EFFECT OF OCULAR DOMINANCE ON THE LATENCY AND AMPLITUDE OF VISUAL EVOKED POTENTIALS

By

Dr. JAGADAMBA. A

Dissertation submitted to

Sri Devaraj Urs Academy of Higher Education and Research. Kolar.

in partial fulfillment of the requirements for the degree of

MASTER DEGREE
IN
PHYSIOLOGY

Under the guidance of

Dr. KARTHIYANEE KUTTY, MD
PROFESSOR

DEPARTMENT OF PHYSIOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE
KOLAR

APRIL 2011

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled "**Effect of Ocular Dominance on the Latency and Amplitude of Visual Evoked Potentials"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. Karthiyanee Kutty,** M.D., professor , Dept of Physiology. Sri Devaraj Urs Medical College, Kolar.

Signature of the candidate

Dr. Jagadamba. A

Date :

Place : Kolar

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation **"Effect of Ocular Dominance on the Latency and Amplitude of Visual Evoked Potentials"** is a bonafide and genuine research work carried out by **Dr. Jagadamba. A** in partial fulfillment of the requirement for the degree of **M.D. in PHYSIOLOGY.**

Signature of the Guide

DR.KARTHIYANEE KUTTY, M.D.,
Professor
Department of physiology
Sri devaraj urs medical college,
Tamaka, Kolar

Date :

Place : Kolar

ENDORSEMENT BY THE HOD, PRINCIPAL HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"Effect of Ocular Dominance on the Latency and Amplitude of Visual Evoked Potentials"** is a bonafide research work done by **dr.jagadamba.a** under the guidance of **Dr. Karthiyanee Kutty,** Professor Department of Physiology.

Seal & Signature of the HOD

Seal & Signature of Principal

Dr. Nachal Annamalai

Dr. M.B. Sanikop

Date :

Place : Kolar

COPY RIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka shall have the rights to preserve use and disseminate this dissertation/ thesis in print or electronic format for academic / research purpose.

Signature of the Candidate

Dr. JAGADAMBA. A

Date :

Place : Kolar

SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR

ETHICAL COMMITTEE

CERTIFICATE

This is to certify that the ethical committee of sri devaraj urs medical college, tamaka, kolar has unanimously approved **Dr. Jagadamba. A** Post-Graduate **S**tudent in the Department of Physiology of Sri Devaraj Urs Medical College to take up the dissertation work entitled **"Effect of Ocular Dominance on the Latency and Amplitude of Visual Evoked Potentials"** to be submitted to the Sri Devaraj Urs Academy of Higher Education and Research, Kolar.

Member Secretary **Principal**

Date :

Place : Kolar

A WORD OF GRATITUDE

To My Respected Teacher and Guide

Dr. Karthiyanee Kutty
Professor
Department of Physiology
Sri Devaraj Urs Medical College,
Kolar.

Though words falter to acknowledge all my debt, it is with a deep sense of gratitude that I express my thanks to my reverend, renowned teacher, guide and professor **Dr. Karthiyanee Kutty**, Department of Physiology whose relentless help, proficient ideas, constant supervision, direction, discussion and inspiration has guided me throughout my post-graduate career.

I am thankful for her act of kindness in taking immense interest in my dissertation work. I once again offer my profound sincere thanks to my guide.

Dr. Jagadamba A.

Acknowledgement

I am grateful to **Dr. Nachal Annamalai**, Professor and Head, Dept of Physiology, Sri Devaraj Urs Medical College, Kolar for her valuable guidance, sincere suggestion, keen interest and constant encouragement.

With an immense sense of gratitude, I thank **Dr. Vinutha Shankar M. S**, and **Dr. Sunanda Nayak**, Professors, Department of Physiology, Sri Devaraj Urs Medical College, Tamaka, Kolar, for their unsurpassable guidance and encouragement in making this study possible.

I am also grateful to **Mrs. Usha Shenoy** and **Dr. Ashwath Kumar,** Assistant Professor, Department of Physiology, Sri Devaraj Urs Medical College, Tamaka, Kolar for their constant guidance, support and invaluable suggestions throughout the study.

I am also thankful to my husband **Dr. Ravi Madhusudhana** and my son **Gaurav** for helping me in all aspects of my post-graduate course.

I express my deepest gratitude to my PG'S **Dr. Kavana G. Venkatappa, Dr Shankarappa, Dr. Raja Reddy, Dr. Padmini Thalanjeri, Dr. Soumya P. Kori, Dr. Ashwini K. Shetty, Dr. Sumit Garg, Dr. Ramya C.S, Dr. Bhanu** for their helping hand in the completion of the dissertation and their valuable support during this study. I express my sincere heartfelt gratitude to all the subjects for giving their willingness to undergo the required

procedure and enabling me to carry out this study. I am also thankful to all the **Non-teaching staff** in the department for their co-operation and help.

I also thank Dr. Girish Bengalorkar, Assistant Professor, Department of Pharmacology, SDUMC, Kolar for his help in statistical analysis.

I thank my parents **Mr. Aswathappa** and **Mrs. Yeshodhamma**, my in-laws **Mr. Madhusudan** and **Mrs. Jayalakshmi** ,my brothers **Mr. Nandagopal**, Mr. Chandrashekar K.N., and **Kumar**, my sister-in-laws **Mrs. Kamala, Mrs Rashmi, Sathwik, Rahul and Brother-in-law Dr. Venugopal, Mrs. Anuradha** for showering their blessings which has helped me throughout for their constant support and encouragement.Above all I thank **The Almighty** for all his guidance and blessings.

Dr. Jagadamba A

Date :

Place : Kolar

LIST OF ABBREVIATIONS

VEP	-	Visual Evoked Potential
PREP	-	Pattern Reversal Evoked Potential
PSVEP	-	Pattern Shift Visual Evoked potential
EP	-	Evoked Potentials
EEG	-	Electroencephalogram
CNS	-	Central Nervous System
SEP	-	Somatosensory Evoked Potential
BAEP	-	Brain Stem Auditory Evoked Potential
DLGN	-	Dorsal Lateral Geniculate Nucleus
GABA	-	Gaba Aminobutyric Acid
(’)	-	Minutes Arc
SD	-	Standard Deviation
DE	-	Dominant eye
NDE	-	Non dominant eye

ABSTRACT

Background: Functional lateralization occurs in the paired organs of the body, such as hands, legs and cerebral hemispheres. Ocular dominance, sometimes called eye dominance or eyedness is the tendency to prefer visual input from one eye to the other. Approximately two-thirds of the population is right eye dominant, however neither eye is dominant in a small portion of the population. VEPs are the electrical potentials recorded from the scalp in response to visual stimuli to assess conduction pathway from the retina to the occipital cortex. In order to enhance the significance of visual evoked potentials in eye dominance it is extremely important to determine normal range of parameters of visual evoked potentials wave forms, especially wave latencies as accurately as possible, which provides electrophysiological evidence of lateralization in the nervous system.

Objectives: To determine ocular dominance. To compare the latency and amplitude of Visual evoked potentials in dominant and non dominant eye.

Materials & methods: Informed consent was taken from each subject. The study was approved by ethical committee of Sri Devaraj Urs Medical College. The study group consisted of 100 normal healthy subjects. Ocular dominance was determined by miles & Porta test & then VEPs was recorded as per the standard protocol described by international federation of clinical Neurophysiology (IFCN) committee by using RMS EMG PK- II machine. Latency & amplitude of individual VEP wave parameters were measured.

Results: Data was analyzed by using descriptive statistics and significance of test done by student t-test.

Conclusion: 75% of the subjects were right eye dominant and 25% were left eye dominant. The latency of N70 & P100 has been found to be lesser in the dominant eye and the amplitude is increased in the dominant eye as compared to non-dominant eye, which provides electrophysiological evidence of lateralization in the central nervous system.

Key words: Visual evoked potentials, ocular dominance.

TABLE OF CONTENTS

Sl. No.	PARTICULARS	Page no.
1	**Introduction**	1
2	**Aims and Objectives**	5
3	**Review of Literature**	6
4	**Materials and Methods**	34
5	**Results and Analysis**	40
6	**Discussion**	47
7	**Summary**	50
8	**Conclusion**	51
9	**Bibliography**	52
10	**Annexures** - **Master Chart** - **Keys to Master Chart**	

LIST OF TABLES

Sl No	Name	Page No.
1	Comparison of eye dominance by Porta & Miles test.	41
2	Percentile distribution of amplitude of Visual evoked Potentials in dominant and non dominant eye in young adults.	42
3	Percentile distribution of latencies of Visual evoked Potentials in dominant and non dominant eye in young adults.	43
4	Comparison of amplitude and Latency in Dominant and Non-dominant eyes.	45
5	Master chart showing Ocular dominance (RE or LE), and Amplitude and Latencies (Dominant and Non dominant Eye).	60

LIST OF FIGURES

Figure	Contents	Page No.
1	Anatomy of Eye Ball	11
2.	Layers of Retina	12
3.	Visual pathway	15
4.	Ocular dominance column	20
5.	Visual Evoked Potential Waveform	29
6.	Method of Recording VEP	39
7.	**Graph 1**: Percentile distribution of amplitude of Visual evoked Potentials in dominant and non dominant eye in young adults.	42
8.	**Graph 2**:Percentile distribution of latencies of Visual evoked Potentials in dominant and non dominant eye in young adults	44
9.	**Graph 3**:Comparison of amplitude in Dominant and Non-dominant eyes	45
10	**Graph 4:** Comparison of N70 latency in Dominant and Non-dominant eyes.	46
11	**Graph 5:** Comparison of P100 latency in Dominant and Non-dominant eyes.	46
12	**Graph 6:** Comparison of N155 latency in Dominant and Non-dominant eyes.	46

INTRODUCTION

The eye has been and continues to be an object of interest and of inquiry for biologists, physicists, chemists, psychologists and others. It is an exemplar of the ingenuity of living systems in adapting to the diverse and changing environment from which mammals has been basic to their success. This has reached a peak in man who is a visual animal, with the ability to guide his own destiny and most influential of all species ever evolved on earth[1].

Of all the sensory system, visual system is a critical feature of adaptation to the environment by registering key information. Thus eye makes vision possible by transforming light energy into neural impulse that travel through the sensory pathway to the cerebral cortex. Where they are further processed, serving as the basis for refined and integrated perceptual judgements[2].

Dominance of one member of a bilateral pair of bodily structures means a functional priority, superiority, or preference of that member. Functional lateralization occurs in the paired organs of the body, such as hands, legs, and cerebral hemispheres. Ocular dominance was first described in 1953 by Giovanni Battista Porta. Ocular dominance, sometimes called eye dominance or eyedness was the tendency to prefer visual input from one eye to the other [14,56]. The eye is a sensory organ and has no conscious proprioception and vision in each eye is represented bilaterally and equally in the occipital lobes. People have no consciousness of using right or left eye, as one is conscious of having left or right hand. One does not see the world from left or right eye but from a single so called cyclopean eye, which combines information from both. Dominance wise eyes work as ones hand. They grab the image

with one eye and pass on to other a start to analyze the object by refining like using their fingertips or balancing objects with two hands[4].

Approximately two-thirds of the population is right eye dominant, however neither eye is dominant in a small portion of the population [5, 6]. Dominance does appear to change depending upon direction of gaze due to image size changes on the retinas [7]. Furthermore, the eye preferred for sighting does not indicate handedness. This is not surprising since each eye projects to both cerebral hemispheres whereas each hand is represented mainly in the opposite hemisphere.

In normal binocular vision there is an effect of parallax, and therefore the dominant eye is the one that is primarily relied on for precise positional information. This may be especially important in sports which require aim, such as Archery, darts or shooting sports[8]. Ocular dominance is an important consideration in predicting patient satisfaction with mono vision correction in cataract surgery, refractive surgery and contact lens wear[9]

Evoked responses measure the electrophysiological response of the nervous system to a variety of stimuli. Almost any sensory modality can be tested; in clinical practice, however, only a few used on a routine basis. The ones most often encountered are the visual evoked responses (VEP, both flash and checkerboard types), short-latency somatosensory evoked responses (SSEP) and short-latency brainstem auditory evoked responses (BAER, BAEP). Late-evoked responses are used for studying higher cortical functions, such as P300 in Alzheimer disease. [10].

Clinical use of evoked potentials (EPs) has changed overtime. Progressive advances in imaging technology have limited the frequency

of evoked-response studies in clinical practice. Current use of MRI technology is mostly responsible for this. The basic difference that persist is that the MRI largely remains an imaging, structural, or anatomical test, while the EP explains the functionality of certain pathways of the nervous system. The MRI scan gives more accurate information about structural problems, while the EP gives us information about the physiology of a certain anatomical pathway with much less spatial or localizing information. Under given circumstances they may be complementary. However, most clinical questions are answered better by MRI of the pertinent neurological structures[10].

Types of EPs in every day clinical use include (1) VEP, usually pattern-shift checkerboard visual evoked responses, (2) SSEP, short latency, somatosensory evoked responses, and (3) BAEP, short latency brain stem evoked responses[10].

The only firm basis for recognizing abnormalities in VEP is a thorough understanding of the normal response and clear delineation of the limits of response variability in healthy population[11].

The visual evoked potential (VEP) is defined as the electrical response, evoked by visual stimulation, from neurones in the visual cortex.The characteristics and stable component of VEP waveform which is most commonly used to investigate the processing of visual information is a positive potential, with a peak latency occurring approximately 100 msec (P100) after onset of a stimulus[12].

The maturity of CNS is studied, based on the changes that latency and amplitude of P100 undergo during development. These changes as documented include decrease in latency and increase in amplitude in childhood, Which is mainly attribute to increase in conduction velocity

due to progressive myelination and an increase in efficacy in synapse, whereas with aging there is delay in latency and decrease in amplitude[13].

VEPs have the advantages that they are less subject to sampling bias, the data are quantitative and they can be recorded chronically to yield information about the kinetics of synaptic plasticity[57].

The focus of this study was proposed to determine the effect of ocular dominance on the latency and amplitude components of visual evoked potentials, in order to lay down norms for visual evoked potentials in young ethnic Indian population. Since many pathological conditions can affect ocular dominance, norms in terms of ocular dominance are important to be defined to be used in clinical practice.

AIMS AND OBJECTIVES OF THE STUDY

Aim

The aim of study is to determine ocular dominance and its effect on the latency and amplitude components of Visual evoked potentials in dominant and non dominant eye in normal healthy subjects.

Objectives

1) To determine ocular dominance.
2) To compare the latency and amplitude of Visual evoked potentials in dominant and non dominant eye.

REVIEW OF LITERATURE

Historical Review

1593 - Giovanni Battista Porta coined the term ocular dominance[58]

The development in clinical neurophysiology is closely linked to the discovery of electricity.

1745-1791 – There were rapid developments in the field of electricity. It became possible to store electricity for experiments which allowed the stimulation of nerves and muscles. The important names during this period that contributed to these achievement included Pieter Van Musschenvorock of Leyden, Benjamin Franklin of Philadelphia and Luigi Galvani of Bologna[15].

1752 - Benjamin Franklin conducted his famous kite experiment, which was electrified due to induction. He charged his Leyden jar by using kite during electrical storms. He postulated the two opposing forces of electricity-positive and negative.

1791 – Luigi Galvani discovered that the nerves were good conductor electricity.

1850 – Helmholtz succeeded in measuring the conduction velocity of nerve in frog.

1851 – Dubois Raymond recorded action potential of voluntarily contracting muscle.

1861 – The method of electrodiagnosis based on faradic and galvanic current was introduced Erb[16].

1873 – Lippman described capillary electrometer which permitted early experiments to observe electrical activity of living tissues.

During World War II, science was given impetus – thermeonic valve was developed which was a trustworthy means for amplifying very small voltages. This marked the beginning of electronics. Thus development of electrophysiology method runs parallel to history of electrical technology.

1875 – The distinction of making the first observation of electrical activity of brain goes to Richard Canton, who reported that he had detected currents from electrodes placed on the skull of exposed brain in rabbits and monkeys. Volta made the first electric battery.

1890 – Waller was the first to demonstrate electrocardiogram[17].

Beginning of 19th century

1903 – Einthoven invented string Galvanometer from which he first recorded electric currents generated by human heart.

1929 – Hans Berger – recorded the first human electroencephalogram from electrodes on the scalp, his original publication were received with scepticism.

1934 – Adrian and Mathew obtained confirmation of his findings and by demonstration to the physiological society ensured their recognition, thus establishing authensity of Berger's findings.

1949 – Lambda waves were reported as saw toothed waves that arise at occiput which are best seen when subject is actively engaged in looking at something that arouses interest. They are random electropositive waves of up to 250 msec. Lambda waves were described by Evans.

1951 – Mundy and Castle detected theta activity in 64% of normal young adult group. ß activity was found in most central regions[18].

1958 – Lennox stimulated the optic nerve and measured latencies of single unit potentials with micropipette recorder from cats.

1960 – Cobb and Dawson showed that pattern response has maximum response and earlier components are best recorded about 6 cms above the inion.

1961 and 1967 – Ciganek and Gastarut introduced the most commonly used nomenclature of VEP waveform the former used Roman numerals the latter used Arabic numerals.

1962 – Hubel and Wiesel demonstrated that the stimulus configuration most effective in eliciting electrical responses from neurons of mammalian visual cortex, were rectangles of relative brightness and darkness and contrast borders between such areas[19].

1967 – Cobb et al found that shifting black and white squares of a chessboard pattern in the visual field of human subjects, produced large and highly reproducible EP's[14].

1970 - Evoked potentials began to have definite clinical utility. This long gestation period resulted in large part from the fact that attention was first focused primarily on long latency components more than 75 msec after the stimulus.

1972 – Halliday et al. first applied the pattern reversal evoked potential clinically to patients with optic neuritis and multiple sclerosis, with striking finding of the latter, was that one half of multiple sclerosis patients had prolonged latency of major positive P100.

1975 – Weale attributed decline in visual acuity with age due to increase latency of P100 to cellular low within the brain.

1976 – Maturational changes in PREPs are described by Sokol and Dobson.

1975, 1977 and 1979 – Studies by Asselman et al., Hennerici and Allison suggested no change in P100 latency until beyond the fifth decade and then an increase of 2 to 5 msec per decade.

1977 – Celesia and Daly were the first to show that segmental demyelination or faulty remyelination of visual pathways would slow visual impulses and attributed this as cause of delays PSVEP.

1978 – Sokol et al. showed that check sizes affected the rate of changes of P100 latency with age and found the rate of increase of P100 was nearly twice as fast for 12' checks as compared with 48'checks.

1979 – Halliday et al. put forth on alternative nomenclature based on latency of each waveform.

1987 – Celesia et al. studied the effect of check size on age changes and found sex differences for the latency and amplitude of P100 of PREP using large checks but not small checks.

1988 – Eggermont in a review of data on the maturation of VEP, SEP and BAEP has proposed that the changes in all three modalities can be described by three exponential functions with time constants of about 4 weeks, 40 weeks and 4 years.

Thus evoked potentials have emerged as an important electro diagnostic technique. There are still continued studies of VEP in relation to number of variables, which determine the maturational processes of visual pathway up to cortex.

Neurophysiology of Eye

Mammalian sensory system is a complex arrangement of interactive pathways that is best appreciated from an evolutionary perspective. They provide organism with information about its environment thus guiding its behaviour accordingly. In addition to qualitative considerations they also have capability to quantify stimulus parameters. Consequently, in depth understanding of the visual system establishes the framework common to other sensory system[21].

The eye is an optical device that organizes light energy such that an image is produced upon the photoreceptive surface. Light which effectively stimulates visual receptors is a form of electromagnetic radiation of which the visible spectrum forms a very narrow band (390 to 760 millimicrons). Objects are perceived when they emit from a source or reflect light that differ in wavelength from immediate surrounding[16].

Anatomy of Eyeball

The principal structures of the eye are shown in figure1. The outer protective layer of the eyeball, the sclera is modified anteriorly to form the transparent cornea, through which light rays enter the eye. Inside the sclera is the choroids, a layer that contains many blood vessels which nourish the structures in the eyeball lining the posterior 2/3rd of the choroids is the retina which is the neural tissue containing the receptor cells[23].

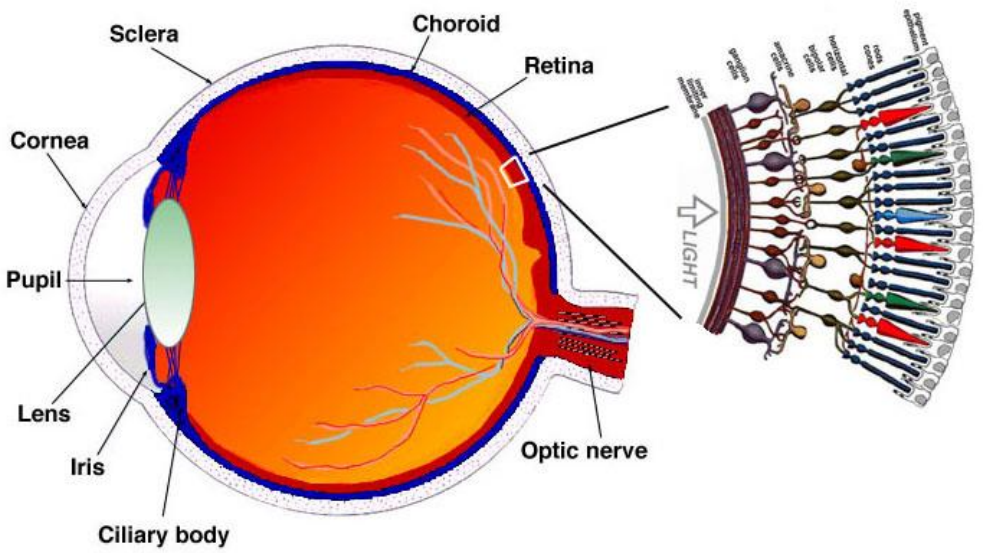


Fig. 1.1. A drawing of a section through the human eye with a schematic enlargement of the retina.

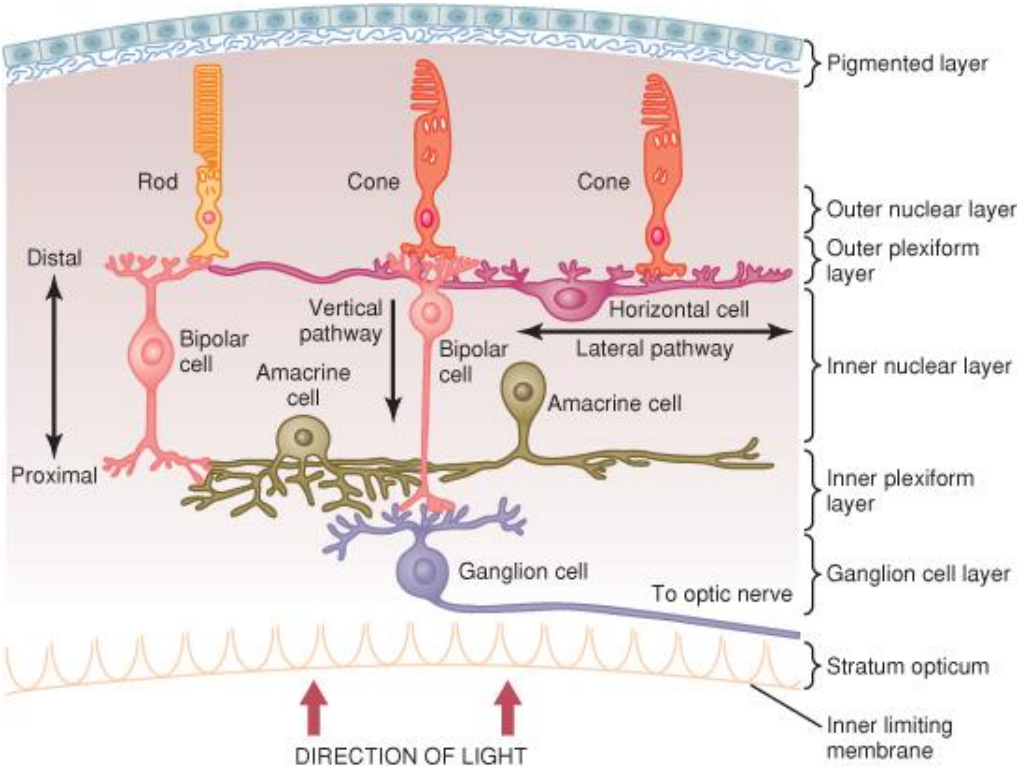
Fig. 1 - Anatomy of Eye Ball

The crystalline lens is a transparent structure held in place by a circular lens ligament (Zonule). The zonule is attached to the thickened anterior part of the choroids, the ciliary body. The ciliary body contains circular muscle fibers and longitudinal fibers that attach near the corneoscleral junction. In front of the lens is the pigmented and opaque iris, the colored portion of the eye. The iris contains circular muscle fibers that constrict and radial fibers that dilate the pupil. Variation in the diameter of the pupil can produce up to fivefold change in the amount of light reaching the retina.

The space between the lens and the retina is filled primarily with a clear gelatinous material called vitreous humor and the aqueous humor fills the anterior chamber of the eye. The visual fields as they appear to the subject are mapped on to the retina in reversal format because of the lens properties of the eye. Right half of the visual field is projected onto the left of the retina and vice versa.

The retina extends anteriorly almost to the ciliary body. It is organized in ten layers and contains rods and cones which are the visual receptors, plus four types of neurons: Bipolar cells, ganglion cells, horizontal cells and amacrine cells. The neural elements of the retina are bound together by glial cells called Muller cells. The processes of these cells form an internal limiting membrane on inner surface and external limiting membrane in the receptor layer.

Figure 2 - Layers of Retina



The receptor layers of the retina rests on the pigment epithelium next to choroids, light rays must pass through the ganglion cell and bipolar cell layers to reach the rods and cones. Horizontal cells connect receptor cells to other receptor cells in the outer plexiform layer. Amacrine cells connect ganglion cells to one another in inner plexiform layer[23].

There are three types of ganglion cells designated W, X, Y cells.

a) **W cells:** they constitute 40% of all ganglion cells having diameter < 10 μm, nerve signals being transmitted signals in the optic nerve fibers at a velocity of 8 m/s. They receive excitation signals from the rods and represent broad fields in peripheral retina because their dendrites spread widely in inner plexiform layer. They detect directional movements and crude rod vision under dark conditions.

b) **Y cells:** They constitute 5% of cells having a diameter 35 μm, nerve signals being transmitted at velocity of 50 m/s. They respond to rapid changes in the visual image like movement and light intensity, thus apprising the CNS almost instantaneously of an abnormal visual event anywhere in visual field but it does not specify the accuracy of location of the event.

c) **X cells:** They are numerous ganglion cells constituting 55%, having a diameter of 10-15 μm and conduction velocity of 14m/s. They represent small fields as their dendrites do not spread widely in the retina; signals represent discrete retinal locations and also receive input from cones transmitting color vision.

There is overall convergence of receptors on bipolar cells and of bipolar cells on ganglion cells and the axons of the ganglion cells converge and leave the eye as optic nerve[24.22].

Visual pathway

The optic nerve leaves the eye and the retinal blood vessels enter it at a point 3 mm medial to and slightly above the posterior pole of the globe. This region is visible through the ophthalmoscope as the optic disc and as there are no visual receptors overlying the disk, this spot is called blind spot.

At the posterior pole of the globe is a yellowish pigmented spot, the macula lutea. This marks the location of fovea centralis, a thinned out rod free portion of retina where the cones are densely packed and there are very few cells and no blood vessels overlying receptors. The fovea is highly developed in humans with greatest visual acuity, as it contains no rods and each foveal cone has a single midget bipolar cell connecting it to a single ganglion cell, so that each foveal cone is connected to a single fibre in the optic nerve[23].

The pathway from the retina to visual cortex contains 1st, 2nd and 3rd order sensory neurons. The somas of the 1st and 2nd-order neurons occupy the retina. The somas of 3rd -order neurons occupy the LGN[20].

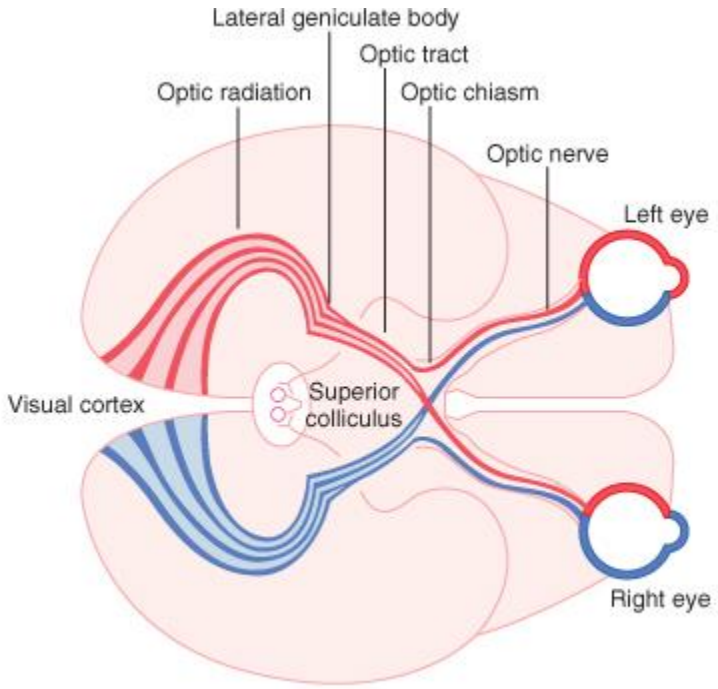


Fig. 3 - Visual Pathway

1st order neurons – The bipolar neurons in the retina are homologous with the bipolar neurons of other special nerves. In the eye, they link the photoreceptor cells to the retinal ganglion cells.

2nd order neurons – The ganglion cells of the retina whose axons give rise to optic nerves and optic tracts. The optic nerves are derived from the retina is embryologically an outgrowth from forebrain and thus consists of forebrain white matter. Histologically the optic nerve contains oligodendrocytes, astrocytes and microglia, whereas true peripheral nerves contain Schwann cells and fibroblasts. Optic nerve does not regenerate after injury. Grossly, the optic nerve is invested with pia, arachnoid and duramater like other parts to CNS[25].

Each optic nerve contains about one million axons, which acquire myelin as they penetrate the sclera. Fibers of macula enter the center of the nerve and they are surrounded by fibers from the four retinal quadrants.

At optic chiasma fibers from the nasal hemi-retina enter contralateral optic tract and those from the temporal hemiretina enter the ipsilateral tract. The ratio of crossed to uncrossed fibers is 53:47 there being more ganglion cells in the nasal hemiretina.

The optic tract undergoes a 90° inward twist carrying fibers from the upper retina to its lateral side. The tracts wind around the midbrain and divides into medial and lateral root. The medial root (10%) enters the midbrain via the superior brachium and lateral root (90%) enters the LGN of thalamus[25].

Medial root of optic tract

Medial root fibers enter the superior colliculus and pretectal nucleus from the superior colliculus:

a) The tectoreticular fibers synapse in gaze centers controlling conjugate movements of the eyes.

b) Tectobulbar fibers activate the sternomastoid to rotate the head and

c) Tectospinal fibers descend in anterior funiculus of cord and excite motor neurons to axial muscles for rotating the trunk. Pretectal nucleus contains the internuncial neurons for light reflex[25].

Lateral root of optic tract

The lateral root terminates in LGN of thalamus entering the principal dorsal nucleus of LGN in line with their positions within the optic tract.

The nucleus serves two principle functions:

1. It relays visual information from the optic tract to the visual cortex by way of optic radiation which is very accurate, point to point transmission with high degree of a spatial fidelity; however the signals from the eyes are kept apart in the DLGN.

2. Nucleus composed of six layers of which layers II, III and V receive signals from uncrossed fibers whereas layer I, IV and VI receive signals from crossed retinotectal fibers.

3. It gates the transmission of signals to the visual cortex.

The DLGN is also divided in another way:

1. Layers I and II are called magnocellular layer as they contain large neurons and receive inputs from Y ganglion cells.

2. Layers III to VI are called parvocellular layer as they contain small to medium sized neurons and receive inputs from X ganglion cells[26].

The principal cells in each lamina (third order neurons) project to primary visual cortex via geniculocalarine tract[25].

Geniculocalcarine tract or optic radiation is of major clinical importance because of its frequent entrapment in lesions of posterior half of the cerebral hemisphere. It travels from LGN to primary visual cortex (area 17) located in the walls of the calcarine sulcus. The fibers destined for the lower half of area 17 begin in the lateral part of LGN and sweep forward into temporal lobe (Meyer's loop) before turning back to join those traveling to the upper half.

The anterior part of optic radiation is supplied by the anterior choroidal branch of the internal carotid artery and its posterior part is supplied by posterior cerebral artery. The geniculocalcarine fibers terminate mainly in layer IV of primary visual cortex[25].

Primary visual cortex

It is also called calcarine cortex because it occupies the walls of the calcarine sulcus which is 10mm deep. Primary visual area is co-extensive with Brodmanns area 17 located in the walls and floor of the calcarine sulcus which is 10mm deep, extends 10 mm onto the medial surface above and below sulcus and 10 mm onto the occipital pole of the brain giving a total area of 25 cm^2. It is defined as visual stria (Stria of Gennari) within grey matter[27].

The cortex has VI layers

Layers II and III are narrow, contains numerous small pyramidal cells. The layer IV which is thick is subdivided by light bands into three sub layers. Upper and lower sub layers are packed with small granule cells and middle layer with fewer small cells scattered between large

stellate cells. Visual cortex contains GABAergic interneurons and fibers in layer II and III exhibit periodicity. Small neurons make symmetrical (inhibitory) synapses upon the apical and basilar dendrites of pyramidal cells. The geniculocalcarine fibers from the magnocellular layer of LGN terminate mainly in layer IV which is organized into four sub-divisions the rapidly conducted signals from Y retinal ganglion cells terminate in layer IV cα and X ganglion cells terminate in layer IV a and IV cß, both transmitted vertically outwards to cortex and inwards to deeper layer[24].

It constitutes of columns of neuronal cells of diameter 30 to 50 μm and represents a functional unit. They are known to decipher separate bits of visual information at successive station and transmit signals to outer layer. Interspersed among these columns are color blobs which decipher color.

The layer four is interlaced with stripes of neuronal columns about 0.5 mm wide and the signals from one eye enter columns of every other stripe alternating with signals from second eye. Thus the cortex deciphers whether the corresponding points from the two retinae are in register with each other[28].

Secondary visual area

Corresponds to Brodmann's area 18 (II visual area) is six layered granular cortex and rostrally merges with area 19. Area 18 interrelates with areas 17 and 19 of the same and opposite hemispheres by association and commissural fibers. It roughly represents the central 50^0 of the visual hemifield and is constituted mainly by complex cells. In area 19 (II visual area) the visual hemifield is represented as a mirror image of area 18 mainly few complex and other hypercomplex cells[27].The secondary visual areas mainly function as pathway for:

a) Analysis of three-demensional position, gross form and motion of objects

b) Analysis of visual detail and color.

Like ganglion cells, the LGN and neurons in layer IV of visual cortex respond to stimuli in their receptive fields with 'on centers' and inhibitory surrounds & with 'off centers' and excitatory surrounds. The visual cortex detects orientation of lines or borders due to linear organizations of mutually inhibiting cells that excite second order neurons. These cells are called simple cells found mainly in layer IV of visual cortex and other neurons which respond to lines displaced to moderate distances laterally or vertically in the filed called complex cells. Thus visual cortex is arranged in vertical columns that are concerned with orientation (orientation columns) of 1 mm diameter with sequential change in orientation from one column to another for every change 5-10 degree. Simple and complex cells called feature detectors because they respond to and analyze certain features of stimuli. Another feature of the visual cortex is the presence of ocular dominance columns.

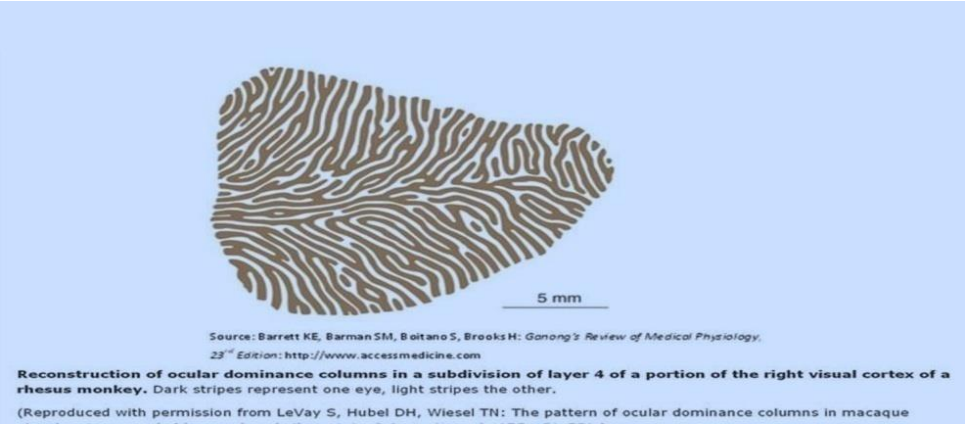


Fig. 4 : Ocular dominance column

The geniculate cells and the cells in layer 4 receive input from only one eye, and the layer 4 cells alternate with cells receiving input from the other eye. If a large amount of a radioactive amino acid is injected into one eye, the amino acid is incorporated into protein and transported by axoplasmic flow to the ganglion cell terminals, across the geniculate synapses, and along the geniculocalcarine fibers to the visual cortex. In layer 4, labeled endings from the injected eye alternate with unlabeled endings from the uninjected eye. The result, when viewed from above, is a vivid pattern of stripes that covers much of the visual cortex and is separate from and independent of the grid of orientation columns[23].

It will be recalled that half the fibers in each optic tract after passing the optic chiasm are derived from one eye and half from the other eye, representing corresponding points on the two retinas. However, the signals from the two eyes are kept apart in the dorsal lateral geniculate nucleus. This nucleus is composed of six nuclear layers. Layers II, III, and V (from ventral to dorsal) receive signals from the lateral half of the ipsilateral retina, whereas layers I, IV, and VI receive signals from the medial half of the retina of the opposite eye.

The respective retinal areas of the two eyes connect with neurons that are superimposed over one another in the paired layers, and similar parallel transmission is preserved all the way to the visualcortex. [28]

Interaction of Visual Signals from the Two Separate Eyes.

Recall that the visual signals from the two separate eyes are relayed through separate neuronal layers in the lateral geniculate nucleus. These signals still remain separated from each other when

they arrive in layer IV of the primary visual cortex. In fact, layer IV is interlaced with stripes of neuronal columns, each stripe about 0.5 millimeter wide; the signals from one eye enter the columns of every other stripe, alternating with signals from the second eye. This cortical area deciphers whether the respective areas of the two visual images from the two separate eyes are "in register" with each other—that is, whether corresponding points from the two retinas fit with each other. In turn, the deciphered information is used to adjust the directional gaze of the separate eyes so that they will fuse with each other (be brought into "register"). The information observed about degree of register of images from the two eyes also allows a person to distinguish the distance of objects by the mechanism of stereopsis[63].

Ocular Dominance

Although eyedness has been the subject of study for centuries, few literatures are available on it. Earliest literature on eyedness is only found after 1593, when Porta for the first time realised existence of a dominant eye. The concept of ocular dominance are based on the use of preferential eye in such tasks as sighting down a telescope, snapping photograph, gun shooting etc.

Some authors have commented that laterality dominance in terms of eyedness is an obvious phenomenon, although its functional significance is not clear. Some studies have indicated that 60% of their sample preferred right eye[58].

Nevertheless, the functional significance of eye dominance remains elusive, & if present, it must differ from hemispheric laterality because brain is not lateralised for the eyes, as it is for the limbs. There is little neurological studies to support the relationship between ocular

and cerebral dominance because there seems no physiological reason to expect that laterality of the limbs should correlate with laterality of eyes, since there is semi decussation of the optic fibres at the optic chiasm. It cannot be said that the right eyed people will demonstrate the dominance of left cerebral hemispheres because the structure of visual system is such that stimulation of any one eye will reach in both cerebral hemispheres.

Types of Ocular Dominance

Sensory dominance may occur when there is a difference in the two retinal images that might lead to rivalry or some binocular interaction. For example, there may be differences in image clarity, brightness or color. Based on these differences, the visual system might find it easier to suppress one eye than the other, or to favor one eye over the other.

Acuity dominance: refers to the measure of visual acuity in which accuracy of the one is more compared to the other. It is the proficiency of one eye where the choice of one eye not allowed. Many investigators are of view that people will sight with that eye with better visual acuity.

Sighting dominance is the most familiar since most of the clinical procedures test for this category of dominance. Sighting dominance is sometimes referred to as Directional dominance.

A directional dominance or sighting test can be done in various ways. For example, a patient will form a hole with his hands and binocularly center an object in that hole. When he alternately occludes either eye, only the dominant eye will still see the same object.

A variant of this test is to have the subject center your (the doctors) right eye in the hole. You will then be looking through the hole at his directionally dominant eye[30].

Due to complexity of the cerebral control of the eyes, there are many types of eye dominance [29, 31]. In this study we have chosen sighting dominance which was described by Porac and Coren. Sighting eye dominance is popularly measured with Portas test &Miles test.

The Portas test that determines which monocular view of near target best matches the binocular view.

Miles test that determines which monocular view of a distant target in an aperture matches the binocular view.

The dominant eye higher degree of myopic refractive error and longer axial length than the non dominant eye especially in patients with high amounts of anisometropia[58].

The repeated suppression of the non sighting eye to avoid physiologic diplopia and the weaker motoric drive may predispose the non-dominant eye to amblyopia ex anopsia[29]

Blepharoptosis is more likely to be present or greater in the non-dominant eye. In unilateral ptosis, the normal eye is almost always dominant. In one study, 54 patients with either unilateral ptosis or asymmetrical ptosis were examined. The right eye was dominant in 33 people and the left in 21people [61]. Ptosis occurred on the side of the dominant eye in 14 individuals and on the non-dominant side in 40 individuals. In this study it suggests that levator muscle tone is influenced by eye dominance when ptosis is present. It may be important to treat first the non-dominant eye in ptosis , which could

look worse than it really is if it affects the dominant eye as a result of retraction of the contralateral levator muscle.

In most children (94%), astigmatic power of the two eyes measured within 1 D of each other. Where the difference was greater than 1.0 D (n _ 30), astigmatism was higher in the nondominant eye in 26 (85%) children. When the interocular difference in astigmatism power was _1.3 D, nondominant eyes were all more astigmatic[69].

In one study fMRI showed that slightly large extent of activation of both occipital lobes when sighting dominant eye was stimulated compared to other eye[31].

Similar conclusions were reached by two VEP studies using monocular checkerboard stimuli[38, 64]

Visual Evoked Potential

The Studies of evoked potentials were the first steps in the neurophysiological exploration of the brain. Evoked potential is an electrical manifestation of the brains reception of and response to an external stimulus. It is an electrical activity appearing in the electroencephalogram but with repetitive stimulation of the eye and averaging by the computer[14]. The EEG activity containing all possible phases of positive and negative wave will slowly approximate to zero. While the activity that is direct result of stimulation will accumulate to produce the evoked response[1].

The VEP tests the function of the visual pathway from the retina to the occipital cortex. It measures the conduction of the visual pathways from the optic nerve, optic chiasm and optic radiations to the occipital cortex. The most important fact to consider is that although

the axons form the nasal half of the retina decussate at the optic chiasm, the temporal axons do not. Therefore, retrochiasmatic lesions may not be detected by full-field checkerboard stimulation. VEPs are most useful in testing optic nerve function and less useful in postchiasmatic disorders. In retrochiasmatic lesions; however, they are not performed routinely in clinical settings. Also note that the macula projects to the occipital pole, while the rest of the retina projects to the mesial calcarine cortex[30].

The VEP is very useful in detecting an anterior visual conduction disturbance. However, it is not specific with regard to etiology. A tumor compressing the optic nerve, an ischemic disturbance, or a demyelinating disease may cause delay in the P100; only additional clinical history and often MRI are needed to uncover the etiology[30].

VEPs are of reliable diagnostic tests that yield reproducible results in routine clinical practice and provide an objective measure of function in the visual sensory systems and tracts. The clinical utility of evoked potentials (EPs) is based on their ability:

1) To demonstrate abnormal sensory system function when the history and/or neurologic examination are equivocal.

2) To reveal the presence of clinically unsuspected malfunction in a sensory system when demyelinating disease is suspected because of symptoms and signs in another area of CNS.

3. To help define the anatomic distribution of a disease process and

4. To monitor changes objectively overtime in a patient's state[20].

VEP's represent mass response of the cortical and sub cortical areas. Normal VEP indicate the intactness of entire visual system and

VEP's can only detect the abnormality but cannot exactly localize the site of lesion in visual pathway[32]. They are non-invasive and have excellent temporal resolution (in range milliseconds) thus permitting study of dynamic changes in nervous system[33].

VEP is only evoked response that is visible without averaging and is highly reproducible as long as patient maintains fixation and normal visual acuity. VEP cannot be seen on routine EEG recording because of low amplitude (0.1-20 μv) and admixture with normal background wave alpha activity > 20 μv. The separation of VEP from EEG is achieved by signal averaging. The practice of repetitive trials is extremely important and is the only means judging waveform consistency[20].

Reversing checkerboard patterns are used as stimuli to the eye, the waveforms derived are called pattern reversal evoked potentials (PREP'S) or pattern shift visual evoked potentials (PSVEP). They provide relevant and reliable information regarding integrity of visual system and are often employed for clinical assessment[20].

PREP's are more reliable (reproducible) overtime and their intersubject variability of major waves is smaller. Since its discovery PREP are subject of intensive research. PREP's has been used to investigate the processing of visual information in man and it has valuable role in detection of pathology of visual system. To increase its usefulness in neurological investigation it has been necessary to define accurately changes in the waveform[34].

Waveform and electrophysiological basis

There are mainly three waveforms: long latency waveforms > 75 msec after stimulation, middle latency 30-75 msec and short latency < 30 msec. The long latency potentials have poor waveform consistency

among subjects as they are easily altered by changes in many psychological variables. The PSVEP uses clinically the first large positive peak that appears 100 msec after stimuli and has high degree of consistency and reliability even though it is long latency waveform.

The differential amplifiers used in neurophysiology record voltage differences between two electrodes input electrode 1 and input electrode 2. Input 1 becomes more positive relative to input 2 and direction of trace movement is dependent on the way the amplifiers respond to a voltage difference between the two electrodes. Thus in Fz and Oz montage, Fz (Frontal zone) is electrode 1 and Oz (occipital zone) is electrode 2. Since there are no conventions in EP's for arrangement of polarity, the most reasonable approach is to copy the method used by most investigators published work. The current recommended conventions of PREP's are positivity of occipital regions (Oz) relative to distant electrodes Fz[20].

VEP waveforms are best identified in normal subjects at Oz (inion) as full field stimulation is greatest at this site. The VEP's to PREP's consist of a set of sequential waveform of opposite polarity. The negative waves denoted by 'N' and positive waves by 'P' which is followed by approximate latency in milliseconds (msec)[32].

There are five principal waves on stimulation of eye, three surface negative waves and two surface positive waves which are labeled according to their polarity and mean latency in normal subjects e.g.P100 where P is polarity and 100 m.sec latency.

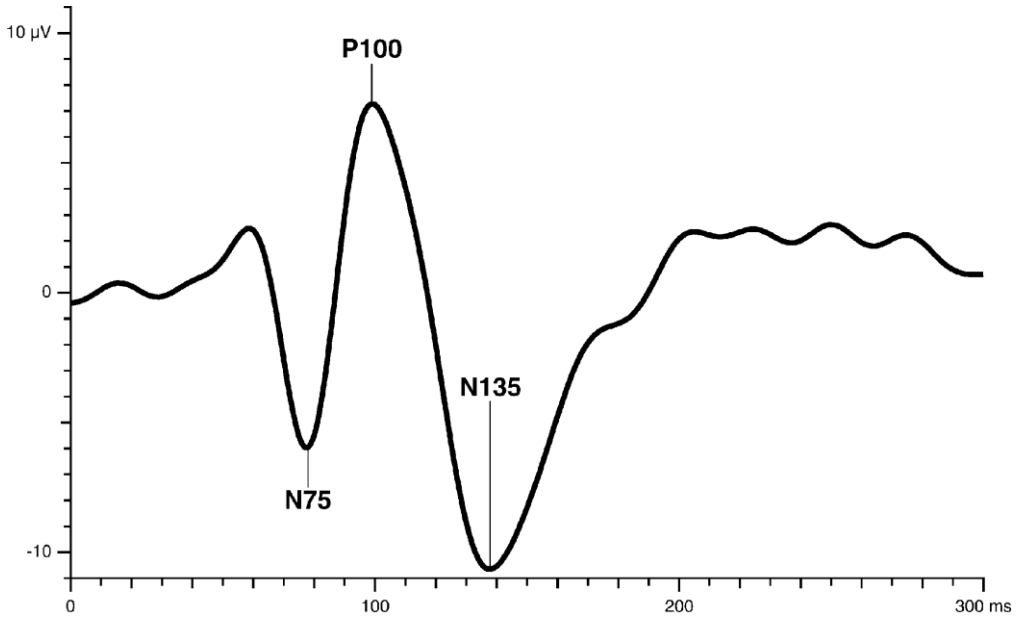


Fig 5: Visual evoked potential waveform

These Waveforms are:

Waveform I =P_1= 55-65 msec (P60)

Waveform II = N_1 75-95 msec (N75)

Waveform III = P_2 =100-120 msec (P100)

Waveform IV =N_2 = 135-160 msec (N135)

Waveform V = P_3 = 200 msec (P200) [1, 34, 35]

Amplitude: 10.1±4.2µvolts.

The earlier waveforms I and II are short latency VEP's elicited by large pattern elements, may reflect the activity of Y cells and latter appearing longer latency VEPs elicited by small checks which reflect X cells.

Sensory evoked potentials in the cortex are sum of potential changes of cortical neurons brought about by synaptic excitation (EPSP's) and inhibition (IPSP's) in the occipital visual areas.

Wave I (P60): small positive deflection reflects activity of afferent geniculocortical on center fibers and primary EPSP's of cortical neurons of striate cortex area 17.

Wave II (N75): Small negative deflection generated within striate cortex area 17.

Wave III and V: large positive deflections which follows short positivity (P100 and P200) lasts nearly 100 msec. It reflects inhibition of large population of cortical neurons cause cellular polarization due to this inhibition seen both in deep neuronal structures (soma) and also superficial structures of extrastriate cortex visual area 18-19.

Wave IV (N145): Reflect less synchronized EPSP's on more superficial structures as apical dendrites and often superimposed on IPSP[1, 34, 35].

Thus the intracortical connections from striate to extrastriate cortex mediate the generation of P100 and N145 which undergo developmental charges reflected in these components.

In the visual field, fovea of retina contributes most of the pattern response P100 component. Retina is divided into central fovea and parafoveal region and peripheral region. Foveal area is said to subtend a visual angle of 4-5^{o} and combined foveal and parafoveal area subtends an angle 8^{o}. This topographical relationship is maintained throughout the visual pathway up to the cortex. The central 5^{o} provided major contribution of fovea to amplitude of P100 and the remaining

contribution is by the peripheral 8-32^{o} of retina. This is because the fovea has large number of cones which are sensitive to patterns.

N75 smaller component predominantly arises from 4-15^{0} annulus of visual field generated by transient movement detector located in peripheral part of retina which has larger and faster conducting axon[36].

Evoked potentials are widely used to quantify the state of maturity of CNS. This is based on the changes that the latency and amplitude of various EP components undergo during development. P60, N75, N145 and P200 are highly inconsistent; though present in most subjects they have little clinical utility and are not analysed[37].

Clinical interpretation of PREP'S based entirely on latency and to less extent the amplitude of major positive peak P100, as they are seen in all normal subjects and has variability small enough to make it reliable in clinical situations. Its latency is not affected by parameters difficult to control in patients such as level of concentration and visual acuity. These waveforms are best recorded with full field stimulation near inion. Majority of P100 is generated by the lower half of the visual field.

P100 waveform is studied with respect to latency and amplitude which are variable with age and check size. Latency is usually stated in milliseconds (1000th of a second) and it refers to the time interval between the stimulus and the waveform peak. It this measures the velocity of nerve conduction and synaptic transmission which are subject to variation with age and check size. Amplitude is usually stated in micro volts (millionth of a volt), however they are less interpretive tools than latencies because of much greater variation in normal subjects[20].

Factors influencing VEP:

1. Age: Age has been reported to influence the latency of P100 at a rate of 2.5ms per decade after fifth decade[19].

2. Gender: The P100 latency is longer in adult males compared to females[19].

3. Eye Dominance: The P100 wave obtained by stimulating the dominant eye is shorter compared to non dominant eye[38].

4. Eye Movement: Eye movement reduces the amplitude of P100 but latency is not affected[20].

5. Visual Acuity: The latency of P100 is reported to be normal with visual acuity as low 20/120[15].

6. Drugs: Drugs producing papillary constriction such as pilocarpine can increase P100 latency[19].

7. Mental activity: During mental activity such as problem solving, the P100 latency has been reported to decrease and amplitude increase[39].

8. Luminance: P100 latency increases as pattern luminance is decreased[40].

9. Contrast: Reductions in the degree of contrast between the black and white squares of the checkerboard pattern cause increased latency[20].

10. Check size: Smaller checks produce larger amplitude in P100[20].

Clinical Applications of VEP

The VEP should be regarded as complementary to clinical examination and neuro-opthalmological investigations. A normal VEP is generally associated with normal visual function. However Brooks & Chiappa[41] showed abnormal VEP may or may not be associated with abnormal clinical findings. Some of the important clinical conditions where VEP is useful are discussed.

1) Multiple sclerosis: A study done by Cant et al.[40] showed the mean latencies in Multiple sclerosis is prolonged by 10-30 ms with definite Multiple sclerosis.Regan[42] showed prolongation of P100 latency is not diagnostic of Multiple sclerosis because it can also occur in a number of other conditions such as spinocerebellar degeneration, compressive lesions, Charcot-Marie Tooth disease, Leber's optic neuropathy glaucoma and amblyopia.

2) Optic neuritis: a study done by Mathews et al[43] showed pattern reversal VEP found to be abnormal in 90% patients.

3) Dysmyelinating diseases: A study conducted by Markand et al[44] the VEP latencies are prolonged in Dysmyelinating diseases such as adrenoleucodystrophy, metachromatic leucodystrophy.

4) Ischemic optic neuropathy: A study by Varga[45] on 43 patients with Ischemic optic neuropathy initial VEP study revealed reduction of P100 amplitude with normal latency of 65% patients & reduced amplitude with prolonged latency in 4.7% patients.

5) HIV infections: Verma and Kearney[46] showed optic nerve and post chiasmal optic pathway may be affected due to HIV per se or due to vasculitis,granuloma.

6) Alcoholics: Kriss et al[47] showed the prolongation of VEP latency in Alcoholics.

7) Friedreich's ataxia: a study by Carroll et al[48] showed the VEP abnormalities in two-third patients with Friedreich's ataxia.

8) Compressive lesions affecting visual pathways: a study done by Gott et al[49] showed the VEP is helpful in detecting the abnormalities even in a patient with pituitary tumors with suprasellar extension.

9) Hysterical blindness: VEP is great value in patients with Hystrical blindness presence of a normal VEP in a patient complaining of visual loss may suggest Hystrical blindness[15].

10) Neurofibromatosis type I: North et al[50] showed VEP was abnormal with normal vision without optic nerve glioma suggesting subclinical abnormality of VEP.

11) Intraoperative monitoring: Moller[51] shown that the VEP has been used to monitor surgery of pituitary & Cavernous sinus tumor & aneurysm surgery.

The amplitude of binocular transient VEP has been found to be larger than monocular for flash stimuli, with a greater difference if the flash is of low intensity[3].

The latency of the transient PVEPs is a robust measure of visual maturation, whereas amplitude is quite variable. Monocular P-VEPs have slightly longer latencies than the binocular PVEPs[71].

Studies have revealed that eye dominance plays a role in determining the hemispheric asymmetry, in VEP recordings.[52]

MATERIALS AND METHODS

MATERIALS

1. Source of data

Study group consisted of 100 normal healthy subjects aged 18-40 years who volunteered. Ethical clearance was obtained from Institutional Ethics Committee.

Inclusion criteria

a. The subjects should be 18-40 years of age.

b. Subjects with normal vision 6/6 with or without glasses.

Exclusion criteria

a. Subjects with visual impairment.

b. Individuals above 40 yrs.

c. Histroy of use of drug intake (mydriatics and miotics).

d. History of chronic medical illness .

2. RMS-EMG MARK II machine was used for recording of VEP with an in built average & amplifier.

Evoked potentials when recorded from electrodes on the surface of the body were very small in the range of -0.5μV to 100μV and these small potentials were recorded from sensitive amplifying and averaging equip ment[20].

Electrode box consists of amplifier sockets for connecting the recording electrodes. They are disc electrodes of 1 cm diameter with silver chloride. They were thoroughly cleaned and disinfected and attached via conducting medium which brought about electrochemical changes in the immediate vicinity of the electrode with ionic migration from the electrode surface into the solution.

The pair of electrodes connected to amplifier is called CHANNEL and an established pattern of connection involving several electrodes and amplifiers is called MONTAGE.

In clinical testing – pattern stimulation is best used as:

a) There is no change in overall luminance.

b) They evoke transient responses which show much greater intersubject similarity in waveform & latency.

When using pattern stimuli the following have to be carefully controlled on the basis of International Federation of Clinical Neurophysiology (IFCN) recommendations[32, 53]:

1) Shape and orientation of the pattern elements-check board is preferred by most as it evokes larger & clearer response than other pattern.

2) Size & sharpness of pattern elements – checks are achromatic patterns whose size were expressed in terms of VISUAL ANGLE subtended by these checks, given by formula.

Visual angle (ß) = Tan^{-1} (W/2D) X120

W=width of checks in mm

D=Distance of the pattern from corneal surface in mm.

Checks are reported in minute of arc (')

3) Size of stimulus field, shape & position in visual space with respect to fixation point. VEP response occurs mostly from central 8^{o}.

4) Stimulus intensity measured as luminance by photometer and expressed as candela per square meter (cd/m^2) should be 100 cd/m^2.

5) Contrast defined as difference between bright & dark portion of pattern should be between 50 to 80%.

6) Rate of presentation of stimulus is of 1 Hz.

7) Mean luminance of center field should be 100 cd/m^2.

8) Background luminance under photopic condition should be atleast 30-50 cd/m^2.

9) Distance between subjects eye and screen is 100 cms.

10) Pupil size affects VEP by altering retinal illumination measured in trolands. Given by formula:

I = L*A

I = Illumination L = mean luminance cd/m^2

A = pupil area in 2mm [54].

METHODOLOGY

During phase of the experiment, subjects were screened as per the criteria laid down under inclusion & exclusion criteria. The informed consent was taken from selected subjects & they were given specific dates to come to neurophysiology lab and hair washed without applying oil.

Data was collected after clinical examination visual acuity of 6/6 with normal visual fields was ensured. On entering lab the subjects were explained about the procedure. Ocular dominance was determined by Miles test and confirmed by Porta test and then the visual evoked potentials were recorded.

Miles Test

In this test the observer extends both arms, brings both hands together to create a small opening, then with both eyes open views a distant object through the opening. The observer then alternates closing the eyes or slowly draws opening back to the head to determine which eye is viewing the object, that is the dominant eye [55].

PORTA TEST

The observer extends one arm, and then with both eyes open aligns the thumb or index finger with a distant object. The observer than alternates closing the eyes or slowly draws the thumb/finger back to the head to determine which eye is viewing the object ,that is the dominant eye [31].

Visual Evoked Potentials were recorded using RMS-EMG MARK-II machine. Visual stimulus was presented to the subject 200 times on computer screen at a distance of 100cm, with checker pattern of size 8x8.

The most important step in recording VEP was fixing of electrode. The electrode was fixed as per 10-20 system which was originally devised for EEG recording. This system specifies the position of scalp electrodes as percentage of distances between definitive landmarks such as nasion, inion and ear tragus.

Using the 10-20 system the reference midfrontal electrode was placed at 30% and recording electrodes at 10% of the whole distance from nasion to occipital protuberance. Thus reference electrode was placed at 10-13 cm above nasion and recording electrodes at 3-5cm above the occipital protuberance (inion).

The electrodes are attached to the surface of scalp with conducting paste after cleaning vigorously keeping skin resistance as low as 5kΩ and these electrodes were connected to electrode box by connecting wires.

One eye was selected, with other eye closed with mask. The subject was asked to see on the fixation point on the center of screen, relaxed, blinking minimally and concentrating on the stimulus pattern. The checks were made to reverse at a rate of 1Hz and 200 responses were recorded. The waveform latency in msec and amplitude in μvolts were measured inbuilt.

Fig 6: Method of recording visual evoked potentials

RESULTS AND ANALYSIS

100 subjects were selected as per the criteria laid down in the materials and methods section for the present study. Ocular dominance was determined and VEP was recorded. The data collected was statistically analyzed and discussed herein after.

Presentation of data

Master chart showing Ocular dominance (RE or LE), and Amplitude and Latencies of Dominant and Non dominant Eye. (Annexure)

Statistical Treatment of the data[71, 72, 73]:

The data was suitably arranged into tables for discussion under different headings.

Descriptive statistical analysis was carried out on this data in the present study. Results on continuous measurements were presented as Mean ± SD (Min-Max) and results on categorical measurements were presented in Number (%). Significance was assessed at 5 % level of significance. Student t test (two tailed, dependent) has been used to find the significance of study parameters on continuous scale within each group. Percentile distribution of latencies and amplitude in dominant and non dominant eye were computed. Conclusions were drawn based on the outcome of this statistical treatment.

Table 1 : Comparison of eye dominance by Porta & Miles test.

Eye dominance	Right Eye	Left eye	Total
Miles Test	75	25	100
Porta Test	75	25	100
Total	150	50	200

Right eye dominant were 75% & left eye dominant 25% in both Portas & Miles test.

In this study we encountered no subject with conflicting results from the Portas & Miles test.

Table 2 : Percentile distribution of amplitude Visual evoked Potentials of μv in dominant and non-dominant eye in young adults

VEP	Min	Max	Mean	SD	Percentile				
					5th	25th	50th	75th	95th
dominant eye	8.06	11.50	9.88	0.79	8.48	9.42	9.76	10.65	11.10
Non dominant eye	7.57	10.89	9.45	0.73	8.19	9.06	9.43	9.72	10.71

Shows amplitude of dominant eye with a minimum value of 8.06 μv & a maximum value of 11.50 μv with a mean value of 9.88 $\pm$ SD of 0.79.

Non dominant eye showed a minimum value of 7.57 μv & a maximum value of 10.89 μv with a mean value of 9.45 $\pm$ SD of 0.73. (Graph 1)

Graph 1 : Amplitute change between Dominant & Non Dominant Eye

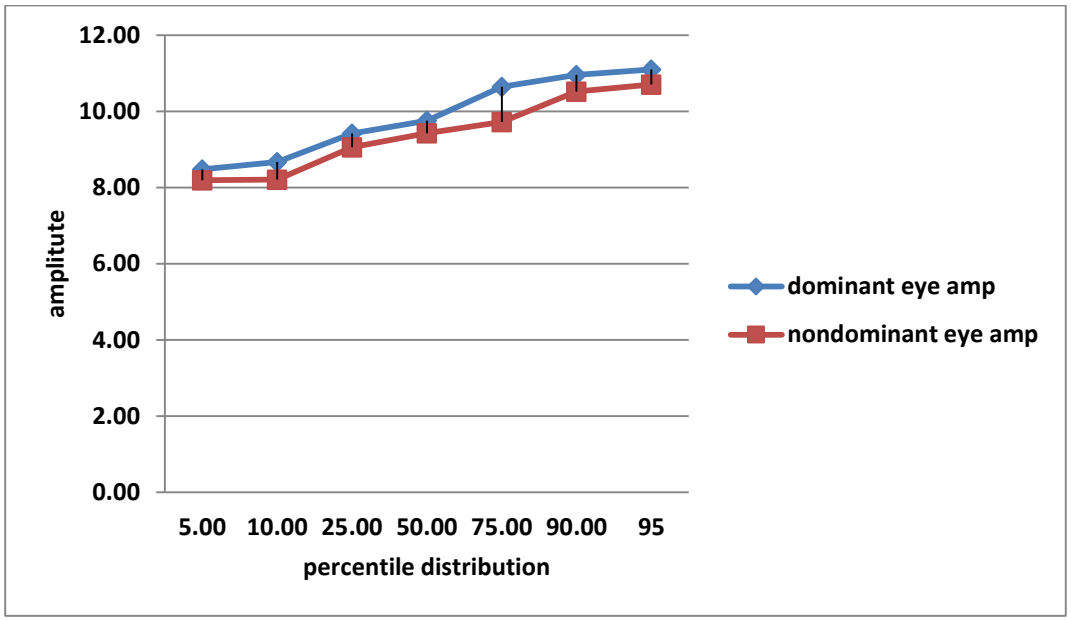


Table 3 : Percentile distribution of latencies of Visual evoked Potentials in dominant and non-dominant eye in young adults

Latencies		Min	Max	Mean	SD	Percentile				
						5th	25th	50th	75th	95th
Dominant eye	**N70**	56.25	80.60	65.27	5.67	60.00	62.50	63.10	66.30	80.60
	P100	80.00	104.38	90.73	7.34	80.00	83.80	91.35	97.88	101.30
	N155	143.75	181.25	161.47	10.20	149.40	151.90	162.50	169.40	180.60
non dominant eye	**N70**	58.80	80.60	65.91	5.40	61.36	63.10	63.80	67.30	80.59
	P100	80.00	119.40	94.29	8.22	80.00	86.30	95.70	101.30	101.90
	N155	123.75	184.38	160.60	12.30	145.66	151.90	158.40	169.40	180.60

Shows dominant eye latency of N70 with a minimum value of 56.25ms & a maximum value of 80.60ms with a mean value of 65.27± SD of 5.67. Dominant eye P100 showed a minimum value of 80.00ms & a maximum value of 104.38ms with a mean value of 90.73± SD of 7.34.Dominant eye N155 showed a minimum value of 143.75ms & a maximum value of 181.25ms with a mean value of 161.47± SD of 10.20.

Non dominant eye latency N70 showed a minimum value of 58.80ms & a maximum value of 80.60ms with a mean value of 65.91± SD of 5.40. Non dominant eye latency P100 showed a minimum value of 80.00ms & a maximum value of 119.40ms with a mean value of 94.29± SD of 8.22. Non dominant eye latency N155 showed a minimum value of 123.75ms & a maximum value of 184.38ms with a mean value of 160.60± SD of 12.30.Also shows the percentile distribution of these measures. (Graph 2).

Graph 2 : Latencies in Dominant and Non-Dominant Eye

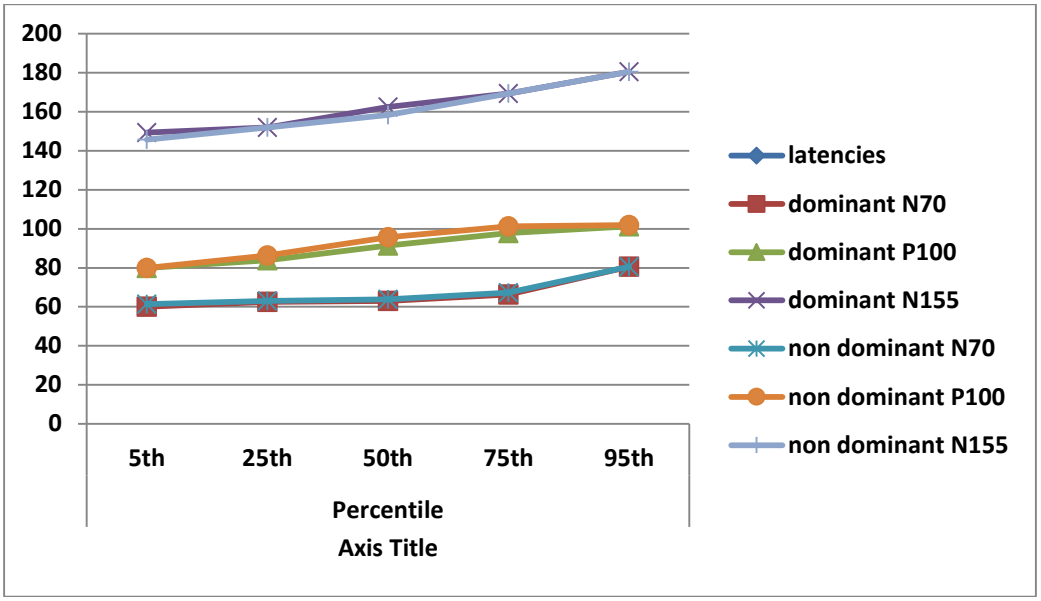


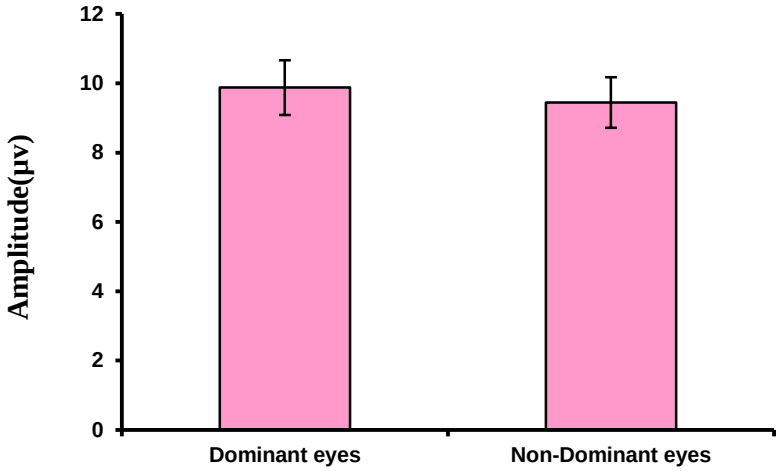
Table 4: Comparison of amplitude and Latency in Dominant and Non-dominant eyes.

	Dominant eyes	Non-Dominant eyes	P value
Amplitude(μv)	9.88±0.79	9.45±0.73	<0.001**
N70	65.27±5.67	65.91±5.40	0.006**
P100	90.73±7.34	94.29±8.22	<0.001**
N155	161.47±10.20	160.6±12.3	0.353

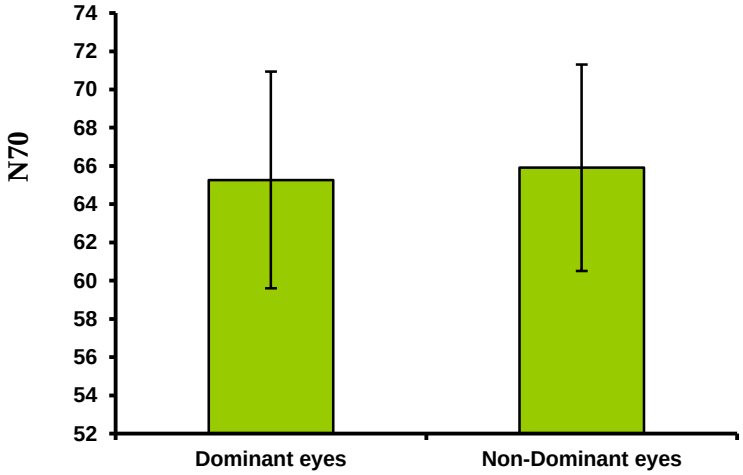
The effect of eye dominance on Amplitude is increased in dominant eye which is statistically significant (P<0.001) compared to the non-dominant eye. (Graph 3)

The effect of eye dominance on latency of N70 and P100 is decreased in dominant eye which is statistically significant (P 0.006 & <0.001) compared to the non-dominant eye. (Graph 4 & 5).

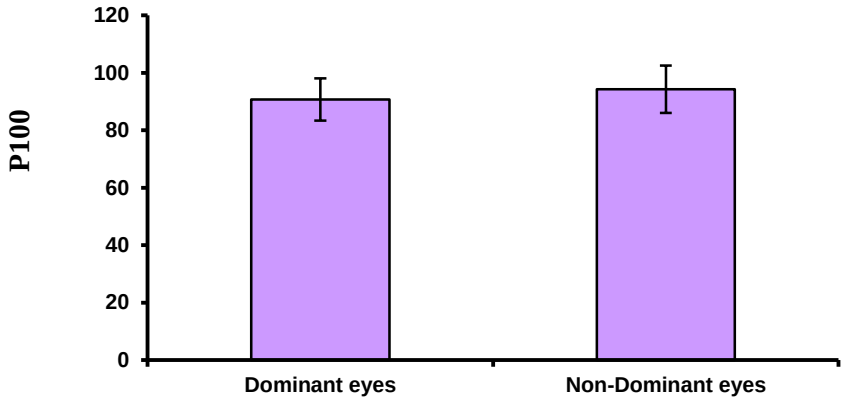
The results of N155 was not statistically significant in the dominant and nondominant eye.(P 0.353). (Graph 6).



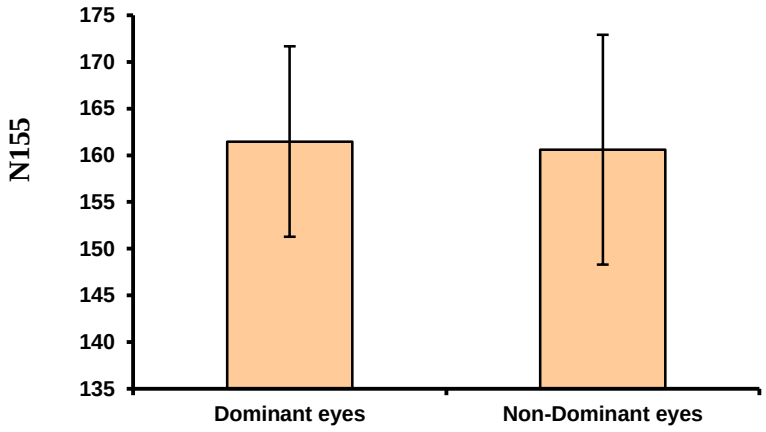
Graph 3: Comparison of amplitude in Dominant and Non-dominant eyes



Graph 4 : Comparison of N70 latency in Dominant & Non-Dominant eyes.



Graph 5: Comparison of P100 latency in Dominant and Non-dominant eyes



Graph 6: Comparison of N155 latency in Dominant and Non-dominant eyes

DISCUSSION

Dominance is mainly influenced by genetics and is defined as physiological priority or preference by one member of any bilateral pair of structures in the body when performing various tasks[55]. The lateralization in eye function is the result of development of binocular vision, with overlapping of the visual fields of the two eyes. The resulting physiologic diplopia is suppressed from the non-sighting eye. This is thought to be the mechanism of development of ocular dominance. The input is favoured from the sighting eye. When a choice is forced between the two eyes, the vast majority of people choose one eye consistently[29].

Approximately 2/3 of the population is right eye dominant. In our study, by sighting dominance we found that 75% were right eye dominant & 25% were left eye dominant, which was consistent with the results of one more study[58]. In our study no subjects were encountered with conflicting results from the Portas & Miles test. Sighting dominance is most important in clinical investigations & most clinical phenomenon are related to this type of dominance in ptosis, cataract, myopia etc. [29, 58, 61].

Visual evoked potentials are the electrical potentials evoked in the brain by visual stimuli and measure the speed of the visual pathways and also the synchronized electrical activity in response to a visual stimulus. In our study, we have measured the activity of monocular stimulation in normal subjects, and have observed the differences between the amount of activity evoked by each eye. These values are compared with sighting eye dominance (DE&NDE). It was revealed in a research that using positron emission test and VEP, integration of visual information begins in the fusiform gyrus as soon as 80 to 130 milli seconds after the visual stimulus in a normal human[59].

In our study the time taken from the stimulus to wave N70 of the visual pathway is shorter in dominant eye compared to the non dominant

eye which is statistically significant. Thus a shorter N70 latency represents faster visual processing in the dominant eye. In a study it was shown that significant visual processing occurs within 150 ms after ball release in sports and all the main features of VEP also occurs within a 150 ms period. A shorter N70 latency therefore represents faster visual processing which is required immediately following ball release in skilled batsmen and would become more critical with high speed deliveries[60].

In our study the time taken from the stimulus to wave P100 latency is also faster in dominant eye, reflecting the activity of visual cortex. In another study it was found that there is a faster P100 latency (~4 ms) in tennis and squash players compared to rowers and sedentary subjects. They related the faster P100 latency, which reflects activity of the visual cortex to a tennis player's ability to rapidly process sensory information[62]. Data of a study showed that, the mean latency of P100 peak was significantly shorter with stimulation of the dominant eye and amplitude were higher in the dominant eye, which provides objective electro-physiological evidence of lateralization in the central nervous system [38].

Another study revealed that the influence of eye dominance scaled by six tests on the parameters (N80, P100latency and N80-P100 amplitude) of the white-black, green-black, red-black and blue-black pattern visual evoked potentials, with normal visual acuity. The P100 latency of the white black PVEPS, for both sexes, significantly shorter in the PVEPs of the dominant eye. The results given were that further electrophysiological evidence for eye dominance as a lateralized CNS phenomenon is not influenced by colour [64].

In our study the Amplitudes were higher in recordings of V. E. P from dominant eye than from the non-dominant eye which is statistically significant. This is because of increased electrical activity in the visual pathway. This is in accordance in a study that showed the visual

processing during the first 100- 150 ms of the ball flight with binocular vision facilitates retinal activation in talented cricketers[65]. On the other hand, one study performed a spectral analysis on steady state V. E. P obtained from dominant and non-dominant eyes found no difference in power values of VEP[66]. Another study reported that eye dominance of dextrals appeared to play a role in determining the hemispheric asymmetry[67].

VEP is very important non-invasive tool in detecting abnormalities of visual system. It is not only useful for clinical neurophysiologist or ophthalmologist but also for neurologist and neurosurgeons. Sighting dominance is most important in clinical investigations .Most clinical phenomenon are related to this type of dominance as in Ptosis, amblyopia, monovision correction in cataract, myopia, hyperopia and sports which require aim such as archery, darts or shooting.

In conclusion, 75% of the subjects were right eye dominant and 25% were left eye dominant. The latency of N70 & P100 has been found to be lesser in the dominant eye and the amplitude is increased in the dominant eye as compared to non-dominant eye, which provides electrophysiological evidence of lateralization in the central nervous system.

SUMMARY

This study was conducted in the department of Physiology, Sri Devaraj Urs Medical College, Kolar, to determine ocular dominance and its effect on the latency and amplitude components of visual evoked potentials in dominant and non dominant eyes in normal healthy subjects. Ocular dominance was determined in selected 100 healthy subjects by Portas and Miles test and visual evoked potentials were recorded. It was observed that 75% of the subjects were right eye dominant and 25% were left eye dominant. The latency of N70 & P100 has been found to be significantly lesser in the dominant eye and the amplitude is significantly increased in the dominant eye as compared to non-dominant eye, which provides electrophysiological evidence of lateralization in the central nervous system.

CONCLUSION

VEPs are used widely in neurophysiology, neurology and ophthalmology and have a wide range of clinical applications. As technology continues to evolve, VEP will likely provide more qualitative and quantitative information regarding the function of the optic nerve & kinetics of synaptic plasticity.

This study of normal ocular dominance is amenable for future studies with VEP using variety of stimuli and such physiological measures are needed to fully understand the functional significance.

In addition, understanding the extent of eye dominance in normal subjects is important for the study and interpretation of monocular clinical eye diseases like ptosis, amblyopia, myopia etc. and also in sports which require aim like archery, darts or shooting.

In conclusion, 75% were right eye dominant & 25% were left eye dominant. The latency of N70 & P100 has been found to be lesser in the dominant eye and the amplitude is increased in the dominant eye as compared to non-dominant eye, which provides electrophysiological evidence of lateralization in the central nervous system.

BIBLIOGRAPHY

1. Gescitelli CA, Eder DDJ, Granda AM. The Visual System in Vertebrates. In: Heidelberg VB, editor. Handbook of Sensory physiology. New York: Springer; 1977. p. 45.

2. Karzman R, Terry R. Normal aging of nervous system. In: Principles of Geriatric Neurology. Philadelphia: Davis company; 1992. p. 18-46.

3. Ciganek L. A comparative study of visual, auditory and somatosensory EEG responses in man. Exp Brain Res 1967;4(2):118-125.

4. Sharp-sighted.org [homepage on Internet]. Brussels: Vandegaer M ;[updated 2009]. Facial masking, Alignment & Dyslexia.0.3 Ocular Dominance; [about 12 screens]. Available from: http://www.sharp-sighted.org

5. Ehrenstein WH, Gahmen BE, Jaschinski W. Eye preference within the context of binocular functions. Graefes Arch Clin Exp Ophthalmol 2005;243(9):926-932.

6. Reiss MR.Ocular dominance: some family data. Laterality 1997;2(1):7-16.

7. Khan AZ, Crawford JD. Ocular dominance reverses as a function of horizontal gaze angle. Vision Res 2001;41(14):1743-1748.

8. Siviero MO, Rysovas EO, Juliano Y, DelPorto JA, Bertolucci PHF. Eye-hand preference dissociation in obsessive-compulsive disorder and dyslexia. Arg Neuropsiquiatr 2002;60(2-A):242-245.

9. Connolly BH. Lateral dominance in children with learning disabilities. Phys Ther 1983;63(2):183-187.

10. Evans AB, Boggs JG. Clinical Utility of Evoked Potentials [monograph on the Internet]. Virginia: Virginia Commonwealth University school of medicine; 2010. Available from: http://emedicine.medscape.com/ article/1137451-overview.

11. Halliday AM. The Visual Evoked potential in healthy subjects. In: Evoked Potentials in Clinical Testing. 2nd ed. Churchil Livingstone; 1993. p .57-113.

12. Shaw NA, Cant BR. Age dependent changes in the Latency of the Pattern Visual Evoked Potential. Electroen Clin Neurophysiol 1980;48:237-241.

13. Eggermont JJ. On the Rate of Maturation of Sensory Evoked Potentials. Electroen Clin Neurophysiol 1988;70:293-305.

14. Chaurasia BD, Mathur BB. Eyedness. Acta Anat 1976; 96(2):301-305.

15. Misra UK, Kalita J. History of Clinical Neurophysiology. In: Clinical Neurophysiology. 2nd ed. Delhi: Elsevier; 2008. p. 1-11.

16. Allison T, Wood CC, Coff WR. Brainstem auditory, Pattern – Reversal Visual and Short-Latency Somatosensory Evoked potentials: latencies in relation to Age, Sex and Brain and Body size. Electroen Clin Neurophysiol 1983;55:619.

17. Cobb WA, Greville GD, Heppenstall ME, Hill DW, Whittridge GWD. Introduction In: Macmillan editor. Electroencephalography. 2nd ed. New York: 1952. p. 3.

18. Kiloh LG, Osselton JW. Normal findings. In: Clinical Electro-encephalography. Long Butter Worths; 1961.

19. Stockard JJ, Hughes JF, Sharbrough FW. Visually Evoked Potential to Electronic Pattern Reversal: latency variations with Gender, Age and technical factors. Am J EEG Technol 1979;19:171-204.

20. Chiappa KA. Evoked Potentials in Clinical Medicine. 3rd ed. New York: Lippincott-Raven; 1997. p. 1-156.

21. Berne RM, Levy MN. The special senses. In: Schmitt WR editor. Physiology. 2nd ed. Washington DC: Mosby; 2004. p. 118-133.

22. Best, Taylor. Vision. In: West JB editors. Physiological Basis of Medical Practice. 12th ed. Baltimore: Williams & Wilkins; 1990. p. 970-996.

23. Ganong WF. Vision. In: Review of medical physiology. 22nd ed. New Delhi: Mc Graw Hill company ; 2005. p. 148-168.

24. Guyton AC, Hall JE. Eye. In: Textbook of Medical Physiology. 11th ed. New Delhi: Elsevier; 2000. p. 613-649.

25. Fitzgerald MJT. Neuroanatomy Basic and applied. 4thed. Bailliere Tindal; 1985. p. 202-209.

26. Park K. Epidemiology of chronic Non – communicable Diseases and conditions. In: Park's Textbook of Preventive & social medicine. 18th ed. Jabalpur : Banarsidas Bhanot; 2005. p. 319-323.

27. Carpenter MB. Core Text of Neuroanatomy. 4th ed. William and Wilkins; 1995.

28. Balazsi AG, Rootman J, Drance SM, Schulzer M , Doulas GR. The effects of Age on the Nerve Fiber Population of Human Optic Nerve. Am J Ophthalmology 1984;97:760-766.

29. Hashim AA. Eye Dominance in Excimer Laser Photorefractive Keratectomy. J Refract Surg 1997;13:461-462.

30. Lecture 11- Ocular Dominance (Steinman Chapter 2, p. 24-27; Chapter 3, p. 56-57)

31. Mendola JD, Conner IP. Eye dominance predicts fMRI signals in retinotopic cortex. Neurosci Lett 2007;414:30-34.

32. Pal GK, Pal P. Visual Evoked Potential. In: Textbook of Practical Physiology. 2nd ed. New Delhi: Ahuja Publishing House; 2007.p. 310-313.

33. Aminoff MJ. Section 19&20. In: Electrodiagnosis in Clinical Neurology, 4th ed. Churchill Livingstone; 1999. p. 421-449.

34. Karzman R , Terry R. Normal aging of nervous system. In: Principles of Geriatric Neurology. Philadelphia :Davis company; 1992. p. 18-46.

35. Cohn NB, Kircher J, Emmerson RY, Dustman RE. Pattern Reversal Evoked Potentials: Age, sex and Hemispheric Asymmetry. Electroen Clin Neurophysiol 1985; 62:399-405.

36. Yiannikasnikas C , Walsh JC. The Variation of the Pattern Shift Visual Evoked response with the Size of the Stimulus Field. Electroen Clin Neurophysiol 1983;55:427-435.

37. Allison T, Ann L. Hume AL, Wood CC, Goff WR. Development and Aging changes in Somatosensory Auditory and Visual Evoked Potentials. Electroen Clin Neurophysiol 1984;58:14-24.

38. Seyal M, Sato S, White BG. Visual evoked potentials and eye dominance . Electroencephalogr Clin Neurophysiol 1981;52:424.

39. Lentz KE, Chiappa KH. Non pathologic (voluntary) alteration of pattern shift visual evoked potentials. Electroencephalogr Clin Neurophysiol 1985;61:30.

40. Cant BR, Hume AL,Shaw NA. Effects of luminance on the pattern visual evoked potentials in multiple sclerosis. Electroencephalogr Clin Neurophysiol 1978;45:496.

41. Brooks EB, Chiappa KH. A comparison of clinical neuro-ophthalmological findings and pattern shift visual evoked potentials in multiple sclerosis. In: Courjon J,Mauguiere F, Revol M ,editors. Clinical Application of Evoked Potentials in neurology. New York: Raven press; 1982. p. 453.

42. Regan D. Human visual evoked potentials. In: Picton TW, editor. Handbook of Electroencephalogrphy and Clinical Neurophysioly. Amsterdam :Elsevier; 1988. p. 159.

43. Matthews WB, Small DG, Small M. Pattern reversal visual evoked potentials in the diagnosis of multiple sclerosis. J Neuro Neurosurg psychiat 1977;40:1009.

44. Markand ON, Garg BP, Demayer WE. Brainstem auditory, Visual and Somatosensory Evoked potentials in leucodystrophies. Electro-encephalogr Clin Neurophysiol 1982;54:39.

45. Varga M. Visual evoked potentials and ultrasonography in Ischemic optic neuropathy. Oftalmologia 2002;53:41.

46. Verma N, Kearney J. Ocular manifestations of AIDS. P.N.G Med J 1996; 39:196-9.

47. Kriss A, Carroll WM, Blumhardt LD. Pattern and flash evoked potential changes in toxic (nutritional) optic neuropathy. In: Courjon J, Mauguiere F, Revol M, editors. Clinical Application of Evoked Potentials in neurology. New York:Raven press; 1982. p. 11.

48. Carroll WM, Kriss A, Baraitser M. The incidence and nature of visual pathway involvement in friedreich's ataxia. Brain 1980;103:413.

49. Gott PS, Weiss MH, Apnzzo M. Checkerboard visual evoked response in evaluation and management of pituitary tumors. Neurosurgery 1979;5:553.

50. North K, Cochineas C, Tang E. Optic glioma in neurofibromatosis type 1; role of visual evoked potentials. Pediatr Neurol 1994;10:117.

51. Moller AR. Evoked potentials. In: Cook SD, editor. Handbook of Multiple Sclerosis. New York :Marcel Dekker; 1990. p. 271.

52. Stoakard JJ, Hughes JF, Sharbrough FW. Visually evoked potentials to electronic pattern reversal: Latency variations with gender, age and technical factors. Am J EEG Technol 1979; 19: 171-204.

53. Kriss A. Recording technique: Evoked potentials in clinical testing. 2nd ed. Los Angeles: Churchill Livingstone; 1993. p. 57-113.

54. Dekaban AS, Salowsky D. Changes in Brain Weights during the span of Human Life: Relation of Brain Weights Body Heights and Body Weights. Ann Neurol 1978;4:345-346.

55. Brackenridge CJ. The contribution of genetic factors to ocular dominance. Behav Genet 1982;12(3):319-325.

56. Khan AZ, Crawford JD. Ocular dominance reverses as a function of horizontal gaze angle. Vision Res 2001;41(14):1743-8.

57. Sawtell NB, Frnkel MY, Philpot BD, Nakazawa K, Tonegawa S, Bear MF. NMDA Receptor – Dependent Ocular Dominance Plasticity in Adults Visual Cortex. Neuron 2003;38:977-985.

58. Cheng CY, Yen MY, Lin HY, Hsia WW, Hsu WM. Association of ocular dominance and anisometropic myopia. Invest Ophthalmol Visual Sci 2004;45(8):2856-2860.

59. Heinze HJ, Mangun G, Burchert W, Bodino G, Gastaud M. Combined spatial and temporal imaging of brain activity during visual selective attention in humans. Nature 2002;372:543-546.

60. Land M, McLeod P. From eye movement to actions: how batsmen hit the ball. Nat Neurosci 2000;3:1340-45.

61. Lyon DB, Gonnering RS, Dortzbach RK, Lemke BN. Unilateral ptosis and eye dominance. Ophthal Plast Reconstr Surg 1993;9(4):237-40.

62. Delpont E, Dolisi C, Suisse G, Bodino G, Gastaud M. Visual evoked potentials: differences related to physical activity. Int J Sports Med 1991;12(3):293-8.

63. Guyton AC, Hall JE. Eye. In: Textbook of Medical Physiology. 11th ed. New Delhi: Elesevier; 2000. p. 640-643.

64. Taghavy A, Kugler CF. A Pattern Reversal Visual Evoked Potentials in Study of eye dominance. Eur Arch Psychiatr Neurol Sci 1987;236:329-332.

65. Thomas NG, Harden LM, Rogers GG. Visual Evoked Potentials, reaction times and eye dominance in cricketers. J Sports Med Phys Fitness.2005;45(3):428-33.

66. Klemm WR, Gibons WD, Allen RG, Richey EO. Hemispheric lateralization and handness correlation of human evoked steady state responses to patterned visual stimuli. Physiol Psycholl 1980;8:409-416.

67. Spinelli D, Mecacci L. Handedness and hemispheric asymmetry of pattern reversal visual-evoked potentials. Brain Cogn 1990;13(2):193-210.

68. Pike J, Polich J, Hemispheric differences for visual evoked potentials from 6 checkerboard stimuli. Neuropsychologia 1988;26(6):947-52.

69. Chia A, Jaurigue A, Gazzard G, Wang Y, Tau D, Stone RA et al. Ocular dominance, Laterality, and Refraction in Singaporean Children. Invest Ophthalmol Vis Sci 2007; 48:3533-36.

70. McCulloch DL, Skarf B. Development of the Human Visual System: Monocular and Binocular Pattern VEP Latency. Invest Ophthalmol Vis Sci 1991;32(8):2372-81.

71. Rosner B. Fundamentals of Biostatistics. 5th ed. Duxbury; 2000.

72. Reddy MV. Statistics for Mental Health Care Research. Bangalore: NIMHANS; 2002.

73. Rao PSS, Richard J. An Introduction to Biostatistics, A manual for students in health sciences. New Delhi: Prentice hall of India. P.86-160.

Table 5 : Master Chart showing Ocular Dominance, wave Latencies and Amplitude

Sl. No.	Age	Sex	Miles Test	Porta Test	Dominant Eye				Non Dominant Eye			
					Amplitude (µv)	Latency(ms)			Amplitude (µv)	Latency(ms)		
						N_{70}	P_{100}	N_{155}		N_{70}	P_{100}	N_{155}
1	18	F	LE	LE	9.09	62.5	97.5	162.5	8.86	62.5	115.63	168.5
2	18	M	LE	LE	9.69	65	80	181.25	9.47	64.38	80	184.3
3	18	F	RE	RE	8.06	60	80	170.6	7.93	64.4	80	170.6
4	18	F	RE	RE	9.27	62.5	83.1	151.3	9.04	62.5	83.8	169.4
5	18	M	RE	RE	10.96	56.25	80.63	168.75	10.52	66.25	100.63	123.7
6	25	F	RE	RE	9.76	69.38	98.13	154.38	9.72	70	98.13	155
7	18	M	RE	RE	9.96	64.4	80.6	170.6	9.46	58.8	80.6	171.9
8	19	M	RE	RE	9.26	63.75	104.3	143.75	8.69	63.13	101.88	145.6
9	28	M	RE	RE	10.96	63.1	90.6	151.9	10.52	63.1	103.8	151.9
10	36	M	RE	RE	11.50	61.3	81.9	162.5	10.89	61.3	93.8	160.5
11	40	M	RE	RE	9.76	63.1	83.8	169.4	9.72	63.1	84.4	169.4
12	39	M	RE	RE	9.94	62.5	93.8	151.3	9.02	62.5	100.6	151.3
13	30	M	RE	RE	9.70	63.1	83.1	151.3	9.10	62.5	85.0	151.4
14	30	M	RE	RE	9.67	70.0	92.1	168.8	9.49	69.5	93.22	168.8
15	39	M	RE	RE	8.17	63.1	101.3	151.3	7.57	62.5	119.4	151.3
16	30	M	RE	RE	9.48	70.0	98.0	162.5	9.06	68.8	100.0	146.3
17	35	M	RE	RE	9.93	63.1	84.4	169.4	9.07	63.1	85.0	169.4
18	20	F	RE	RE	8.48	63.1	84.4	169.4	8.19	63.1	86.3	151.9
19	18	F	RE	RE	10.28	63.1	84.4	151.9	10.15	63.1	85.6	151.9
20	38	M	RE	RE	11.10	63.8	100.0	151.3	10.71	63.8	101.5	151.3
21	18	M	RE	RE	9.95	80.6	101.3	151.9	9.14	80.6	101.9	151.9
22	18	M	RE	RE	9.42	80.6	83.8	169.4	9.25	80.4	101.3	169.4
23	29	M	RE	RE	10.81	62.5	94.4	169.4	9.51	63.1	95.7	169.4
24	28	M	RE	RE	9.61	62.5	93.8	155.0	9.21	65.0	95.0	152.5

25	18	M	RE	RE	8.67	64.0	85.0	180.6	8.21	64.5	87.0	180.6
26	40	M	RE	RE	10.32	62.5	100.6	169.4	9.41	65.5	101.3	169.4
27	18	F	LE	LE	10.65	66.3	90.6	149.4	10.28	67.3	91.2	149.4
28	32	M	RE	RE	9.69	68.1	98.8	156.3	9.47	69.2	100.9	156.3
29	28	M	RE	RE	9.27	63.1	84.4	169.4	9.04	63.2	85.7	169.4
30	33	M	RE	RE	9.84	62.5	93.1	168.8	9.43	62.5	100.6	168.8
31	30	M	RE	RE	9.61	63.1	95.0	151.9	9.59	63.1	97.3	151.9
32	29	M	RE	RE	9.75	62.5	92.5	168.8	9.33	62.5	93.4	168.8
33	30	M	RE	RE	11.00	62.5	96.9	152.5	10.64	62.5	101.9	152.5
34	18	F	LE	LE	9.69	65	80	181.25	9.47	64.38	80	184.3
35	19	F	LE	LE	8.06	60	80	170.6	7.93	64.4	80	170.6
36	18	M	LE	LE	9.27	62.5	83.1	151.3	9.04	62.5	83.8	169.4
37	18	M	LE	LE	10.96	56.25	80.63	168.75	10.52	66.25	100.63	123.7
38	19	F	LE	LE	9.76	69.38	98.13	154.38	9.72	70	98.13	155
39	19	M	LE	LE	9.96	64.4	80.6	170.6	9.46	58.8	80.6	171.9
40	19	M	RE	RE	8.48	63.1	84.4	169.4	8.19	63.1	86.3	151.9
41	20	M	RE	RE	10.28	63.1	84.4	151.9	10.15	63.1	85.6	151.9
42	19	F	RE	RE	11.10	63.8	100.0	151.3	10.71	63.8	101.5	151.3
43	30	M	RE	RE	9.95	80.6	101.3	151.9	9.14	80.6	101.9	151.9
44	19	F	RE	RE	9.42	80.6	83.8	169.4	9.25	80.4	101.3	169.4
45	18	M	RE	RE	10.81	62.5	94.4	169.4	9.51	63.1	95.7	169.4
46	27	M	RE	RE	9.61	62.5	93.8	155.0	9.21	65.0	95.0	152.5
47	18	M	RE	RE	8.67	64.0	85.0	180.6	8.21	64.5	87.0	180.6
48	19	F	RE	RE	10.32	62.5	100.6	169.4	9.41	65.5	101.3	169.4
49	19	M	LE	LE	10.65	66.3	90.6	149.4	10.28	67.3	91.2	149.4
50	19	F	LE	LE	10.65	66.3	90.6	149.4	10.28	67.3	91.2	149.4
51	18	F	RE	RE	9.69	68.1	98.8	156.3	9.47	69.2	100.9	156.3
52	19	F	RE	RE	9.27	63.1	84.4	169.4	9.04	63.2	85.7	169.4
53	19	M	RE	RE	9.84	62.5	93.1	168.8	9.43	62.5	100.6	168.8

54	19	M	RE	RE	9.61	63.1	95.0	151.9	9.59	63.1	97.3	151.9
55	18	M	RE	RE	9.75	62.5	92.5	168.8	9.33	62.5	93.4	168.8
56	19	M	RE	RE	11.00	62.5	96.9	152.5	10.64	62.5	101.9	152.5
57	18	M	LE	LE	9.69	65	80	181.25	9.47	64.38	80	184.3
58	19	M	LE	LE	8.06	60	80	170.6	7.93	64.4	80	170.6
59	20	M	LE	LE	9.27	62.5	83.1	151.3	9.04	62.5	83.8	169.4
60	19	M	LE	LE	10.65	66.3	90.6	149.4	10.28	67.3	91.2	149.4
61	19	M	RE	RE	11.10	63.8	100.0	151.3	10.71	63.8	101.5	151.3
62	19	F	RE	RE	9.95	80.6	101.3	151.9	9.14	80.6	101.9	151.9
63	18	M	RE	RE	9.42	80.6	83.8	169.4	9.25	80.4	101.3	169.4
64	18	F	RE	RE	10.81	62.5	94.4	169.4	9.51	63.1	95.7	169.4
65	19	F	RE	RE	9.61	62.5	93.8	155.0	9.21	65.0	95.0	152.5
66	18	F	RE	RE	8.67	64.0	85.0	180.6	8.21	64.5	87.0	180.6
67	19	F	RE	RE	10.32	62.5	100.6	169.4	9.41	65.5	101.3	169.4
68	18	M	RE	RE	10.65	66.3	90.6	149.4	10.28	67.3	91.2	149.4
69	30	M	RE	RE	9.69	68.1	98.8	156.3	9.47	69.2	100.9	156.3
70	27	M	RE	RE	9.27	63.1	84.4	169.4	9.04	63.2	85.7	169.4
71	32	F	LE	LE	9.84	62.5	93.1	168.8	9.43	62.5	100.6	168.8
72	28	M	RE	RE	9.61	63.1	95.0	151.9	9.59	63.1	97.3	151.9
73	26	M	RE	RE	9.75	62.5	92.5	168.8	9.33	62.5	93.4	168.8
74	26	M	RE	RE	11.10	63.8	100.0	151.3	10.71	63.8	101.5	151.3
75	29	M	RE	RE	9.95	80.6	101.3	151.9	9.14	80.6	101.9	151.9
76	32	F	RE	RE	9.42	80.6	83.8	169.4	9.25	80.4	101.3	169.4
77	26	F	RE	RE	10.81	62.5	94.4	169.4	9.51	63.1	95.7	169.4
78	28	M	RE	RE	9.61	62.5	93.8	155.0	9.21	65.0	95.0	152.5
79	32	M	RE	RE	8.67	64.0	85.0	180.6	8.21	64.5	87.0	180.6
80	34	M	RE	RE	10.28	63.1	84.4	151.9	10.15	63.1	85.6	151.9
81	19	M	LE	LE	11.10	63.8	100.0	151.3	10.71	63.8	101.5	151.3
82	20	M	LE	LE	9.95	80.6	101.3	151.9	9.14	80.6	101.9	151.9

83	19	M	LE	LE	9.42	80.6	83.8	169.4	9.25	80.4	101.3	169.4
84	19	F	LE	LE	10.81	62.5	94.4	169.4	9.51	63.1	95.7	169.4
85	33	F	LE	LE	9.61	62.5	93.8	155.0	9.21	65.0	95.0	152.5
86	19	F	LE	LE	8.67	64.0	85.0	180.6	8.21	64.5	87.0	180.6
87	19	F	RE	RE	10.32	62.5	100.6	169.4	9.41	65.5	101.3	169.4
88	20	F	LE	LE	10.65	66.3	90.6	149.4	10.28	67.3	91.2	149.4
89	20	M	LE	LE	10.65	66.3	90.6	149.4	10.28	67.3	91.2	149.4
90	20	M	RE	RE	9.69	68.1	98.8	156.3	9.47	69.2	100.9	156.3
91	19	M	LE	LE	9.27	63.1	84.4	169.4	9.04	63.2	85.7	169.4
92	18	M	RE	RE	9.84	62.5	93.1	168.8	9.43	62.5	100.6	168.8
93	19	M	RE	RE	9.27	62.5	83.1	151.3	9.04	62.5	83.8	169.4
94	19	M	RE	RE	10.96	56.25	80.63	168.75	10.52	66.25	100.63	123.7
95	18	F	RE	RE	9.76	69.38	98.13	154.38	9.72	70	98.13	155
96	18	F	RE	RE	9.96	64.4	80.6	170.6	9.46	58.8	80.6	171.9
97	19	M	RE	RE	9.26	63.75	104.3	143.75	8.69	63.13	101.8	145.6
98	19	M	RE	RE	10.96	63.1	90.6	151.9	10.52	63.1	103.8	151.9
99	19	M	RE	RE	11.50	61.3	81.9	162.5	10.89	61.3	93.8	160.5
100	32	M	RE	RE	9.27	62.5	83.1	151.3	9.04	62.5	83.8	169.4

KEY TO MASTER CHART

LE - LEFT EYE

RE - RIGHT EYE

F - FEMALE

M - MALE