the diminution of amplitude noticed in the older subjects.

Whereas, Von Wedel (1979) stated that amplitude values show no significant age dependency.

In the normal group, wave V amplitude for females subjects showed a very slight decrease (0.025 uv) from the youngest to oldest age groups. For male subjects the amplitude decrease was twice as great (0.050 uv) from the youngest to the oldest age groups.

Female amplitude exceeded male amplitude by amounts ranging from 0.080 uv in the youngest group to 0.120 uv in the oldest group.

In the SN group, there was a slightly greater age effect on wave V amplitude for females, amplitude decreased by about 0.050 uv. For males,- amplitude decreased by about 0.020 uv (Jerger and Hall, 1980).

Soucek and Mason (1987) found a significant reduction of amplitude for all component of the ABR in the elderly subject when compared to controls.

Wave replicability deteriorate with age (Rosenhamer, Lindstroa and Lundborg, 1980).

The recognizability was the same upto 45yrs, in older individuals waves II and -IV were more difficult to identify (Rosenhall et al, 1885). Kjaer (1980) reported better recognizability for waves II and IV.

SUMMARY

TABLE-II

Sl. NO.	Year	Investigator	Subjects No.	Age range	Inclusive criteria	Stimulus Type	No.of stimulus	Rate	Level	Findings
1	2	3		5	6	6	7	8	9	10
1	1978	Beagley & Sheldrake	70 5M+5F for each decade	20-80	Normal hearing	Click	1024 to 2048	20/sec 10/sec	60,70 80dB BL	Mininal increase in latency of wave Y as a function of age F had shorter latency than M. Diminution of amplitude with age.
2	1978	Rowe	50 25 young 25 old	17-33 51-74	Hg. thre. to click stimli within 6dB for young & within 14dB for old. No h/o neurologic disease.	Click	1024 to 8192	10/sec 30/sec	30,60 d8 8L	Wave II 4 1V absent from one or both sides, most reprodacible peaks are III 4 Y. Old subjects showed longer latency. IPI 1-111 and 1-Y increased with age.
3	1978	Thomsen	70		Studied better ear in Meniere's pts. No conductive impairtent.	Tone burst 2KHz	Rise fall time0.3msec plateau 1msec.	10/sec	98dB SPL	Latency increasing approximately 0.lmsec/decade.
4	MM	YonWedel	142 old 33 young	59-80 20-32	Confirsed normal hearing with audiogram		2048		30,50 4 70dB Hi,	Decreasing wave occurrence with growing age. The possibility of the absence of wave IInd, IIIrd and Yth increased with age. 1st and lYth wave latency increased with age. Amplitude values show no significant age dependency.
5	MM	Jeger & hall	319 182m 137f	20-79	98 subject had 8g. sensitivity with in normal limits. 221 had vary degree of SM hearing loss.		2048		90-100 4BHL & 10-20dB decreased until ABR waveform absent.	In normal group, latency increased as a function of age for both sexes. The average latency in the oldest group was 0.2msec longer than the youngest group. Wave Y amp. for F showed slight decrease (0.025uv) as compared to tales (0.050uv) with age. In the SH group, latency showed little change as a functions of age. Amp. of wave V decreased by 0.050uv in F and 0.020 uv in M with increasing age.

1	2	3	4	5	6	6	7	8	9	10
6	1980	Kjaek								Pronounced increase of EPL with increased age for sales.
7	1980	Rosenhamer et al	62 21 youngF 10 old F 20 young M 11 old M	20-37 50-63 23-40 50-65	Normal pure tone audiogram	Click	2000-3000	22.5/ sec	80,60 MOdBSL	Found significant difference of all peak latencies between young A old females, but not between young & old males. No significant latency difference between males and females among the old subjects.Gender related 1-F EPL difference in young indhidulas ditinished with increasing age. Wave replicability deteriorated with age.
8	1982	Maurizi								Significant correlation between age & IPL 1-Y in males.
9	1982	Otto & Mc Candles:	90 30 old 30 young M 30 hg.imp.	60-90 18-31 17-15	Normal hearing on battery of tests like sp. audiometry impedance SSI, TDT, STAT, SISI etc.	Click	1024	10/sec	8 M 5 M B nSL	When advanced age and high frequency hearing loss interact, high frequency loss found to be greater factor in morphological & latency changes
10	1983	Allison et al								Significant increase in IPL 1-111 & 1-V with age.
11	1985	Chu								Stall increase in 1PI III-V & f-V with age.
12	1985	Rosenhall, Bjorkman Pederson & Kall	268 153F 115M	515	Normal hearing with the exception that a slight high freq. loss in some cases.	Clicks	1021 & 2019	25/sec	80dB !L	Have II & IV tore difficult to identify in older group. Prolongation of latency of wave 1 upto 0.1-0.2msec 4 of wave V rep to 0.2msec, seen with increasing age. No difference latency between female and male.

1	2	3	1	5	6	6	7	8	9	10
13	1987	Soucek & Mason	31 old 15 young	67-97 19-30	Normal otoscopic findings No H/o COM, noise exposure brainstem lesions.	Click	2018		90dBnHL	Ho significant delay in brainstem conduction times for the elderly. Significant reduction of amplitude for all exponents of the ABE in elderly.
14	1987	Sturebecher & Werbs	69F 86M	Mean age 42.7 39.1	Normal hearing	Clicks	1096	10/sec	115dbSPL	A correlative linear dependence on age for the latencies of wave 1, III, V in M and for the vave V in P.
15	1988	Jerger & Johnson	412 325 with coch hg.loSS 87with retro coch disease	M15-79 F14-80 M5-75 F-18-61	SN loss ranged from minimal to severe No ME pathologi 3 Retrococh. invol-Vement confirted surgically & Radio-logically.	Clicks	1000-1500	21.1/- 11.1/ SMS	On the basis of PTA_2	Wave V latency increased as high frequency loss increased. In elderly group gender difference was not apparent at high click levels but as the click level decreased, latency increased for M but notfor F.
16	1988	Pstta & Matei	50	l-70yrs 5subg. 1,10,30 50470 res.10 subj in each.	a)Normal hearing & low click hearing threshold (lOdBSPL) b) Equal representation ofH & F. c} Sate age in subgrps. studied with a deviation of only i6 montus. In old age an elevation of lOdB 8L threshold Has accepted.	Clicks	2000	10/sec	80 4 6MB 8L tasking by50dB8L	Components 1-V undergo a significant variation in amplitude with age over a life time.

1	2	3	4	5	6	6	7	8	9	10
13	1990	Spivak & Nalinoff	80 4grps	A19-35 B55-64 CM-74 B75-80	Nornal hearing for their age as defined by the I960 United State: Dept. of Health Education and Welfare survey. Ho H/o stroke, psychiatric or neurologic proble.	Cliks	2000	11.1/ sec	60dB SL	Increa:e in low frequency energy concentration and poor Morphology a: a function of age.

CONCLUSION

It is apparent that most of the studies show significant difference in auditory brainstem responses in older individuals as compared to youngers. But controversies are still prevailing. It is a fact that ABR are affected by many variables due to changes in stimulus and recording parameters as well as criteria adopted to select subjects. Also it is very difficult to control factor such as noise exposure presbycusis and other silent diseases in older individuals.

While recording and analysing ABB of older individuals one should bear in mind the normal variations and as suggested by Spivak and Maniloff (1990), it is mandatory to modify recording parameters through optimizing SN ratio through signal averaging to use greater number of sweeps and analog or digital filtering during response acquisitions.

BIBLIOGRAPHY

Allison T, Hume A.I,., Wood C.C., Goff W.R. (1984): Developmental & aging changes on somatosensory, auditory and visual evoked potentials. Electroencephalogr. Clin Neurophysiol, 58, 14-24.

Basatra, A., & Russolo, M., (1982): Comparison between central tonal tests and central speech tests in elderly subjects. Audiology, 21, 334-341.

Beagley, H.A. & Sheldrake J.B. (1978): Differences in brainstem auditory response latency with age & sex. Br. J. Audiol. 12, 69-77.

Bezold, F., (1894): Cited by Kopra, L.L. in "The Auditory, communicative Manifestations of presbycusis" in Rehabilitative Audiology (Ed). Hull, R.H., 243-269, Grune and stratton Inc. New York, 1982.

Bijay Kumar, (1990): Audiological findings in Geriatrics: A comparative study. An Unpublished Master's Dissertation, University of Mysore.

Birren, J.E., (1955): Cited by Ronch, J.L., in "Who are these aging persons?" in Rehabilitation Audiology (Ed). Hull, R.H., 183-213, Grune and Stratton Inc., New York, 1982.

Bunch, C, (1929): Cited by Kopra, L.L., in "The audiotory communicative manifestations of presbycusis in Rehabilitative Audiology (Ed). Hull, R.H., 243-269, Grune and Stratton Inc., New York, 1982.

Busse, E., Pfeiffer, E., (1977): Cited by Ronch J.L.,, "Who are these aging persons?" in Rehabilitation Audiology (Ed). Hull, R.H., 243-269, Grune and Stratton Inc., New York, 1982.

Butter, R., & Lewis, M., (1977): Cited by Ronch, J.L., "Who are these Aging persons?" in rehabilitative Audiology (Ed). Hull, R.H., 243-269, Grune & Stratton Inc., New York, 1982.

Carhart R , (1956): "Peculiarities of auditory function in conjuction with pusbycusis, Laryngoscope. 68, 253-277.

Carhart.R.,Jerger J.,(1959): Preferred method for clinical determination of pure tone threshold. J. of Speech and Hearing Disorders. 24,330-345.

Carhart.R.Tillman, J.W., (1970):Interaction of competing speech signals with hearing losses. Arch of Oto.77, 273-279.

Chu, N. (1985): Age related latency changes in the brain-stem and evoked potentials.Electroencephalogr. Clin Neurophysiol 431-436.

Corso, J.E., (1963): Age and sex variation in pure tone thresholds and A survey of hearing levels treats 18 to 65 years. Arch Oto. 77, 385-405.

Dayal, V.S., and Nussboun, M.A., (1971): Patterns of pure tone loss in presbycusis, Acta. Oto. 71, 382-384.

Fowler, E., (1940): The aging ear, Arch.Oto 10, 475-480.

Fria T.J. (1984) Maturation of the ABR: Additional perspectives. Ear Hear. 5, 361-365.

Gaeth, J., (1948): A study of phonemic regression associated with hearing loss. An Unpublished Doctoral Dissertation, North Western Univ., Evonston.

Glorig, A., Davis, H., (1961): Age, Noise and Hearing loss. Anna1s *of* Otology Rhinology and Laryngology. 70 553-557.

Goetzinger, C,, (1961): "A study of hearing in advanced age". Arch, of Oto. 73, 662-674.

Hall, J.W., (1982): Acoustic reflex amplitude, Effects of age and Sex. Aodiology. 21, 294-309.

Hecox, K., Galambos, R., (1974): Brain-stem auditory evoked responses in human infants and adults. Archives of. Otolaryngology. 99: 30-33.

Hull, R.H., (1978): Cited by Maurer, J,F., "The psychological aspects of presbycusis" in Rehabilitative Audiology (Ed) Hull, R.H., 271-281, Grune & Stratton Inc., New York, 1982.

Indrani, R., (1981): Normal hearing by air conduction as a function of age and sex in Indians. A dissertation submitted to the University of Mysore.

Jeny (1991): A comparative study of masking level difference in geriatrics. An Independent Project Submitted to the University of Mysore.

Jerger, J., (1973): Audiologic findings in aging. Ady Otorhinolaryngol. 206, 115-124.

Jerger, J & Hall J. (1980): Effects of age and sex the ABR. Arch. Otol 106, 382-391.

Jerger, J., and Johnson, K. (1988): Interaction of age, gender and SN hearing loss on ABR latency. Ear Hear... 9, 168-176.

Jewett, D., & Willston, J.S., (1971): Auditory evoked far fields averaged from scalp of human. Brain 96, 681-696.

Jokinen and Kaiya (1976): Presbycusis: Forward Vs backward frequency sweep audiometry. Acta Otolaryngology 76, 227-231.

Kapur, Y.P., et al., (1967): Hearing in Todas of south India, Arch. of Oto. 74, 56-62.

Keith W.J. , Greville K.A. (1987): Effects of audiometric configuration on the ABB. Ear Hear, 8, 49-55.

Kelly-Ballweber D., Dobie R.A. (1984): Binaural interaction measured behaviourally and electrophysiologically in young and old adults. Audiology, 23, 181-194.

Kirikar, I.S., & Shitara, T., (1964): A study of hearing in advance age. Laryngoscope. 74,, 265-220. .

Kirikar, T., (1969): Audiotory function in advanced age with reference to histological changes in central auditory system. Audiology. 8, 221-232.

Konig, E., (1957): Pitch discrimination and age. Acta Otolaryngol. 48.

Konkle, D., Beasley, D & Bess, F., (1977): Intelligibility of time altered speech in relation to chronological aging. J. Of. Speech and Hearing. res., 20, 108-115.

Krishnamurthy, B.N., (1985): The brainstem evoked responses in geriatric population. An Independent Project Submitted to the University of Mysore.

Lutermann, D.M., Welsh, O.L., Mabrosi, J., (1966): Response of aged male to time altered speech stimuli. J.Speech Hear res., 9, 226-239.

Margolis, R.H.,-(1978): The effect of age on acoustic reflex threshold in normal hearing subjects. G.R., Popelka (Ed). Detecting hearing loss from acoustic reflex thresholds, ASHA Monograph.

Marshall, L., (1981): Auditory processing in aging listeners. Journal of Speech and Hearing Disorders. 46, 226-240.

Maurer, J.F., and Rupp, R.R. (1979): "The psychological aspects of presbycusis", in Rehabilitative Audiology (Ed) Hull, R.H., 271-281,Grune & Stratton Inc., New York, 1982.

Maurizi M., Altiasimi G., Ottavian, F. (1982): ABR in the aged Scand. Audiol. 11, 213-221.

Miller, M.H., (1967): Audiological rehabilitation of the geriatric patients. MAICO. *2(1),* 1-31.

Milne, J.S., Lander, I.J., (1975): Pure tone audiometry in Older People. British. J. of Audiol. 50-57.

Milne, J., (1977): A longitudinal study of hearing loss in older people. British J. Audiol. 11, 7-14.

Moller & Janneta (1981): Auditory evoked potentials in "The pro-ed series in communicative disorders" Series Ed. Halpern. H,, Pro-ed, Austin, Texas.

Nerbonne, M.A., Bliss, A.T., and Schow (1978): Acoustic impedance values in the elderly. Journal of Ameriran Audiological Society. 4, 57-59.

Nixon, J., Glorig, H., High, D.W., (1962): Changes in air and bone conduction thresholds as functions of age. Annals of Otology. Rhinology *And* Laryngology. 74, 288-298.

Otto W.C. and McCandless G.A. (1982) : Aging and the ABR. Audilology. 21, 466-473.

Pestalozza, G., *&* Shore, I., (1955): Clinical evaluation of presbycusis on the basis of different tests of auditory function. Laryngoscope. 65, 1136-63.

Psatta D.M. and Matei M (1988): Age dependent amplitude variation of brain-stem auditory evoked poteritials. Electroencep. and Clinical Neurophysiology. 71, 27-32.

Punnan, B., (1976): Audiological findings in presbycusis. A Master's Dissertation Submitted to the University of Mysore.

Rosen, S., (1962): Cited by Kopra, L.L. in "The auditory - communicative manifestation of presbycusis" in Rehabilitative Audiology (Sd) Hull, R.H., 243-269, Grune & Stratton Inc., New York, 1982.

Rosenhamer H.J., Lindstrom B., Lundborg T. (1980): On the use of click-evoked electric brainstem responses in audiological diagnosis: Scand. Audiol. 9, 93-100.

Rosenhamer, H.J., Lindstrom, B. , Lundborg, T., {1981): On the use of click-evoked electric brainstem responses in audiological diagnosis, III. Latencies in Cochlear hearing loss. Scand Audiol. 10, 3-11.

Rosenhall, U., Bjorkman, 6., Pedersen, K., Kall A., (1935): Brain-stem auditory evoked potentials in different age groups. Electroencephalogy Clin Neurophysiol 62, 426-430.

Rosenwasser, H., (1964): Otitic problems of the aged. Geriatrics. 19, 11-17.

Rowe, M.J., (1978): Monaural variability of the brain-stem auditory evoked response in young and old subjects. Electroencephalogr. Clin. Neurophysiol. 44, 459-470.

Sataloff, J., Vasselo, L., & Manuduke, H., (1965): Presbycusis air and bone conduction threshold, Laryngoscope. 51: 889-901.

Schuknecht, H., (1955): Presbycusis, Laryngoscope 65. 402-409.

Schuknecht, H., (1967): The effect of aging on the cochlea (Ed) B. Graham. Sensorineural hearing loss Process and disorders. Boston, Little Brown.

Schuknecht, H., (1967): Further obserations on the pathology of presbycusis. Archives of otolaryngology. 80, 369-392.

Seth, S.M., & Kacker, S.K., (1977): Contours of normal hearing in Delhi as factor of aging. Silent World. 6(1), 21-23.

Shanas, E., and Maddox, G., (1976): Cited by Kopra L.L., in "the auditory-communicative manifestation of presbycusis" in Behabi1itative Audiology (Ed) Hull, R.H., 243-269, Grune & Stratton Inc., New York, 1982.

Soucek S, Mason S.M. (1987) : A study of hearing in the elderly using non-invasive ECOG and ABR. J. Otolary 16(6), 345.

Soucek S, Michaels, L., Frohlich A (1986): Evidence for haircell degeneration as the primary lesion in hearing loss of the elderly. J_ Otolaryngol 15: 175-183.

Spivak L.G. & Malinoff R. (1990): Spetral differences in the ABRs of old and young subjects. Ear Hear. 11(5), 351-358.

Stockard, J.J., & Stockard, J.E, (1981): Auditory Evoked potentials in "The, pro-ad series on communication disorders" Series (Ed). Halpern, H., Proed, austin, Texas.

Sturzbecher & Werbs (1988): Influence of age, sex and hearing loss on ABR latencies. Scandinavian audiology. 17, 248-250.

Thompson, D.J., Sills, J.A., Recke, K.S., Bui, D. M., (1980): Acoustic reflex graph in the aging adults. Journal of Speech and Hearing Research. 23, 405-418.

Thomson J. , Terkildsen K, Osterhammel P. (1978): Auditory brainstem responses in patients with acoustic neuromas. Scand. Audio]. 7, 179-183.

Von Wedel, H. (1979): Differences in brainstem response with age and sex. Scandinavian Audioiogy. Supply, 9, 205-209.

Weinstein and Ventry (1983): Audiologic Correlates of Hearing Handicap in the Elderly. Journal of Speech and Hearing Research. 26, 148-151.

Audiologic Correlates of Hearing Handicap in the Elderly. Weinstein and Ventry JSHR, 1983, 26, 148-151.

Zwaardemaker, H., (1891): Cited by Kopra L.L., in "The Auditory communicative manifestations of presbycusis". in Rehabilitative Audiology (Ed) Hull, R.H., 243-269, Grune & Stratton Inc., Hew York, 1982.