Original Research Article

# Effects of age on visual evoked potentials

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## Abstract

**Background:** Pattern-reversal visual evoked potential (PRVEP) is an objective, sensitive and non-invasive neurophysiological test that can prove to be a useful clinical tool in investigating the physiology and pathophysiology of human visual system. But results of PRVEP are affected by different physiological parameters like age, gender, head size, BMI. The present study was undertaken to find out the effect of age on pattern –reversal visual evoked potential (PRVEP). **Materials and Methods:** PRVEP was recorded in 60 healthy volunteers in the age-group of 18-75 years. They were divided into 2 age groups below and above 40 yrs. Mean P100 latencies and N75-P100 amplitudes were compared in 2 groups **Result:** The present study demonstrated that mean P100 latency is significantly prolonged in older subjects ($p<0.001$) while mean N75-P100 amplitude is significantly reduced. **Conclusion:** Prolonged PRVEP P100 latency with age reflects electrophysiological alterations in visual pathways. PRVEPs are useful objective measures to investigate the involvement of neural elements of visual system in the elderly individuals.

**Key Words:** Ageing, N 75-P100 amplitude, P100 latency, Visual evoked potentials.

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## INTRODUCTION

Visual evoked potentials (VEPs) provide important diagnostic information regarding the functional integrity of the visual system. VEPs record visually evoked electrophysiological signals extracted from the electroencephalographic activity in the visual cortex. Responses evoked by patterned stimuli constitute pattern visual evoked potentials and pattern reversal is the preferred stimulus for most clinical purposes because of its relative simplicity and reliability with less intra-individual and inter-individual variability. However, certain physiological and physical factors like age, gender, body mass index (BMI) and head size can influence the PRVEP (pattern reversal visual evoked potential) waveforms. Hence, it is necessary to evaluate the role of these physiological factors on the visual evoked responses of normal subjects.[1,2] Each sensory system has its own time of maturation and aging. One of the first obvious signs of aging is the failure of an individual to read the fine prints, such as smaller font on the label of a medicine bottle. This visual decline cannot be wholly explained by senile miosis and media opacities encountered during aging. It could probably be due to the changes in neuronal pathway concerned with vision.[3] Visual evoked potentials (VEPs) can be a productive research methodology for studying such age-related visual declines owing to its objective and sensitive nature. They provide a measure of normal functioning of the visual system and also for assessing the changes during different stages of life. Visual evoked potentials can serve as a window into the central nature of neural processing and the pattern of age-related signal transmission delays in the visual system can be measured**.** Patterned visual evoked potential testing detects minor visual pathway abnormality with much greater sensitivity and accuracy than unpatterned stimuli. Of the various VEP components described in normal subjects- the N75, P100, and N145, P100 is the most consistent and least variable peak and the most clinically useful measurements on the responses to monocular full-field stimulation are

1. P100 latency and
2. N75-P100 Amplitude

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Additional latency, duration, and amplitude are highly variable measures and generally add little to clinical interpretation.[4] The maturation and senescence of different sensory system reflects different patterns. In a study by Allison T *et al* (1983), VEP P100 latency did not change between 20 and 59 years[5]. Glimore R (1995) who studied the process of senescence in sensory system found that the latencies of visual evoked potentials prolong by 2-4 ms/decade after age 40 years[6]. The present study hence, is an attempt to contribute to the researches and share our investigations and findings by performing an objective evaluation of the visual functions in the subjects with the older age-group by way of pattern reversal visual evoked potentials (PRVEPs).

## MATERIALS AND METHODS

The study was conducted on 60 healthy adults in the age-group of 18-70 years with normal visual acuity. They were divided into 2 age groups of less than and more than 40 years. Approval from the Institutional Ethical committee was taken to carry out the research work. A complete neuro-ophthalmologic examination of each subject was done after obtaining a written informed consent and a detailed clinical history.

**Inclusion Criteria:** Adult subjects with visual acuity 6/6(With or without corrective glases), normal fundus and visual field examinations.

**Exclusion Criteria:** Subjects with metabolic, endocrine or demyelinating pathologies; glaucoma, strabismus, amblyopia, optic neuropathies, inherited or acquired neurological disorders, compressive lesions of anterior visual pathways, HIV infections, history of drug-abuse and history of cerebro-vascular accidents. For the best results of VEP testing, subjects were advised to come without applying oil or any hair chemical to the scalp, asked to put on their usual glasses. Subjects were instructed to have an adequate sleep the previous night to prevent the effect of drowsiness on the responses. Subjects were explained about the test to ensure full cooperation. Subjects were also instructed to avoid any mydriatic or miotic drug 12 hours before the test. Preparation of scalp skin was done before electrode application.

**VEP recording:** VEP was performed on RMS EMG.EP machine in a specially equipped sports physiology lab. Subjects were seated comfortably about 100 cm away from a video-monitor with a 30 cm screen. The video-monitor presented a black and white checker-board pattern with a fixation spot in the centre of the screen. The checks/pattern elements reversed alternately at the rate of 1.71 Hz. Standard disc surface electrodes were placed according to the International 10/20 system of electrode placement, with active electrode at Oz, reference electrode at Fz and ground electrode at Fpz.

Volunteers were instructed to fix the gaze on a small red square at the centre of the screen of video-monitor. Monocular stimulation was done with an eye-patch covering the other eye. With the preset stimulus and recording conditions as mentioned above and keeping the electrode impedance <5 kΩ, the recording procedure was started. Parameters for the study were P100 latency, N75-P100 amplitude. All the data was expressed as mean ± S.D. The significance of difference between groups was calculated by using unpaired t-test.

## OBSERVATIONS AND RESULTS

This study comprised of 60 healthy adults in the age-group of 18-70 years. The subjects were distributed into 2 groups according to their age: <40 years and >40 years. Mean P100 latency was significantly prolonged in older age group. And N75-P100 amplitudes were found to be significantly reduced in older subjects.

**Table 1:** Mean P 100 latencies in all subjects

| Sr no | Age groups (Years) | Number of subjects (n) | P 100 Latency (ms) | |
|---|---|---|---|---|
| | | | Right eye (Mean ± S.D.) | Left eye (Mean ± S.D.) |
| 1 | < 40 | 32 | 100.75 ± 4.36 | 100.44 ± 4.38 |
| 2 | > 40 | 28 | 104.62 ± 2.76 | 104.12 ± 2.68 |

**Table 2:** Mean N 75 - P 100 Amplitudes in all subjects

| Sr no | Age groups (Years) | Number of subjects (n) | N 75 - P 100 Amplitude (μv) | |
|---|---|---|---|---|
| | | | Right eye (Mean ± S.D.) | Left eye (Mean ± S.D.) |
| 1 | < 40 | 32 | 6.25 ± 3.05 | 6.24 ± 3.12 |
| 2 | > 40 | 28 | 4.52 ± 1.62 | 6.24 ± 1.28 |

**Table 3:** Comparison of VEP Parameters between two groups

| | P 100 Latency (ms) (Mean ± S.D.) | N75 - P100 Amplitude (μv) (Mean ± S.D.) |
|---|---|---|
| <40 Yrs | 100.6 ± 4.37 | 6.24 ± 3.15 |
| >40 Yrs | 104.37 ± 2.73 | 4.66 ± 1.47 |
| P value | 0.0002 | 0.0182 |
| Significance | ++ | + |

## DISCUSSION

Physiologic ageing, a universal and natural phenomenon of gradual deterioration of physiologic functions with age has been of particular interest to the researchers studying the mechanism of ageing and age-related diseases. The effects of ageing are widespread in the body with brain as no exception. Slowing in visual processing speed is a common characteristic of ageing and has been a well-established phenomenon.[7] These age-related declines cannot solely be explained on the basis of the changes in various optical characteristics in the older subjects, but neural elements of the visual system and visual pathway affection can be important factors in the aged. Visual evoked potentials are objective measures investigating the

functional integrity of the visual system and can provide important information regarding the physiologic and pathologic changes in the visual system. The study hence, included healthy subjects in a wider age-group including the elderly subjects in an attempt to find the electrophysiologic pattern of variations with ageing by pattern reversal visual evoked potentials. The aging differences demonstrated in the present study could be due to anthropometric, environmental, dietary, and genetic differences. The aging changes in the P100 latencies and amplitude may also be explained by age-related visual declines. Changes in ocular media lead to reductions in illuminance of the visual stimulus and neurons showing senile changes with age, but an important determinant of retinal aging are the cumulative exposure to high energy photons from solar radiation which may accelerate the process of aging[8]. A long-term cumulative exposure to high energy photons from solar radiation cause apoptotic damage or death of photoreceptors and neurons in retinal diseases due to the hyper‑excitation toxicity of the visual cells.[9-12] Besides this environmental light stress, dietary stress in the forms of deficiency, especially of Vitamin A appears to activate photo-transduction at a higher rate and in a Continuous manner which may result in prolonged lower concentration of the calcium ions causing death of rods and neurons. A possible role of difference in individual genetic constitution may also be considered. Studies have reported the possible role of certain genes such as neuronal *Rac‑1*[13] and *rdy*[14] to increase the photo‑oxidative stress and damage, whereas arrestins[15] and 1,3‑dimethyl thiourea (DMTU)[16] reduces the photo‑oxidative stress, in experimental animals. Therefore, a high radiation exposure, a still rampant Vitamin A and/or protein deficiency or it could be genetic constitution of our Indian population that may contribute to the aging differences in the visual system. The present study shows that there are certain age related changes in the latencies of all the three waveforms. We can see changes in N75, P100 and N145 with variations in age, out of this P100 latency is more useful. In present study we can see that P100 latency is prolonged in older subjects. Larsen JS. explained this increase in latencies by the gradual lengthening of the visual pathway with the growth and increase in the head circumference[17]. After 60 years, it shows gradual prolongation may be explained by degenerative changes of aging. These findings are in line with those reported by previous workers.[1,5] Allison T *et al*[5] assumed that latency changes are a valid measure of the speed of axonal and synaptic conduction and the rise time of post synaptic potentials in sensory pathways and cortex. A decrease in latency with age reflects increasing conduction velocity or maturation of the nervous system. An increase in the latency with age reflects a decrease in conduction velocity or degenerative processes associated with aging. Plonsey, 1969[18] suggested Impedance of the body is mainly resistive and changes with age in the conductive media surrounding the nervous system likely do not produce artifactual changes in latency. Balazsi AG *et al*, 1984[19]; Wisniewski and Terry, 1976[20]reported aging changes in the human brain particularly in the calcarine fissure and optic nerve and visual pathways like axonal dystrophy. Demyelination and defective myelin regeneration in the aging brain which may thereby reduce the conduction velocity in the visual pathways., Vrabec F, 1965[21] reported degeneration of the retinal ganglion cells with increased deposit of lipofuscin and agyrophilic granules in the cell body, loss of dendrites and tortuosity of dendrites. McGeer, DI. and McGeer,P.1976[22]; Samorajaski T, 1977[23], suggested a deranged metabolism and function of neurotransmitter in the aging brain leading to an increased synaptic delay. Ordy JM and Brizzee KR, 1979[24], and Devaney KO and Johnson, H.A,1980[25]. reported an age-related neuronal loss in the lateral geniculate and striate cortex., Samuel *et al*, 1983[26] showed that vascular and biochemical changes occurring in the elderly brain which may adversely affect various processing in the CNS.

**Amplitude**: The present study found an inverse relationship between age and the N75-P100 amplitude. The mean N75-P100 amplitude observed here in present study is however in close agreement with those reported by O.P.Tandon[27]. These changes in amplitude can be attributed to, At early age when neuronal density is highest in the human visual cortex, at 25-60 it reaches to adult level mental performance and in older ages due to degenerative changes in brain. Nicholas R. Galloway[28] and Robert E.Dustman *et al*[29] in two different study observed the same age related changes in P100 amplitude as in present study.

## CONCLUSION

Aging documents increase in PRVEP P100 latency with significant influence. Effects on P100 latency are stronger in comparison with N75-P100 amplitude changes. Neuronal loss, changes in cell membrane composition and senile plaques present in older subjects has been speculated. Reduction in retinal illuminance due to the decrease in pupillary diameter with age has also been suggested. Few other age-related changes documented in the neural elements of the aging visual system such as age-related loss of rods and cones, reduction in the number of cells in the primary visual cortex to about 25 % at the age of 60 and atrophy of the retinal ganglion cells can also be involved in the electrophysiologic alteration in the visual pathways. We believe that this study has contributed to

the idea that visual electrophysiology might be useful in objectively testing the CNS aging processes that influence visual perception. This finding suggests that a more complex electrophysiological examination at several levels of visual information processing could differentiate normal and pathological aging processes.

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