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# THE INFLUENCE OF AGING ON AUDITORY EVOKED POTENTIAL IN ADVANCED AGE GROUP

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**ABSTRACT**
Each sensory system has its own time of maturation. The individuation of the exact period of life when brain aging starts is difficult to defined. Normally, the amplitude of evoked potentials decreases, and their latency increases from adult to elder life. The absolute latencies of Waves I, III, and V are 0.1 to 0.2 ms longer for subjects aged 50 years and older than for those aged 20–30 years. The influence of age on interwave latency is inconclusive but also suggests that there may be an age-related prolongation of 0.1 to 0.2 ms for the I–V interpeak interval. Age related changes may be confounded by the presence of sensory hearing loss. Since age effects on central conduction time in the acoustic pathway are still debated, the present study was conducted to assess the effects of aging on central conduction time & compared with the young adult. Auditory evoked potential were studied in twenty five normoacoustic elderly subjects & results were compared with twenty five age and sex matched controls (young adults between 18-25 years age group). The older adults had prolonged wave III & wave V latencies. The interpeak latency I-III and I-V are also prolonged which suggest that aging process is central phenomenon.

**Keywords:** Auditory evoked potential, Advance age, Peak latency, Interpeak latency, Amplitude

## 1. Introduction:

Evoked potentials provide a measure of the function of sensory systems that change during the different stages of life. Each sensory system has its own time of maturation. The individuation of the exact period of life when brain aging starts is difficult to defined. Normally, the amplitude of evoked potentials decreases, and their latency increases from adult to elder life. Many authors speculate that these modifications depend on neuronal loss, changes in cell membrane, composition or senile plaques present in older patients, but there is no evidence that these changes modify the cerebral function in healthy aged individuals.[1]

Age related neuronal and structural changes within the human brainstem predict brainstem auditory evoked response differences. Findings regarding cell loss are contradictory but degenerative changes such as cell size and cell shape irregularities and accumulation of lipofuscin pigment observed in the ventral cochlear nucleus, superior olivary nucleus, inferior colliculus, medial geniculate body, and inferior olive. Degenerative changes in the myelin sheaths and axis cylinders of the above structures are also reported.[2]

The relation between age and decrease in volume of the cochlear nucleus that was not associated with neuronal count, but possibly to changes in axon size and degree of myelination also reported.[3] Hansen (1965) suggested progressive neural atrophy within peripheral and central auditory system with advanced age.[4]

The absolute latencies of Waves I, III, and V are 0.1 to 0.2 ms longer for subjects aged 50 years and older than for those aged 20–30 years. The influence of age on interwave latency is inconclusive but also suggests that there may be an age-related prolongation of 0.1 to 0.2 ms for the I–V interpeak interval.[5,6] Age related changes confounded by the presence of sensory hearing loss & they have opposite effects on interwave latency and similar but nonadditive effects on absolute latency.[7]

The latency of an auditory evoked potential is influenced directly both by the point of maximum motion of the basilar membrane and by synchrony of neurons contributing to the response.[8] In case of high-frequency loss, the peak of basilar membrane motion may occur at a point of hair cell loss. Thus, hair cells located apically to the peak of membrane motion respond to the signal, resulting in an increase in response latency. Furthermore, primary degeneration of spiral ganglion cells may alter the probability of a response in a central auditory neuron because of the reduction in the number of auditory nerve fibres that innervate the neuron.

The aim of our study is to provide a supplementary contribution concerning the dependence of brainstem potential on advance age.

**2. Material and Method:**

Twenty five normoacoustic subjects in the age group of 60-80 years were randomly selected from both sexes. A detailed history was obtained & otolaryngological examination carried to exclude ear discharge, vertigo, trauma, operation and intake of ototoxic drugs. Patients with history suggestive of diabetes, hypertension, ischemic heart disease, renal disorder and smoking were excluded from the study. Specific history was also taken to rule out any prolonged exposure to noise. The Rinne & Weber test were done to rule out any abnormality of hearing defects. The test was done with due permission of ethic committee of institute along with written consent from the subjects. BAEP recording was done in a quiet air conditioned room with the help of RMS EMG EP MARK II Machine manufactured by RMS recorder & Medicare system, Chandigarh. Recordings were obtained using silver cup electrodes filled with contact gel. The electrodes were fixed on vertex (Cz, 10-20 international electrode placement system) & on the mastoid process. The ground electrode was placed on forehead (Fz). Impedance of electrode was kept below 5 k ohms. A band pass of 100-3000 Hz was used to filter out undesirable frequencies in the surroundings. Responses to 2000 click presentation were averaged for 10 msec.

The subject's hearing threshold was determined for each ear at the time of testing. The acoustic stimulus was rarefaction clicks, which were generated by passing 0.1 ms square pulses through shielded headphones. Clicks of intensity 60 dB above the hearing threshold were delivered at the rate of 10 pulses per second. Monaural stimulation was used & contralateral ear was masked by white noise at 30 dB below the click intensity. The peak latencies of waves I, II, III, IV & V were measured. The interpeak latencies I-III, I-V, III-V was computed. Amplitudes of waves were also measured from peak to following trough of the wave.

**3. Results:**

Auditory evoked potential were studied in twenty five elderly subjects (60-80 years) & results were compared with twenty five age and sex matched controls (young adults between 18-25 years age group). The mean and standard deviation of the absolute latency and interpeak latency in milliseconds are shown in Table 1, 2 & 3.

A significant main effect for age indicated that older adults had prolonged wave III & wave V latencies than young adults. Older individuals had longer I-III and I-V interpeak latency than young adults. The older males had significantly prolonged latencies of waves III and V as compared to older females. Also, the interpeak latencies of the wave's I-III, I-V & III-V had a significantly increased value in older males than in the older females.

**4. Discussion:**

Changes in interpeak intervals reflect changes in neural conduction time in the auditory system and are used diagnostically in acoustic neuromas and demyelinating diseases**.** Absolute latencies of auditory brainstem response waves tend to increase in older adults (Allison *et al* 1984; Allison *et al* 1983; Jerger & Hall 1980; Martini *et al* 1991; Ottaviani *et al* 1991; Otto & McCandless 1982; Rowe 1978).[9-15] Interpeak intervals may also increase in the aging human (Rowe 1978; Oku & Hasegewa 1997; Rosenhall *et al* 1986),[15-17] although not all studies have found evidence for age related increases in interpeak intervals (Martini *et al* 1991; Ottaviani *et al* 1991; Otto & McCandless 1982; Beagley & Sheldrake 1978; Costa *et al* 1990; Harkins 1981).[12-14,18-20]

The latency prolongation of the ABR components showed that the cognitive processing was affected with aging. Cognitive alterations observed with aging related to the dopaminergic and the cholinergic systems which play an important role in the process of cognition, because the number of mascarinic Ach receptors in the CNS and the activity of choline acetyltransferase in the nerve terminals were decreases with aging. On the other hand, nigrostriatal axons, nigrostriatal dopaminergic neurons and strial endogenous dopaminergic concentration in the human brain and in the D2 dopamine receptor binding sites were found to decrease with age. So, the cognitive decline is caused by the deterioration of the dopaminergic and the cholinergic systems. Thus, cognitive decline occurs as age advances, which may be the reason for the changes in the BAEPs as age advances.[21]

The prolonged Wave III and wave V latencies which were found in the present study supported by H S Johannsen (1984)[22] who found significant long latency in older subjects for III, IV, V, and VII at all levels of stimulus intensity. The

difference in latency time between two consecutive responses shows that potentials IV and VI followed more quickly in the older than in the younger persons. In our study prolonged latencies due to age were also found at I-III and I-V interpeak latency is in accordance with Uziel A *et al* (1980)[23] who reported prolonged I-III, I-V and III-V interpeak latencies in advance age when compared to young adult subjects (about 0.2 ms).

Our findings are opposed by Oku and Hasegewa (1997)[16] who found the latencies of Waves I, III, and V were progressively delay in the older participants, might be due to increased high-frequency thresholds in older participants.

In contrast with studies suggesting that age has a direct effect on ABR latencies and interpeak intervals, other studies suggest that threshold elevation is more of a factor.

Beagley and Sheldrake (1978)[18] did not find latency abnormalities in older adults with normal hearing. Otto and McCandless (1982)[14] observed no significant difference in ABR latencies in young and older participants with similar degrees of high frequency hearing loss. Martini *et al* (1991)[12] reported that older adults had increased latencies for Waves I, III, and V compared to young participants with normal sensitivity due to the mild hearing loss and not specifically to aging. Ottaviani *et al* (1991)[13] found the latencies of Waves III and V were significantly prolonged in old relative to control participants only for the groups with PTAs of greater than 30 dB HL. Older had significantly increase I-V interpeak intervals, but when the participants were regrouped by hearing levels, no significant changes were observed in the IPIs. Thus, the authors concluded that age-related changes in the absolute latencies and IPIs were due to threshold changes rather than aging per se.

Our study present with significant prolongation of III and V latency & I-III and I-V interpeak latency & this finding is opposed by Costa *et al* (1990)[19] who found age related prolongation of wave I whereas wave III do not show significant change. Interpeak latencies I-III decrease and I-V and II-V (considered true "central conduction time" through the acoustic pathway) do not show a significant change.

Our study demonstrate reductions in ABR amplitudes as a function of age supported by Beagley & Sheldrake 1978; Costa *et al* 1990; Harkins 1981; Kjaer 1980; Psatta & Matei 1988; Sand 1991.[18-20,24-26] Even when threshold elevation is accounted for, most studies suggest a reduction in ABR amplitudes in older participants. Typically, the amplitude of Wave I or the electrocochleogram is more affected by age than Wave V (Costa *et al* 1990; Psatta & Matei 1988).[19, 25]

Finally, significantly shorter latencies were found for older females than older males at Waves III and V. In young groups with normal hearing shorter Wave V latencies have observed in females than in males.[27]

Using interpeak latencies, Stockard *et al* (1978)[28] reported shortened Wave I-V IPLs in females compared to males. We found comparable trends in Wave I-V IPLs for females. It has been suggested that the latency advantage for females may be due to differences in the anatomical distances between the various segments of the auditory pathway.

**Conclusion:**

The interpeak latency represents conduction time through relay stations of auditory pathway in the brainstem. Thus IPL I-III is a measure of conduction from acoustic nerve to pontomedullary region, III-V conduction in the more rostral pontine and midbrain portion of the pathway and I-V reflects the total brainstem conduction time (Starr A 1976).[29] The significant prolongation of I-III and I-V interpeak latency in our study suggests that the aging process affect the central part of auditory pathway on which does not involve the peripheral part of the acoustic pathway.

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**Table 1: Comparison of Absolute Latency in Young & Older Adults**

| Absolute Peak Latency | Young Adult | Old Adult | P Value |
|---|---|---|---|
| I | 1.73 ± 0.18 | 1.70 ± 0.22 | 0.602 (NS) |
| II | 2.72 ± 0.19 | 2.70 ± 0.24 | 0.604 (NS) |
| III | 3.25 ± 0.24 | 3.81 ± 0.12 | <0.001 (HS) |
| IV | 4.82 ± 0.30 | 4.85 ± 0.20 | 0.88 (NS) |
| V | 5.64 ± 0.28 | 5.88 ± 0.26 | 0.005 (S) |

**Table 2: Comparison of Interpeak Latency in Young & Older adults**

| Interpeak Latency | Young Adult | Older Adult | P Value |
|---|---|---|---|
| I-III | 1.89 ± 0.21 | 2.24 ± 0.17 | <0.001 (HS) |
| I-V | 3.85 ± 0.38 | 4.39 ± 0.25 | <0.001 (HS) |
| III-V | 1.98 ± 0.38 | 2.15 ± 0.28 | 0.082 (NS) |

**Table 3: Comparison of Absolute Peak & Interpeak Latency in Older Male & Females**

| BAEP Waves | Older Male | Older Female | P Value |
|---|---|---|---|
| I | 1.65 ± 0.23 | 1.69 ± 0.22 | 0.620 (NS) |
| II | 2.72 ± 0.22 | 2.78 ± 0.23 | 0.220 (NS) |
| III | 3.82 ± 0.15 | 3.66 ± 0.17 | 0.005 (S) |
| IV | 4.85 ± 0.18 | 4.88 ± 0.16 | 0.550 (NS) |
| V | 5.86 ± 0.29 | 5.61 ± 0.25 | 0.004 (S) |
| I-III | 2.23 ± 0.16 | 1.99 ± 0.21 | < 0.001 (HS) |
| I-V | 4.38 ± 0.25 | 3.91 ± 0.38 | < 0.001 (HS) |
| III-V | 2.16 ± 0.29 | 1.91 ± 0.28 | 0.004 (S) |