INFLUENCE OF REFRACTIVE ERROR AND OCULAR DOMINANCE ON PATTERN REVERSAL VISUAL EVOKED POTENTIAL – A COMPARATIVE STUDY

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

in partial fulfillment of the regulations

for the award of the degree of

M.D. (PHYSIOLOGY) BRANCH – V

CHENGALPATTU MEDICAL COLLEGE,

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI – TAMILNADU

MAY 2018

CERTIFICATE

This is to certify that this dissertation titled "**INFLUENCE OF REFRACTIVE ERROR AND OCULAR DOMINANCE ON PATTERN REVERSAL VISUAL EVOKED POTENTIAL – A COMPARATIVE STUDY**'' is a bonafide record of work done by **DR.M.RADHIKA**, during the period of her Post graduate study from 2015 to 2018 under guidance and supervision in the Department of Physiology, Chengalpattu Medical College and Hospital, Chengalpattu – 603 001 in partial fulfillment of the requirement for **M.D. PHYSIOLOGY** degree Examination of The Tamil Nadu Dr. M.G.R Medical University to be held in May 2018.

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DECLARATION

I declare that the dissertation entitled "**INFLUENCE OF REFRACTIVE ERROR AND OCULAR DOMINANCE ON PATTERN REVERSAL VISUAL EVOKED POTENTIAL – A COMPARATIVE STUDY**'' submitted by me for the degree of M.D. is the record work carried out by me during the period of June 2016 to June 2017 under the guidance of **Dr.A.ANITHA, M.D., DCH.,** Head of the Department of Physiology, Chengalpattu Medical College, Chengalpattu. This dissertation is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of the University regulations for the award of degree of M.D., Physiology (Branch V) examinations to be held in May 2018.

Place: Chengalpattu **Signature of the Candidate**

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LIST OF ABBREVIATIONS

ABBREVIATION	EXPANSION
WHO	**World Health Organisation**
EEG	**Electroencephalogram**
VEP	**Visual Evoked Potential**
MS	**Multiple Sclerosis**
LGB	**Lateral Geniculate Body**
LGN	**Lateral Geniculate Nucleus**
CDR	**Cup Disc Ratio**
RE	**Refractive Error**
PRVEP	**Pattern Reversal visual evoked potential**
VA	**Visual Aquity**
SD t-VEP	**Short-Duration transient visual evoked potential**

INSTITUTIONAL ETHICAL COMMITTEE

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The request for an approval From the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 11.05.2016 at the Medical Education Unit, Government Chengalpattu Medical College, Chengalpattu at 12.00 PM.

The Members of the committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

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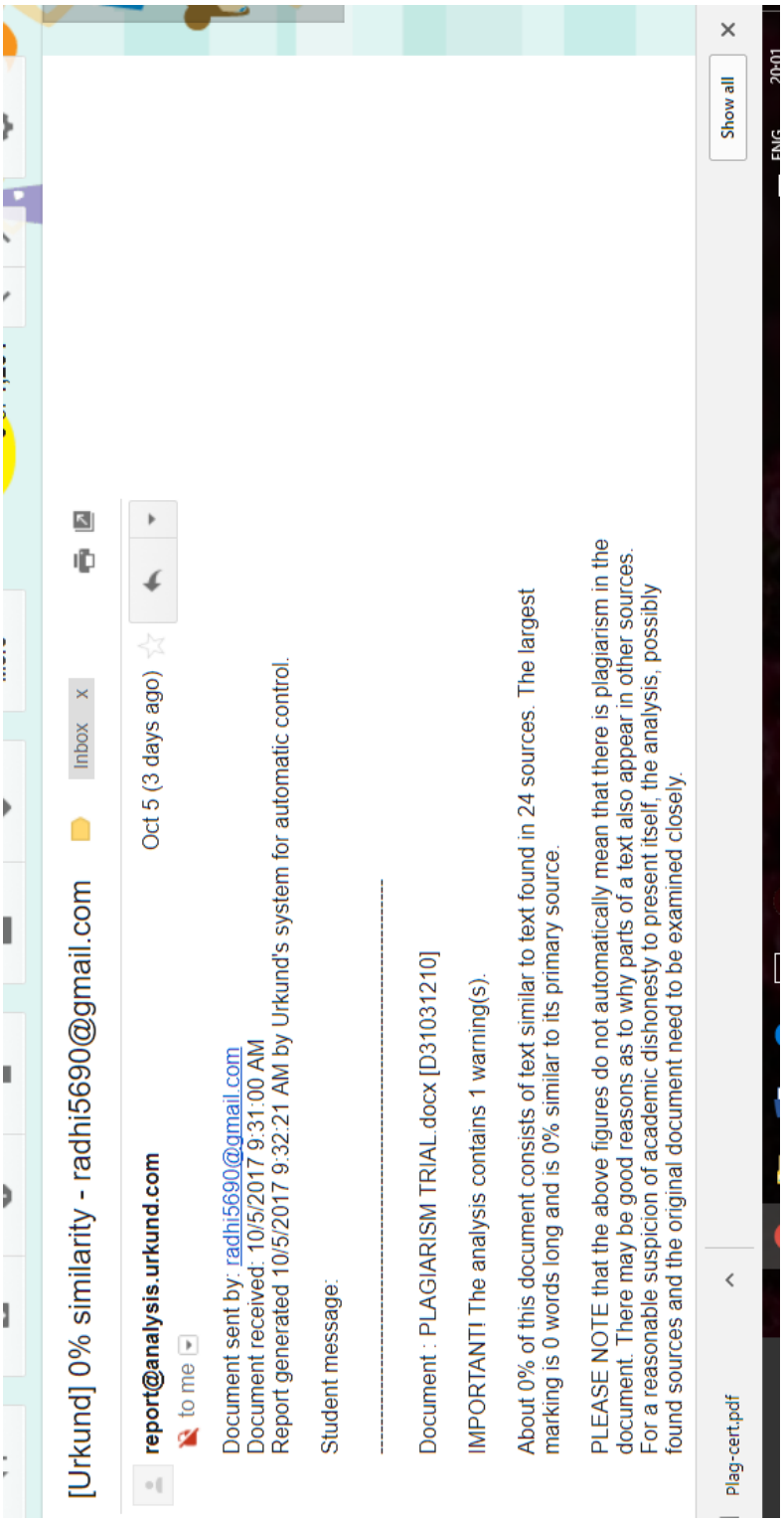

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This is to certify that this dissertation work titled "**INFLUENCE OF REFRACTIVE ERROR AND OCULAR DOMINANCE ON PATTERN REVERSAL VISUAL EVOKED POTENTIAL – A COMPARATIVE STUDY**'' of the candidate **DR.M.RADHIKA** with registration Number **201515502** for the award of **degree of M.D.** in the branch of **PHYSIOLOGY - BRANCH – V.** I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **0%** percentage of plagiarism in the dissertation.

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INTRODUCTION

Vision is one of the most important special senses in humans and is the basis for most human communication. The visual system detects and interprets electromagnetic waves between 400 and 750 nm long, which constitutes visible light. The human eye is a precise system which comprises components that must be optimally maintained so that a clear image is seen. Transparency, surface regularity and smoothness, and a stable ocular anatomy are important for sight. (Parson)

The WHO estimates that 153 million people worldwide live with visual impairment due to uncorrected refractive errors. This does not include the people living with uncorrected presbyopia, which is likely to be quite significant, according to some early evidence.

The number of people globally with refractive errors that have not been corrected was estimated at 660 million (10 per 100 people) in 2013. The number of people globally with refractive errors has been estimated from 800 million to 2.3 billion.[4] Visual impairment was estimated to affect 161 million people globally in 2002, of whom 37 million were blind

It also has serious social implication for school children as it make the child drop out of school. An estimated 2.3 billion people worldwide have a refractive error in which about 500 million people do not have access to refractive error services who live in developing countries and are mainly children. The latest global estimates of visual impairment suggest that among

children aged 5-15 years, 12.8 million are visually impaired due to refractive error representing prevalence of 0.97% with higher prevalence reported in china and urban areas of South East Asia.[6]

Visual System:

Human eye has a lens system, a variable aperture system (the pupil), and a retina that corresponds to the film.

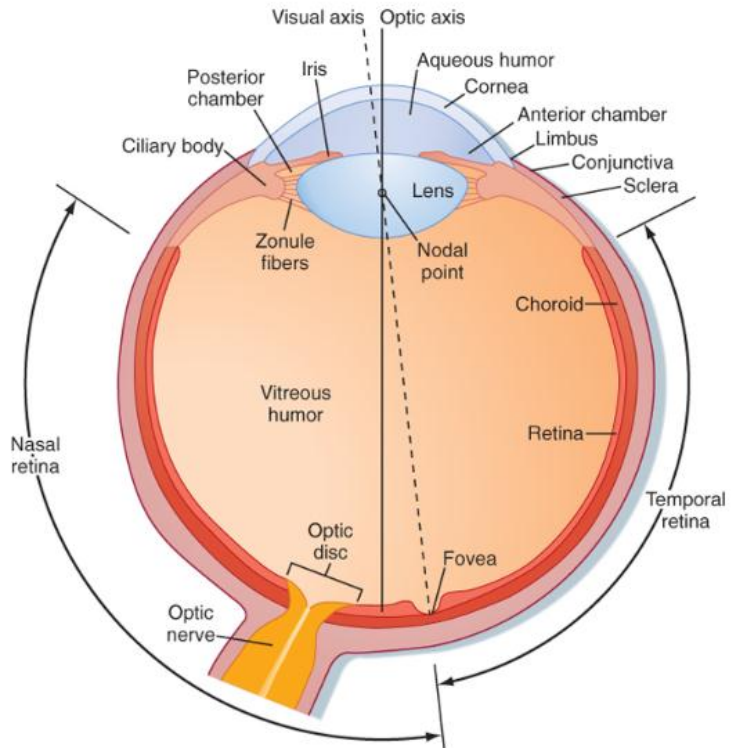


Figure 1: The Human Eye

The lens system of the eye is composed of four refractive interfaces: (1) the interface between air and the anterior surface of the cornea, (2) the interface between the posterior surface of the cornea and the aqueous humor, (3) the interface between the aqueous humor and the anterior surface of the lens of the eye, and (4) the interface between the posterior surface of the lens

and the vitreous humor. The internal index of air is 1; the cornea, 1.38; the aqueous humor, 1.33; the crystalline lens (on average), 1.40; and the vitreous humor, 1.34.

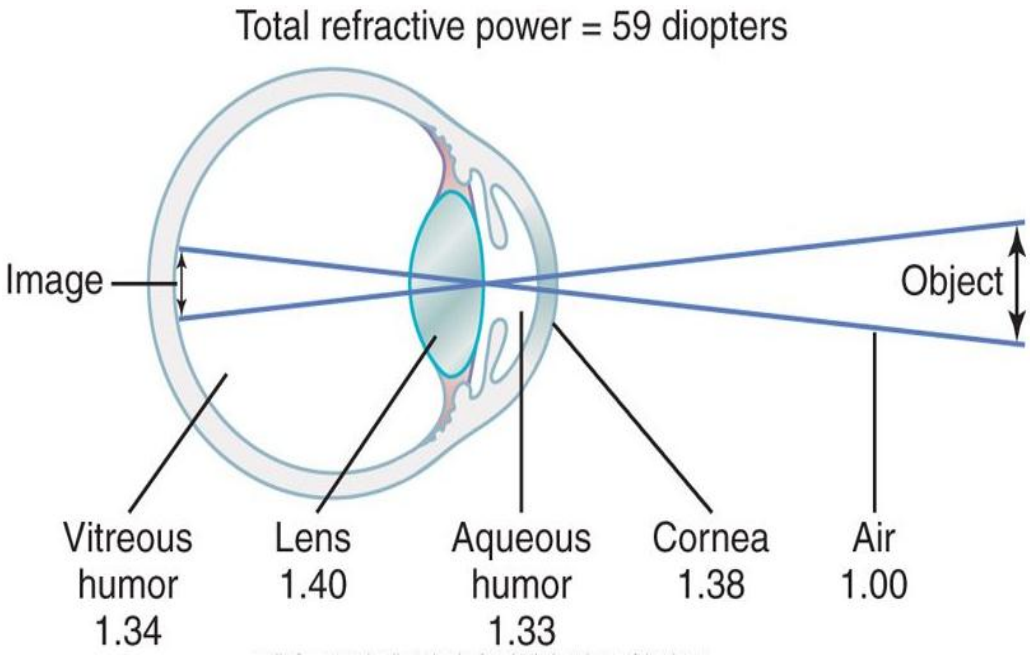


Figure 2: Refraction in Human Eye

Two thirds of the 59 diopters of refractive power of the eye is provided by the anterior surface of the cornea. The principal reason for this is that the refractive index of the cornea is markedly different from that of air, whereas the refractive index of the eye lens is not greatly different from the indices of the aqueous humor and vitreous humor.

The refractive power of the internal lens of the eye is only 20 diopters, about one-third the total refractive power of the eye. But the importance of the internal lens is that, in response to nervous signals from the brain, its curvature can be increased markedly to provide "accommodation".

Handedness and Ocular dominance:

In physiological aspect dominance indicates predominance, priority and preferential activity of one side of the body to other side. Often one have a tendency to write with same hand which is the dominant hand and might as well a kick a ball with same foot which is the dominant foot. Functional lateralization occurs in the paired organs of the body, such as hands, legs, and cerebral hemispheres.

In 1593, Giovanni Ballista Porta is the one who described Ocular dominance first. Ocular dominance, otherwise called eye dominance is the tendency to prefer visual input from one eye to the other. As an individual is conscious of having right and left hand use, no one is conscious about which eye is predominantly used ,either right or left eye. An individual does not see the world from right or left eye but gets information from both, which is so called cyclopean eye.[8]

Ocular dominance is a complex phenomenon. Approximately two-thirds of the population is right-eye dominant and one-third is left-eye dominant; whereas in a small portion of the population neither eye is dominant. Dominance wise eyes grab the image with one eye and pass on to other and start to analyze the image (Jagadamba and Karthianee Kutty, 2012).

Sub-types of Ocular dominance: (Taghavy and Kugler, 1987).

1) Sighting dominance,
2) Sensory dominance
3) Acuity dominance.

The conduction in the dominant eye is very fast. So while playing the games in which player have to focus high speed ball like cricket and tennis it is shown that as they have to maintain the ability to make controlled contact with ball at high speed with bat or racquet.

Tests to detect dominant eye:

1. The Miles 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 (i.e. the dominant eye).
2. The Porta test.
3. The Dolman method,
4. The convergence near-point test
5. The pinhole test.
6. The ring test.
7. Lens fogging technique.
8. A dichoptic motion coherence threshold test
9. Look at your nose tip to notice near sight dominance

Measure of binocular vision:

Binocular was derived from latin words where ‘bini’ means double and ‘oculus’ means eye. Binocular vision indicates a type of vision in which two eyes perceives a single three-dimensional image of its surroundings. It gives a wider field of view and. It is responsible for stereopsis in which binocular disparity (or parallax) provided by the two eyes different

positions on the head gives precise depth perception of an image. Apart from binocular summation of visual impulse, the two eyes influences each other in pupillary diameter difference, accommodation, interocular transfer and ocular dominance.

Fusion of images occurs only in a small volume of visual space. Running through the fixation point in the horizontal plane is a curved line which is called the empirical horizontal horopter. Here the objects there fall on corresponding retinal points in the two eyes. Empirical vertical horopter, is effectively tilted away from the eyes above the fixation point and towards the eyes below the fixation point. These empirical horizontal and vertical horopters mark the centre of the volume of singleness of vision. Within this thin, curved volume, known as Panum's fusional area, the objects nearer and farther than the horopters are seen as single. Outside this Panum's fusional area , double vision occurs. It is impossible to align images outside of Panum's fusional area , when each eye has its own image of objects [14]. Inside this panum’s fusional area in which an eye is moving faster to the object and stay fixated on it is more likely to be termed as the dominant eye.[14]

Emmetropization

Emmetropia is the condition in which there is considered to be an absence of any refractive error because parallel beams of light come to focus on the retina, with the eye at rest.

Eye of a newborn is hypermetropic . At birth the average axial length is 18 mm, the cornea will be curved and anterior chamber will be slightly shallow. Rapid growth occurs in the first few years of life to reach an axial

length of about 23 mm by the age of 3 years, the cornea becomes slightly flat, the anterior chamber deepens and the degree of hypermetropia gradually reduces. From 3 to 14 years of age, the axial length increases further by 1 mm and the power of the crystalline lens changes to achieve and maintain emmetropia. This entire process from birth onwards is known as emmetropization.

AMETROPIA

The condition in which incident parallel rays of light do not come to a focus upon the light-sensitive layer of the retina is called Ametropia. It may be due to one or more of the following conditions:

1. Axial ametropia: Abnormal length of the globe—very long in myopia, very short in hypermetropia. A 1 mm elongation produces approximately 3 D of myopia and 1 mm shortening 3 D of hypermetropia.

2. Curvature ametropia: Abnormal curvature of the refracting surfaces of the cornea or lens—more curved in myopia, slightly curved in hypermetropia. A 1 mm change in the radius of curvature of the cornea produces a 6 D change in refraction.

3. Index ametropia: Abnormal refractive indices of the media. In index myopia the refractive index, either of the cornea, the aqueous or of the lens is too high, and that of the vitreous maybe too low as in a vitrectomized eye with silicone oil in the vitreous cavity. In index hypermetropia the opposite conditions are operative, and the

error is high when the lens is absent as in aphakia or absence of the lens from the pupillary plane[2].

4. Abnormal position of the lens: Displacement forwards causes myopia, displacement backwards hypermetropia.

Refractive errors:

A refractive error is a very common eye disorder. It occurs when the eye cannot clearly focus the images from the outside world. The result of refractive errors is blurred vision, which is sometimes so severe that it causes visual impairment.[1] Refractive errors occur when the shape of the eye prevents light from focusing directly on the retina. The length of the eyeball (longer or shorter), changes in the shape of the cornea, or aging of the lens can cause refractive errors.

Types of refractive errors:

1. Myopia (short sightedness): difficulty in seeing distant objects clearly;

2. Hyperopia/ Hypermetropia (long sightedness): difficulty in seeing close objects clearly;

3. Astigmatism: visual disturbances resulting from an irregularly curved cornea, the clear covering of the eyeball.

4. Presbyopia: which leads to difficulty in reading or seeing at arm's length because of ageing and occurs almost universally.

Refractive errors affects a large proportion of the population worldwide, irrespective of age, sex and ethnic group. It can be diagnosed by an eye examination and treated with corrective glasses, contact lenses or refractive surgery, but cannot be prevented. Correction is provided in different forms according to the type of error, the age of the person, the requirements in terms of work of activity performed. If corrected in appropriate time and by eye-care professionals, they do not impede the full development of good visual function. If, however, they are not corrected or the correction is inadequate, refractive errors become a major cause of low vision and even blindness

Because of the increasing realization of the enormous need for correction of refractive error worldwide, this condition has been considered one of the priorities of the recently launched global initiative for the elimination of avoidable blindness: VISION 2020 — The Right to Sight[7]

History:

The ancient Egyptians and Mesopotamians are those who began optics with the development of lens. Then the Theories on light and vision was developed by ancient Greek philosophers, and the development of geometrical optics in the Greco-Roman world. The word optics is derived from the Greek word meaning "appearance, look".

In 1621, Snellius Willebrord discovered the law of refraction. It expresses the relationship between the path of a ray of light passing through the boundary of two adjacent substances and their respective refractive indices

In 1637, Descartes published Dioptrique in which he announced his formulation of the laws of reflection and refraction, using analogies of moving balls and the walking stick of a blind man. In 1657, Marin Cureau de la Chambre, in his book “light” states his disagreement with those who seek to explain the motion of light ballistically (as Descartes does), saying instead that in the case of reflection, equal angles are not made due to some principle that creates equal angles as such, but rather because nature does everything by the simplest means, and that the equality of angles in reflection was merely a necessary result of light taking the simplest (shortest) path.

Ibn Sahl's 984 treatise “On Burning Mirrors and Lenses” explains his understanding of how curved mirrors and lenses bend and focus light. Therby Ibn Sahl discovered the law of refraction, usually called Snell's law.the first person to differentiate myopia and hypermetropia what we call now was Aristotle (in 384BC). Galen used the word myopia first in 138-201AD. The written record is in “Libris Pandectorum”, a complication of roman law.

In 1755, Hypermetripic condition was first suggested by mathematician Kastner. But he was failed to differentiate between hyperopia and presbyopia. In 1864 Donders ,the ophthalmologist of Uteecht clarified the difference between hyperopia and presbyopia.

Myopia / 'short sightedness' :

Myopia is a significant and prevalent disease in children with increasing rates of progression. With over 80 million reported myopic children world wide there are considerable public health and socioeconomical concerns. A substantial amount of research has been done to determine

the etiology of myopia, the risk factors associated with myopia, techniques to prevent myopia and ways to treat myopia.(Original article, Gregory I. Ostrow et al)

Myopia is a type of refractive error in which, parallel rays of light coming from infinity are focused anterior to the light- sensitive layer of the retina, with the accommodation at rest.

Myopia is derived from the term "muopia" which, in Greek, means to close the eyes.(Optometry: The Primary Eye Care Profession). Severe myopia is associated with potentially blinding complications such as glaucoma, retinal detachment, and myopic macular degeneration.

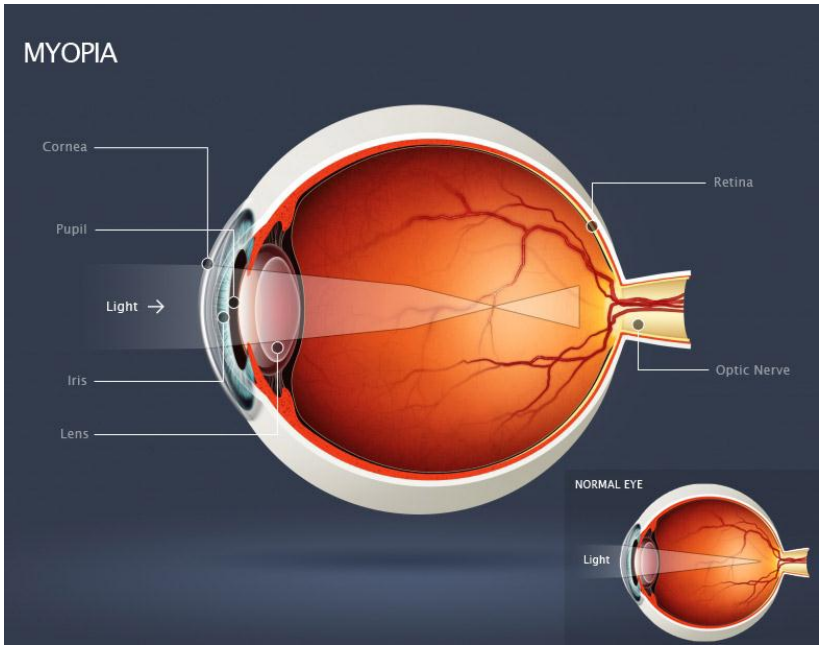


Figure 3: Myopia / 'short sightedness'

Table 1

Classification of Myopia:

Type of Classification	Classes of Myopia
Clinical Entity	Simple myopia Nocturnal myopia Pseudomyopia Pathological myopia Acquired myopia
Degree	Low myopia (<3.00 D) Medium myopia (3.00 D-6.00 D) High myopia (>6.00 D)
Age of Onset	Congenital myopia (present at birth and persisting through infancy) Youth-onset myopia (<20 years of age) Early adult-onset myopia (2-40 years of age) Late adult-onset myopia (>40 years of age)

1. Simple Myopia

In simple myopia, the refractive status of the eye is dependent on the optical power of the cornea and the crystalline lens, and the axial length. An eye with simple myopia is either too long for its optical power or, less commonly, too optically powerful for its axial length. Simple myopia, which is common than the other types of myopia, is generally less than 6 diopters (D); in many patients it is less than 4 or 5 D.

2. Nocturnal Myopia

Occurs in dim illumination, nocturnal or night myopia is due to increased accommodative response associated with low levels of light

3. Pseudomyopia

Pseudomyopia is the result of overstimulation of the eye's accommodative mechanism or ciliary spasm leading to an increase in ocular refractive power. The condition is named so because the patient only appears to have myopia due to an inappropriate accommodative response

4. Pathological Myopia

A high degree of myopia associated with degenerative changes in the posterior segment of the eye is known as Pathological or degenerative myopia. The degenerative changes can result a decrease in best corrected visual acuity or changes in visual fields. Sequelae such as retinal detachment and glaucoma are relatively common.

5. Acquired Myopia

Induced or acquired myopia is because of exposure to various pharmaceutical drugs, blood sugar level variation, nuclear sclerosis of the crystalline lens, or other anomalous conditions. This myopia is often reversible and temporary.

Other type of classification:

1. Non-pathologic
2. Pathologic myopia.

Each group have separate disease processes, clinical features, and prognoses.

1. Non-pathological myopia:

Non-pathological myopia is also called as physiological, simple or school myopia. In non-pathologic myopia the refractive power of the eye does not correlate with the axial length. The degree of non-pathologic myopia is usually minimal to moderate (< 6 D) and onset usually begins during childhood or adolescence.

2. Pathologic myopia:

Pathologic myopia / high myopic refractive error is progressive and generally presents very early in childhood. Pathologic myopia is usually defined as spherical equivalent > 6.00 diopters or axial length >26.5mm[3]. Patients with high axial myopia are at a greater risk of developing progressive retinal degeneration and other vision threatening pathology. Pathological curvature myopia is seen typically in keratoconus. The condition is strongly

hereditary, commoner in women than in men. It has a racial predilection, being common among Jews and Japanese and most cases are of genetic origin. (parson)

Possible Risk Factors for Myopia Development:

1) Family history of myopia
2) Presence of myopia on noncycloplegic retinoscopy in infancy, decreasing to emmetropia before entry into school
3) Refractive error of emmetropia to 0.50 D of hyperopia
4) Against-the-rule astigmatism
5) Decreased accommodative function or near point esophoria
6) Substantial amount of near work on a regular basis
7) Steep corneal curvature or high axial length to corneal radius ratio
8) Conditions temporarily obscuring the retina from clear imagery during infancy

Etiology of Myopia:

Myopia is a complex disease with a multi-factorial etiology. It is well documented that pathological non-syndromic high myopia and associated syndromic high myopia show evidence of familial inheritance. Although non-syndromic high myopia is most commonly inherited in an automosomal dominant pattern, multiple chromosomal loci have been identified which suggests genetic heterogeneity.

High myopia is also a symptom of several multi-system complex diseases. The genetic mutations for these syndromes have been identified and

the subsequent structural defects of the eye are most commonly related to connective tissue and retina. This type of myopia is only a small proportion of the overall myopic population and to date, there is no known isolated gene associated with physiologic myopia.

Table 2

Etiologies of Myopia

Type of Myopia	Etiologies
Simple Myopia	Inheritance Significant amounts of near work Unknown
Nocturnal Myopia	Significant levels of dark focus of accommodation
Pseudomyopia	Accommodative disorder High exophoria Cholinergic agonist agents
Pathological Myopia	Inheritance Retinopathy of prematurity Interruption of light passing through ocular media Unknown
Acquired Myopia	Age-related nuclear cataracts Exposure to sulfonamides and other pharmaceutical agents Significant variability in blood sugar level

Associated Disease:

The power of the lens can be increased by osmotic changes (diabetes, galactosemia, uremia, sulfonamides), nuclear sclerotic cataracts, anterior lenticonus, and changes in lens position or shape (miotics, anterior lens dislocation, excessive accommodation). Changes to the cornea secondary to keratoconus, congenital glaucoma, and contact lens-induced corneal warpage can also cause myopia. Myopia can also be the result of increased axial length secondary to retinopathy of prematurity, posterior staphyloma, scleral buckle surgery and congenital glaucoma.

Clinical picture:

Symptoms:

- ✓ Poor vision for distance (short sightedness)
- ✓ Asthenopic symptoms
- ✓ Half shutting of the eyes
- ✓ Muscae volitantes (floating black opacities in front of eyes due to degenerated liquified vitreous) in patients with pathological myopia.

Signs :

- ❖ Prominent eyeballs
- ❖ Anterior chamber is slightly deeper than normal
- ❖ Fundus is normal; rarely temporal myopic crescent is seen.

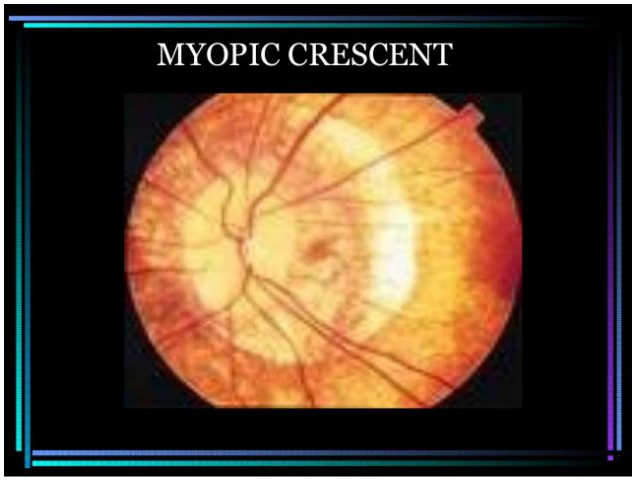


Figure 4: Myopic Cresent

- In case of pathological myopia fundus examination shows
- large optic disc with characteristic myopic crescent,
- degenerative changes in retina and choroid,
- foster fuch's spot (dark red circular patch due to sub-retinal neovascularisation and choroidal haemorrhage)

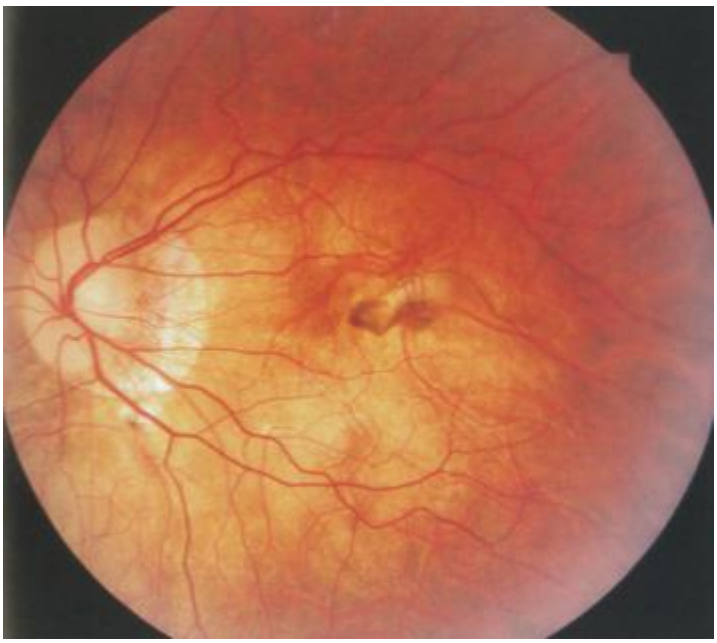


Figure 5: Foster Fuch's Spot

- ❖ Cysoid degeneration may be seen in peripheral retina
- ❖ Posterior staphyloma due to ectasia of sclera at posterior pole.
- ❖ Subnormal electroretinogram

Complications:

- Retinal detachment
- Choroidal haemorrhage
- Vitreous haemorrhage
- Compicated cataract
- Strabismus fixus convergence.

Hypermetropia:

The term Hypermetropia denotes, Hyper meaning excess, met meaning measure and opia meaning the eye. Hypermetropia is a refractive error in which parallel rays of light come to a focus behind the retina, when accommodation is at rest is called Hypermetropia or Hyperopia.

Classifications of Hypermetropia:

Depending by anatomical features:

1. Axial Hypermetropia:

It occurs when antero-posterior diameter of the eye is less than the normal causes hypermetropia (Normal axial length is 24mm). 1mm shortening will cause +3D hypermetropia.

2. Curvatural Hypermetropia:

Normally the radius of curvature of cornea anteriorly and posteriorly is 7.8mm and 6.5mm respectively. Normal radius of curvature of lens anteriorly and posteriorly is 10mm and 6mm respectively. If the curvature of cornea and lens is less than the normal causes hypermetropia. 1mm flattening of curvature of cornea will cause +6D hypermetropia.

3. Index Hypermetropia:

It occurs if refractive index of cornea and lens is less than the normal.

4. Displacement of lens:

If the lens is displaced backwards, it results in hypermetropia.

5. Absence of lens:

Absence of crystalline lens (Aphakia) causes hypermetropia. It may be surgical removal or posterior dislocation.

Classification by degree of hypermetropia:

Low Degree: +0.25 to +3D
Medium Degree: +3 to +5D
High Degree: >+5D.

Classification by the action of accommodation:

1. Latent Hypermetropia:

It is the amount of hypermetropia which is corrected normally by the normal tone of ciliary muscle. It is more in young children than in adults. It can be manifested only after (atropine) cycloplegic medication.

2. Manifest Hypermetropia: Is of two types.

(A) Facultative Hypermetropia: It is a type of hypermetropia which can be corrected by the effort of accommodation.

(B) Absolute Hypermetropia: It is a type of hypermetropia which cannot be overcome by the effort of accommodation.

3. Total Hypermetropia:

It can be found out by abolishing the tone of ciliary muscle by cycloplegics like atropinization. Total hypermetropia= Latent hypermetropia+ Manifest hypermetropia (Facultative+Absolute).

Clinical test to find out different type of manifest hypermetropia:

1. Manifest Hypermetropia:

The strongest convex lens with which the patient can still maintain full distance vision 6/6 ,indicates manifest hypermetropia.

2. Absolute Hypermetropia:

If the patient can not normally see 6/6 without a lens then the weakest convex lens that will allow him to read this line ,indicates absolute hypermetropia.

3.Facultative Hypermetropia:

Facultative hypermetropia=Manifest hypermetropia -Absolute hypermetropia.

Classification into Physiological and Pathological hyperopia:

1. Physiological Hypermetropia:

It is normal biological variation which includes axial and curvatural hypermetropia. It may be hereditary.

2. Pathological Hypermetropia:

A reduction in axial length due to any space-occupying lesion within the eye such as retinal detachment, Central Serous Retinopathy, orbital tumours, retinal tumour etc.

Optical variation:

1. Parallel rays of light come to focus behind the light sensitive layer of retina and the image which are formed here are blurred and indistinct.
2. Far point of hypermetropic eye is beyond infinity that is behind the retina and far point of eye is virtual point behind the eye.
3. Image size is smaller when compared with emmetropia.

4. In hypermetropia the fomation of clear image is impossible ,unless the converging power of the optical system is increased. This may be done in two ways-

   a) by the effort of accommodation.

   b) by placement of a convex lens in front of the eye.

5. In hypermetropia rays coming from a point on the retina will be divergent than corresponding rays of the emmetropic eye.

Clinical picture:

1 Blurring vision –more for near than for distance.

2. Convergent Squint –due to continuous effort of accommodation

   Excess of convergence

   Dissociation of muscle balance

   A convergent squint

3. Early onset of presbyopia.

4. Accommodative asthenopia. These include:

   a) Tiredness of eyes.

   b) Frontal or fronto-temporal headache.

Signs:

1 Small eye ball.

2. Small cornea.

3. Anterior chamber is shallow and the angle is narrow.

4. Visual acuity varies with the degree of hypermetropia and the power of accommodation.

5. Apparent divergent squint

6. Ophthalmoscopically-

   a) Optic disc is smaller, hyperaemia with less defined cup disc ratio.

   b) It may show a characteristic appearance which may resemble optic neuritis or papilloedema.

   c) Tortuosity and abnormal branching of blood vessels.

Complications:

1. Recurrent styes, blepharitis or chalazion may occur due to frequent rubbing of the eyes to get clear vision.

2. Accommodative Convergent squint may develop in children due to excessive use of accommodation.

3. Amblyopia.

4. Angle closure glaucoma due to small eye with shallow anterior chamber and narrow anterior chamber angle

Visual evoked potentials :

Evoked potential is nothing but any neuronal response triggered by stimulating sensory receptors or peripheral nerves, and also any neuronal activity time-related to cognitive processes or motor programming. (Binnie, 2010). Recording of evoked potential are non-invasive techniques and have excellent temporal resolution as minute as in milliseconds, thus permitting to study the dynamic changes that occur in the nervous system (Michael aminoff, 2005).

Specific objectives of Evoked potentials :

(i) provides recorded evidence of abnormality,

(ii) heplful in detecting subclinical lesions,

(iii) finding the level of impairment along an anatomical pathway,

(iv) providing clues about the pathology of disorders and

(v) prognostic monitoring of management of disorder.

History (John Heckenlively, 2006)

The study of evoked potentials arose an interest in spontaneous electrical activity of brain, recorded in EEG. Beck marked the position of electrodes that gave him response to light stimului and sound stimuli[9]. Pravdich-Neminsky took the first photograph of an evoked potential recorded of the cortex of a dog on stimulation of the sciatic nerve.[9]

In 1875, Richard Caton first recorded the electrical activity in the animal brains. 50 years later, Hans Berger discovered resting α rhythm of the EEG in man. VEP, that has been useful for clinical and research purposes since more than five decades was first introduced by Adrian and Matthews. The rhythm from the occipital region of the brain was demonstrated by Lord Adrian and said that and its appearance was associated with visual inattention. Among different evoked potentials, apart from brain stem auditory evoked potentials and somatosensory evoked potentials, visual evoked potentials (VEPs) were reported to be the most commonly used EP (Nuwer et al., 1998).

Halliday clinically utilized pattern reversal VEPs in the diagnosis of patients of optic neuritis, and it gave way to VEPs for being particularly used in the assessment of cases having suspected multiple sclerosis(MS). Pattern reversal VEPs than flash VEPs are more sensitive to optic nerve lesions. This commonly used method of VEP, pattern reversal VEP was developed and popularized in the early 1960s [9].

The visual evoked potential is an electrical signal recorded from the occipital region of cerebral cortex in response to the change in visual input such as a pattern stimuli or a flash light. It measures the time taken for conduction of neuronal activity from the retina to the occipital cortex. It is used clinically as a measure of the integrity and function of visual pathway (Andrew blum, 2007). VEPs recorded with pattern reversal VEP are exquisitely sensitive to disorders of the optic nerves and optic chiasm, which is very useful clinically.

The evoked responses can measure peak latencies and amplitudes, in the millisecond domain, which are analyzed and they provide numerical data which are quantitative extensions of neurological examination (Walsh et al., 2005).

VEP and Stereoscopic Vision : (Michael aminoff, 2005) (Misra, 2010)

The visual system recieves information and processes it along multiple parallel channels. The neuronal circuitry of the retina starts discriminating the visual information, where particular features such as contrast, colour, luminance, and other parameters of the stimulus are processed.

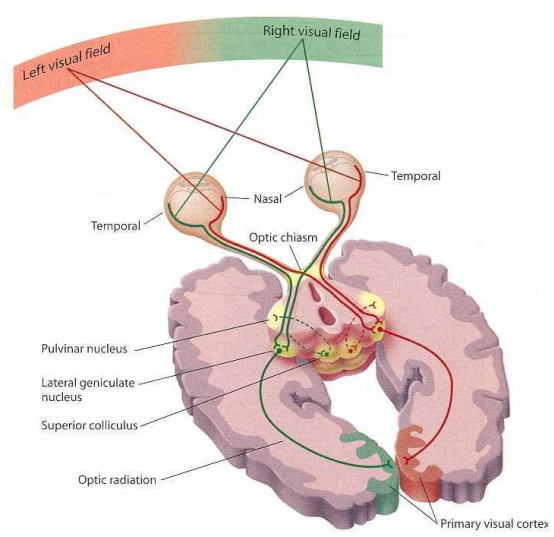


Figure 6: Stereoscopic Vision and Visual Pathway

Different types of retinal ganglion cells are X, Y, and W types. The X cells are small ganglion cells that transmits visual information from cones via small diameter axons with small receptive field. It incorporates lateral inhibition and concentrate in central visual field, low sensitivity to motion and providing the substrate for pattern VEPs via the geniculate pathway. The Y

cells are large retinal ganglion cells that transmits the information from rods, via large diameter axons with wide receptive field, peripheral retinal location, high sensitivity to motion, and without lateral inhibition. It possibly generates flash VEP via an extrageniculate pathway.

These retinal neurons send central projections of visual signals to the lateral geniculate body (LGB). Two separate classes of ganglion cells have been described in the macaque monkey:

(1) P cells (or midget cells) and

2) M cells (or parasol cells).

These two neuronal groups in the retina have its own physiologic properties and project separately to segregated brain regions. M (magnocellular) pathways can be activated independently of the other pathways. This system is involved mainly in motion analysis. P cells projections to the parvocellular laminae of the LGB, whereas M cells send their projections to the magnocellular laminae. From the LGB, visual information is transmitted to the primary visual cortex (striate area 17) and secondary visual cortex, area 18 and 19.

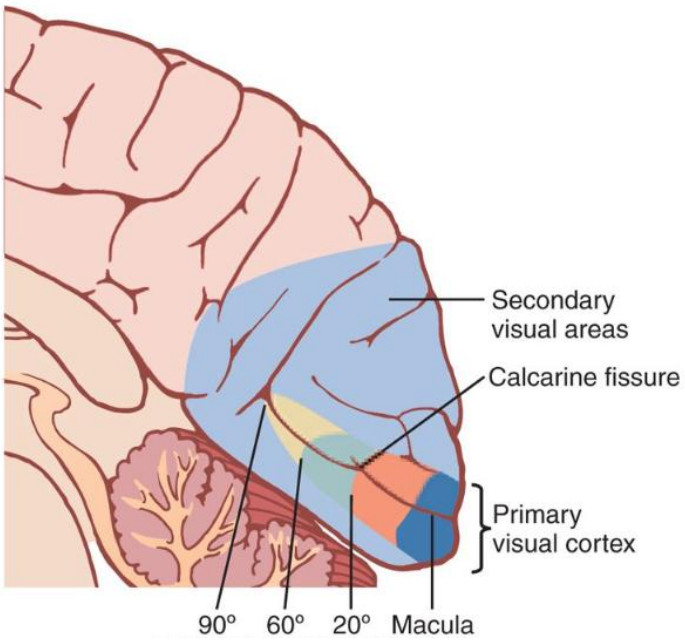


Figure 7: Visual Cortex

The parvocellular system shows a preference for high spatial frequency stimuli and involved in colour selectivity. Over one-third of the cerebral cortex is devoted primarily to visual function and atleast 10 cortical visual areas have been described in humans. Visual stimuli also involve large areas of the temporal and parietal lobes and not only the occipital lobes. This indicates that VEPs can be recorded from a wide region of the scalp, from the vertex to the inion. So , the reference electrode should be located anterior to the vertex or distant from these active regions.

By changing the characteristics of the visual stimulus, different structures of the retina and visual pathways can be specifically activated. This indicates the importance of selecting the proper visual stimulus for evaluating specific clinical disorder. The P100 waveform of VEP is generated in the peristriate and striate cortex of occipital lobe due to activation of primary cortex and thalamocortical volleys. On giving pattern or flash stimulation, not

only there is increased metabolism in primary visual area but also in the secondary visual areas 18 and 19 (Phelps et al., 1981). VEP is primarily focuses the activity originating in the central visual field (3-6°), which is relayed to the surface of occipital lobe. Peripheral retina stimulation projects to the regions deep within the calcarine fissure. Thus VEPs are attenuated or even unrecordable on peripheral retinal stimulation.

Normal Pattern Reversal VEP :

The VEPs represents the response of cortical and possibly the subcortical areas. The transient VEPs consist of series of waveforms with opposite polarity. The negative waveform is denoted as 'N' and positive deflection as 'P', and the latency is recorded in millisecond. The peak latency and peak- to- peak amplitudes of these waves can also be measured (Misra, 2010)

Pattern VEPs are recorded in the mid-occipital region when the pattern is presented in the central part of the visual field.

Components of VEP:

- N75
- P100
- N145

Of these three components P100 has the largest amplitude which is the positive one peaking at a latency of about 100ms and can be obtained with a

wide range of patterns, even though its amplitude and latency are affected by changing the stimulus parameters.

Table 3

Normal Values of P100 wave

Parameters	Shahrokhi et al (1978) Mean + SD	Misra and Kalita Mean + SD
P100 latency(ms)	102.3 + 5.1	96.9 + 3.6
Amplitude (mv)	10.1 + 4.2	7.8 + 1.9
Duration	63.0 + 8.7	55.9 + 7.7

The clinical interpretation of pattern VEPs depends mainly on the P100 latency. The P100 latency is the time from the onset of the stimulus to the main positive peak. Amplitude is more variable and less specific than latency. It is recommended to measure the amplitude of P100 from the preceding N75 peak.

Clinical Applications of VEP:

VEP, rather than primarily reflecting neuroanatomic lesions, it is a measure of physiological function. VEP should be regarded as complementary evidence to clinical examination and neuro-ophthalmological investigations. The VEP studies is a sensitive method for documenting the abnormalities in visual pathways especially anterior to optic chiasma. Abnormalities in the waveform, latency, or amplitude, or of the VEP indicates the impairment in the conduction in the retino-striate pathway.

Evoked potentials have been proved to be clinically helpful in following ways:

- Helpful in detection of demyelinated diseases, particularly useful in supporting the diagnosis of non-organic visual loss
- Applied to investigate purely subjective symptoms and detect whether they are related to any dysfunction of organic origin.
- to assess the degree of functional recovery and to appraise the causative mechanisms of neurological deficits or
- helps in monitoring various cerebral functions when the patient's condition is critical or at risk in the operating theatre or during intensive care.

A large number of studies combining EPs and brain-imaging techniques in finding the sites of lesions in patients with focal pathology of the nervous system and various degrees of sensory deficit. VEP studies are helpful in various neurological disorders for diagnostic purpose and also in detecting subclinical abnormalities. Without detectable alteration in visual acuity, contrast senstitivity, pupillary reactivity, colour desaturation, fundoscopy, or perimetry, prolonged P100 latencies denotes any disease of the anterior optic pathways. Abnormalities missed by MRI may also be revealed in VEP. But VEPs are not useful in investigating lesions posterior to optic chiasma.

Intraoperative VEP recording minimizes postoperative visual deterioration and it can be avoided. So during orbital and pituitary surgery, monitoring VEP intra-operatively prevents damage to optic nerves or chiasm,

and to help identify these structures if they are embedded in, or infiltrated by tumour. If any abnormality occurs, alteration or reversal of procedure may prevent or minimize the damage.

Impact of Refractive error and Ocular dominance on VEP: (Misra, 2010)

The visual evoked potential is defined as the electrical response, evoked by visual stimulation, from neurons in visual cortex[11]. A normal VEP is generally indicates normal visual function, however an abnormal VEP study may or may not be associated with normal clinical findings. Various studies shows that P100 wave latency is one of the major parameter known to find any abnormality in the visual Pathway.

Different variables like pupil diameter, refractive errors, age, sex hormones, eye dominance, illumination, type of stimulus, electrode position, and anatomical variations can influence the recording of VEP.

Eye dominance:

While playing the games like cricket, tennis it is shown that conduction in the dominant eye is very fast as they have to maintain the ability to make controlled contact with ball at high speed with bat or racquet[8]. Due to neuro anatomic asymmetries of human visual cortex, amplitude of P100 is greater in dominant hemisphere and the P100 wave obtained by stimulating the dominant eye is shorter and amplitude greater compared to the nondominant eye (Seyal et al., 1981).

Visual Aquity: -

P100 amplitude decreases with reduction of visual acuity and the latency is reported to be normal with visual acuity as low as 20/120. P100 latency is insensitive to refractory errors, with a checkerboard containing large checks, but with smaller checks represented foveally, it increases when the retinal image is defocused. Therefore visual aquity must be measured and refractive errors should be corrected before recording VEPs and its ideal to ask patients to wear corrective glasses during testing. Poor visual aquity causes low amplitude and prolonged pattern VEPs.

The pattern-reversal visual evoked response (VER) technique is very useful in the investigation of various neurological disorders, particularly multiple sclerosis (MS). Absolute or relative increases in the latency of the VEP is almost invariably found in patients with demyelinating optic neuropathy, and attenuation and desynchronisation of the response is a common feature of non-demyelinating lesions of the visual pathways. But various changes in VEP occurs because of the influence of refractive error and ocular dominance. Hence, In view of the increasing use of this technique in neuro-ophthalmological diagnosis, we are interested in studying the influence of refractive error and ocular dominance in pattern-reversal VEP.

REVIEW OF LITERATURE

Visual System :

The visual system is a salient division of the central nervous system which makes us able to process the visual information, as well as enabling the formation of several non-image photo response functions. It helps in detection and interpretation of information from visible light and forms images representing the surrounding environment.

The visual system carries out many important functions such as

- Reception of light and
- Formation of monocular representations;
- Building up of binocular perception from a pair of two dimensional projections;
- Identification and categorization of visual objects;
- Assessment of distances between objects; and adjusting body movements in relation to the objects seen.

Visual perception is the psychological processing of visual information. If visual perception is impaired it is regarded as blindness. Vision is generated by photoreceptors present in the neurosensitive layer of retina. The information generated leaves the retina by way of the optic nerve, and only the nasal fibres cross at the optic chiasm, whereas the temporal fibres passes as such. Leaving the optic chiasm, the informations are carried via the optic tract and to the lateral geniculate nucleus (LGN), where all the axons synapses. From there, the LGN axons as the optic radiations,

radiate through the deep white matter of the brain, which will ultimately reach the primary and association visual cortex.

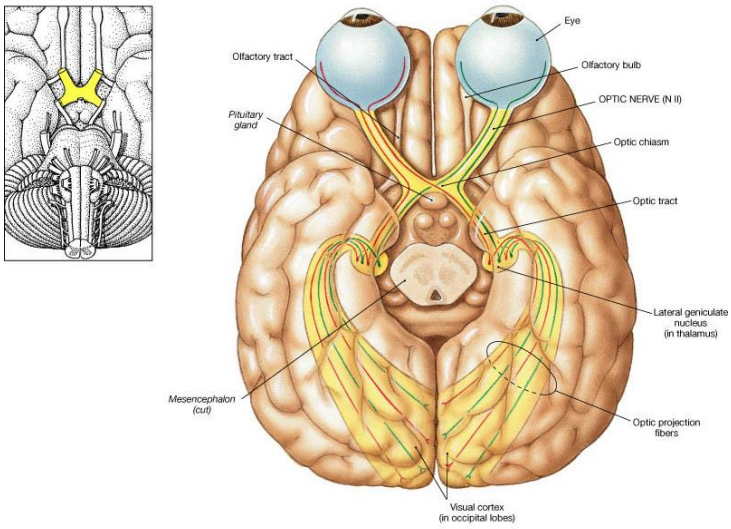


Figure 8: Visual Pathway

According to the International Classification of Diseases -10, there are 4 levels of visual function:

1. Normal vision
2. Moderate visual impairment
3. Severe visual impairment
4. Blindness.

The term “low vision” refers to group having moderate visual impairment combined with severe visual impairment. Low vision and blindness represents all visual impairment.

Major causes of visual impairment

Globally the major causes of visual impairment are:

- Uncorrected refractive errors (myopia, hyperopia or astigmatism), 43 %
- Unoperated cataract, 33%
- Glaucoma, 2%.

According to statistical analysis by World Health Organization and its fact sheets updated in 2014,

- ✓ Around 285 million people worldwide are estimated to be visually impaired, among which 39 million people are blind and 246 million people have low vision.
- ✓ Uncorrected refractive errors are the main cause of moderate and severe visual impairment globally; the leading cause of blindness in middle- and low-income countries remains to be cataract.
- ✓ About 90% of the visually impaired population worldwide belong to low-income group.
- ✓ 82% of people living with blindness are in the age group of 50 and above.
- ✓ The number of visually impaired people affected from infectious diseases has reduced significantly in the last 20 years according to global estimates.

- ✓ The most important fact is that 80% of all visual impairment can be prevented or cured.

It is estimated that there are 1.4 million children with blindness in the world. Two thirds of them live in developing countries and among them 2, 70,000 live in India. The prevalence of refractive error in this study population was 7.03% in 2013 (36), in New Delhi its 6.4 %.in 2002 (37) , in Lucknow 7.4% (38) and in Tamil Nadu 7% (39). Myopia found the most common type which constitutes for 69.72% of the refractive errors. 22.02% people are having myopic astigmatism and 8.25% of the cases have hypermetropia.

Binocular vision:

According to **Rahul Bhola et al** says that binocular vision is one of the hallmarks of the human race that has given it a supreme position in the hierarchy of the animal kingdom. With the normal alignment of the both eyes, binocular vision is an asset. But when the alignment is lost, it becomes a liability. Binocular Single Vision may be defined as the state of simultaneous vision, which is achieved by the coordinated use of both eyes, so that separate and slightly dissimilar images arising in each eye are appreciated as a single image by the process of fusion. Thus binocular vision implies fusion of visual informations from both the eyes for the perception of single visual image.[34]

Ocular Dominance:

As we have a dominant hand which is preferred to write, or a dominant foot for kicking a ball, the concept of having a dominant eye has been around for a very long time

Coren and Kaplan's defining criteria are

(a) The eye with better contrast sensitivity, visual acuity or other measure of visual functioning.

(b) The dominant eye will be most often the eye in which a rivalling stimulus is perceived, and

(c) The eye with which one looks at a distant object through a ring held in both hands at arm's length with both eyes open is used for sighting. [26]

In 1903,it was Rosenbach who discovered that even though each of the two eyes in isolation may provide equal vision, most people have a dominant eye. **Guntram Kommerell et al** tested with a simple sighting test in which subjects are requested to aim with one of their index fingers at a distant target, with both eyes open. Since the finger is imaged outside Panum's area, it appears doubled.

In 1927, Hillemanns confirmed Rosenbach's finding by doing a study on non-strabismic subjects. In his study about 40% showed a dominance of the right and about 20% of the left eye.

In 1958, Sachsenweger presented two targets at a stereo-disparity of 7' in the midline between the two eyes. Sachsenweger quantified the prevalence of one eye over the other by adjusting the position of the two targets until the subject perceived them aligned.

In1994, Lang proposed that partial suppression of the one eye is the reason for predominance of the other eye which renders double images at the border of Panum's areas.

In 1995, Haase on the other hand, suggested that prevalence of one eye is due to fixation disparity that is a small deviation of the other eye from the fixation point. In patients who suffer from eye strain, it can be eliminated by phoria-correcting prisms.

Clinical significance of Ocular dominance:

Ocular dominance is significant in general public, especially by those who have to use a monocular instrument, for example, a microscope. It is also more important in those who have participated in activities where one eye needs to be selected over the other, such as in shooting or archery.

Einat Shneor et al found that ocular dominance is not only useful for monocular tasks, but the dominant eye is also for binocular tasks. Images of dominant eye seem to be more salient than those of the non-dominant eye.[24]

In day today life Ocular dominance is more important in the field of sports and ophthalmic refractive/surgical intervention. For reliable and accurate shooting it is important to ensure that the eye which is over the rib of the gun is fixed on the target[19]

Knowing the dominant eye, an athlete adopt better strategies for improving athletic performance. Most athletes know their dominant hand and foot, and they adjust body movements accordingly. But many are unaware of that their dominant eye may process visual information more fully and

accurately than their non-dominant eye. An athlete can achieve better head and eye positioning according to their ocular dominance which helps to interpret fast action in sports such as basketball or baseball.[20]

Some individuals are cross-dominant, that is a right-handed person being left-eye dominant or a left-handed person being right-eye dominant. This may a serious disadvantage in sports such as archery and target shooting where one side of the body is used to both aim and shoot.[20]

The evaluation of ocular dominance is more important in clinical decision-making process when considering certain ophthalmic refractive and surgical interventions. **Pointer et al also** says that the most common such clinical scenario is the likelihood of adaptation to monovision correction, where one eye is corrected for distance vision and the other eye is in corrected for near vision.[22]

Determination of Ocular Dominance:

1) The Miles test:

In 1928, Miles W.R established the basis of determination of ocular dominance.

i. Ask the observer to extend both the arms and to bring both hands together to create a small triangular opening between both hands, then with both eyes open views a distant object through the opening.

ii. Then the observer should close the eyes alternatively and slowly draw the opening back to the head to determine which eye is

viewing the object. The eye with which the observer can view is the dominant eye.

2) The Porta test:

i. Ask the observer to extend one arm, then with both eyes open aligns the thumb or index finger with a distant object. Then the observer should close the eyes alternatively and slowly draw the thumb/finger back to the head to determine which eye is viewing the object. The eye with which the observer can view is the dominant eye.

3) The Dolman method (Hole-in-the-card test):

i. A card with a small hole in the middle is given to the subject and instructed to hold it with both hands. Then ask the subject to view a distant object through the hole with both eyes open. Then the observer should close the eyes alternatively and slowly draw the opening back to the head to determine which eye is

viewing the object. The eye with which the observer can view is the dominant eye.

4) Lens fogging technique:

   i. The subject is asked to fix a distant object with both eyes open and appropriate correction in place. Then in front of each eye, a +2.00 or +2.50 lens is alternately introduced, which blurs the distant object. The subject is then asked to state in which eye the blurring is more noticeable and that eye is considered as the dominant eye.

5) The convergence near-point test:

   a. The subject fixates an object that is moved toward the nose until divergence of one eye occurs (i.e. the non-dominant eye). It is an objective test of ocular dominance.

6) A dichoptic motion coherence threshold test is a quantitative test which indicates ocular dominance.

7) Certain stereograms.

8) The pinhole test.

9) The ring test.

10) Ask the subjest to look at the nose tip to notice nearsight dominance.

The association between ocular dominance and refraction were investigated by **Eser L et al**. They found right and left eyes were dominance

in 67% and 33% of patients, respectively. Results showed that males had a higher right eye dominance (70%) than females (65%) with a mean cycloplegic spherical equivalent refraction (SE) of -2.12 diopters (D) and -2.38 D, respectively. This study revealed that the hole-in-the-card dominance test is a method that is easy to perform for both patients and clinicians.

Melissa L. Rice et al studied the ocular dominance with a near ocular dominance test modelled after the distance hole-in-the card test and with four other test of ocular dominance. Their study assessed both test-retest reliability of all the four tests of ocular dominance and agreement between tests. They found that there was excellent test-retest reliability of each ocular dominance test, and there was moderate agreement between tests.[35]

Among the whole population, approximately two-thirds is right eye dominant, however in a small portion of the population, neither eye is dominant. **Jagadhamba Ashwathapa et al** says that dominance is found to change depending upon direction of gaze due to image size changes on the retinas. The eye that is preferred for sighting does not indicate handedness. This is not surprising since signals from each eye projects to both cerebral hemispheres whereas each hand is represented mainly in the opposite hemisphere.[16]

The study on ocular dominance done by **Audrey Chia et al** suggested that 65% of subjects are right eye dominant , 32% are left eye dominant, and no consistent preference in 8%(18). There appears to be a higher prevalence

of left-eye dominance in those with Williams–Beuren syndrome, and in patients with migraine[17]

Ocular Dominance and VEP:

Anju Thakur Jha et al studied the effect of ocular dominance on VEP by recording with LED goggle in candidates of both sex. They found that 70-75% of subjects are right eye dominant and 25-30% are left eye dominant. The latency of N75 & P100 is lesser in dominant eye when compared to nondominant eye and there is a disparity in amplitude and latency in both eyes because of ocular dominance. In this study they emphasize that before recording the VEP of any patient for evaluation of any neuro-pathological defect, determination of ocular dominance should be done.

Comparison of the P100 latency and amplitude between dominant and non dominant eye was done and analysed by **Vinodha et al**. And they found that majority of the patients are right eye dominant and on VEP analysis, it was found that P100 Latency obtained by stimulating the dominant eye was significantly shorter and Amplitude was significantly greater when compared with nondominant eye.[23]

Refractive error

Refractive error is a major public health problem worldwide and one of the most common cause of visual impairment (43% of all cause: WHO 2010)[11.] It is a remediable cause of visual impairment, with correction of significant refractive error being a priority of Vision 2020: The Right to Sight,

the World Health Organization's (WHO) Global Initiative for the Elimination of Avoidable Blindness.[12]

Uncorrected refractive error has serious economic and social effects on individuals and communities, restricting educational and employment opportunities of affected individuals.[12] **Rupert.R.A.Bourne et al** also states that it also has serious social implication for young children as it make the child drop out of school because of detrimental effect on academic performance and also on mental ability[11]. Refractive error causes visual impairment (<6/12 in the better eye) in 81.7% of children in the urban Indian population and in 61% in the rural Indian population.

Serge Resnikoff et al emphasises that Visual impairment from uncorrected refractive errors can cause immediate and long-term consequences in children and adults, such as loss of employment and educational opportunities, loss of economic status of individuals, families and societies, leading to impaired quality of life.

Factors responsible for uncorrected refractive errors:

(Serge Resnikoff et al)

1) Lack of proper awareness and recognition of the problem at all the levels, personal and family level, as well as at community and public health level;

2) Inadequacy of refractive services for testing;

3) Insufficient provision of affordable corrective lenses appropriate for refractive errror; and

4) Cultural discrepancies to compliance.

In the International statistical classification of diseases, injuries and causes of death, 10th revision (ICD-10), H54, the definition of visual impairment is based on “best-corrected” vision, i.e. visual acuity obtained with the best possible refractive correction[13]

By checking best-corrected vision, visual impairment was estimated to affect 161 million people globally in 2002, of whom 37 million were blind[2]. The main cause of blindness and low vision was considered to be cataract; but considering uncorrected refractive errors to be one among the causes, visual impairment at global level was significantly underestimated.

Global Burden of Disease Study 2013 quotes that the individuals suffering from visual impairment, increased from 518·6 million in 1990 to 774·1 million in 2013, taking the availability of visual aids into account. In 2013, of this total 72·4% was uncorrected presbyopia. In 1990 there were 137·0 million individuals with moderate or severe vision loss has increased to 178·8 million in 2013 excluding presbyopia. And there was an increase in number of blind people from 23·1 million to 33·0 million over the same period. The most important cause of visual impairment was uncorrected refractive error accounting for over 85% prevalence of all cases and 56% of YLDs.

Types of refractive error:

Refractive errors are most commonly four types such as

1) Myopia:

a) Simple

b) Acquired

c) Noctural

d) Pseudomyopia

e) Pathological

2) Hyperopia:

a) Axial

b) Curvatural

c) Index

d) Displacement of lens

e) Absence of lens

3) Astigmatism:

a) Regular

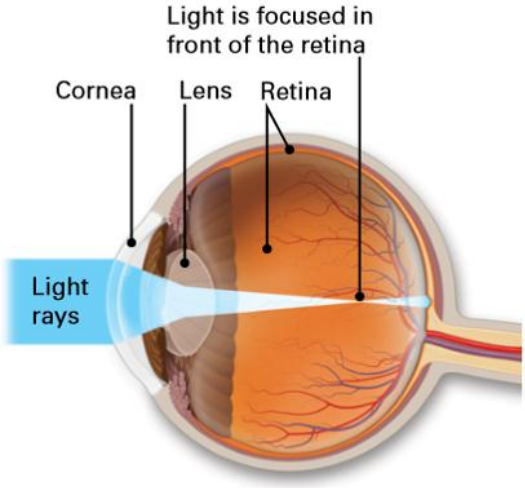
b) Irregular

4) Presbyopia

Pathophysiology of refractive error:

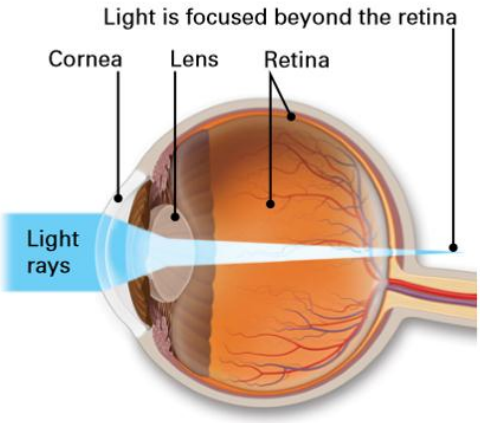
Myopia (nearsightedness): A type of refractive error where objects far away appear blurred, while objects nearby appear clearly. In myopic patients,

images comes to focus in front of the neurosensitive layer retina instead of on the retina.



In myopia, the eye is too long or the cornea is too steep. Distant objects appear blurry because images focus in front of the retina instead of on it.

Hyperopia (farsightedness): A condition where objects far away appear clearly, while objects nearby appear blurred. In Hyperopic individuals, images comes to focus behind the neurosensitive layer retina instead of on the retina.



In hyperopia, the eye is too short. Close objects appear blurry because images focus beyond the retina.

Astigmatism is a condition in which the visual disturbances resuls from an irregularly curved cornea, and where the eye does not focus light evenly onto the the light-sensitive layer of retina.

Presbyopia is an age-related condition in which the lens can no longer change its shape adequately to allow the eye to focus close objects clearly.

Symptoms:

- Blurred vision (most common)
- Haziness
- Double vision
- Eye strain
- Headaches
- Glare or halos around bright lights
- Squinting

Complications [33]:

1) Retinal detachment:

Myopia is a definite risk factor for retinal detachment and is more common in younger individuals. The risk increases with higher degrees of myopia[44]

Pathophysiology of RD in Myopia

Severe Myopia (above 5 – 6 diopters)

Excessive stretching

Retina more thinned out

Posterior vitreous detachment

Retinal breaks

Equatorial separation of retinal layer

Retinal Detachment

[Equatorial type]

In High Myopia / Pathological Myopia, severity in disease leads to formation of macular holes. It is usually limited to posterior pole within the temporal vascular arcades. These changes will lead to retinal detachment of macular type.[46]

2) Cataract formation:

Myopia when left untreated leads to the formation of

i. Posterior subcapsular cataract (PSC) ,

ii. Cortical and Nuclear cataract.

iii. myopia has been linked

iv. to damage of rod outer segments and increased

v. production of potentially cataractogenic lipid peroxida-

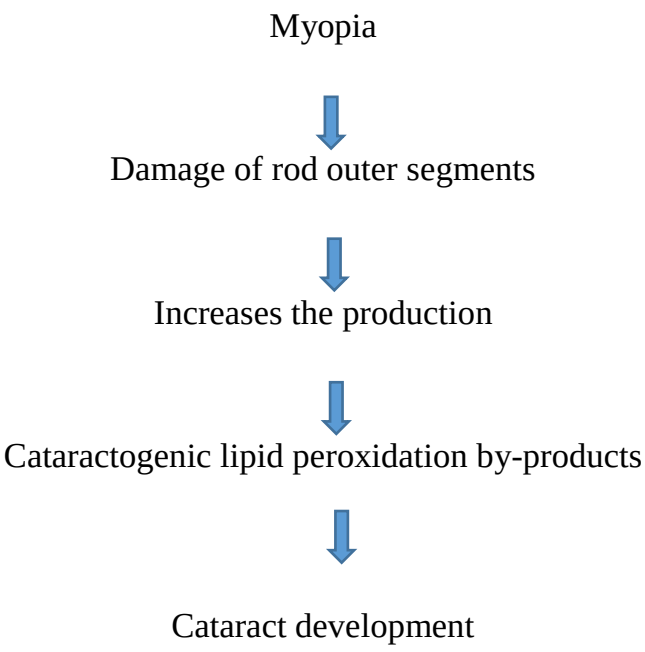
vi. tion by-products

vii. myopia has been linked

viii. to damage of rod outer segments and increased

ix. production of potentially cataractogenic lipid peroxida-

x. tion by-products



High myopia leads more commonly to nuclear cataract. The myopia and cataract relationship had been found in opposite direction also and the progression of opacity in the lens nucleus may initiate the development of myopia.

3) Glaucoma:

Myopic eyes have many structural difference when compared with emmetropic eyes. Myopic eyes axial length will be longer and the vitreous chamber will be deep.

Myopic Eyes

Increased axial length

Higher Cup–Disc Ratios (CDRS)

Increased optic nerve fibre layer defects

Greater deformability of the lamina cribrosa

Glaucomatous optic disc changes

Myopic eyes are structurally different from emmetropic eyes: myop ic eyes have longer axial lengths and vitreous chamber depths (Scott and Grosvenor, 1993). Eyes with increased axial length appear to have higher cup–disc ratios (CDRs), increased optic nerve fibre layer defects and possibly greater deformability of the lamina crib-rosa, leading to higher susceptibility to glaucomatous optic disc changes.

4) Chorioretinal abnormalities:

Mechanical stretching and thinning of the choroid and retinal pigment epithelium leads to concomitant vascular and degenerative changes. These

changes occurs mainly due to the increased axial elongation in myopics. These abnormalities include lacquer cracks, retinal breaks, lattice degeneration, chorioretinal atrophy, Fuch's spot, pigmentary degeneration, posterior staphyloma.

Marr et al done a retrospective study in 112 children with age less than 10 years presenting over 3 years who were found to have high myopia (defined as one or both eyes demonstrating 6D spherical equivalent or more of myopic RE on retinoscopic examination). They concluded that high myopia in early childhood is strongly associated with systemic and ocular problems[41]. Marr et al reviewed 114 consecutive children of age under 10 years with high hypermetropia (greater than + 5.00 D) during a 5-year period and reported that high hyperopia has a similar incidence of associated ocular abnormalities as high myopia[42]

Refractive error and VEP:

Electrical response are evoked in the corresponding cortical receptive areas and in a number of subcortical relay stations on the stimulation of particular sense organs or peripheral nerves. And lot of computer techniques and averaging methods have been tried in evaluating these electrical responses. Electrophysiological responses of the nervous system in response to various stimuli has been measured as Evoked potentials.

As clearly pointed out by **Nuwer et al,** in the past half century, Evoked potentials had evolved as a challenging scientific tool and an advanced technique commonly applied in neurophysiology.

Visual evoked potentials (VEPs) reflect the electrical signaling occurring during the visual processing and are a graphic representation of the cerebral electrical potentials evoked by a defined visual stimulus and which are generated in the occipital cortex[27].

It has been one of the most important clinical tools to be developed in neurophysiological research and it is an objective method of identifying abnormalities of the afferent visual pathways.

Anju Thakur Jha et al studied the effect of refractive error on VEP by using LED goggle as stimulation source. Mean value of latency of P100 for patients with refractive error was prolonged when compared to the mean value of latency of P100 for subjects without refractive error and found to be statistically significant. And the N75-P100 amplitude difference was also a statistical decreased in the group with refractive error but not in candidates without refractive error. They also state that decreased amplitude and prolonged latency of P100 are often seen in patients with Multiple Sclerosis, Optic Neuritis, Ischemic Optic Neuropathy and so many other neuropathic diseases involving visual pathway. So while doing VEP for visual pathway evaluation, refractive error should be kept in mind to minimize false positive results[10].

The effect of introduced REs on the VEP on subjects of both gender aged 19-45 years were studied by **Collins et al** and they found a pronounced effect on the P100 component of the VEP with these introduced REs. The P100 latency was abnormally prolonged in 31% of subjects with introduced RE with the (−2.00 DS −2.00 DC × 90°) diopter lens and in 87.5% of

subjects with introduced RE with the (+2.00 DS + 2.00 DC × 90°) diopter lens. And they also found a reduction in amplitude of the P100 component, especially for the convex lenses[30].

Zislina et al studied component P100 of Pattern Reversal visual evoked potentials (PRVEPs) in patients with myopia. Their study revealed that latencies fell outside the normal limits with decreased VA (Visual Aquity). The refractive errors cause deterioration of the visual capacity to recognize objects and the reduction of VA or of the contrast of the stimulus causes prolongation of the pattern reversal visual evoked potential (PR-VEP) latencies and decreased amplitude with a reduction in VA was established in their study[31].

VEP in Myopia and Hyperopia:

P100 latency was increased and amplitude decreased with or without the correction of refractive error and this is proved by the evaluation done by **Ruchi Kothari et al** over the influence of refractory error on Pattern Reversal Visual Evoked Potential (PRVEP) recordings of a Indian patients having myopia and hypermetropia. There was significant difference in latency of P100 and amplitude of P100 between controls and myopics with glasses and its highly significant between controls and myopics without glasses. However they had an insignificant difference in P100 latency and amplitude between controls and hyperopics with glasses and in those without glasses[28].

Considering the effects of uncorrected refractive errors (RE) in a short-duration transient visual evoked potential (SD t-VEP) system and regarding their role in the objective measurement of RE, **Aashish Anand et al** study

used an automated post-signal processing algorithm, and P100 amplitude (N75 trough to P100 peak) and latency were identified. Induced hypermetropia and myopia found to be strongly correlated with both P100 amplitude and latency. Their study showed that using VEP scores, a single VEP response had a high sensitivity and specificity for discerning emmetropia.

Dobrila karlica et al performed visual evoked potential (VEP) examination in children and evaluated the possibility of best correction for hyperopia depending on the VEP results. VEP was recorded before correction and after correcting with a range of +1.0 and +6.0 d convex lens increasing with 1 d step. When the VEP curves with highest amplitude and shortest P100 wave latency were recorded, that corrective values of lens that caused it were noted. And thus they obtained the hyperopic refraction values in retinoscopy in cooperative children corresponded to the values obtained by the best VEP curve method. Hence they suggest that hyperopic correction could be objectively determined in children with developmental difficulties using VEP[33].

VEP changes and gender differences:

The influence of refractive error on Visual evoked potential (both P100 amplitude and latency) in myopic among different gender (boys and girls) also showed changes. **Vinodha.R et al** after measuring refractive error by autorefractometry, VEPs were performed by checkerboard pattern reversal stimuli system. Their results showed significant prolongation ofP100latency [P100L] in myopic subjects and reduction in P100

amplitude was found and it was also significant. But when the results were compared separately between boys and girls, reduction in amplitude was observed significantly in boys whereas insignificant reduction in amplitude for girls. Pearson's correlation study revealed negative correlation for VEP-L and weak positive correlation for VEP-amplitude.

Ruby Sharma et al determined the normative values and investigated the effect of gender difference and anthropometric parameters on visual evoked potentials. Results showed that the N70, P100 and N145 latencies were significantly longer in males when compared to females and the P100 amplitude was higher in females in both left and right eye as compared to males.

VEP in correlation with Ocular Dominance and Refractive error:

The correlation between Ocular Dominance and Refractive error done by **Ching Yu Cheng et al**, obviously gives the impression that the dominant eye, when compared to nondominant eye, had a greater myopic refractive error and longer axial length, especially in subjects with higher amounts of anisometropia. When anisometropia exceeds beyond 1.75 D, the dominant eye was always more myopic than the nondominant eye. And thus assessing ocular dominance in patients helps to assess the efficacy of myopia interventions [21].

Quing Wang et al studied association between ocular dominance and degree of myopia in patients (1771 young myopia cases) with anisometropia and also investigated visual evoked potential (VEP) in high anisometropias. They found that there was no significant difference between dominant and

nondominant eyes in patients with anisometropia 1.0–1.75 D. But in patients with anisometropia ≥1.75 D group, the degree of myopia was significantly higher in nondominant eyes than in dominant eyes. This change was more significant in anisometropia ≥2.5 D group. Hence their study reveals that nondominant eye had a tendency of higher refraction, with the increase of anisometropia, and in high anisometropias N75 wave latency of nondominant eye was longer than that of dominant eye.

Vinodha et al sought to analyze the correlation between ocular dominance and anisometropic myopia. After measuring refractive error by autorefractometry, Ocular dominance was tested by miles test and then VEPs (Visual Evoked Potentials) were recorded by checker board pattern reversal stimuli system. And they have confirmed that there was no significant association between ocular dominance and anisometric myopia with the mean interocular difference of 0.4897D and also in anisometropia of <0.5D to >0.5D[43]

Currently methods of electro neurophysiology has gained so much importance in clinical practice. The visual evoked potential (VEP) is one among those non-invasive electro neurophysiological technique which has got growing clinical significance and advantages in evaluating clinical disorders. Evoked potentials are that, when the retina is stimulated with light, the bioelectric signals are generated in the striate and extrastriate cortex which can be recorded from the scalp electrodes.

Review of literatures highlights that visual evoked potentials are definitely influenced by ocular dominance by reduction in latencies meaning

the rapid conduction in visual pathway. Studies conclude that refractive error definitely affect the evoked potentials by prolongation of latencies. The visual evoked potentials in correlation with ocular dominance and refractive error is inconclusive. So the aim of the present study is to evaluate the influence of ocular dominance and refractive error on pattern reversal visual evoked potential.

AIM AND OBJECTIVES

Aim:

To determine and analyse the effect of refractive error and ocular dominance on pattern reversal visual evoked potentials.

Objectives:

1) To compare the latencies and amplitude of P100 and N75 of both eyes in control group, myopic and hypermetropic patients.

2) To evaluate the difference in P100 latency of both eyes between myopic and hypermetropic patients.

3) To assess the prevalence of ocular dominance in study population

4) To compare the P100 latency and amplitude between dominant eye and non – dominant eye in study population

5) To compare the P100 latency and amplitude between dominant eye and non – dominant eye of myopic and hypermetropic patients.

MATERIALS AND METHODS

This study has been conducted after getting approval from the Institutional Ethics Committee, Chengalpattu Medical College, Chengalpattu

Study Design

Visual evoked potentials are recorded in patients with Refractive error who are included in the study group and compared with the normal subjects.

Type of study :

Cross-sectional study.

Place of study:

Neurophysiology Lab,
Department of Physiology,
Chengalpattu Medical College, Chengalpattu.

Duration of study:

June 2016 to June 2017(12 months)

Study population:

Study group

Study group consists of eighty patients with a clinical diagnosis of Refractive error (forty one Myopics and thirty nine Hypermetropics). The patients were recruited from the Outpatient department of Ophthalmology, Chengalpattu Government Hospital, Chengalpattu. All the participants were informed about the study and written consent was obtained from them. The

study was conducted in the Department of Physiology, Chengalpattu Medical College, Chengalpattu. The age of the study group subjects ranged between 18 - 45 years. The diagnosis of Refractive error was made based on the Autorefractometer.

Control Group

Control group consists of forty five corresponding age matched normal subjects.

Inclusion criteria:

- Age group : 18 - 45yrs of both gender
- Patients with Refractive error (diagnosed within 2 years)
- Subjects with normal visual acuity

Exclusion criteria:

- H/O ocular surgery
- H/O color-blindness.
- H/O seizures.
- H/o Diabetes mellitus , hypertension or any cardiovascular illness
- H/O CVA, demyelinating diseases (multiple sclerosis)
- H/O Cataract & Glaucoma
- Abnormality in retina and of optic changes
- Proliferative diabetic retinopathy
- Known Smoker / Alcoholic /tobacco chewing
- Subjects taking long-term medications which affect vision like barbiturates, aminoglycosides, frusemide, miotics and mydriatics.

History Taking:

History of the Refractive error patients regarding their age, occupation, socioeconomic status, educational status, family history, duration of disease, mode of onset of symptoms, treatment, history of diabetes mellitus, and for visual problems. Similar history was also elicited in normal subjects

Clinical Examination:

For both the study and control group vision was assessed by testing Height, weight, blood pressure were measured. BMI were calculated using Quetelets index (weight in kg/ height in m^2)

Field of vision by perimetry,

Colour vision by Ishihara's chart,

Visual acuity using Snellen's chart and Autorefractometer, refractive power of both eyes were assessed.

Testing for Ocular Dominance:

Miles test:

1. The observer is made to extend both arms in front.
2. Asked to bring both hands together to create a small opening in front of the eyes.
3. Then both eyes are made open to view a distant object through the opening.

4. The observer then made to alternatively close the eyes or slowly draws opening back to the head to determine which eye is viewing the object (i.e. the dominant eye).
5. The other eye is considered to be non- dominant eye.

Figure 9 : Miles test:

Patient Evaluation:

1. Patients and controls were explained about the test to ensure full co-operation.

2. Subjects were asked to avoid hair spray or oil after the last hair wash.

3. Subjects were asked to remove their usual glasses for refractive errors if any during the test.

4. Subjects must be alert and awake during the test.

5. Results of ophthalmological examination like visual acuity, field of vision and colour vision, pupillary symmetry were reviewed before starting the test.

6. Subjects were instructed to avoid any miotics or mydriatics 12 hours before the test.

7. Gaze fixation: Unlike the recording of auditory and somatosensory potentials, pattern VEP studies require active cooperation of the subject, who is usually instructed to gaze at a dot placed in the centre of the pattern, for full-field stimulation. The most elaborate means of controlling fixation is to reject all sweeps contaminated by eye movement potentials.

Evoked Potentials :

The VEP testing was performed using Physiopac (Neuroperfect EMG-2000)

Figure 10: VEP Recorder -Physiopac (Neuroperfect Emg-2000)

- ❖ Electrodes pick up the electrical signals.
- ❖ Differential input of amplifier deletes noise sources common to active & reference inputs but passes those that are different at the two inputs.
- ❖ Isolated ground serves as reference to differential inputs which improves isolation mode rejection ratio.
- ❖ Synchronous switch prevents the stimulus artifact.
- ❖ Gain stage amplifier increases the signal to convenient amplitude.
- ❖ Filters separate the excess noise from signal.
- ❖ Sampling (sample & hold) circuit captures & freezes the signals at frequent intervals.
- ❖ Digitizer converts continuously variable analog signals to a number proportional to its amplitude.
- ❖ Storage provided for digital values needed for single sweep.
- ❖ Averager (10a) adds all the sweeps together and it scales (10b) the results.
- ❖ Data are displayed (along with graticule, cursors, and numerical readouts for certain values).
- ❖ A speaker provides the auditory representation of signal.
- ❖ Timing circuitry generates the start-of-sweep, amplifier switch control signals and start-of-stimulus.
- ❖ Stimulus generator produces auditory or visual stimulus, electric shock as appropriate.

Electrodes :

Standard disc EEG silver – silver chloride surface electrodes were used

for both VEP recording. Skin should be prepared by abrading and degreasing. Electrode paste is used to fix the electrodes in position. It conducts electrical potential and reduces movement artifacts by stabilizing the electrode. Electrode impedance should be kept below 5 KΩ.

Figure 11: VEP Monitor Displaying Checker Board Pattern

VEP Recording :

I) Electode placement:

10 – 20 International system of EEG electrode placement:

* Active electrode (Oz) is placed 3cm above inion.

* Reference electrode (Fz) is placed 12cm above nasion.

* Ground electrode (Cz) is placed over the forehead.

II) Settings:

1. Amplification range: 20,000 – 1,00,000
2. Low cut filter: 2 Hz
3. High cut filter: 100 Hz
4. Sweep speed: 350 ms

5. Sweep duration: 20 ms/D
6. Sweep sensitivity: 10 μV
7. Number of epochs: 200
8. Distance between subject's eye and screen: 100 cm

Recording Of VEP:

It normally consists of 3 waves

- The transient VEPs consists of series of waveforms of opposite polarity
- N denotes the negative wave form
- P denotes the positive wave form
- Commonly used waveforms are N75,P100,N145
- The peak latencies and peak to peak amplitudes of these are measured
- P100 & N75 peak latency, its duration and amplitude are used for VEP analysis

RESULTS

Out of 125 population who participated in the study, 45 were control group, 41 were myopics and 39 were hypermetropic individuals. Pattern reversal VEP was done, latency and amplitude was taken for analysis

Data analysis was done using SPSS software version 20 using independent samples T-Test and paired T-Test, ANOVA and Chi square test.

P value < 0.05 was considered significant.

P value <0.01 was considered highly significant.

Table 4

Confounding Variable

Parameter	Control n=45	Myopics n=41	Hypermetropics n=39	p Value
Age	34.89	34.41	35.92	0.532
WEIGHT	63.89	64.71	65.31	0.757
HEIGHT	156.33	156.88	156.92	0.587
BMI	24.73	26.2757	26.58264	0.06
SBP	116.40	114.15	114.62	0.482
DBP	74.89	73.66	73.59	0.437

$p \leq 0.05$ is significant; $p \leq 0.01$ is highly significant; $p \leq 0.001$ very highly significant.

Table shows no statistically significant difference in control and refractive error groups regarding age distribution, height, weight, and body mass index.

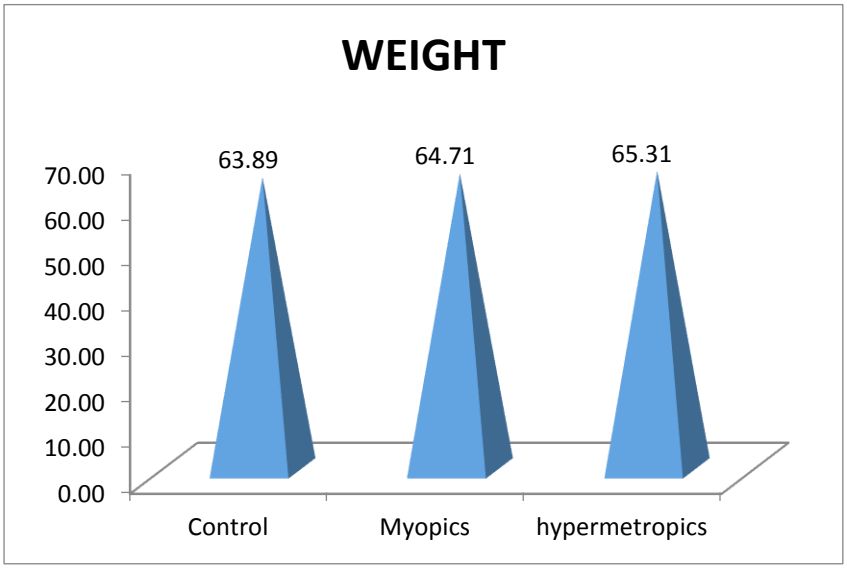


Figure 12 : Weight Distribution

Mean values of weight between three groups

In control group it is 63.89 kg, Myopics – 64.71 kg, and Hypermetropics – 65.31 kg.

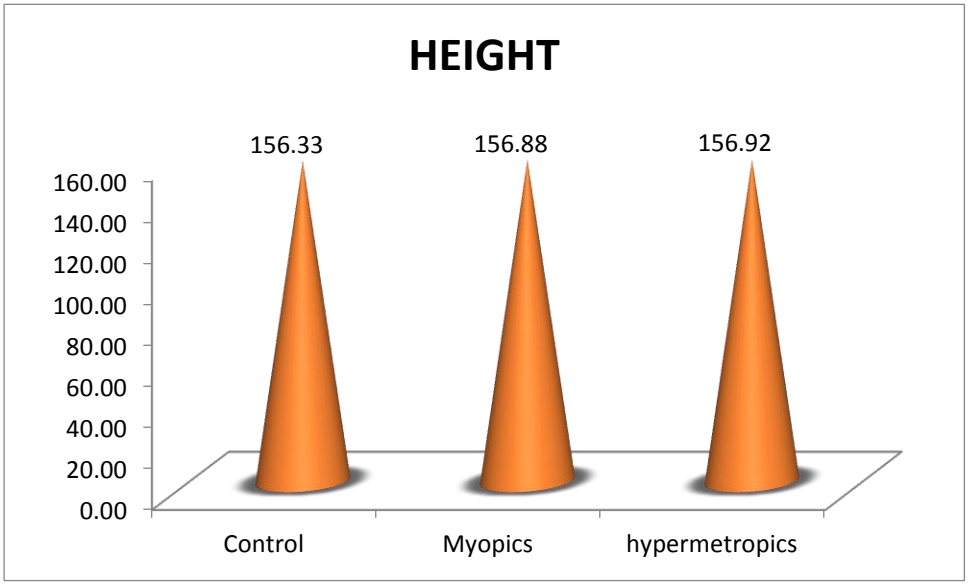


Figure 13 : Height Distribution

Mean values of height between three groups

In control it is 156.33cm, Myopics – 156.88 cm, and Hypermetropics – 156.92 cm.

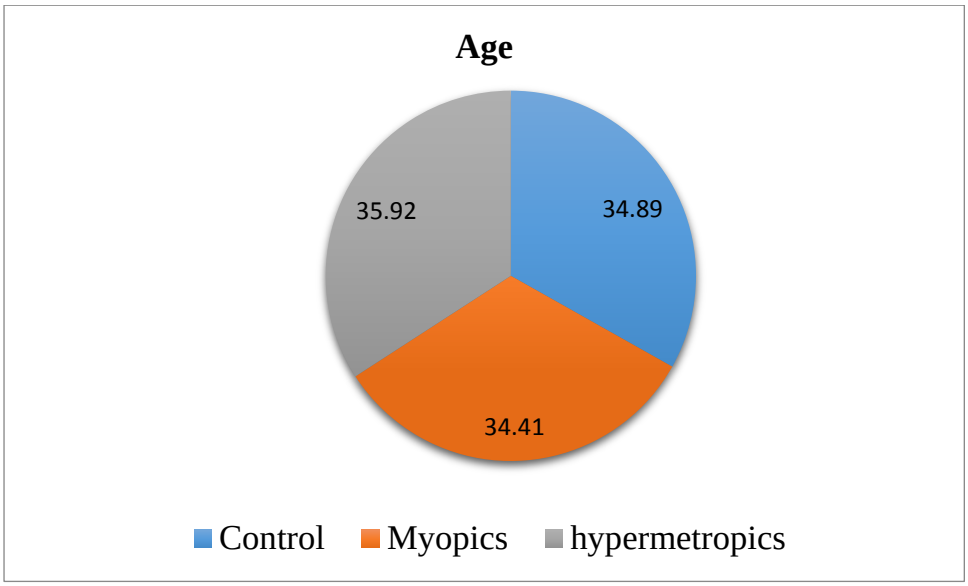


Figure 14: Age Distribution

Mean values of age between three groups

In control it is 34.89, Myopics – 34.41, and Hypermetropics – 35.92.

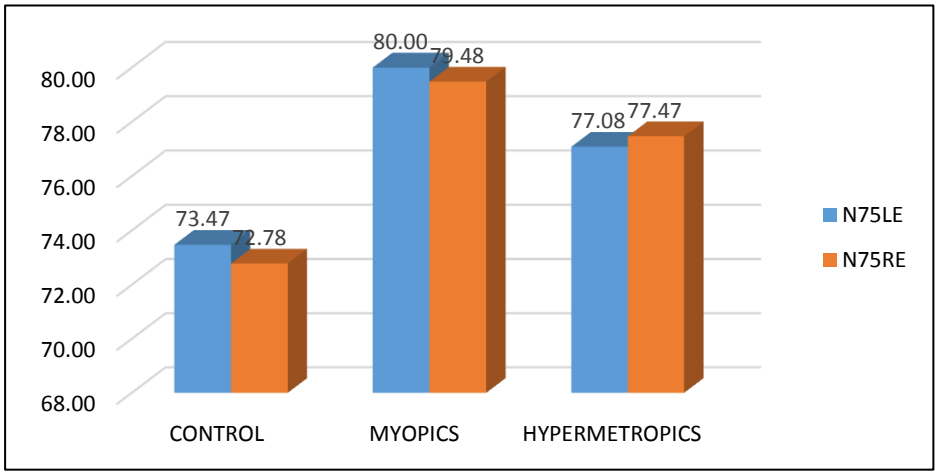


Figure 15: Comparison Of N75 Latency In Both Eyes Between Groups

The mean value of N75 latency in both eyes is compared between controls, myopics and hypermetropics and it is found that there is a highly significant (p value 0.001) prolongation of N75 latency in myopics and hypermetropics comparing with controls.

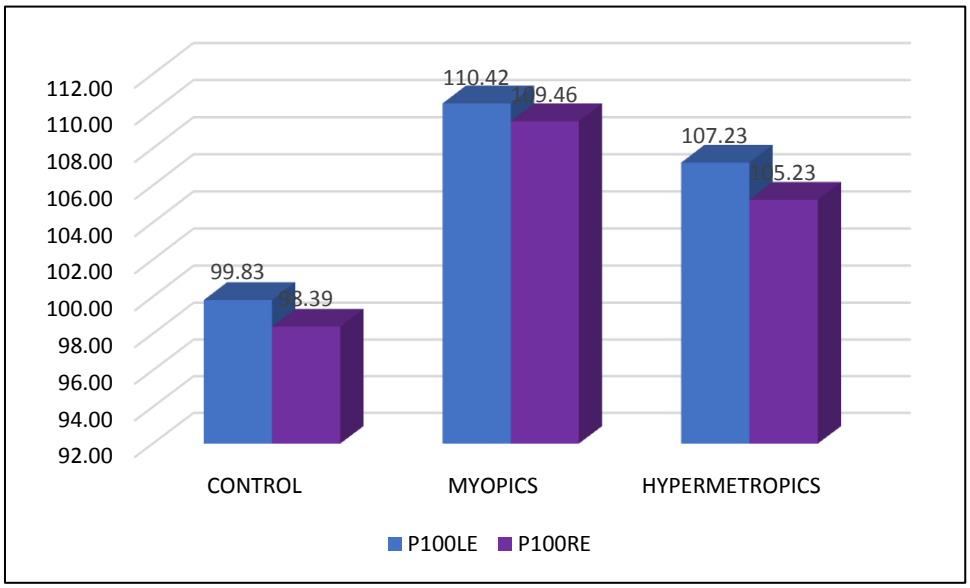


Figure 16: Comparison Of P100 Latency In Both Eyes Between Groups

Comparing P100 latency in both eyes between controls, myopics and hypermetropics it is found that there is a significant (p value 0.01) prolongation of P100 latency in myopics and hypermetropics

Table 5

Mean Latency and Amplitude Values of VEP

Parameter	Control n=45	Myopics n=41	Hypermetropics n=39	P value
	Mean ± sd	Mean ± sd	Mean ± sd	
N75 Latency	72.78 ± 8.32	79.48± 6.29	77.47± 7.87	0.01*
P100 latency	98.39 ± 8.01	109.46± 8.94	105.23± 18.61	0.0001**
N145 Latency	138.77 ± 18.52	153.54± 14.18	151.54± 16.64	0.001**
N75 Amplitude	0.96± 0.55	0.85± 0.39	0.68± 0.34	0.01*
P100 Amplitude	2.15± 1.46	1.84± 0.72	1.58± 0.54	0.03*

$p \leq 0.05$ is significant; $p \leq 0.01$ is highly significant; $p \leq 0.001$ very highly significant.

The table shows that there is significant difference in N75 latency and amplitude and P100 amplitude among controls, myopics and hypermetropics. Also there is highly significant difference in P100 latency and N145 latency among controls, myopics and hypermetropics

N75 and P100 latencies are significantly prolonged in refractive error when compared to controls. N75 and P100 amplitude is shortened significantly in refractive error when compared to controls.

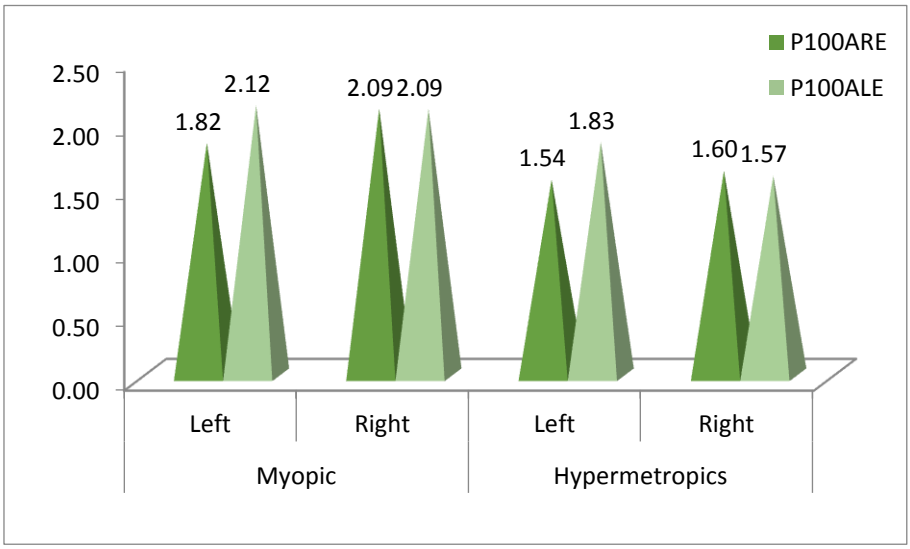


Figure 17: P100 Latency in Myopics and Hypermetropics

The graph shows insignificant difference in P100 latencies myopics and hypermetropics in both left and right eye with p value of 0.289 and 0.29 respectively.

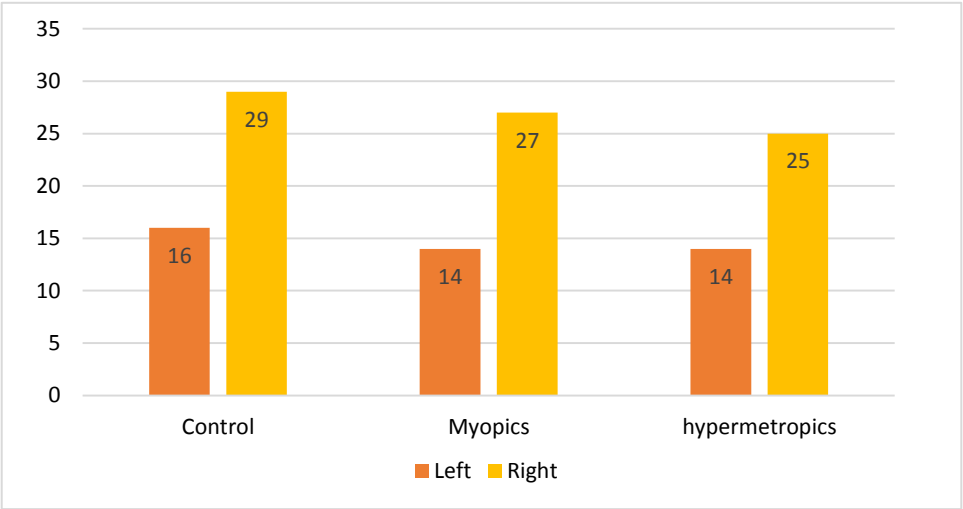


Figure 18: Prevalence of Ocular Dominance Among Groups

The graph shows that among all three groups (Controls, Myopics and Hypermetropics) the right eye dominance is more prevalent than left eye.

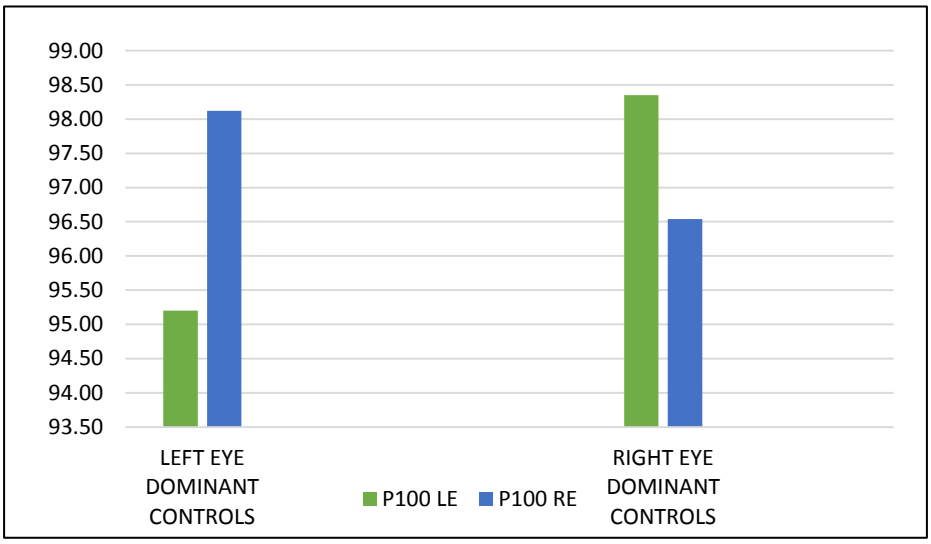


Figure 19: P100 Latency and Ocular Dominance in Control Group

The graphs shows that there is a significant decrease in P100 latency in dominant eye when compared to non-dominant eye. The p value is 0.01 in left eye dominant controls and 0.03 in right eye dominant controls.

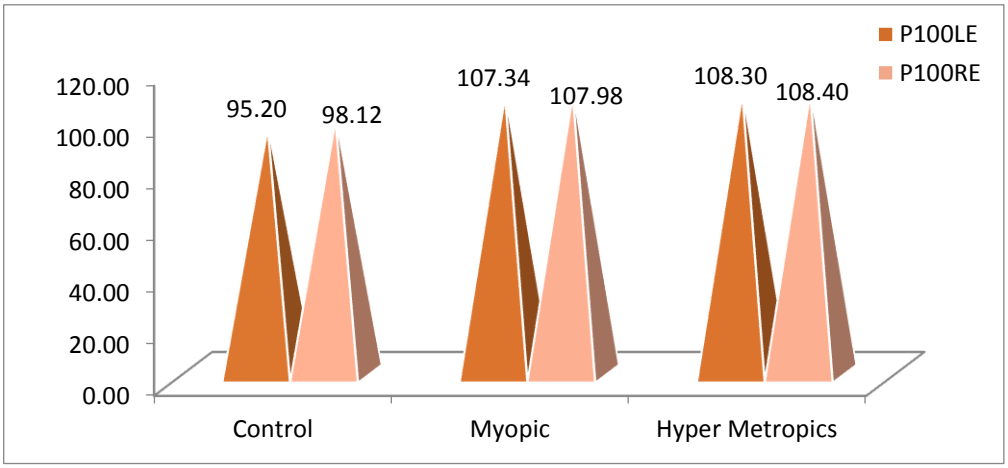


Figure 20: P100 Latency In Left Eye Dominant Individuals

The graph showed that there is decrease in P100 latency in all three groups (Controls, Myopics and Hypermetropics) in dominant eye (left eye) when compared to non-dominant eye (right eye).

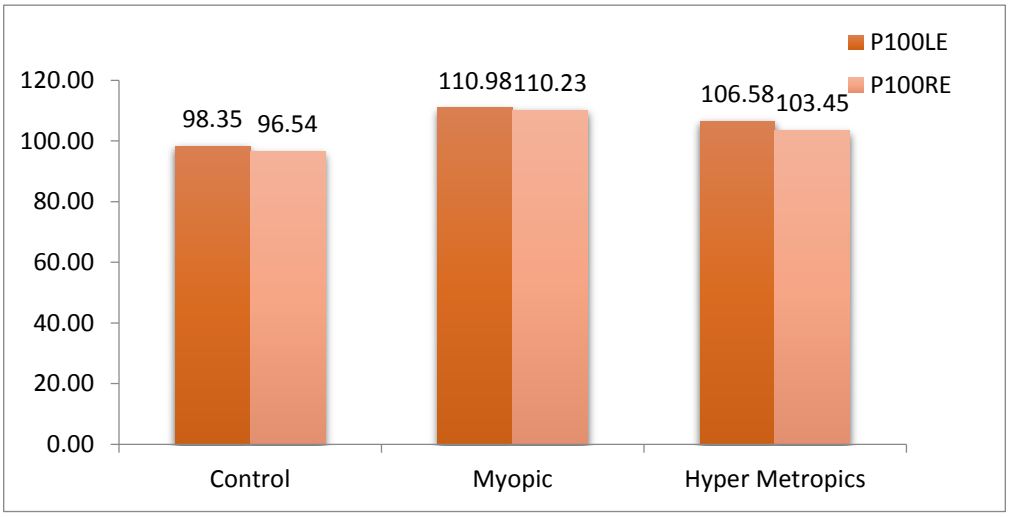


Figure 21: P100 Latency In Right Eye Dominant Individuals

The graph showed that there is decrease in P100 latency in all three groups (Controls, Myopics and Hypermetropics) in dominant eye (right eye) when compared to non-dominant eye (left eye).

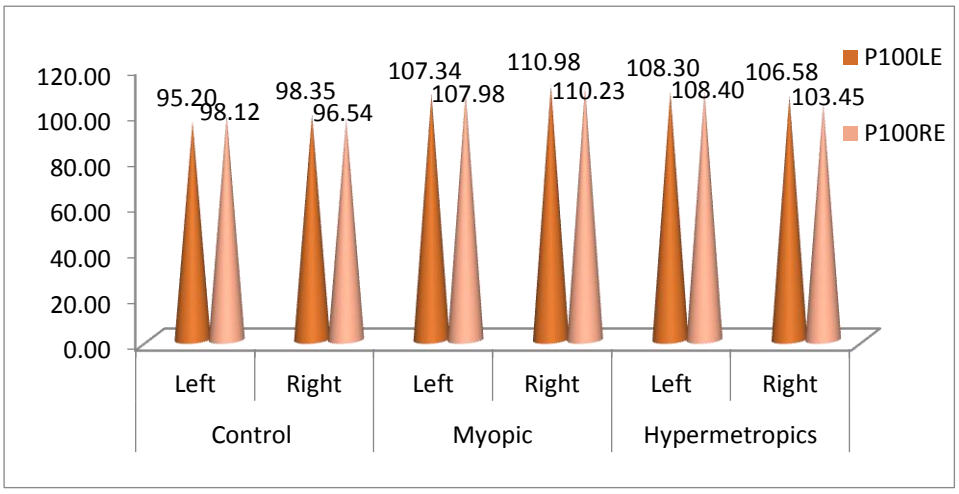


Figure 22: P100 Latency and Ocular Dominance in Refractive Error

The graph shows that there is a decrease in P100 latency in dominant eye when compared to non-dominant eye in all three groups (Controls, Myopics and Hypermetropics)

Table 6

P100 Latency and Ocular Dominance in Refractive Error

GROUP	Left Eye dominant			Right Eye dominant		
	P100 LE	**P100 RE**	**P VALUE**	**P100 LE**	**P100 RE**	**P VALUE**
Myopic	107.34	107.98	0.18	110.98	110.23	0.33
Hypermetropics	108.30	108.30	1.00	106.58	103.45	0.50

The table shows that there is a decrease in P100 latency in dominant eye when compared to non-dominant eye. It is not statistically significant in both myopic and hypermetropic group.

DISCUSSION

The visual system is one of the most important division of the central nervous system which helps in detection and interpretation of information from visible light and forms images representing the surrounding environment. The cortical visual evoked potential is an established investigating technique used for assessing the visual pathway function[45]. The pattern reversal VEP is especially used in detecting visual function disability and objective visual aquity. The pattern reversal VEP is affected by various variables like age, refractive errors, eye dominance and sex hormones etc. And we narrowed our objective to evaluate the influence of refractive error and ocular dominance on VEP[46].

The P100 waveform of VEP is generated in striate and peristriate occipital cortex not only due to activation of primary cortex but also due to thalamocortical discharge. P100 wave is one of the major discriminator between normality and abnormality of visual Pathway[10]. In refractive error the visual pathway is definitely involved since the exact focusing of the image on the neurosensitive layer of retina depends on the visual aquity of the individual.

Ocular dominance which refers to unequal functioning of two eyes, influences the visual pathway in a way that the dominant eye has visual perceptual processing priority. Vinodha et al[23] study says that P100 latency and amplitude is altered in dominant eye when compared with non-dominant eye.

In the present study VEP was done on a study population of 125, 41 were myopics , 39 were hypermetropic individuals and , 45 were age, weight, height and BMI matched healthy control group and analyzed using Neuroperfect EMG. The commonly used waveforms are N75,P100 and NI45, of which P100peak latency is the most reliable form of latency and hence P100 duration and amplitude are used for VEP analysis and there is significant prolongation of P100 latency in the study group which included myopics and hypermetropics.

The amplitude of VEP can be modified by attention, cranial shape, distribution of sulci of brain and size of brain. P100 amplitude changes occur in individuals at particular age, at 10-15 years there is decrease in amplitude then it is stabilized and remains constant up to 40 years, there after shows gradual decline to almost half of adult value at age of 25 years. Age must be considered an important factor when conducting VEP studies as it has been shown in previous studies that because of age related changes in retina and rostral part of visual system, P100 peak time increases with age.

In the present study the mean values of age showed statistically insignificant difference with mean age of control group of 34.89, in myopics it is 34.41, and hypermetropics it is 35.92. The mean values of BMI also showed statistically insignificant difference with mean BMI in control group of 24.73, in myopics it is 26.28, and in hypermetropics it is 26.58. The age groups and BMI of the subjects and the controls were matched in view of the fact that age and BMI related changes could influence the latencies in VEP.

The mean value of N75 latency in both eyes is compared between controls, myopics and hypermetropics and it is found that there is a highly significant (p value 0.001) prolongation of N75 latency in myopics and hypermetropics comparing with controls. Comparing P100 latency in both eyes between controls, myopics and hypermetropics it is found that there is a significant (p value 0.01) prolongation of P100 latency in myopics and hypermetropics. Consistent with this result, Collins et al[13] also a significant prolongation of P100 latency in refractive error when compared with controls. The principal cause for prolongation of P100 latency in myopics and hypermetropics is the degree of defocusing and the blurring of image produced by the RE.

The mean values of N75 amplitude in controls, myopics and hypermetropics 0.96, 0.85 and 0.68 repectively which showed a significant decrease in refractive error. And the mean values of P100 amplitude in controls, myopics and hypermetropics 2.15, 1.84 and 1.58 repectively which showed a significant decrease in refractive error. Anju Thakur jha et al[10], Ruchi Kothari et al[29] and Ashish anand et al study had shown statistical significant difference in latency of P100 in group with refractive error and that there is also a qualitative degradation of the VEP waveform with degradation of P100 peaks and N75 troughs with increasing refractive errors.

The amplitude of the response in a pattern reversal VEP is dependent on the visual system's ability to resolve the pattern and on the degree of retinal image focus. Even small errors of refraction tend to reduce the average amplitude of the waves of VEP.

In this study there is an insignificant difference in P100 latencies of myopics and hypermetropics in both left and right eye with p value of 0.289 and 0.29 respectively. Contrastly Ruchi Kothari et al[29] study showed that VEPs seem to be more affected by myopia than hypermetropia and the changes persisted even after application of correction and there is greater prolongation of VEP latency for small amounts of plus lens defocus than for minus and it was believed that subjects partially accommodated for minus lens. The reason behind the indifference between myopics and hypermetropics in the present study could be due to early recruitment.

Analysing the prevalence of ocular dominance among three groups, this study shows that among all three groups (Controls, Myopics and Hypermetropics) the right eye dominance is more prevalent than left eye. Li Yuan et al[48], Nida tariq et al[56] and D.Lopes-Ferreira et al[40] also got predominant right eye dominance in their study. Unanimous result had been published in the literature report that the right eye is the dominant eye in the majority of patients.

In the control group the present study shows that there is a significant decrease in P100 latency in dominant eye when compared to non-dominant eye with a p value of 0.01 in left eye dominant controls and 0.03 in right eye dominant controls. These results were consistent with Jagadamba.A et al[16] and Anju Thakur Jha et al[8] study in which significant shorter P100 latency is documented in dominant eye when compared with non- dominant eye. Lesser latency represents faster visual processing and rapid transmission of sensory information in the dominant eye.

There is a decrease in P100 latency in dominant eye when compared to non-dominant eye among all three groups but statistically insignificant among both myopic and hypermetropic group. Qing Wang et al[25] study found that there was no significant difference between dominant and nondominant eyes in patients with anisometropia 1.0–1.75 D. But in patients with anisometropia ≥1.75 D group, the degree of myopia was significantly higher in nondominant eyes than in dominant eye. It reveals that nondominant eye had a tendency of higher refraction, with the increase of anisometropia.

Whereas Ching Yu Cheng et al[21] study shows that when anisometropia exceeds beyond 1.75 D, the dominant eye was always more myopic than the nondominant eye. In the present study since the anisometropia is less than 1.75 D there was no significant difference between dominant and nondominant eyes in patients. Literatures evidences that only in case of high anisometropia, the delayed electrical activity in visual pathway might play a role in the development and progression of myopia.

VEP, a non invasive technique is used to examine the activity of visual system and the effect of nerve conduction change can be assessed by noting the waveforms of VEP [57.] The P100 latency prolongation represents the defocusing of image on to retina and therefore VEP provides an insight to the underlying refractive error and it also proves that ocular dominance does not contribute for the progression of refractive error, when there is low anisometropia. It is so obvious that there is influence of refractive error and ocular dominance on VEP. The precise and rapid sensory visual processing occuring in dominant eye is evident and clearly highlighted at an early stage

by VEP. Hence it this non-invasive and simple procedure would be mandated not only while screening for demyelinating disorders like optic neuritis and multiple sclerosis and also for early intervention to reduce the morbidity associated in visual errors.

LIMITATIONS AND FUTURE SCOPE

1. Sample size could have been increased
2. The study was cross-sectional in nature and not longitudinal which limited the ability to attribute causation.
3. The study population could have included patients with high anisometropia which would have helped in analysing the hypothesis better and with stronger evidence.
4. In future study the handedness of individual should also be considered and patients with high anisometropia also to be included.

CONCLUSION

The human eye is a precise system which comprises components that must be optimally maintained to form a clear image. Refractive error is a serious disorder with many social implication. Globally the major causes of visual impairment is uncorrected refractive errors, 43 %. An estimated 2.3 billion people worldwide have a refractive error in which about 500 million people do not have access to refractive error services who live in developing countries.

The VEP technique is a sensitive method for documenting the abnormalities in visual pathways at early stage. We narrowed our objective to study the influence of refractive error and ocular dominance on pattern reversal VEP.

The present study was done with 125 subjects including myopics, hypermetropics and normal subjects of age 18-45 years and we found a significant prolongation of N75 and P100 latency and decrease in N75 and P100 amplitude in refractive error when compared with controls. And the P100 latency in controls is decreased in dominant eye when compared to non-dominant eye, and not among myopic and hypermetropic group.

The study revealed that there is a definite changes in VEP pattern due to refractive error and ocular dominance and it also proves that ocular dominance does not contribute to the progression of refractive error, when there is low anisometropia and it should be taken in account while screening for demyelinating disorders like optic neuritis and multiple sclerosis and for early intervention and reduce the morbidity associated in visual errors.

SUMMARY

- The present study was done with125 subjects both male and female of age group 18-45 years to analyse the influence of refractive error and ocular dominance on Pattern reversal VEP.
- VEP recording was done using physiopac (Neuroperfect EMG 2000)after institutional ethical committee clearance and informed consent.
- There is a significant prolongation of N75 latency and P100 latency in both eyes and decrease in N75 and P100 amplitude in myopics and hypermetropics when compared with controls.
- Among the study population right eye dominance is more predominant.
- P100 latency in controls is decreased in dominant eye when compared to non-dominant eye among all three groups(Controls, Myopics and Hypermetropics). and but statistically insignificant decrease in both myopic and hypermetropic group.
- It proves that ocular dominance does not contribute to the progression of refractive error, when there is low anisometropia.
- Influence of refractive error and ocular dominance on VEP were analysed and results showed a strong association.
- The precise and rapid sensory visual processing occuring in dominant eye is evident and clearly highlighted at an early stage by VEP. Hence it this non-invasive and simple procedure would be mandated not only while screening for demyelinating disorders like optic neuritis and multiple sclerosis and also for early intervention to reduce the morbidity associated in visual errors.

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DATA COLLECTION FORM

Name :

Age :

Sex :

Height :

Weight :

Occupation :

Socioeconomic status :

Present complaints with duration :

Past history :

- H/O hypertension/H/O cardiovascular disease
- H.o seizures
- H/o drugintake
- H/O treatment of any eye problem

Clinical examination : Vital signs:

- General examination
- Examination of CNS
- Ophthalmological examination-

INVESTIGATION:

Investigations	Right eye	Left eye
Visual Acuity		
Hole in the card test		
Dilatation and fundoscopy		
Visual evoked potential(VEP)		

PATIENT CONSENT FORM

STUDY DETAIL :

"**INFLUENCE OF REFRACTIVE ERROR AND OCULAR DOMINANCE ON PATTERN REVERSAL VISUAL EVOKED POTENTIAL – A COMPARATIVE STUDY"**

STUDY CENTRE:

Department Of Physiology Chengalpattu Medical College , Chengalpattu.

PATIENT NAME: AGE: SEX:

IDENTIFICATION NUMBER:

I confirm that have understood the purpose of procedure for the above study.

I have the opportunity to ask question and all my questions and doubts have been answered to my satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw anytime without giving any reasons, without my legal rights being affected.

I understand that my investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study, I understand that my identity

will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arrives from the study.

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team.

I hereby give consent to participate in this study.

I hereby give permission to undergo complete clinical examination and diagnostic test.

Signature of investigator Signature/Thumb impression of participant

Date:

Place: Participant's Address:

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு : கண் உபாதைகள் உள்ளவர்களுக்கும் மற்றும் சாதாரண மனிதர்களுக்கும் ஆன கண் பார்வை திறனுக்கு உகந்த மூளையின் செயல்பாட்டு பரிமானம்.

ஆய்வு செய்யப்படும் இடம் : உடல் இயங்கில் துறை

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் வயது : பங்கு பெறுபவரின் எண் :

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விபரங்கள் எனக்கு விளக்கப்பட்டுள்ளது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கின்றேன். எந்த காரணத்தினாலோ எந்த சட்டச்சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும்போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவை இல்லை என அறிந்த கொள்கிறேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ முடிவையோ பயன்படுத்திக்கொள்ள மறுக்கமாட்டேன்.

இந்த ஆய்வில் பங்குகொள்ள ஒப்புக் கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் சாட்சியாளரின் கையொப்பம்.

இடம்: இடம்:

தேதி: தேதி:

MASTER CHART - CONTROL GROUP

Sl. No.	NAME	AGE	SEX	DOMINANT EYE	WEIGHT	HEIGHT	BMI	SBP	DBP	REF RE	REF LE	N75LE	N75RE	P100LE	P100RE	N145LE	N145RE	N75ARE	N75ALE	P100ARE	P100ALE	N75P100R	N75P100L
1	Kanniappan	40	F	Left	58	158	23	110	70	0	0	79	81	101	109	164	176	0.50	2.06	1.62	6.71	2.12	8.77
2	Gopal	33	F	Left	70	155	21	120	80	0	0	80	83	100	98	129	120	0.63	0.40	1.19	0.65	1.82	0.69
3	Nambi	34	F	Left	70	155	25	110	70	0	0	79	81	104	109	173	173	0.00	2.02	0.00	5.76	0.00	8.18
4	Dhanalakshmi	32	M	Left	65	155	27	110	70	0	0	83	86	135	108	163	124	0.69	1.17	0.44	4.14	4.31	3.12
5	Kala	27	F	Left	65	157	26	110	70	0	0	78	69	100	99	124	118	0.66	0.86	1.52	2.17	2.18	3.03
6	Shakunthala	30	F	Left	65	154	27	110	70	0	0	68	74	101	100	141	143	1.61	2.50	3.75	5.98	5.36	8.45
7	Sarasu	40	F	Left	65	156	27	110	70	0	0	69	79	100	99	138	143	0.68	1.75	3.16	3.50	3.84	5.31
8	Karigalan	30	F	Left	56	155	23	120	80	0	0	74	75	100	99	133	138	0.82	1.71	1.45	2.13	2.27	3.84
9	Kaniappan	40	F	Left	60	154	25	110	70	0	0	85	76	100	99	148	147	0.65	0.60	1.94	0.64	2.59	0.70
10	Rani	35	F	Left	65	155	27	110	70	0	0	63	80	100	99	151	143	0.15	0.76	0.95	1.91	1.10	2.67
11	Rajeshwari	29	F	Left	68	156	24	130	80	0	0	88	81	100	96	133	141	0.35	0.69	0.69	1.12	1.84	1.81
12	Vanitha	32	F	Left	64	155	22	110	70	0	0	83	78	100	76	131	98	0.39	0.16	0.02	0.95	0.41	1.11
13	Vasantha	40	F	Left	68	154	25	120	80	0	0	83	63	100	93	133	116	0.70	0.01	0.57	1.93	1.27	1.94
14	JAYA	34	M	Left	60	157	24	130	80	0	0	69	68	99	96	156	148	1.44	1.57	2.79	2.31	4.23	3.88
15	Vijaya	30	M	Left	65	160	25	110	70	0	0	88	81	100	100	136	133	0.50	0.47	0.50	1.86	1.65	2.33
16	Ramesh	45	M	Left	60	156	25	110	70	0	0	64	78	100	93	129	123	0.09	0.55	2.27	1.93	2.96	2.48
17	isakiammal	38	M	Right	60	158	24	130	80	0	0	75	80	100	100	190	156	0.05	0.00	2.14	1.64	2.79	0.64
18	Senthil	34	F	Right	60	155	25	110	70	0	0	79	75	116	98	166	181	1.76	1.25	2.03	2.39	3.79	3.64
19	Rani	24	F	Right	64	155	24	120	80	0	0	66	68	99	93	139	138	1.23	1.29	4.54	4.54	0.06	5.83
20	Muniammal	34	M	Right	62	158	25	110	70	0	0	69	65	95	95	135	138	0.40	1.05	4.66	4.71	5.06	5.16
21	Kalaiarasi	39	M	Right	68	155	24	110	70	0	0	84	82	101	101	127	122	1.52	0.82	4.80	2.99	6.32	3.81
22	Perumal	32	F	Right	60	156	25	110	70	0	0	83	83	102	106	159	158	1.08	1.23	6.06	7.02	7.14	8.25

Sl. No.	NAME	AGE	SEX	DOMINANT EYE	WEIGHT	HEIGHT	BMI	SBP	DBP	REF RE	REF LE	N75LE	N75RE	P100LE	P100RE	N145LE	N145RE	N75ARE	N75ALE	P100ARE	P100ALE	N75P100R	N75P100L
23	Kanniammal	30	M	Right	66	156	23	120	80	0	0	83	81	100	100	114	118	1.81	1.41	2.25	2.30	4.06	3.71
24	Thangam	40	f	Right	60	159	24	130	90	0	0	66	69	100	100	129	129	0.31	1.23	2.26	2.55	2.57	3.78
25	Rukumani	31	f	Right	70	156	25	120	80	0	0	71	69	100	100	146	140	1.50	1.75	3.32	3.77	4.82	5.52
26	Uthamalingam	28	F	Right	65	158	24	130	80	0	0	73	73	95	98	134	143	1.21	1.80	1.27	2.22	2.67	4.08
27	Kamalam	39	F	Right	65	155	27	110	70	0	0	73	69	98	100	141	130	1.68	1.68	0.90	1.17	2.58	2.85
28	Karthick	40	F	Right	65	160	25	110	70	0	0	71	70	99	100	144	134	1.14	2.05	0.86	1.65	2.00	3.70
29	Damayanthi	30	M	Right	70	158	28	120	80	0	0	53	58	76	81	104	105	1.87	1.96	0.36	0.71	2.23	2.67
30	Murali	35	M	Right	65	157	26	120	80	0	0	55	56	78	78	103	110	1.90	2.07	0.54	0.45	2.44	2.52
31	Vanajarani	23	M	Right	70	155	25	110	70	0	0	78	61	106	123	144	155	1.77	1.73	1.77	2.15	2.75	3.81
32	Kanaga	41	M	Right	65	156	27	130	80	0	0	65	74	108	119	150	163	1.06	1.03	1.84	2.29	2.90	3.32
33	Sherif	40	F	Right	55	156	23	120	80	0	0	71	70	100	100	140	151	0.83	0.89	2.68	3.75	3.51	4.64
34	Saritha	38	M	Right	75	160	29	110	70	0	0	76	59	100	96	123	139	0.90	0.92	1.28	0.58	2.18	1.00
35	Muneeswari	40	F	Right	56	155	23	110	80	0	0	61	79	99	93	123	130	0.44	0.77	2.34	3.08	2.78	3.85
36	Kishorekumble	35	M	Right	60	156	25	130	80	0	0	70	69	99	99	145	149	0.94	0.97	2.82	2.52	3.76	3.49
37	Rahul	36	F	Right	58	156	21	110	70	0	0	71	69	96	99	164	160	0.83	0.98	2.41	2.74	3.24	3.72
38	Uma	40	F	Right	63	155	26	110	70	0	0	68	73	100	100	130	131	0.95	0.64	2.56	2.64	3.51	3.28
39	Poonguzhali	36	F	Right	60	156	25	120	80	0	0	76	78	100	99	131	133	0.95	0.64	2.56	2.64	3.51	3.28
40	Karkuzhali	30	F	Right	70	155	23	120	80	0	0	79	50	100	100	130	133	0.57	0.65	2.23	1.87	2.80	2.52
41	Raghuvaran	36	F	Right	69	157	24	110	70	0	0	85	84	100	96	129	125	0.92	0.04	3.32	1.65	4.24	1.69
42	Andal	33	F	Right	71	156	25	110	70	0	0	69	78	95	99	151	159	1.37	1.10	1.86	2.63	3.23	3.73
43	Raji	40	M	Right	60	160	23	130	80	0	0	77	78	100	100	177	171	1.99	2.56	0.63	1.59	2.62	4.15
44	Ranmkumar	34	F	Right	60	158	24	118	80	0	0	66	65	95	96	131	134	1.32	1.35	5.51	5.72	6.86	7.04
45	Indra	43	M	Right	59	156	24	120	70	0	0	65	65	98	93	136	135	1.23	1.41	4.29	4.37	6.15	5.78

MASTER CHART - MYOPIC GROUP

Sl. No	NAME	AGE	SEX	DOMINANT EYE	WEIGHT	HEIGHT	BMI	SBP	DBP	REF RE	REF LE	N75LE	N75RE	P100LE	P100RE	N145LE	N145RE	N75ARE	N75ALE	P100ARE	P100ALE	N75P100R	N75P100L
1	Mohana	40	F	Left	60	157	24	110	80	-1.5	-2	78	74	98	96	145	126	0.84	0.29	1.95	2.89	2.77	2.55
2	Rukumani	45	F	Left	70	165	26	110	70	-1.5	-2.25	83	75	104	103	139	136	0.56	1.15	2.08	2.49	4.33	2.72
3	Vanathi	35	F	Left	69	155	29	100	70	-2.5	-3	85	86	120	110	180	161	1.23	0.96	1.87	0.00	1.98	3.46
4	Mohan	35	M	Left	65	155	27	110	70	-1.5	-1.25	76	75	109	109	145	163	0.14	0.87	2.56	3.76	3.53	2.46
5	Dhivya	27	F	Left	68	155	28	110	70	-2.25	-1.5	78	75	105	101	166	146	1.01	0.20	3.00	2.13	3.09	3.64
6	Kanaga	34	F	Left	70	158	28	100	70	-1.75	-0.75	80	75	101	101	150	149	0.90	0.52	1.52	2.71	3.22	4.42
7	Kantha	33	F	Left	65	157	26	130	70	-2.5	-1.75	81	81	110	109	149	151	0.32	0.96	2.53	2.15	3.87	1.97
8	Ammu	24	F	Left	58	154	24	140	80	-2.75	-3.5	83	78	110	106	149	149	1.54	1.06	1.80	1.57	3.35	2.21
9	Gayathri arumugam	29	F	Left	55	156	23	120	90	-1.25	-2.5	70	81	105	106	153	151	1.70	0.96	2.24	2.73	3.10	3.86
10	Vishnu priya	27	F	Left	80	155	33	110	80	-1.75	-1.5	74	80	116	115	160	158	1.73	1.42	2.47	2.93	3.13	3.33
11	Sumathi	39	F	Left	60	150	27	130	80	-2	-1.75	74	73	118	113	173	156	1.41	0.96	0.16	0.96	3.54	3.97
12	Vaishnavi	22	F	Left	70	160	27	110	70	-4	-3.25	75	84	117	119	160	158	0.78	1.85	1.42	2.42	3.58	4.04
13	Tejashwini	36	F	Left	75	157	30	120	80	-2.5	-1.75	81	80	111	117	155	153	0.68	1.04	1.00	1.95	3.23	3.10
14	Karthick	43	M	Left	65	160	25	110	70	-2.5	-1.25	76	70	108	108	160	156	0.34	0.98	0.93	0.94	3.02	2.58
15	Meenakshi	40	F	Right	60	155	25	110	80	-0.25	-1.25	78	85	113	105	153	153	0.54	1.21	2.90	0.00	4.04	4.46
16	Keerthana	28	F	Right	55	156	23	110	70	-1.75	-2.5	84	71	113	115	159	158	0.18	1.24	3.30	1.65	2.43	3.80
17	Akshaya	33	F	Right	60	155	25	100	80	-2.5	-3	81	86	111	111	151	150	0.79	0.00	0.93	1.99	2.65	2.82
18	Krithiga	28	F	Right	60	150	27	120	70	-0.75	-1.5	81	79	109	109	158	140	1.00	0.89	1.62	1.08	1.34	2.93
19	Ashwitha	27	F	Right	65	157	26	110	70	-2.5	-3.25	81	79	109	109	168	181	1.35	1.39	2.79	2.26	3.58	3.06
20	Hanisha	35	F	Right	60	160	23	120	80	-3.25	-2.75	80	76	108	109	186	165	0.62	0.84	1.53	2.49	1.11	2.14
21	Mini	30	F	Right	65	163	24	100	70	-1.75	-2.25	75	76	101	101	153	129	0.74	0.24	1.38	2.48	5.69	0.00
22	Maligai	36	F	Right	69	155	29	100	70	-2.5	-1.5	84	84	105	105	125	125	0.84	0.94	2.04	2.30	1.93	2.88

Sl. No	NAME	AGE	SEX	DOMINANT EYE	WEIGHT	HEIGHT	BMI	SBP	DBP	REF RE	REF LE	N75LE	N75RE	P100LE	P100RE	N145LE	N145RE	N75ARE	N75ALE	P100ARE	P100ALE	N75P100R	N75P100L
23	Chandra	43	F	Right	70	158	28	110	70	-2.25	-3.25	83	81	111	105	156	159	0.30	0.42	1.96	2.21	2.97	3.00
24	Manoj	38	M	Right	60	166	22	110	70	-2.5	-1.75	80	75	108	106	159	149	0.84	0.53	1.86	1.77	1.18	1.92
25	Krishna	30	M	Right	59	157	24	100	70	-1.25	-2.5	80	79	106	118	150	144	0.45	0.00	1.70	3.38	4.00	3.45
26	Kani	31	F	Right	65	154	27	120	70	-2.5	-3.25	80	80	101	101	168	154	0.65	1.39	1.74	3.24	2.25	3.31
27	Christina	25	F	Right	60	156	25	110	70	-1.75	-2.5	84	84	113	110	149	153	0.72	0.83	1.60	2.93	2.10	1.08
28	Gayathri	29	F	Right	60	156	25	130	80	-3.25	-2.25	83	80	110	109	147	149	0.61	0.62	0.75	1.82	2.86	2.83
29	Kannama	42	F	Right	60	157	24	110	70	-2.5	-3.25	83	83	109	109	148	150	0.80	0.73	1.50	2.71	2.10	3.08
30	Manjula	42	F	Right	70	155	29	120	70	-2.25	-1.5	76	81	106	106	146	148	0.98	1.11	1.74	3.35	2.32	0.00
31	Valli	43	F	Right	80	160	31	130	70	-2.5	-1.5	81	81	105	110	154	151	0.77	1.42	2.99	2.68	3.46	2.28
32	Kamala	42	F	Right	65	157	26	130	80	-1.25	-0.5	69	76	118	115	159	154	0.57	1.00	0.97	1.78	1.07	2.80
33	Chandrasekar	40	M	Right	60	155	25	120	70	-2	-1.25	79	71	118	115	156	161	1.23	1.24	0.78	1.66	3.27	2.13
34	Kavia	39	F	Right	70	158	28	110	80	-2	-1.5	81	75	107	109	166	158	1.00	1.38	3.41	2.06	4.28	2.40
35	Venugopal	44	M	Right	60	160	23	110	70	-0.75	-1.25	109	108	159	156	206	204	1.60	0.09	2.04	1.85	4.27	4.11
36	Kesavan	45	M	Right	65	158	26	130	80	-2.25	-2.5	78	85	113	105	153	153	1.00	0.64	1.54	1.19	3.30	2.53
37	Ajith	32	M	Right	70	160	27	120	70	-1.25	-2	78	78	106	104	159	158	0.64	0.69	1.86	1.50	0.40	2.20
38	Elakkiya	29	F	Right	80	160	31	110	70	-2	-3.25	78	76	108	109	170	145	0.91	1.00	1.62	1.61	1.84	2.75
39	Dhakshinamoorthy	36	M	Right	45	145	21	100	70	-3.25	-4	81	81	110	111	178	173	1.03	1.07	1.24	2.69	2.50	3.70
40	Anjana	25	F	Right	60	155	25	120	80	-2	-1.25	89	88	116	109	168	181	0.74	0.77	2.16	1.16	2.18	1.88
41	Kishore	30	M	Right	70	160	27	110	70	-1.75	-2.25	75	75	108	106	145	143	0.62	0.84	1.86	2.50	2.28	3.00

MASTER CHART - HYPERMETROPIC GROUP

Sl. No.	NAME	AGE	SEX	DOMINANT EYE	WEIGHT	HEIGHT	BMI	SBP	DBP	REF RE	REF LE	N75LE	N75RE	P100LE	P100RE	N145LE	N145RE	N75ARE	N75ALE	P100ARE	P100ALE	N75P100R	N75P100L
1	poongodi	45	F	Left	100	153	43	110	70	2.25	3	71	74	99	100	128	130	0.54	0.59	1.53	1.66	2.07	2.25
2	Madhu	32	F	Left	80	154	34	100	70	2.5	2.5	76	74	105	101	136	135	0.90	0.39	1.39	1.94	2.29	2.33
3	Ramani	39	F	Left	60	155	25	100	80	2.25	3	83	78	111	104	139	138	0.29	0.48	0.88	1.95	1.43	1.17
4	Shakunthala	37	F	Left	58	162	22	120	80	2.25	3.25	79	69	99	94	144	145	0.00	1.58	0.00	1.88	0.00	2.46
5	Kanniyama	41	F	Left	70	160	27	120	70	2.75	3	74	60	95	91	131	126	0.98	1.00	1.40	2.14	1.54	2.03
6	vasanthi	29	F	Left	65	165	24	110	80	2.75	2	71	74	99	100	128	130	0.95	1.00	1.16	1.45	3.18	2.79
7	Kalai	43	F	Left	55	156	23	120	80	2.75	3.25	76	78	119	109	158	158	0.87	0.42	1.95	2.13	3.82	3.55
8	Kavi	40	F	Left	60	158	24	120	70	2.5	2	75	75	114	113	161	160	0.71	0.31	1.40	1.67	3.11	2.98
9	Vijayalakshmi	40	F	Left	60	156	25	110	70	2.25	3.25	75	89	113	118	159	159	0.96	0.95	2.00	1.90	2.96	2.55
10	Archana	33	F	Left	60	158	24	110	80	3.25	2	75	73	113	113	159	156	1.03	1.53	2.17	1.41	2.20	3.94
11	Saraswathy	39	F	Left	69	160	27	120	70	3.25	2.75	80	74	110	114	161	164	0.54	0.80	2.06	1.76	2.60	2.56
12	Agalya	25	F	Left	65	157	26	110	80	3	2.5	84	83	113	118	166	161	0.49	1.30	2.12	1.98	1.78	0.80
13	Nandha kumar	45	M	Left	59	156	24	130	70	2.5	2.25	68	99	110	131	166	168	0.51	0.09	2.06	1.05	1.57	4.14
14	Ilakiya	29	F	Left	60	156	25	110	70	2.75	3	79	76	120	114	161	158	0.43	0.93	1.37	2.70	2.80	1.63
15	Rajeshwari	40	F	Right	65	154	27	100	70	2.5	2.5	81	80	103	108	146	154	0.62	0.58	1.60	0.96	2.22	2.54
16	Janani	28	F	Right	68	155	28	120	70	2.75	3	79	75	104	103	159	163	0.92	0.38	2.21	1.14	2.13	2.52
17	Govindan	45	M	Right	79	160	31	110	80	2.25	3.25	94	88	136	136	184	180	0.81	0.10	1.46	1.29	2.27	2.39
18	Vaibavan	28	M	Right	65	157	26	110	70	3	2.25	83	75	115	115	155	160	0.92	0.83	2.09	1.82	3.01	2.65
19	Raja	37	M	Right	60	155	25	100	70	3	2.5	70	81	105	106	153	151	0.50	0.24	1.57	2.94	2.07	2.18
20	Suseela	39	F	Right	60	154	25	110	80	2.75	2.25	74	60	95	91	131	126	0.63	0.58	1.90	1.41	1.53	1.99
21	Giri	34	M	Right	120	152	52	120	70	3	2.5	71	74	99	100	128	130	0.47	1.00	1.55	0.97	2.02	2.01
22	vijaya	34	F	Right	70	156	29	120	70	3.25	2.25	78	78	100	101	140	136	0.63	1.00	1.37	1.80	2.00	2.89
23	Kanniyamma	45	F	Right	65	157	26	110	80	3	3	74	60	95	91	131	126	0.10	0.59	1.29	1.62	3.60	2.21
24	Arun kumar	40	M	Right	60	165	22	110	70	2.75	2.5	74	74	116	105	171	138	0.47	1.00	1.25	1.50	3.00	3.03
25	Janani	30	F	Right	60	160	23	110	70	2.25	3	79	75	104	104	159	163	0.95	1.01	2.39	1.11	2.50	2.12
26	vijaya	38	F	Right	80	160	31	130	70	2.75	3	78	78	100	101	140	136	0.15	0.70	1.42	2.12	1.57	3.82

Sl. No.	NAME	AGE	SEX	DOMINANT EYE	WEIGHT	HEIGHT	BMI	SBP	DBP	REF RE	REF LE	N75LE	N75RE	P100LE	P100RE	N145LE	N145RE	N75ARE	N75ALE	P100ARE	P100ALE	N75P100R	N75P100L
27	Sarala	45	F	Right	50	156	21	110	70	2.5	3	76	77	106	102	147	146	0.32	0.96	0.75	1.82	1.07	2.80
28	Anjali	30	F	Right	48	155	20	120	70	2.75	2.25	61	81	81	101	95	136	0.92	0.58	2.10	1.64	3.09	4.22
29	Ramya	26	F	Right	60	150	27	110	80	2.5	2.5	70	75	91	99	131	120	0.74	1.02	1.16	2.53	3.00	3.55
30	Swetha	24	F	Right	64	155	27	120	70	2.75	3	75	77	108	110	160	161	0.73	1.01	1.74	1.53	3.47	3.54
31	Pushpanjali	43	F	Right	50	155	21	130	70	2.25	2.25	66	74	114	11	159	165	0.18	0.13	0.03	0.02	0.21	0.15
32	Suganthi	42	F	Right	60	155	25	100	80	2.25	3.25	80	81	111	115	165	165	0.96	0.65	2.00	2.41	3.00	3.09
33	Vinodhini	27	F	Right	59	156	24	140	80	3.25	3	84	84	114	103	180	165	0.88	1.00	2.31	1.65	2.19	2.72
34	Nandhini	29	F	Right	65	160	25	110	80	2.5	2	75	75	111	119	156	179	0.98	0.71	1.60	2.07	2.86	2.78
35	Vimala	36	F	Right	59	156	24	130	70	2.5	3	90	90	116	106	180	150	0.44	0.08	1.78	1.90	2.20	2.00
36	Agasthiya	24	F	Right	55	155	23	120	80	3	2.25	84	84	118	111	155	153	0.53	0.40	1.91	2.38	2.44	2.78
37	Kanagambal	45	F	Right	65	158	26	110	70	2.5	2.5	75	80	85	116	108	169	1.87	1.00	1.61	0.33	1.82	1.33
38	Vinoth	39	M	Right	70	158	28	110	70	3.25	2.25	93	88	131	130	180	181	0.90	0.98	1.83	1.14	3.00	2.12
39	Vani	36	F	Right	69	160	27	120	70	3	2.75	80	88	108	101	169	171	0.73	0.74	1.20	1.04	2.93	2.78