CLINICAL STUDY OF ETIOPATHOGENESIS OF ISOLATED OCULOMOTOR NERVE PALSY

DISSERTATION SUBMITTED TO

In partial fulfillment of the requirement for the degree of

M.S. DEGREE EXAMINATION OF

BRANCH III OPHTHALMOLOGY

of

THE TAMIL NADU DR. M. G. R MEDICAL UNIVERSITY

CHENNAI- 600032

DEPARTMENT OF OPHTHALMOLOGY

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI- 11

APRIL 2015

CERTIFICATE

This is to certify that this dissertation entitled **"*Clinical Study Of Etiopathogenesis Of Isolated Oculomotor Nerve Palsy*"** submitted by ***Dr. Saranya.K.V*** to the faculty of Ophthalmology ,The Tamil Nadu Dr. MGR Medical University, Chennai in partial fulfillment of the requirement for the award of M.S Degree Branch III (Ophthalmology), is a bonafide research work carried out by her under my direct supervision and guidance.

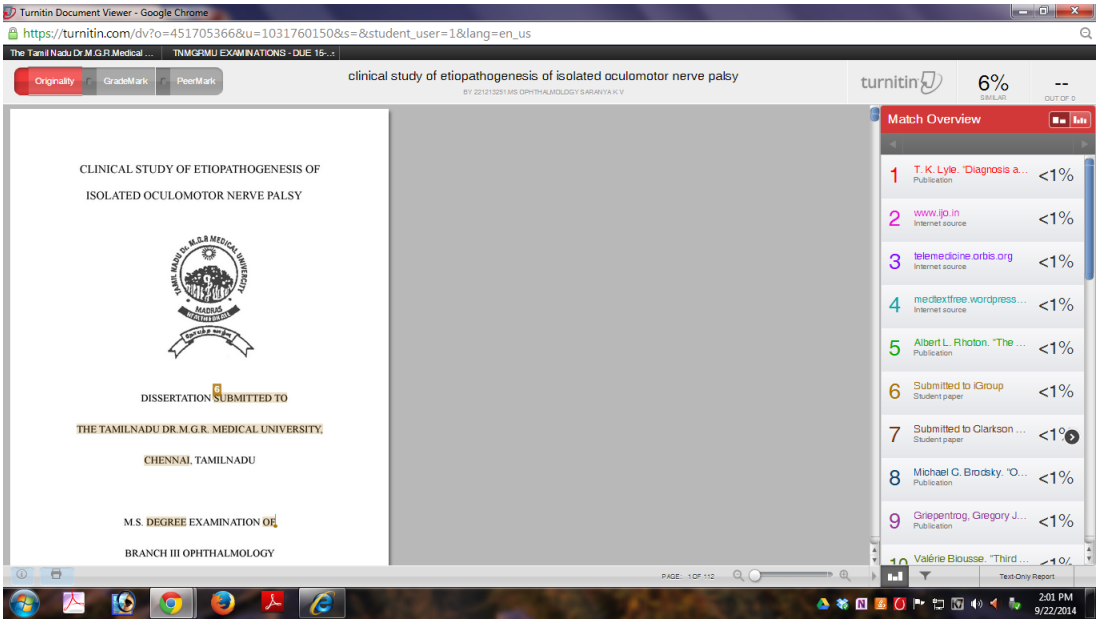
Dr. L.D.THULASI RAM MS. (Ortho)
The Dean
Tirunelveli Medical College,
Tirunelveli

Dr A.YOGESWARI.
Professor & Head of the Department
Department of Ophthalmology
Tirunelveli Medical College,
Tirunelveli.

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled ***"Clinical Study Of Etiopathogenesis Of Isolated Oculomotor Nerve Palsy"*** is a bonafide and genuine research work carried out by me under the guidance of **Dr. RITA HEPSI RANI .M**, Assistant Professor of Ophthalmology, Department of Ophthalmology, Tirunelveli Medical College, Tirunelveli

Dr. Saranya.K.V
Post Graduate In Ophthalmology,
Department Of Ophthalmology,
Tirunelveli Medical College,
Tirunelveli.


Turnitin Document Viewer - Google Chrome
https://turnitin.com/dv?o=451705366&u=1031760150&s=&student_user=1&lang=en_us
The Tamil Nadu Dr.M.G.R.Medical ...
TNMGRMU EXAMINATIONS - DUE 15-...
Originality
GradeMark
PeerMark
clinical study of etiopathogenesis of isolated oculomotor nerve palsy
BY 221213251 MS OPHTHALMOLOGY SARANYA K V
turnitin
6%
SIMILAR
--
OUT OF 0
CLINICAL STUDY OF ETIOPATHOGENESIS OF
ISOLATED OCULOMOTOR NERVE PALSY
DISSERTATION SUBMITTED TO
THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY,
CHENNAI, TAMILNADU
M.S. DEGREE EXAMINATION OF
BRANCH III OPHTHALMOLOGY
Match Overview
1 T. K. Lyle. "Diagnosis a... Publication <1%
2 www.ijo.in Internet source <1%
3 telemedicine.orbis.org Internet source <1%
4 meditextfree.wordpress... Internet source <1%
5 Albert L. Rhoton. "The ... Publication <1%
6 Submitted to iGroup Student paper <1%
7 Submitted to Clarkson ... Student paper <1%
8 Michael G. Brodsky. "O... Publication <1%
9 Griepentrog, Gregory J... Publication <1%
Text-Only Report
2:01 PM
9/22/2014

ACKNOWLEDGEMENT

I express my sincere gratitude and thanks to **The Dean,** Tirunelveli Medical College, Tirunelveli, for providing all the facilities to conduct this study.

I sincerely thank **Dr.A.Yogeswari** Professor and HOD, Dept of Ophthalmology for her valuable advice, comments and constant encouragement for the completion of this study.

I am highly thankful to **Dr.A.R.Anabarasi and Dr.Chittibabu** former HOD's ,Department of Ophthalmology, TVMCH for their valuable comments.

I am highly indebted to **Dr.R.Kumaraswamy, Dr. D.Anandhi** and **Dr. S.B.Sivathanu,** and **Dr.M.Rita Hepsi Rani,** Assistant Professors, Department of Ophthalmology , TVMCH who helped me by offering their valuable suggestions and for being with me throughout the study .

My special thanks to my Co-Postgraduate colleagues **Dr.Rohini.A. and Dr.M. Rekha Sravya** for their help and immense support.

My Special Thanks to My Teachers in Department Of Neuro medicine and Neuro surgery.

To my patients, I extend my sincere thanks for having participated in the study in spite of their illness.

I owe my thanks to **Mr.Heuber,** for his immense help in analyzing the data and preparing the manuscript.

I am also thankful to my family and friends for their constant support and encouragement.

Above all, I am thankful to The Almighty God who gave me wisdom and health to complete my postgraduate course and to make this thesis a reality.

CONTENTS

TABLES

FIGURES

ABBREVIATIONS

BSV	-	Binocular Single Vision
CCN	-	Central Caudal Nucleus
CT	-	Computed Tomography
IO	-	Inferior Oblique
IR	-	Inferior Recti
LPS	-	Levator Palpebrae Superioris
LR	-	Lateral Recti
MLF	-	Median Longitudinal Fasciculus
MR	-	Medial Recti
MRA	-	Magnetic Resonance Angiography
MRD	-	Mariginal Reflex Distance
MRI	-	Magnetic Resonance Image
NPA	-	Near Point of Accomodation
PPRF	-	Paramedian Pontine Reticular Formation
RAF	-	Royal Air Force
SO	-	Superior Oblique
SR	-	Superior Recti

INTRODUCTION

THIRD CRANIAL NERVE PALSIES may be partial or complete, congenital or acquired, isolated or accompanied by other neurological signs. They can result from lesions anywhere from the nucleus to extraocular muscles[1].

- Complete third nerve palsy present with symptoms of complete drooping of upper eyelid and diplopia as lid is elevated. Ocular signs include complete ptosis due to paralysis of levator palpabrae, exotropia due to palsy of all extraocular muscles except lateral rectus and superior oblique, mydriasis with ipsilateral loss of both direct and indirect pupillary reflexes due to paralysis of sphincter pupillae.
- A partial form or paresis causes incomplete involvement of muscles.
- Superior division may be involved alone resulting in double elevator palsy with true or pseudo ptosis.
- Inferior division may be involved alone sparing superior rectus and levator palpabrae.[2]

The clinical course of occulomotor nerve palsy mainly depends on the etiology. The commonest etiology is ischemia due to diabetes mellitus, which is usually pupillary sparing and resolves completely with

in 3 months .Compressive or traumatic third nerve palsy have an indolent course worsening slowly with or without simultaneous features of aberrant regeneration and resolves incompletely over 6 month[2].

HISTORY

- ✓ In ancient mythology, condition of squint considered an affliction sent by an angry god or devil spirit.
- ✓ The word ‘strabismus’ was derived from aprominent geographer from Greece called‘ ’Strabo’’ who had peculiarly prominent squint[3]
- ✓ In early times treatment was fanciful. In Eberyus papyrus it is referred that “turning of eyes” should be treated with brain of tortoise and oriental spices[3]
- ✓ Hippocrates differentiates paralytic from concomitant squint[3].
- ✓ Paul of Aegina was first to rationally treat the squint by advising to wear perforated mask between eyes to tempt them look straight[3]
- ✓ George bartisch adviced mask of different types for convergent and divergent squint [3]
- ✓ Chevalier John Taylor(1703-72) realised squint was a disturbance in muscle equilibrium[3]
- ✓ .Johann Freidrich Dieffenbach was first done a myotomy(1839) and later advancement surgery(1842)[3]

- ✓ Von Graefe, practiced more conservative method of partial tenotomy[3]
- ✓ George L de Buffon (1743) was first to advise treatment by Occlusion of the sound eye[3] for strabismic ambylopia.
- ✓ Meckenzie (1854) and Javal (1868-1896) stressed that squint was eventually an anomaly of binocular vision[3]
- ✓ Claud Worth (1903) proposed the fusion theory[3] of squint .
- ✓ In 1873, Tergast specified that nerve supply to the extrinsic ocular muscles is in proportion to the bulk of the muscle[3]
- ✓ C Cuppers, in 1974, popularised anew surgical procedure termed faden operation[3] (retroequatorial myopexy).
- ✓ Sir William Richard Gowers (1845-1915) was the first to pointOut differences between supranuclear and infranuclear ocular Palsies[4]

ANATOMY OF EXTRAOCULAR MUSCLES

There are six extraocular muscles, in each orbit, meant for movement of the eyeball as, media recti (MR) lateral recti (LR), superior recti (SR) and inferior recti (IR),superior oblique and inferior oblique muscles[6]

Tab 1Origin,Insertion,Action And Innervations Of Extraocular Muscles

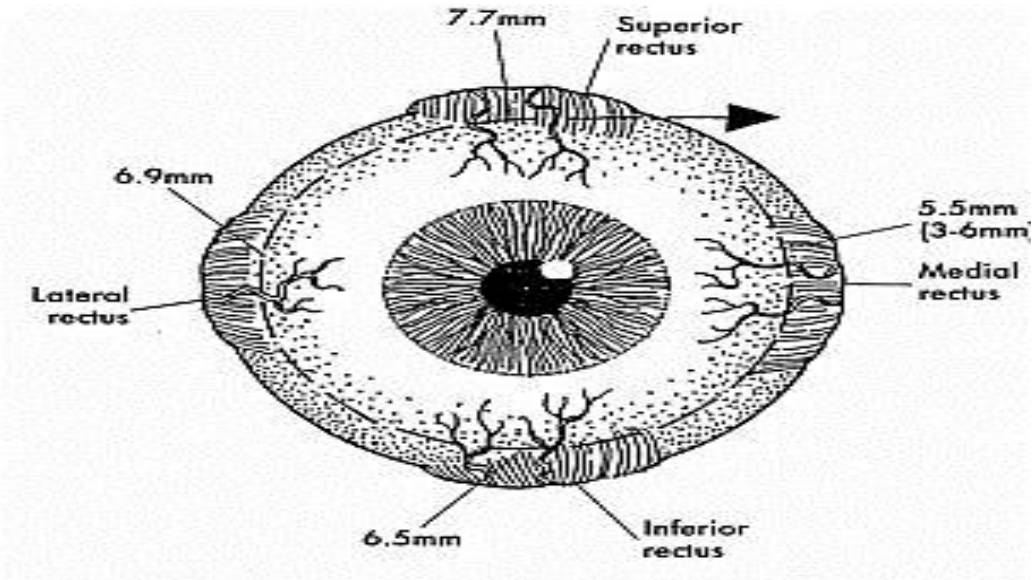
Muscle	**Origin**	**Insertion**	**Action from primary position**	**Innervation**
Medial rectus	Annulus of zinn	5.5mm from medial limbus	Adduction	Lower Oculomotor
Lateral rectus	Annulus of zinn	6.9mm from lateral limbus	Abduction	Abducen
Superior rectus	Annulus of zinn	7.7mm from superior limbus	Elevation , Intorsion , Adduction.	Upper Oculomotor
Inferior rectus	Annulus of zinn	6.5mm behind inferior limbus	Depression, Extorsion, Adduction.	Lower Oculomotor
Superior oblique	Orbital apex above Annulus of zinn	Posterior to equator in superotemporal quadrant	Intorsion , Depression , Abduction.	Trochlear
Inferior oblique	Behind inferior orbital rim lateral to lacrimal fossa	Macular area	Extorsion, Elevation , Abduction.	Lower Oculomotor

The annulus of zinn, where all the extraocular muscles, except the inferior oblique have their origins in the apex of the orbit, is a ligamentous structure which surrounds the optic foramen and part of the superior orbital fissure.

Structures passing through the annulus include the optic nerve, ophthalmic artery, superior and inferior divisions of the oculomotor nerve, abducens nerve and the nasociliary branch of the trigeminal nerve

After their origin at the orbital apex, the medial and lateral rectus muscles follow the corresponding walls of the orbit forwards until the equator of the globe. The inferior rectus follows the floor of the orbit posteriorly, while the superior rectus muscle is separated from the roof of the orbit by the levator palpebrae superioris

Fig 1 Insertion Of Eom- Spiral Of Tillax



Anterior to the equator, the recti muscles follow the curve of the globe and insert on the sclera by tendinous expansions. The curve formed by connecting the tendinous insertions of each of the recti muscles, in relation to the corneal limbus, called the spiral of Tillaux.

In primary position, the visual axis of the eye is aligned with the medial wall of the orbit and forms a 45° angle with the lateral wall. This means that, the insertions of vertical recti muscles form an angle of 23° with the visual axis of the eye and the angle of insertion of (superior and inferior) oblique muscles forms an angle of 55° with the visual axis.

Blood supply[5,7]:

Arterial system:

The vascular supply of extraocular muscles is via muscular branches from the ophthalmic artery.

i. ***Lateral muscular branch***:

Supplies the superior rectus, lateral rectus ,superior oblique and levator palpabrae.

ii. ***Medial muscular branch***:

Supplies the inferior rectus, medial rectus and inferior oblique muscle.

Seven anterior ciliary arteries arise from muscular branches as two each from medial, superior and inferior rectus and one from lateral rectus.

Venous system:

Venous drainage through superior and inferior orbital veins.

LEVATOR PALPEBRAE SUPERIORIS:

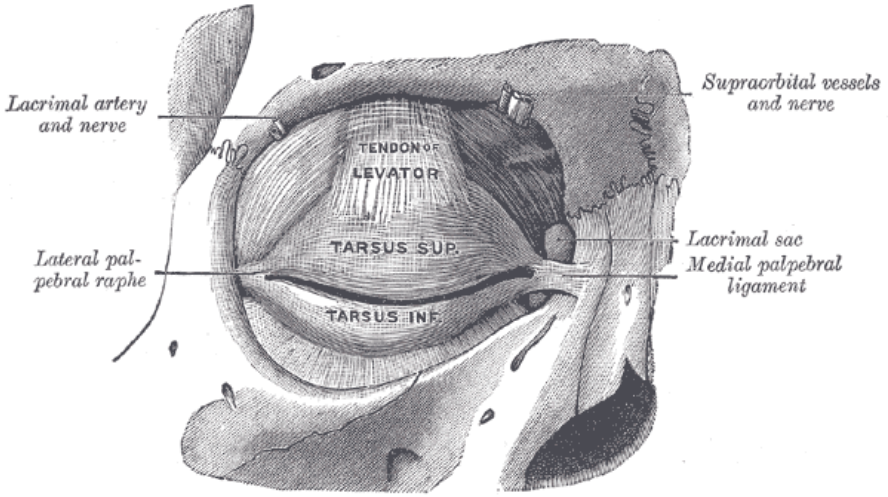
Origin:

- The LPS muscle takes origin on the under surface of the lesser wing in the sphenoid bone above and anterior to the optic foramen[5]

Course:

Then it passes forward and below the orbital roof and about 1 cm behind the orbital septum, it ends in a fan shaped membranous expansion or aponeurosis[5]

Fig 2 Levator Palpebrae Superioris



Insertion:

- The aponeurosis inserts primarily in the inferior half of the upper lid into the septa that separates the orbicularis oculi and some fibers inserts to the anterior surface of the superior tarsal plate[5].
- The lateral horn which is the lateral extension of the aponeurosis is inserted in the orbital tubercle and lateral canthal ligament[5]
- The medial horn which is the medial extension of the aponeurosis, is inserted in the medial canthal ligament.
- The muscle fibers are similar to those of the superior rectus muscle, except that they are slightly larger, about 30-50 μ in diameter

Blood supply:

- The vascular supply[5] is from the lateral muscular branch of the ophthalmic artery, the supraorbital artery, the lacrimal artery.

Nerve supply:

- The nerve supply is from branches of superior division of the oculomotor nerve[5] that reach the muscle either by piercing the medial edge of the superior rectus or by winding round its medial border.[5]

Embryology of extraocular muscles:

The extraocular muscles are derived from two sources of cephalic mesodermal cells; the prechordal plate and cranial paraxial mesoderm[7]

Pecularity of extraocular muscles[8]:

On comparing with other skeletal muscles,extraocular muscles has

- High degree of differentiation
- Rich blood supply
- High resistance to fatigue
- More fibroelastic tissue.
- Absent stretch reflex
- For precise control ofocular movements, there is high ratio of nerve fibers to muscle fibers in the extraocular muscles (1 :3-1 :5) when compared with skeletal muscle (1 :50- 1: 125).

- Two types of fibres are in extraocular muscles are

a) Felderstruktur[8]:

- Slow, tonic fibres
- Innervated by grape like nerve endings(en grappe)
- Used in smooth pursuit

b) Fibrillinstruktur[8]:

- Fast, twitch fibres

- Have plate like nerve endings(en plaque)
- Used in saccades.

Organisation of extraocular muscles (EOM):

Cross section through the rectus muscle shows two distinct regions as

a. **an outer orbital layer,**

- It is in close relation to the periorbita and orbital bone[5,7]
- consists of small diameter fibers producing slow (tonic) activity

b. **an inner global layer**,

- it is in close relation to the optic nerve and eye ball[5,7]
- consists of larger fibers producing fast (twitch) activity.

Before the muscles become tendinous, orbital layer ends but global layer extends to the full length of muscle. Sometimes the orbital layer of the oblique muscles encircles the global layer completely. There is no layered organisation for levator palpebrae superioris

The orbital layer apparently acts only on the extraocular muscle Pulleys, whereas the global layer inserts directly on the sclera to move the globe.

EXTRA OCULAR MOVEMENTS

Ocular movements can be classified in to uniocular and binocular. Uniocular movements are termed ductions while Binocular movements are termed versions and vergences.

(1) Uniocular movements (Ductions)

- Horizontal eye movements towards the nose are termed adduction and away from the nose are termed abduction.
- Vertical eye movements upwards are termed sursumduction or elevation and downwards are termed deosursumduction or depression.
- The torsional movements of the eye, along the anteroposterior axis where the eye moves towards the nose and away from the nose are called Intorsion (incycloduction) or extorsion (excycloduction) respectively
- An agonist is any particular extraocular muscle producing a specific extraocular movement,
- Antagonists are the muscles having opposite action in the same eye. E.g. Ipsilateral MR and LR.
- Synergists are the two muscles of the same eye moving the eye in the same direction. E.g. Ipsilateral SR and IO muscles for elevation .

- Each extra ocular muscle has two synergists and two antagonists with the exception of medial and lateral rectus muscles which have two synergists and three antagonists.
- Yoke muscles (i.e. contralateral synergists) refer to a pair of muscles, one from each eye, which act simultaneously during version movements. E.g. Right LR and left MR muscles for dextroversion.
- Contralateral antagonists (antagonist of the yoke muscle) refers to pair of muscles (one from each eye) having opposite action. E.g. Right LR and left LR.

2,Binocular movements

These are of two types

- Versions are conjugate synchronous ,symmetric movements of both eyes in the same direction.eg, Dextroelevation dextrodepression, levoelevation and levodepression
- Vergences are disjugate, synchronous and symmetric movements of the two eyes in opposite direction. The vergence movements of clinical significance are convergence and divergence.

COURSE OF THE OCULOMOTOR NERVE

The larger oculomotor nerve which is more complex compared to other cranialnerves supplying the extraocular muscles[5]consists of ,

i. **Somatic motor fibers**[5]

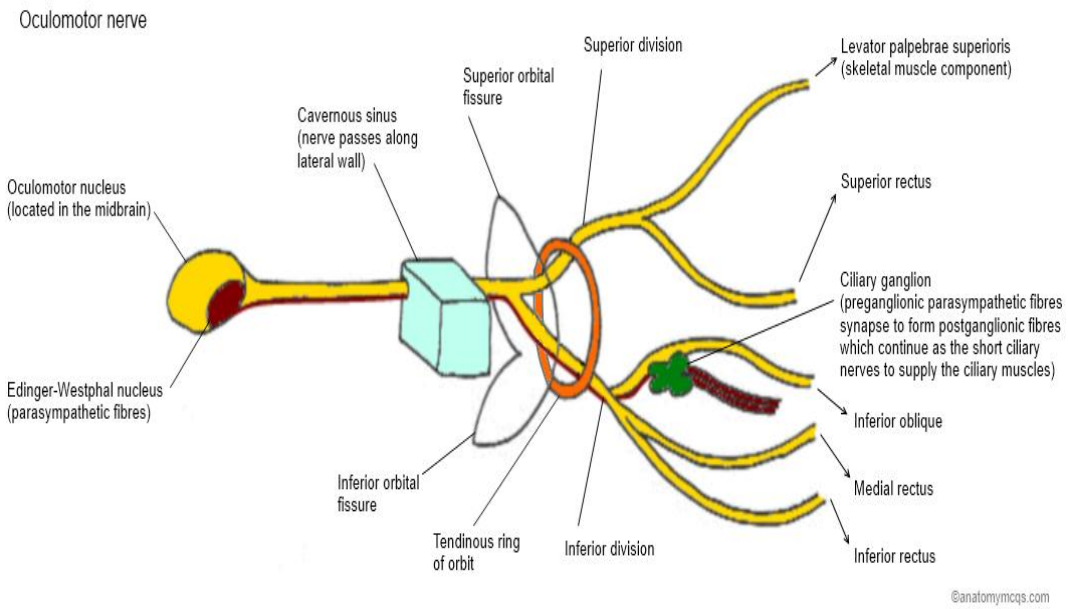
Innervating the superior, inferior and medial rectusmuscles, the inferior oblique muscle and the levator palpebrae superioris.

ii. **Visceral (parasympathetic) fibers**[5]

Innervating intrinsic muscles in the eye, i.e. the ciliary muscle and the iris sphincter muscle.

In humans, the oculomotor nerve has about 15,000 axons, four times the number in the abducens nerve and seven times the number in the trochlear nerve, most of which are distributed to about 40,000 muscle fibers.

Fig 3 Course of oculomotor nerve



The oculomotor nucleus

The oculomotor nucleus consists of mass of cells lying in the inferior periaqueductal grey matter in the rostral midbrain at the level of superior colliculus[5]. The oculomotor nucleus has both midline unpaired and lateral paired nuclei[5]

It contains three types of neurons as

1) Motor neurons:

Each muscle is served by motor neurons from single, circumscribed mass of cells called subnucleus, except the medial rectus, which has three subnuclei[9]

The motor neuron pools are arranged in longitudinal columns on top of each other, with the inferior rectus subnucleus located most dorsally and laterally in the caudal region and the medial rectus subnuclei located most ventrally with inferior oblique subnucleus located between them.

The neurons innervating the superior rectus crosses the midline and supply muscle on contralateral side

The levator palpebrae muscle of right and left eye gets origin from single midline caudal part of the nucleus, i.e.central caudal nucleus (CCN)[5]

The trochlear nucleus is present at the caudal aspect of the oculomotor nucleus and its axons runs dorsally to cross the anterior

medullary velum and innervate the contralateral superior oblique muscle.

Therefore the superior rectus and superior oblique muscles are supplied by contralateral nucleus. In other words, each cyclovertical muscle and its corresponding yoke muscle pair have nuclei on the same side of the brain.

Majority of oculomotor neurons are post mitotic by the time of formation of eye muscles. All the nerve fibes will reach te muscle by 8th week of gestation.

2) Internuclear neurons :

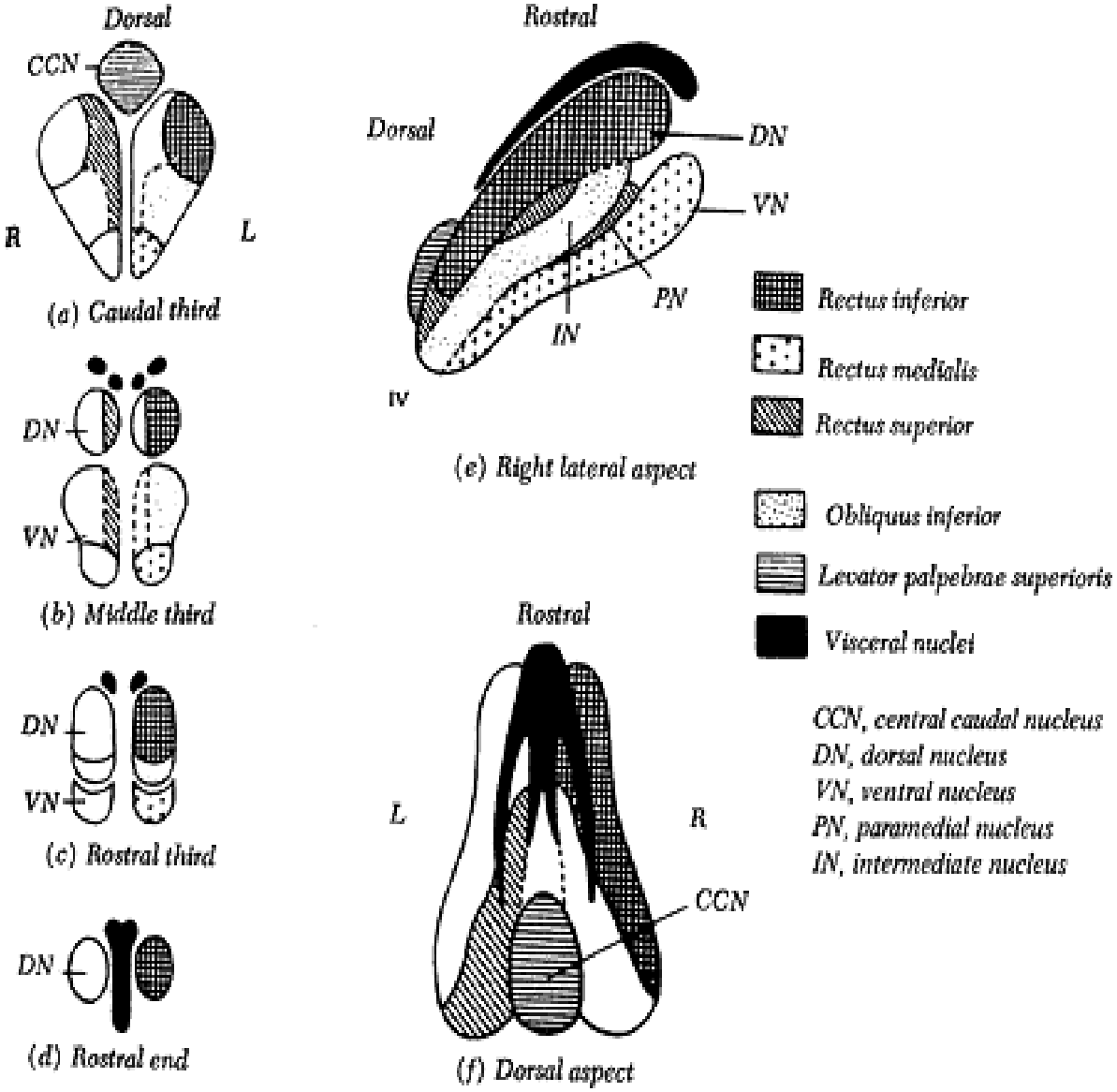
Projects to ipsilateral and contralateral nucleus[5]

3) **Parasympathetic neurons:**

Arises from the Edinger-Westphal nucleus[10], anterior median nucleus and Perlia's nucleus[5.]

Controls papillary constriction and accommodation via ciliaryganglion[11].

Fig 4 Warwick's representation of topographic arrangement of nucleus[5:]



.

The Fascicular part of oculomotor nerve:

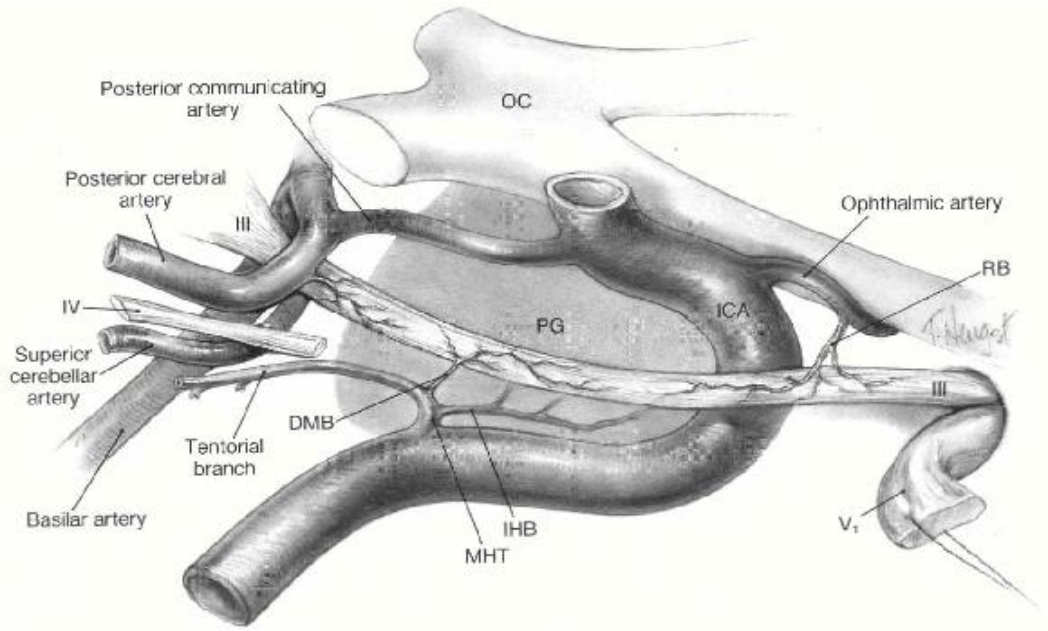
- It emerges from the ventral side of the nucleus as a sheet and lies within the substance of the brainstem[5,9]
- Then descend ventrally and pass through the red nucleus to partially penetrate the medial part of the cerebral peduncle[5,9]
- Topographically differentiated into superior and inferior divisions.
- Then the fibers emerge on both sides ofthe interpeduncular fossa, and coalesce to form two large nerve trunks.
- The blood supply of the oculomotor nuclei and fascicle is by median group of arteries from the basilar artery[5.]
- Obstruction of these vessels may produce lesions of the oculomotor nuclei and their fascicles with virtually no other neurological signs or there may be associated signs and symptoms from involvement of the reticular formation, the red nucleus and/or the cerebral peduncle[5]

Oculomotor nerve in subarachnoid space:

- The majority of axons in the oculomotor nerve are myelinated and divided as small (3-6 μ) and large(6-18 μ) by the diameter of axons.

- The large caliber axons originate from the motor neurons supplying the extraocular muscles
- The smaller axons transmit parasympathetic impulses to ciliary body and iris.
- The nerve is invested by pia and passes through the subarachnoid cistern in obliquely downward, forward and lateral direction at thelevel of the tentorial incisura[5,9]
- Then it pass through the lateralpart of posterior clinoid process[5] and pierces the dura.
- Then it runs between the superior cerebellar artery and the posterior cerebralartery[5].

***Fig 5 Vascular supply of oculomotor nerve in subarachnoid space*:**

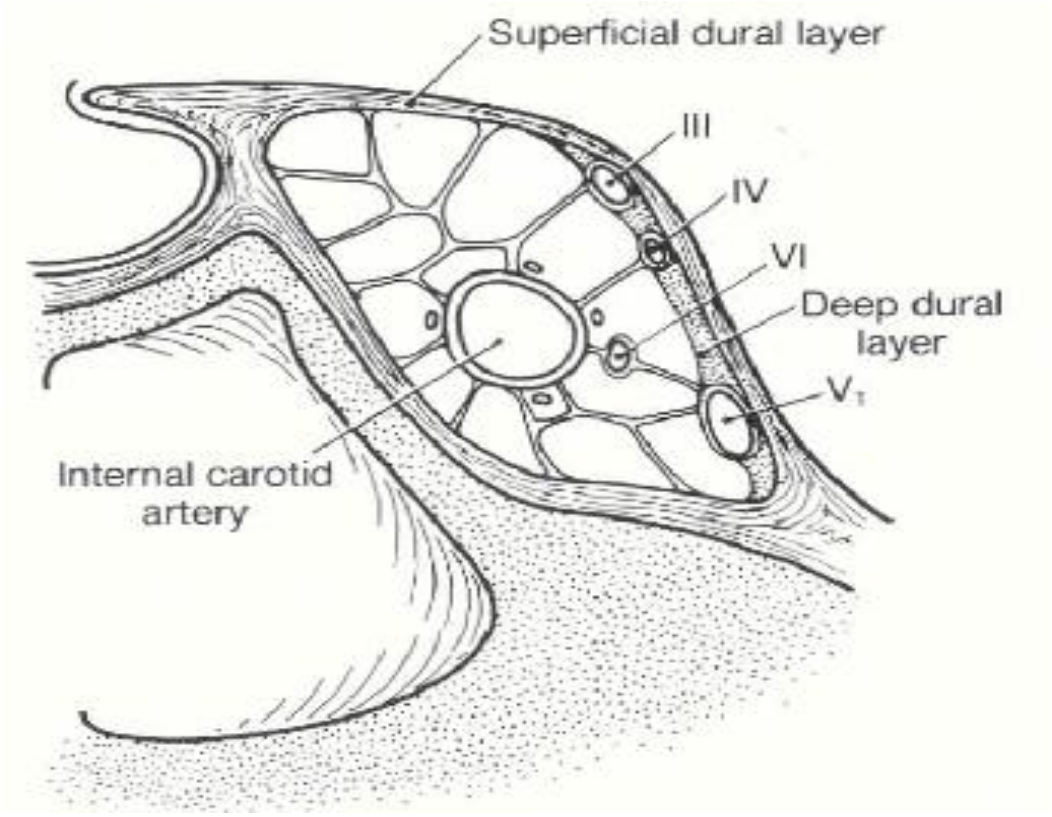


- As it runs distally, the nerve lies lateral to the posterior communicating artery for about 0.5 cm
- Here pupillary fibres are on the dorsomedial aspect of nerve making it vulnerable for compression
- Then it enters the cavernous sinus by piercing the dura.
- .The blood supply by meningohypophyseal trunk of the internal carotid artery , the posterior cerebral artery and superior cerebellar artery [5,12]

Oculomotor nerve in cavernous sinus:

- Enters the cavernous sinus through its roof, and lie in its lateral wall and below it lies the trochlear nerve[5,12]
- In the anterior part of cavernous sinus, it divides into superior and inferior parts (5 mm behind the superior orbital fissure.)
- On the anterior part of cavernous sinus, oculomotor nerve receives sympathetic fibers[5]from the carotid trunk.
- The vascular supply is derived from branches of the meningohypophyseal branch[5,11] along with branches of ophthalmic artery.

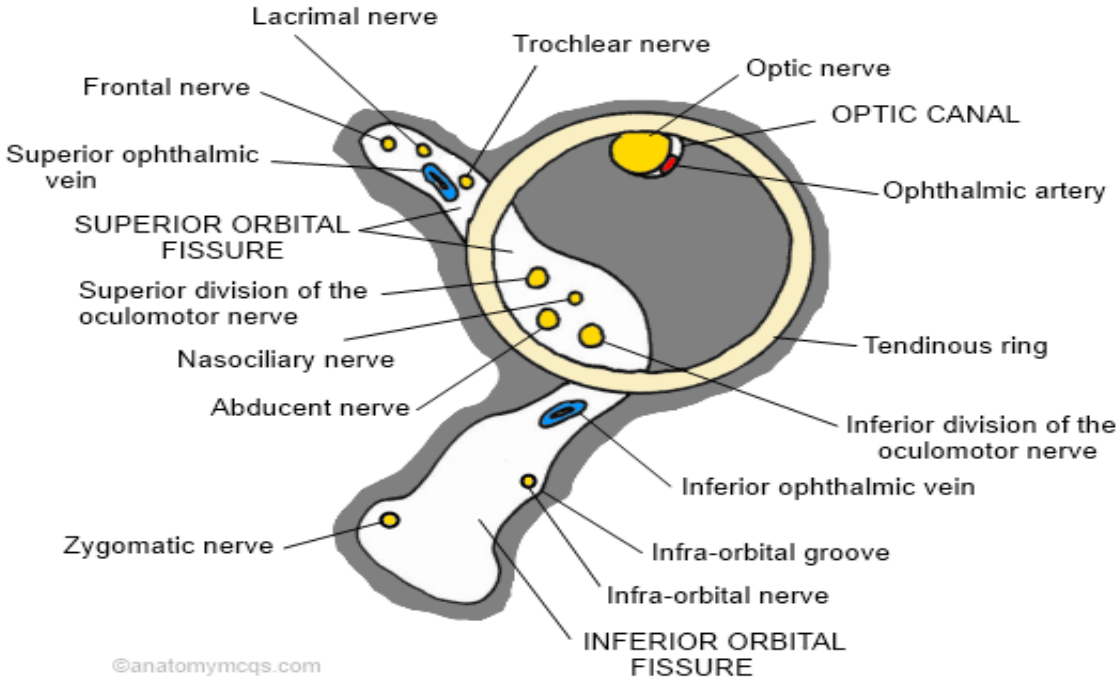
Fig 6 Oculomotor Nerve In Cavernous Sinus:



Oculomotor nerve in superior orbital fissure:

- Both superior and inferior divisions[5,9] enters orbit via the middle part of superior orbital fissure
- It is crossed superiorly by the trochlear and ophthalmic division of trigeminal nerve[11,12]

Fig 7 Oculomotor nerve in superior orbital fissure:



Oculomotor nerve in orbit:

Each division has separate course in orbits

a) Superior division:

- The superior division has fiber count of about one-third that of the inferior division.
- The smaller superior division, passes up and over the lateral aspect of the optic nerve,
- Divides into multiple small branches and then supply superior rectus and levator palpebrae superioris muscle from its under surface[5]

b) Inferior division:

- The inferior division divides into multiple branches in posterior orbit.
- It innervates medial and inferior rectus muscles and through the inferior rectus muscle itself to penetrate the inferior oblique muscle[5]
- The inferior division transmits parasympathetic axons to the ciliary ganglion[5.]
- Within the cavernous sinus and orbit, the pupillary fibers are located in the inferior division of the nerve[4].Thus, the relative pupil sparing in cavernous sinus or orbital apex lesions may reflect preservation of the inferior branch.

SUPRANUCLEAR COMPONENTS OF THE OCULAR MOTOR SYSTEM:

For proper functioning of oculomotor systems ,there should be normal motorneurons, premotor structures and internuclear pathways.

a) Internuclear system:

- Consists of median longitudinal fasiculus[5],which extends from oculomotor nucleus in mesencepalon to spinal cord.
- It serves as both ascending and descending pathways and formed mainly by fibres of vestibular system.

- ❖ Ipsilateral projection of MLF is from superior vestibular nucleus and contralateral projection of MLF is from medial vestibular nucleus.
- ❖ Lesion in MLF cause both horizontal and vertical gaze problems because MLF fibres affect all 3 ocular motor nuclei.

b) Immediate premotor structures of the brainstem[5]:

- ❖ For horizontal gaze—abducens nuclei and paramedian pontine reticular formation(PPRF).
- ❖ For vertical gaze---mesencephalic reticular formation (MRF) which includes interstitial nucleus of Cajal and rostral interstitial nucleus of MLF(riMLF).

c) Paramedian pontine reticular formation[5]:

- ❖ Located near to abducens nucleus and controls horizontal eye movements.
- ❖ Afferent fibres are from vestibular nuclei,cerebellum and superior colliculus.
- ❖ Efferent fibres to abducens nulei on ipsilateral side and riMLF
- ❖ It also has efferent fibres to reticular formation, vestibular nuclei, spinal cord and cerebellum.

d) Rostral interstitial nucleus of MLF[5]:

- ❖ It is a oval group of cells situated on either side of midline in rostral mesencephalon.
- ❖ It is the immediate premotor area responsible for vertical saccades.

e) Nucleus of posterior commissure[5]:

- ❖ It consists of crossing fibres intermingled with scattered cells that constitute its nucleus.
- ❖ Lesions in this part selectively affects upward vertical eye movements

OCULOMOTOR NERVE PALSY

Can present as congenital or acquired nerve palsy

Congenital nerve palsy

Signs :

- NO DIPLOPIA.
- Usually unilateral with no other neurological abnormality[5,9,13]
- Have ptosis, ophthalmoparesis and miotic pupil

Etiology :

- Absence of oculomotor nucleus or nerve
- Birth injury

Associated features:

i. **Congenital adduction palsy with synergestic divergence**:

Presents with unilateral adduction palsy with both eyes

Abducted during gazeevoked in the direction of paralysed medial rectus[5,9]

ii. Vertical retraction syndrome:

Presents with limitation of movement of involved eye on vertical movements with globe retraction and narrowed palpaberal fissure,[5,9]

iii. **Oculomotor paresis with cyclic spasms**:

Presents with mydriasis,ptosis,ophthalmoparesis and reduced accommodation associated with cyclic spasms which include elevation of ptotic lid,adduction,increase in accommodation and pupillary constriction[5,9]

Childhood oculomotor nerve palsy[13] :

Etiology :

- ❖ Vascular
- ❖ Post traumatic
- ❖ Opthalmoplegic migraine
- ❖ Inflammatory
- ❖ Congenital
- ❖ Cryptogenic
- ❖ Neoplastic

Acquired Nerve Palsy:

It is more common than congenital palsy.

A. Lesions in oculomotor nucleus:

Signs :

- ❖ Symmetric bilateral ptosis,contralateral elevator palsy(SR),ipsilateral paresis of other extraocular muscles(SR,IR,MR,IO)[5]
- ❖ Pupils are involved only in dorsal, rostral lesion which is usually bilateral[5]

Etiology :

- ❖ Ischemia, usually from occlusion of isolated perforating paramedian arterioles[5,9]or the basilar artery.
- ❖ Other etiologies include hemorrhage , tumor , inflammation , brain stem compression and degeneration as machado-joseph disease[5].

Special features:

- ❖ Focal lesions of the oculomotor nucleus occasionally causes isolated extraocular muscle palsies[5] (e.g. isolated inferior rectus palsy).
- ❖ Lesions of paired medial rectus nuclei causes wall eyed bilateral intranuclear ophthalmoplegia (WEBINO)[12]

Mauthner's rule[14]states that in oculomotor nerve palsy if lesionis in the level of nucleus, muscles supplied will have an progressive paralysis,with normal pupillary reaction.But this rule is not proved to be correct in all cases.

B. **Lesions in oculomotor fascicle**:

Signs :

- ❖ Ipsilateral oculomotor nerve palsy[5] with pupillary involvement.Rarely pupillary sparing occurs
- ❖ Diagnosis of oculomotor fascicle lesions depends on other co-existing neurological signs[5]

Etiology[5]:

- Ischaemic, infiltrative, compressive, inflammatory or demyelinating diseases.

Associated features:

- **Benedict's syndrome**:

  Involves superior cerebellar peduncle, red nucleus and substantia nigra resulting in ipsilateral oculomotor nerve palsy and involuntary movements[5] on the contralateral side.

- **Weber's syndrome:**

  Involves the ventral mesencephalon includingthe cerebral peduncle, which contains pyramidal tract fibers.It consists of oculomotor nerve palsy with other side hemiplegia[5,14].

- **Nothnagel's syndrome**

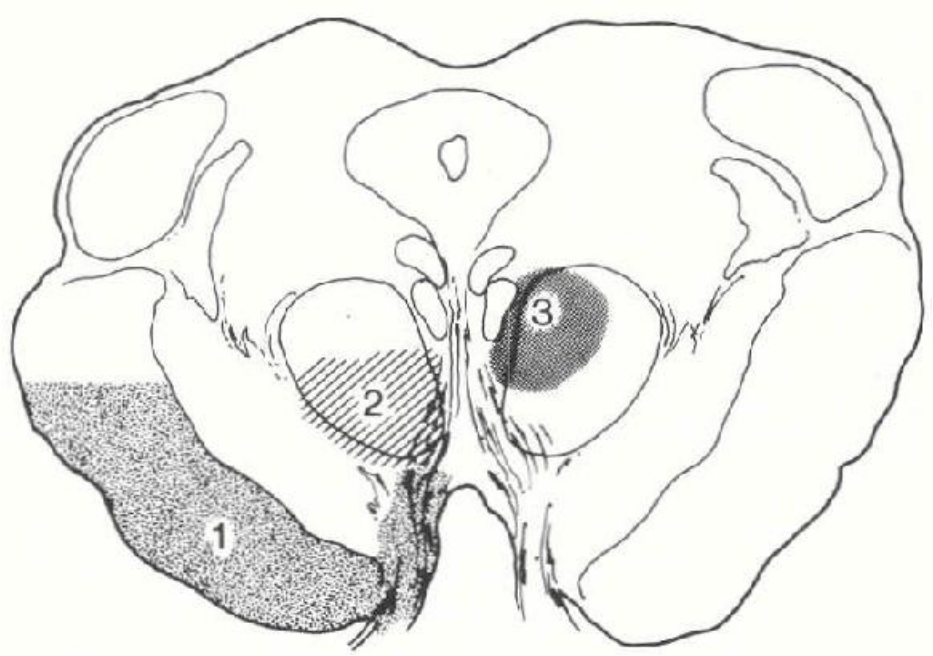
  Due to lesion in dorsal aspect of the midbrain, superior and inferior colliculus, and brachium conjunctinumresulting in partial oculomotor paresis combined with cerebellar ataxia[5]

- **Claude's syndrome**

  Due to simultaneous damage of red nucleus and the brachium conjunctivum with featuresof both Benedict's andNothnagel's syndrome[5] such as oculomotornerve paresis, contralateral asynergia, ataxia, dysmetria and

dysdiadochokinesia. It is due to thrombosis of medial interpeduncular division[5]in posterior cerebral artery

Fig 8 Lesions in oculomotor fascicle:



1-WEBER SYNDROME

2-BENEDICT SYNDROME

3-CLAUDE SYNDROME

C. Lesions of the oculomotor nerve in the subarachnoid space:

- Most common site for isolated oculomotor nerve palsy.May be complete,incomplete or progressive.
- Oculomotor nerve lesions produced by damage to its subarachnoid space may present as[5]

(i) Ophthalmoplegia with pupillary involvement.

(ii) Ophthalmoplegia with pupillary sparing

(iii) Isolated internal ophthalmoplegia

(i) Subarachnoid oculomotor nerve palsy with pupillary involvement:

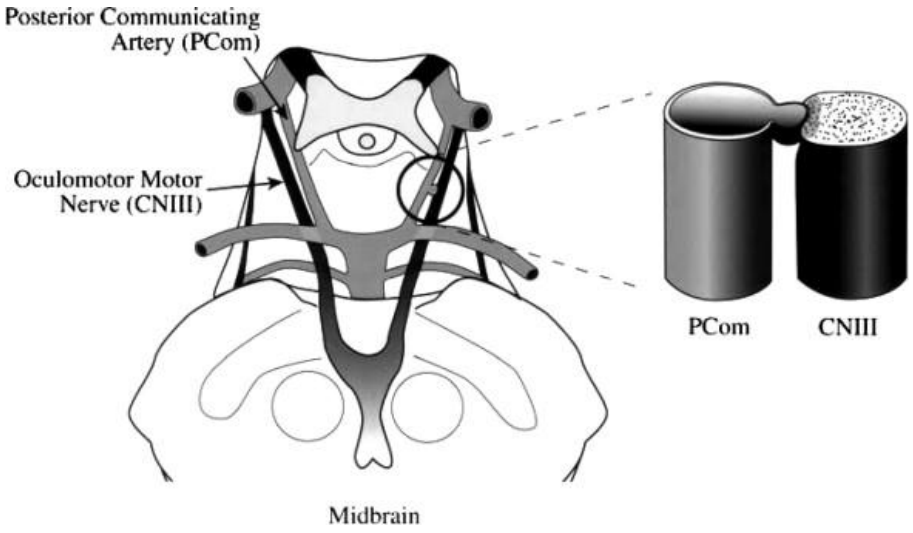
Signs:

- Ipsilateral isolated oculomotor nerve palsy with no other neurological signs[5]

Etiology :

- Intracranial aneurysms[3]usually arising from the junction of the internal carotid and posterior communicating arteries but can also arise from the basilar artery[5,14]
- Tumours located in the interpeduncular fossa[5]
- Neurinomas[5]
- Severe cranial trauma, associated with skull fracture and concussion.
- Viral syndromes or following immunisation[13]
- Carotid cavernous fistula
- Other causes like basal Meningitis, vascular or ischaemic (diabetic) and pseudotumour cerebri

Fig 9 Aneurysm In Posterior Communicating Artery Causing Compression Of Oculomotor Nerve:



(ii) Subarachnoid oculomotor nerve palsy with pupillary sparing:

Signs :

- ❖ Pain around eye is common.
- ❖ Usually resolves with in 6-12 weeks without aberrant regeneration[5,18]

Etiology :

- ❖ Ischaemia is the common reason[2,5,9] for isolated, pupil sparing oculomotor nerve palsy. Though diabetes mellitus is attributed as frequent cause[15], others include systemic hypertension, atherosclerosis, migraine and systemic lupuserythematosis,.

- ❖ Subarachnoid compressive lesions particularly aneurysms[3,9]which always cause incomplete palsy
- ❖ Other causes include viral inflammation, systemic lymphoma and l infiltration from chronic lymphocytic leukemia[5]

(iii) Isolated internal ophthalmoplegia:

Signs :

- ❖ Normal extraocular movements with fixed ,dilated pupil[5]
- ❖ usually associated with headache and loss of consciousness

Etiology :

- ❖ Intracranial aneurysms[3,5], particularly from posterior communicating artery.
- ❖ Transtentorial uncal herniation[16] which is an surgical emergency
- ❖ Trauma
- ❖ Tumors as schwanomma
- ❖ Infections as leprosy,measles,herpes zoster[5] etc

It should be differentiated from ciliary ganglion involvement or direct pharmacological blockade by pharmacological testing.

D. Leisons of the oculomotor nerve within the cavernoussinus and superior orbital fissure:

i. **Sphenocavernous syndrome** :

As there is anatomical continuity, lesions involving cavernous sinus andsuperior orbital fissure can be considered as a single entity[5]. However it can also cause isolated oculomotor nerve dysfunction[5, 9]

Signs:

- Multiple ocular motor nerve palsiesinvolvingall ocular motor cranial nerves along with ophthalmic division of trigeminal nerve.
- Severe periorbital pain
- Forehead numbness,proptosis,lid edema ,conjunctival chemosis
- Horners syndrome with miotic and poorly reacting pupil[5,17]

Etiology:

- Common lesions include aneurysms, pituitary tumours ,metastatic tumours , meningiomas, vascular causes, craniopharyngiomas nasopharyngeal tumours and infectious inflammatory processes.

ii. **Tolosa hunt syndrome**:

- Usually unilateral.

- ❖ Presents with boring retroorbital Pain andophthalmoplegia caused by idiopathic granulamatous inflammation[2,5,15]
- ❖ Most commonly involved nerve is oculomotor nerve .
- ❖ In MRI enlargement of cavernous sinus with abnormal material and convexity on the wall of cavernous sinus will be seen.
- ❖ It responds well to steroids[5].

iii. Isolated oculomotor nerve involvement in the cavernous sinus:

Signs:

- ❖ Isolated oculomotor nerve dysfunction, with or without pupillary involvement[19].

Etiology :

- ❖ Pituitary adenomas, craniopharyngiomas,suprasellar aneurysms and infiltrative lesions as multiple myeloma and nasopharyngeal carcinoma[5,]
- ❖ Also, in some diabetic patients ischaemic oculomotor nerve palsy[5] isdue to lesion in the intracavernous part of oculomotor nerve.

E. Lesions of the oculomotor nerve within the orbit:

a) **Incomplete oculomotor paresis**:

- ❖ Both superior and inferior division can be affected separately due to lesion in either the spheno-cavernous region or the orbital apex[5]

b) **orbital apex syndrome**

- ❖ Cause oculomotor nerve dysfunction along with other ocular motor palsies , visual loss and significant proptosis[5]

c) Spheno-cavernous syndrome, with pain as a principle feature can be distinguished from the orbital apex syndrome in which proptosis and optic neuropathy are characteristic features.

d) **Pseudo-orbital apex syndrome**[5]

- ❖ Due to large intracranial mass lesions which on expansion compresss the intracranial optic nerve and either subarachnoid or cavernous portionof the ocular motor nerves, along with impending venous drainage in the orbit.

F. Lesions of the oculomotor nerve of uncertain Or variable location:

- In many cases of isolated oculomotor nerve palsy, the exact site oflesion is unclear[5] and in certain diseases many sites of nerve get affected.
- In diabetes, the oculomotornerve may be affected either in mesencephalic, subarachnoid or even in cavernoussinus portion of the nerve [5,17]
- In some cases of double elevator palsy, i.e. ipsilateral inferior oblique and superior rectus paresis, , the lesion will be either nuclear or orbital in location[5]

PATHOGENESIS OF ACQUIRED OCULOMOTOR NERVE PALSY:

A. Ischaemic ("Diabetic") oculomotor palsy:

Studies by Weber et al[14] states that following changes occurs in ischaemic ("diabetic") oculomotor palsy:

- Focal demyelination with minimal axonal degeneration.
- Remyelination which is thought to be responsible for recovery without aberrant Regeneration.
- Necrotising angiopathy is thought to cause the demyelinative lesion

HbA1c is found to be frequently higher [20]in patients with ischaemic oculomotor palsy indicating poor control of diabetes . Moreover left ventricular hypertrophy (LVH) and elevated haematocrit[20] is considered as the important determinants of ischaemic ocular motor nerve palsy

B. Traumatic oculomotor palsy:

Occurs due to lesion in the oculomotor nucleus or peripheral portion of thenerve.

- In nucleus , the mechanism is due to the blow creating a fluid wave in the third ventricle which breaks around the anterior end of the aqueduct of sylvius, and causing oedema and petechial haemorrhages within the nucleus[4].

- In the peripheral portion of the nerve, the mechanism of injury is tearing and bruising near a basal skull fracture [4]

C. Intracranial aneurysm:

Intra cranial aneurysm can cause ocular motor palsy in following ways as

- Miliary aneurysms in smaller cerebral vessels causing lesions in ocular motor nuclei[4].
- Rupture of Larger aneurysms causing subarachnoid haemorrhage.
- Aneurysms acting as space occupying lesions and causing palsy by pressure upon the nerve.

D. Neoplasms:

Intracranial space-occupying lesions cause ocular motor palsy by following mechanisms [4]

- Direct compression by tumour.
- Indirect displacement of the brain by the tumour

RECOVERY FROM ACQUIREDOCULOMOTOR NERVE PALSY:

Many patterns of recovery[5], are noted after oculomotor nerve palsy such as,

1,Complete recovery:.

- Microvascular diseases has good recovery compared to undetermined causes, trauma and neoplasms.
- Isolated palsies have highest recovery rate compared to multiple cranial nerve palsy
- The average time to recovery is 4 to 6 weeks in microvascular causes and six months in traumatic palsy.

2. Persistent oculomotor palsy:

- In some patients the paralysis may persist life long.
- Usually due nerve transection by trauma or infiltration by tumour[16]

3. Partial recovery:

- Particularly seen after damage to fasicular portion of oculomotor nerve[5]

4. Partial recovery with oculomotor nerve synkinesis:

OCULOMOTOR SYNKINESIS

- Injury to the oculomotor nerve at any portion along its whole pathway can results in Oculomotor synkinesis[2]

The clinical features of oculomotor synkinesis include[2,5,13]

A. Elevation of upper lid on attempted depression (pseudo Von-Graefe sign) or adduction (reverse Duane's syndrome).
B. Pseudo-Argyl Robertson pupils:

   Pupil reacts poorly to light stimulus butconstricts well during attempted adduction.
C. On attempted downward or upward gaze there is adduction of eye.
D. Retraction of globe on vertical movements
E. Monocular vertical optokinetic responses.

Pathogenesis of aberrant regeneration:

- In General, after damage to a nerve more axons are regenerated than normal[5]
- It sprouts from the terminal end of damaged nerve and ends in the Schwann tubes in the end organ
- In oculomotor nerve damage, nerves destined for one muscle reaches other muscle or ciliary ganglion.
- If axons reaches other muscle, different action is produced for example, if LPS receives fibres for medial rectus any attempt for adduction of eye results in elevation of eyelid
- If misdirected axons reaches ciliary ganglion, on attempted movement of eye there may be pupil constriction, increased myopia or increase in intraocular pressure.
- Apart from the possible mechanisms of aberrant regeneration or misdirection fibers for oculomotor synkinesis many other alternative mechanisms are proposed .

- They include

a) Ephatic transmission[5]:

Defined as an electronic mode of transmission between fibres resulting in Axo-axonal “cross-talk”.

b) Synaptic reorganization of the Oculomotor Subnucleus[5]:

Chromatolysis occurs resulting in various structural and metabolic changes in nerves.

Oculomotor synkinesis can be either,

(i**) “Primary” oculomotor synkinesis**:

- Occurs with out oculomotor nerve palsy[5]
- Caused by slow growing masses in thecavernous sinus, usually meningiomas or aneurysms .

.(ii**) Secondary oculomotor synkinesis**,

- Occurs after congenital palsy and recognized acquired oculomotor palsy[5]
- Most commonly occur in association with trauma or compressive lesions, rarely in ophthalmoplegic migraine but never in ischaemic (diabetic) ophthalmoplegia or demyelinating syndromes

CLINICAL EVALUATION

History :

- ❖ Major complaints of patients are drooping of eyelids,diplopia and near vision difficulty
- ❖ History about headache,periorbital pain,fatiguability and other neurological symptoms should be recorded

Clinical examination:

1) **Clinical evaluation of ptosis, extraocular movements, diplopia charting and hess charting**:

A. ***Blepharoptosis :***

- ❖ Ptosis is an important feature of oculomotor nerve palsy and its evaluation helps in the follow up as well as in the surgical management.
- ❖ Evaluation includes various measurements[21] such as,

a. **Palpebral fissure height**

Should be measured in primary position, upward and downward position.

b. **Margin reflex distances (MRD**)

MRD1is thegap between corneal light reflex and centre of the upper lid margin with the eyes in primary position(normal +4.5)[21]

MRD2 is the gap between corneal light reflex and lower eyelid margin with the eyes in the primary position (normal=+5.5mm)[21]

c. **Levator muscle function**

Normal Levator muscle excursion is about15 to18mm.

B. ***Extraocular movements****:*

i. Head posture[6]:

- In muscle paralyses the patient turn his head towards the action of the paralysed muscle to prevent diplopia it is known as the compensatory head posture.
- Head posture is adapted mainly to
    a) Obtain binocular single vision
    b) Obtain wide separation of images
- Three components for a compensatory head posture[22] are
    i. Face turn to the right or left.
    ii. Chin elevation or depression.
    iii. Head tilt.
- If horizontally acting muscles are paralysed, the compensatory head posture usually consists of a simple face turn.
- In vertical muscle palsy ,the head posture gets complicated and in such cases it should be remembered that

    1. Elevator muscle palsy causes chin elevation and depressor muscle palsy causes chin depression.

2. Face turn towards the same side of involved muscle in vertical rectus palsies and towards opposite side in oblique muscle palsies.

3. The intorters (superiors) palsycauses head tilt in same direction of faceturn and extorters (inferiors) palsy, the head tilt is in opposite direction of face turn.

- *In oculomotor nerve palsy no compensetary head posture is achieved as more muscles are paralysed.*

ii,Ocular Movements*:*

- Both Ductions and versions are examined.
- Ductions are quantitated with a grade 0 to –4 scale with –1 denotes minimal limitation and –4denotes severe limitation of movement[23]
- Versions should evaluated in all nine cardinal positions of gaze.
- Abnormal versions should be marked from +4 to –4 with 0 denotes normal and +4 denotes severe over action while –4 indicates severe underaction[6,23]
- Any convergence/ divergence excess or insufficiency should be evaluated.
- In unconscious patients assessment of ocular movements requires some special BRAIN STEM tests. This tests are absent in supranuclear lesions[5,9].

1. Oculo-cephalic reflex ("Doll's eye movements")

In normal individuals, deviation of eyes to one side occurs when head is turned to other side.

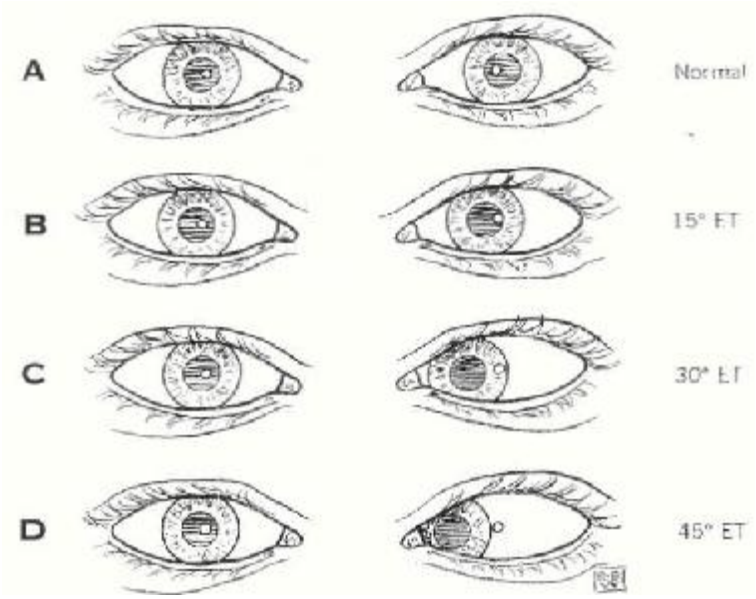
2. Oculo-vestibular reflex:

This test is performed by syringing with cold water (30° C) or warm water (44° C) in ears. When cold water is used, the eyes will deviate to the same side and nystagmus occurs in other direction. With water of 44° C, the eyes will deviate to the opposite side and the direction of nystagmus will be to the side of the syringing.(COWS-Cold water opposite side nystagmus and warm water same side nystagmus)

iii, The Hirschberg test*:*

- It is a simplest method to estimate the angle of deviation.
- A small spot light is held in front of patients face at a distance of 33 cm with patient looking directly at the light and the first Purkinje image is observed in relation to the corneal center[6,23]
- Corneal reflex is displaced in the nasal aspect from its original position in exotropia, in temporal aspect in esotropia, in the inferior cornea in hypertropia, and superior corneain hypotropia.
- In Hirschberg test 1 mm of disposition of reflex in cornea corresponds to 7°[5] (or 15$\angle$)of angle of deviation.

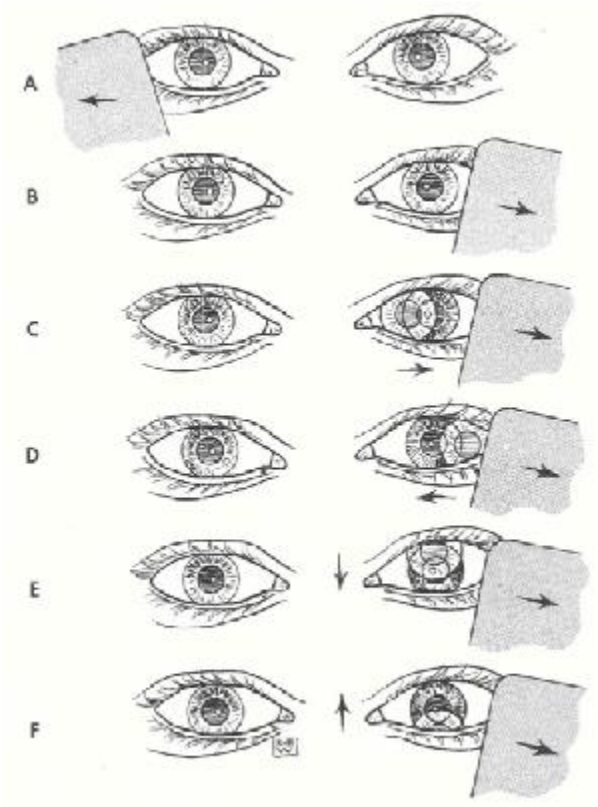
Fig 10 Hirschberg Test:



iv. **Cover test:**

- Patient is asked to fix on a test object and an occluder is held in front of fixating eye .
- It consists of three types of tests, namely the uniocular cover test ,cover-uncover test and alternate cover test[6]

Fig 11Simple Cover Test:



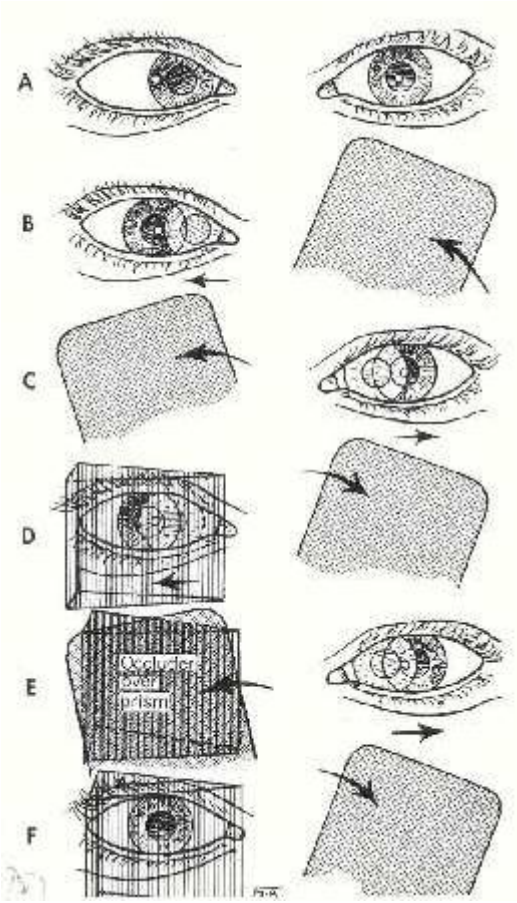
- The simple cover and cover-uncover tests are to determine if there is deviation , type of deviation and its direction[6].
- The alternate cover test is used determine presence of any latent phoria and to differentiate concomitant or incomitant squint [23]
- If the secondary deviation(deviation of the sound eye when the affected eye is fixing) is greater than the primary deviation (deviation of the affected eye when the sound eye is fixing), it is an incomitant squint[6].

- ❖ If secondary deviation is equal to primary deviation, it is a concomitant squint.

v. The prism and cover test method:

- ❖ It is performed for near and distant fixation.
- ❖ Patient is asked to fix on a test object and an occluder is held alternatively on each eye. By doing this, the unoccluded eye moves opposite of the deviation[6,23]
- ❖ After estimating the angle of deviation, a low strength prism is held before one eye.
- ❖ The apex of prism is held in direction of deviation
- ❖ The prism strength is increased until the deviation is corrected[6]
- ❖ It is impossible to measure cyclo deviation by this method , but must be subjectively determined (Maddox Double Rod Test).
- ❖ Other objective Tests are prism reflex test (krimsky's), the Lister's perimeter and synaptophore.
- ❖ Subjective tests used are Maddox rod, the Maddox double rod test ,synaptophore and Maddox wing.

Fig 12 The Prism And Cover Test Method



C,Assessment of diplopia:

- Usually performed in a semi dark room with the patient seated with his head perfectly still throughout the investigation.
- A red-green goggles are used with red glass is placed before right eye (red over the right eye) [6,23].
- The source of light is held at a distance of one in front of patient. (preferably the Armstrong "barlite", which gives a slit of light 1½ inches long).
- The light is shown in all nine cardinal positions of gaze[6]

- The presence or absence of diplopia and the type of diplopia are noted in the nine cardinal directions of gaze
- The true image is the image that corresponds to the fixing eye and it lies on the macula.
- The false image is the image of the deviating eye which is less distinct and being perceived by the peripheral retina
- Following points are noted during charting [6] as

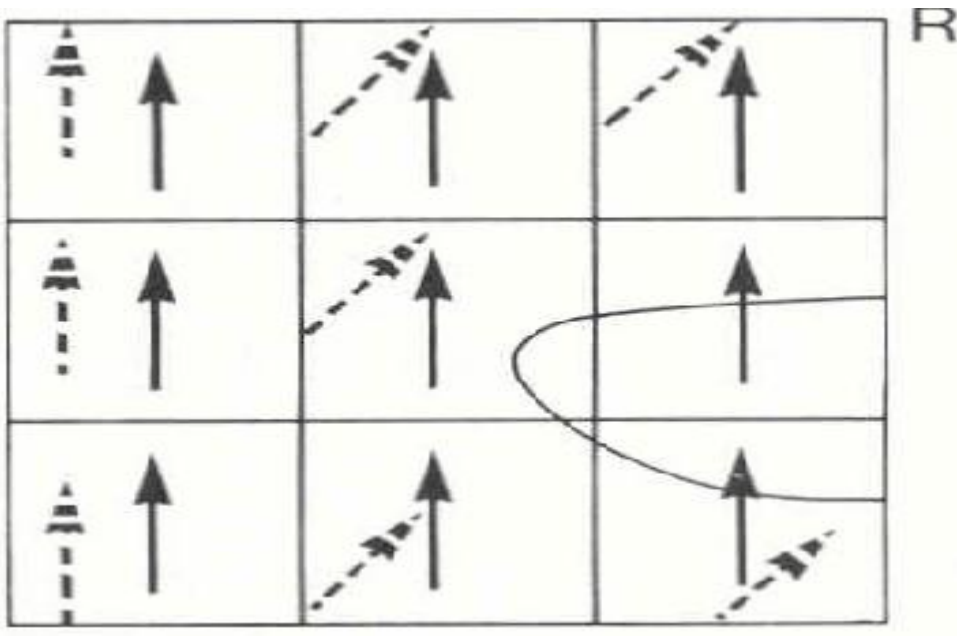
A. whether diplopia is horizontal or vertical

B. if horizontal, whether crossed or uncrossed

C. whether images are right over left or left over right.

D. which direction, maximum seperation of the images is noted.

Fig 13 Diplopia Charting In Right Third Nerve Palsy:



- ❖ Maximum separation of the images is seen in the position of the normal action of the paralysed muscle..
- ❖ The horizontal diplopia is uncrossed when the false image is on the same side as the deviating eye, and crossed when it is on the opposite side.
- ❖ Uncrossed diplopia is seen in lateral rectus, superior oblique and inferior oblique paralysis. All other muscle paralyses will result in crossed diplopia
- ❖ Disadvantages:
    - Not possible in colour blind patints
    - Only an qualitative assessment
    - Can be done only in active, intelligent patient

C. ***Hess screen examination:***

Description[6,23]:

- ❖ The Hess screen is a black screen, which is three feet wide and three and a half feet long.
- ❖ It consists of rows of red lines forming an of 5° angle in between.
- ❖ In the middle of the screen there is zero point and there is a red dot at point of intersection of the 15° and 30° lines with one another and with the corresponding horizontal and vertical lines,

- These red dots form an inner square of eight dots and an outer square of sixteen dots.
- A knot with three green cords together in form of letter 'Y' considered as indicator[6]

Procedure[6] :

- The patient, seated in front of screen at 50 cm and wears red and green glasses
- Through the red glass he is able to see only the red marks on the screen, and through the green glass he can see only the green cords.
- Patient is asked to keep the knot connecting green cords on each dot in turn, keeping his head still and directed straight ahead.
- The test is first carried out with the red glass before the right eye. The glasses are then reversed to change the fixation and the examination repeated

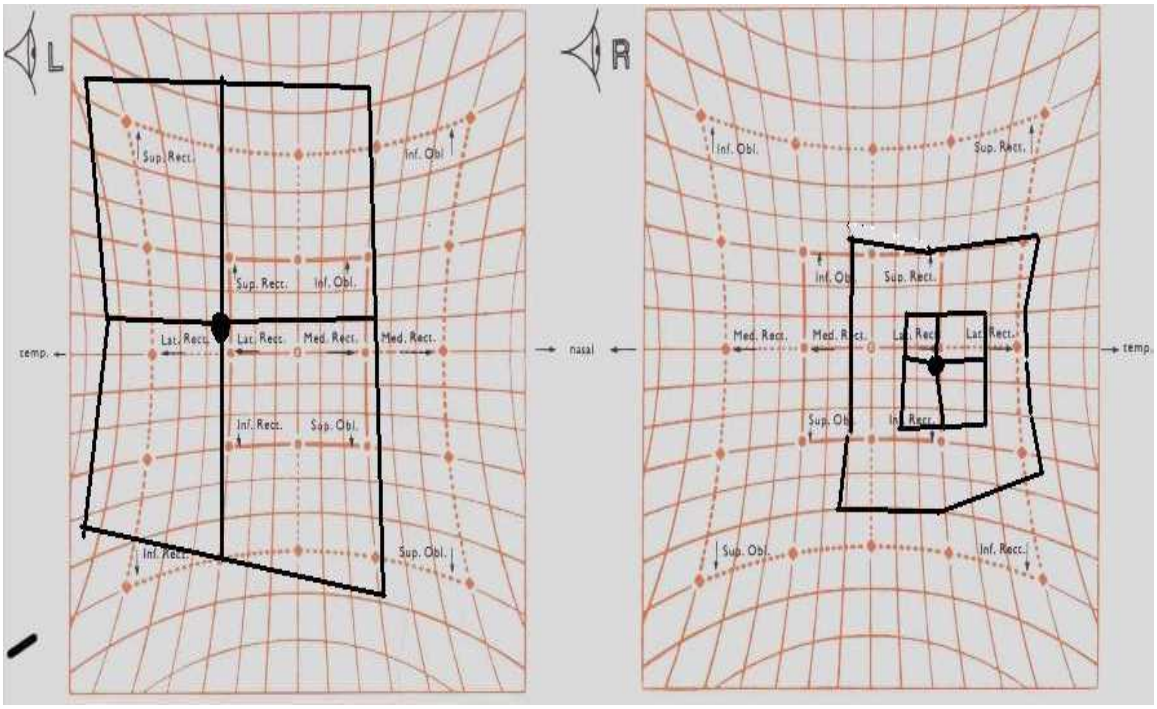
Clinical relevance:

- Used to compare oculomotor innervations of the two eyes.
- It helps to differentiate between paralytic squints from concomitant squints.
- To demonstrate the progression of palsy
- To demonstrate the recovery pattern from an ocular palsy.

Interpretation of a Hess chart:

- The areas of the two measuring fields[6] should be compared.
- The smaller field belongs to the eye with the paretic muscle.
- The larger field belongs to the eye with the overacting muscle[23].
- The smaller field will show the greatest compression in the direction of action of the paretic muscle. Due to contracture of the muscle there is displacement of the field in the direction of action of the direct antagonist .
- Due to over action of the yoke there is greatest expansion in the main direction of action of the yoke of the paretic muscle in the larger . There may also be a displacement of the field away from the direction of the antagonist the yoke muscle due to secondary inhibitional palsy.

Fig 14 Hess Charting Of Complate Right Oculomotor Nerve Palsy:



2) Examination of the pupils:

- Any difference in size must be noted (anisocoria).
- Both light and near reflexes are tested .

Most common pupillary findings[5,9] are,

- ✓ Dilated pupil—in pupil involving third nerve palsy
- ✓ Normal pupil-- in ischemic third nerve palsy
- ✓ Constricted –in cavernous sinus lesion
- ✓ Pseudo Argyll robinson—in aberrant regeneration

3) Visual acuity:

Distant vison by Snellens chart

4) Colour vision:

Colour vision assessed with ishiharas pseudochromatic chart.

5) **Visual fields**:

Assessed with Bjerrums tangential screen and if necessary with automated perimeter

6) Accommodation:

Is tested by using the RAF ruler.

In total third-nervepalsy the near point of accommodation (normally 7-10 cm in front of the eye) recedes farther as the power of accommodation is lost[5,22]

7) Fundus examination:

Done to evaluate for papilloedema, diabetic and hypertensive retinopathy .

8**) Central nervous system:**

All other cranial nerves, motor and sensory system should be thoroughly examined

MANAGEMENT OF THIRD-NERVE PALSY

Five clinical patterns of oculomotor nerves involvement may be determined for effective management as

Table.2 Appropriate investigations for different etiologies of oculomotor nerve palsy[5]

S .no	Presentation	Probable etiology	Investigation
1	Only pupil involvement	Supratentorial mass or aneurysm	MRA
2	Pupil involvement ,EOM palsy and ptosis	Lesion may be anywhere from nucleus to oculomotor nerve,commonly aneurysm	Conventional MRA
3	EOM palsy and ptosis with out pupillary involvement	Most common is ischemia due diabetes and hypertension. If compressive lesion suspected	Blood pressure, random glucose level, and erythrocyte sedimentation rate MRI
4	EOM palsy, ptosis , miotic pupil	Cavernous sinus lesion suspected	MRI with gadolinium
5	Aberrant regeneration	Mostly due to mass lesions	MRI

According to anatomical presentation[2] investigation modalities differ as,

- Innucleus orfascicular lesions is suspected ,MRI[24] or spiral CT is indicated.

- If meningeal signs are presentor is bilateral, CSF examination to be done
- If lesion to be in cavernous sinus, MRI with gadolinium is mandatory[19]
- If lesion to be in orbit, then a spiral CT with contrast, both coronal and axial views is advised

According to age of presentation , investigation modalities differ as[2],

- Patients <50 yrs CT or MRI [24]with MRA[2] should be done.
- Patients >50 yrs, investigation vary with pupillary involvement as
  - In pupil sparing, patient is closely observed with assessment of blood pressure, random glucose level, and erythrocyte sedimentation rate[2,].
  - In pupillary involvement[25],assessment of blood pressure, random glucose level, and erythrocyte sedimentation rate along with CT or MRI with MRA[2] should be done.

TREATMENT

Treatment modalities of isolated oculomotor nerve palsy vary with mode of presentation and probable etiology.

I. Observation:

- Palsy due to ischemic causes usually resolves within 3 months[2,5]
- Due to trauma, usually take longer time to resolve and resolution may be complete or incomplete. mostly it resolves with aberrant regeneration.
- Once paralysis gets stabilised ,resolution after 6 months rarely occurs[2]

II. Specific treatment:

a) Intracranial aneurysm:

After identifying the concerned artery, aneurismal clipping is done.

b) Tolosa hunt syndrome:

High dose Oral corticosteroids [19]for 2-4 weeks followed by gradual tapering over several months is advised. Usually symptoms subside within 72 hours. Other drugs used are cyclosporine, azathioprine and methotrexate.

c) Neurotuberculosis

Resulting in tuberculoma or basal meningitis should be treated with anti tuberculosis treatment(ATT)[2] drugs.

d) Neoplasms[2] :

Excision of mass along with chemotherapy or radiotherapy is considered.

III. **Short term management for squint:**

a, Prismatic correction:

- Fresnel prisms[26] are useful in small deviations
- Advantages of Fresnel press on membranous prisms are easy to apply on the back of spectacles, light weight and cosmetic acceptance.
- Response is not so good due to presence of both horizontal and vertical deviation [1]

b, Botulinum injection:

- Poor response due to more muscle involvement[26].
- Injection into same side, opposing, non paralysed muscle is advisable.
- Diagnostic information about the history of the deviation is masked after Botulinum injection
- Usually response lasts for six months[1]

IV. Surgery for strabismus and ptosis:

A,Complete third palsy[26]

- With complete third-nerve palsy, motility disturbance is corrected first and later ptosis correction is done.
- Lateral rectus recession and Medial Rectus resection should be done if there is medial rectus recovery.
- Supra maximal recession–resection may be required for obtaining a cosmetically acceptable position.
- SO transposition [2] is done to provide an anchoring, adducting force, but the innervation pattern of SO is not appropriate for an adductor and the pattern does not change when muscle is transposed.
- This transposition is effected in one or more ways,namel[1,26]
  i. Trochlea is fractured , the superior oblique tendon is shortened and it is joined with sclera above the medial rectus insertion.
  ii. In Scott procedure, SO is transformed into an adductor and abduction and hypotropic effect is eliminated by suturing SO tendon anterior to insertion of SR muscle .

B, Partial third-nerve palsy that affects chiefly the MR muscle,

- Can be treated by surgery on all four horizontal recti. [1]
- If there is associated vertical recti weakness, the paresis of vertical recti cancel each other and there is purely an exotropia in the horizontal plane.
- In these cases resection of paretic MR muscle with recession of other three horizontal recti muscles are done
- In functional terms, this amounts to weakening of ipsilateral antagonist (LR), the contralateral yoke (LR) and yoke's antagonist (i.e. contralateral MR). This gives good surgical results if adjustable sutures are used which will allow post-operative adjustment for BSV

C, In paresis resulting from involvement of lower division of third-nerve:

- Treatment is by transferring the functioning SR to MR and LR to IR and by tenectomising the SO in the affected eye[1]

D, Ptosis surgery[26]

Initially ocular alignment should be corrected and eventually ptosis correction surgery should be done. Ptosis surgery, principally involves Frontalis sling suspension either using autogenous fascia lata or synthetic materials like supramid, Gore-tex,or silicone.

REVIEW OF LITERATURE

- Warwick (1953) [5] proposed the organisation of the oculomotor nuclear complex into different motor pools serving individual extraocular muscles
- Kerr (1964)[3] proposed that pupillomotor fibers are located superficially in the superior and medial portion of the nerve in the subarachnoid space**.**
- In 1958, CW Rucker[27], published his classic paper reviewing the incidence and aetiology of extraocular muscle paralysis in 1000 patients seen at the Mayo clinic. In his study incidence of isolated nerve palsy was 68.5% and multiple cranial nerve palsies was 31.5% o. In his study 12.4% of cases are due to trauma 20.1% due undetermined etiology,19.3% due to vascular lesion,18.3% due to neoplasms,18.3% due to aneurysms and 13.9% are due to other causes.
- Green et al [28]conducted a study on neuroophthalmic evaluation on oculomotor nerve palsy in 130 patients on 1964 in Wills eye institute. He did his study on only isolated oculomotor nerve palsy and states that right eye involved in 43.1%, left eye in 55.4% and bilateral in1.5%. They noted equal sex distribution of cases. The commonest etiology was aneurysms(29.2%) followed by

undetermined (23.8%) also in this study authors compared the aetiology with pupillary involvement

- Vimala menon et al [29](1984) conducted a study to analyse the etiology of acquired occulomotor nerve palsy in 63 patients at Dr. RP. Centre for ophthalmic sciences, AIIMS, New Delhi and concluded that third cranial nerve was the second most commonly injured nerve next to abducens. The maximum incidence was noted between 11-40 years of age and the common causes attributed are undetermined in 30%, and post traumatic in 22.2% patients. They noted the emergence of idiopathic inflammatory psuedotumors of orbit as an important cause of third nerve palsy.
- A study was conducted by Rush and Younge[30] about paralysis of third ,fourth and sixth cranial nerve palsy in 1981 in 1000 patients. In his study right side paralysis was noted in 47.8% of cases and left side palsy in 48.1% cases.23.1% are due to undetermined cause,16.2% due to trauma, 20.7% due to microvascular ischemia,11.7% due to neoplasms,13,8% due to aneurysms and 14.5% are due to other causes. They concludes that recovery rate from palsy due to microvascular ischemia (71.2%) was far better than post;traumatic (38.9%) and undetermined causes (48.1%)

- An observational case series study was conducted by Abdul-Reza Tabassi[31] et al on etiology of oculomotor nerve palsy in 2001. In their study commonest presenting symptom was lid ptosis in 78.6% cases and mean age of presentation was 50.5 years. They concluded that microvascular ischemia(42.8%) was commonest cause of palsy in which diabetes was noted in 32.1% of cases. Trauma was noted in 14.3% cases, aneurysms in 7.1% cases and tumours in 7.1% of cases. He observed pupil involvement in 11% of ischemic cases.
- Rama et al[32] conducted a study about paralysis of third, fourth and sixth cranial nerve in government hospital, Andhra pradesh. He concluded that the incidence of third nerve palsy is 30%. 18% of palsies are due to trauma, 3 % are due to undetermined cause, 1.1% is due to aneurysms. and 17% due intracranial neoplasms. They noted that incidence of multiple nerve involvement is more than isolated nerve involvement. Neurotuberculosis was noted in 21% of total cases. rare causes as herpes zoster, tolosa hunt syndrome and myasthenia are noted in their study in 16.7% of cases.
- A Study conducted by Richards and Jones[33] about Causes and prognosis in 4278 cases of paralysis of the oculomotor, trochlear and Abducen's cranial nerves in 1992.In their study incidence of

isolated nerve palsy wes 68.1% and multiple nerve palsies was 31.9%.They noted 23.9% of cases are due to undetermined causes,14.7% due to trauma,3.2% due to microvascular ischemia,12.5% due to neoplasms,15.8% due to aneurysms and 13.2% are due to miscellaneous causes

- VP Singh et al[34] conducted a study on Causes and prognosis of paralysis of oculomotor, trochlear and Abducen's cranial nerves and noted over all recovery rate in 50 of cases.

AIM OF THE STUDY

INTRODUCTION:

It is a Case control study conducted on 36 patients of isolated oculomotor nerve palsy in Tirunelveli medical college from January 2013-july 2014 for duration of 18 months.

Patients were followed for 3-6 months to assess the recovery pattern and classified as cases (patients recovered) and controls (patients not recovered)

PRIMARY OBJECTIVE :

- To assess the factors influencing recovery of isolated oculomotor nerve palsy

SECONDARY OBJECTIVE :

- To identify common etiologies of isolated oculomotor nerve palsy

MATERIALS AND METHODS

INCLUSION CRITERIA:

- Isolated oculomotor nerve palsy
- Both congenital and acquired palsy
- Both males and females

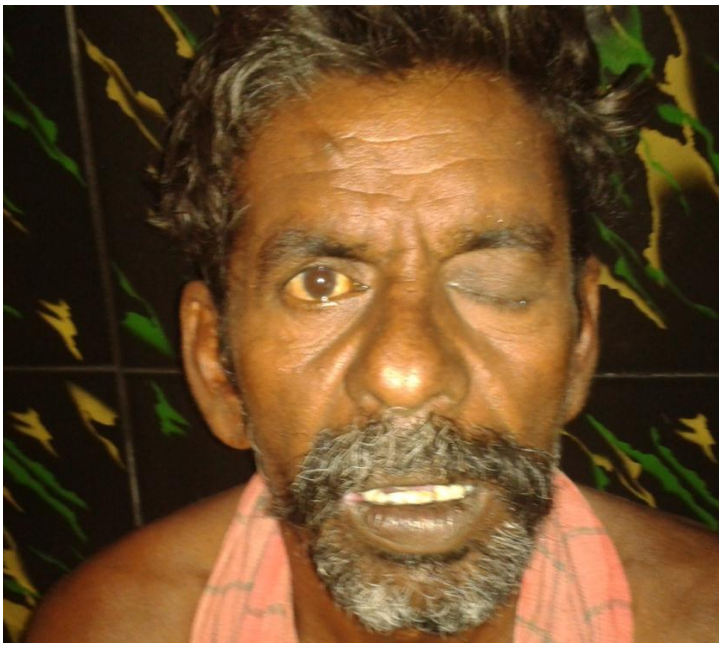
EXCLUSION CRITERIA:

- Terminally ill patients
- Trochlear, abducent nerve involvement

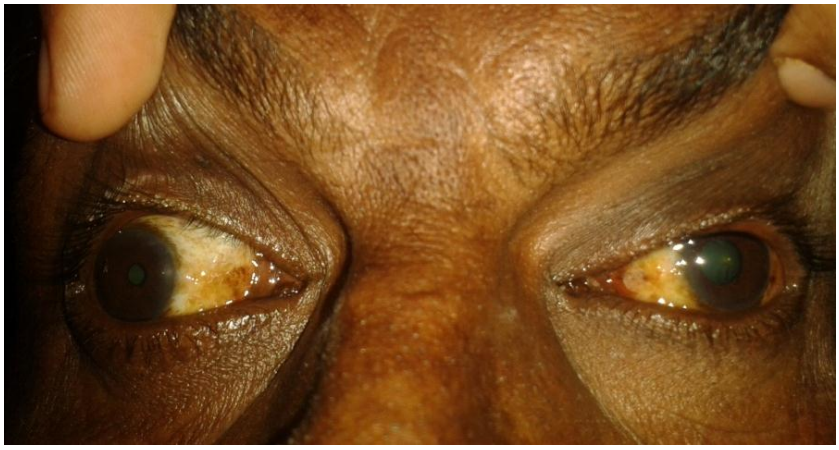
- A detailed history regarding oculomotor nerve palsy was taken as diplopia, headache and drooping of eye lids
- Main emphasis was given to past history of diabetes,hypertension and trauma.
- Examination under oblique illumination included assessment of head posture , ptosis evaluation, extraocular movements, Hirschberg test, Cover test, Prism cover test ,Pupil involvement and Aberrant regeneration.
- Visual acuity and colour vision were assessed in all patients
- Slit lamp to rule out Adies pupil was done.
- Fundus examination should be done to assess presence of optic nerve involvement, papillodema and diabetic retinopathy.

- CNS evaluation was done to assess presence of other cranial nerve palsy, motor, sensory ,autonomic and cerebellar signs.
- Systemic evaluation including blood pressure and random blood sugar was done.
- Neuroimaging was done in the form of Computed tomography of brain and orbit with / without constrast or Magnetic resonance imaging according to etiology suspected.
- Patients were followed up 6 months to assess recovery.

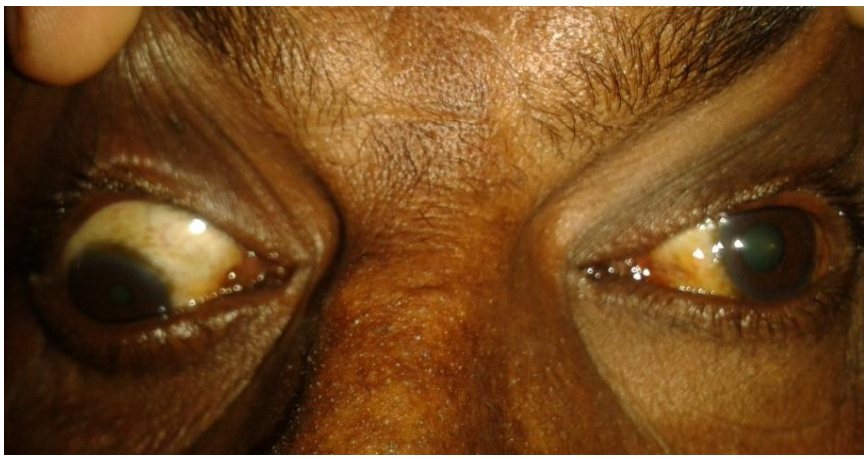
Left complete ischemic oculomotor nerve palsy



Restricted Adduction in left eye

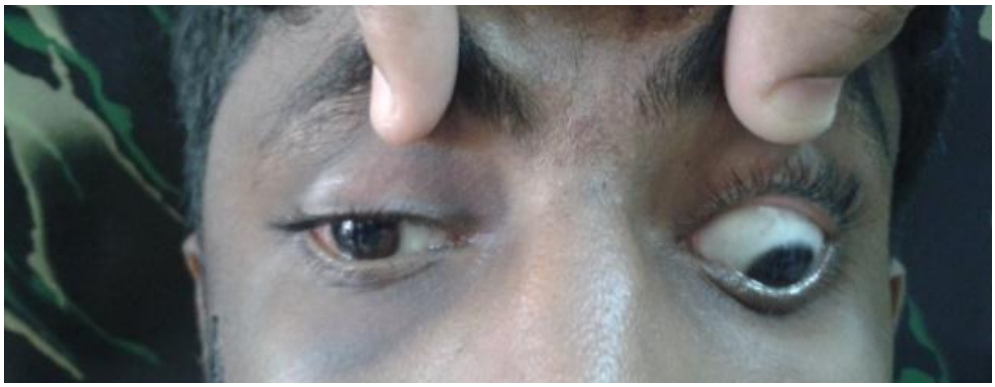


Restricted depression in left eye

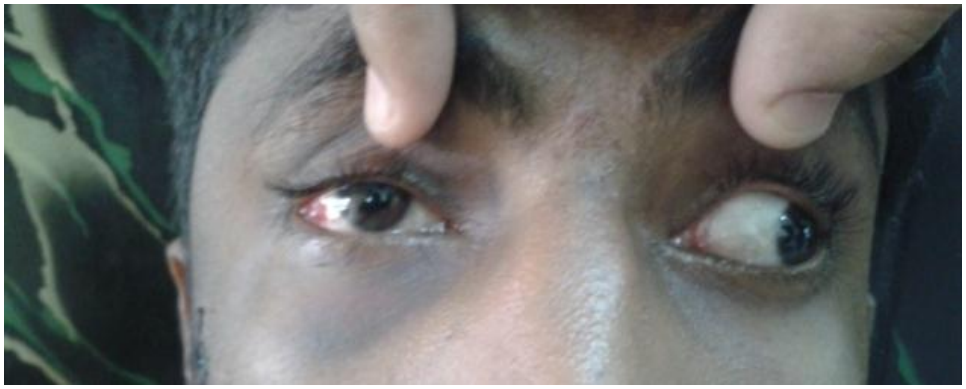


Right incomplete oculomotor nerve paresis due to Trauma

Restricted depression in Right eye

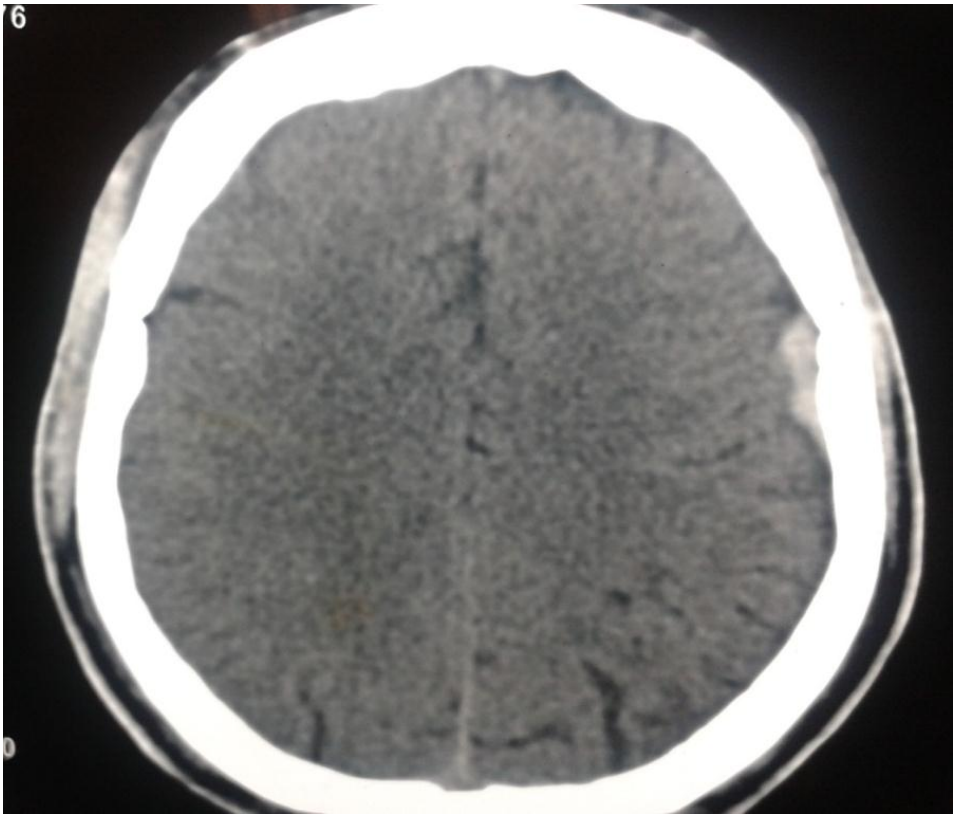


Restricted Adduction in right eye



Right Pupil sparing oculomotor nerve palsy

Right subdural hematoma



RESULTS

Table 3 Age Distribution

Age	Number	Percentage
21-30yrs	**3**	**8.3%**
31-40yrs	**6**	**16.6%**
41-50yrs	**10**	**27.7%**
51-60yrs	**8**	**22.2%**
61-70yrs	**7**	**19.4%**
>71yrs	**2**	**5.5%**

The Age Distribution showed isolated oculomotor palsy in 3 patients in 21-30yrs age group, 6 in 31-40yrs age group,10 in 41-50yrs age group,8 in 51-60yrs age group,7 in 61-70yrs age group and 2 in>71yrs age group. (Table 3 , Fig 15)

Fig 15 Age Distribution

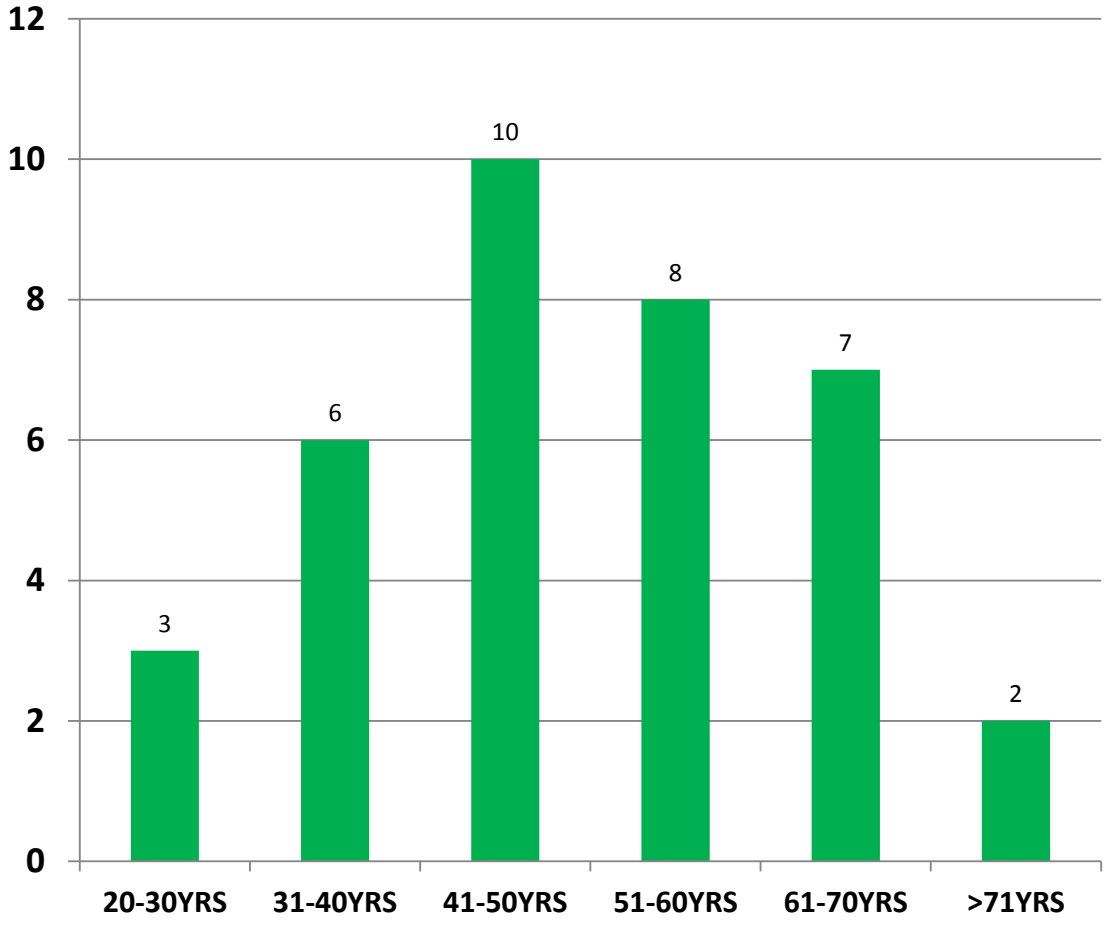
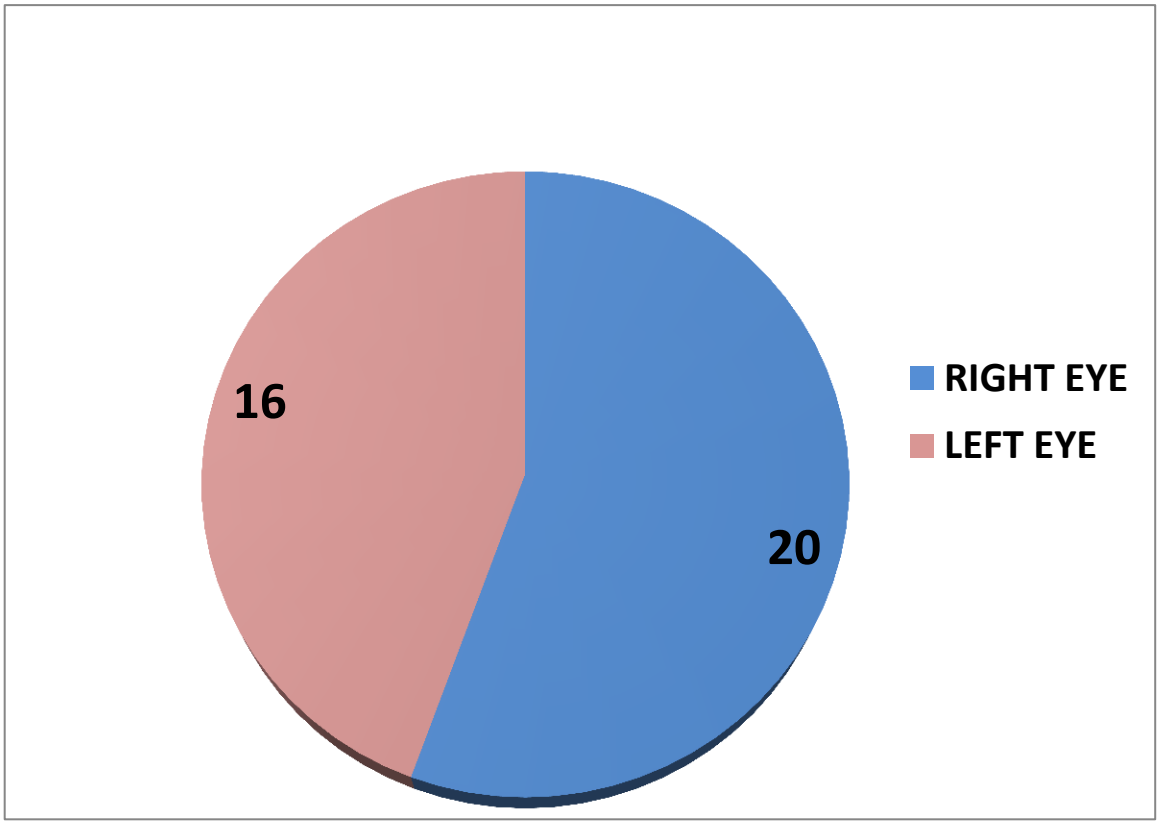
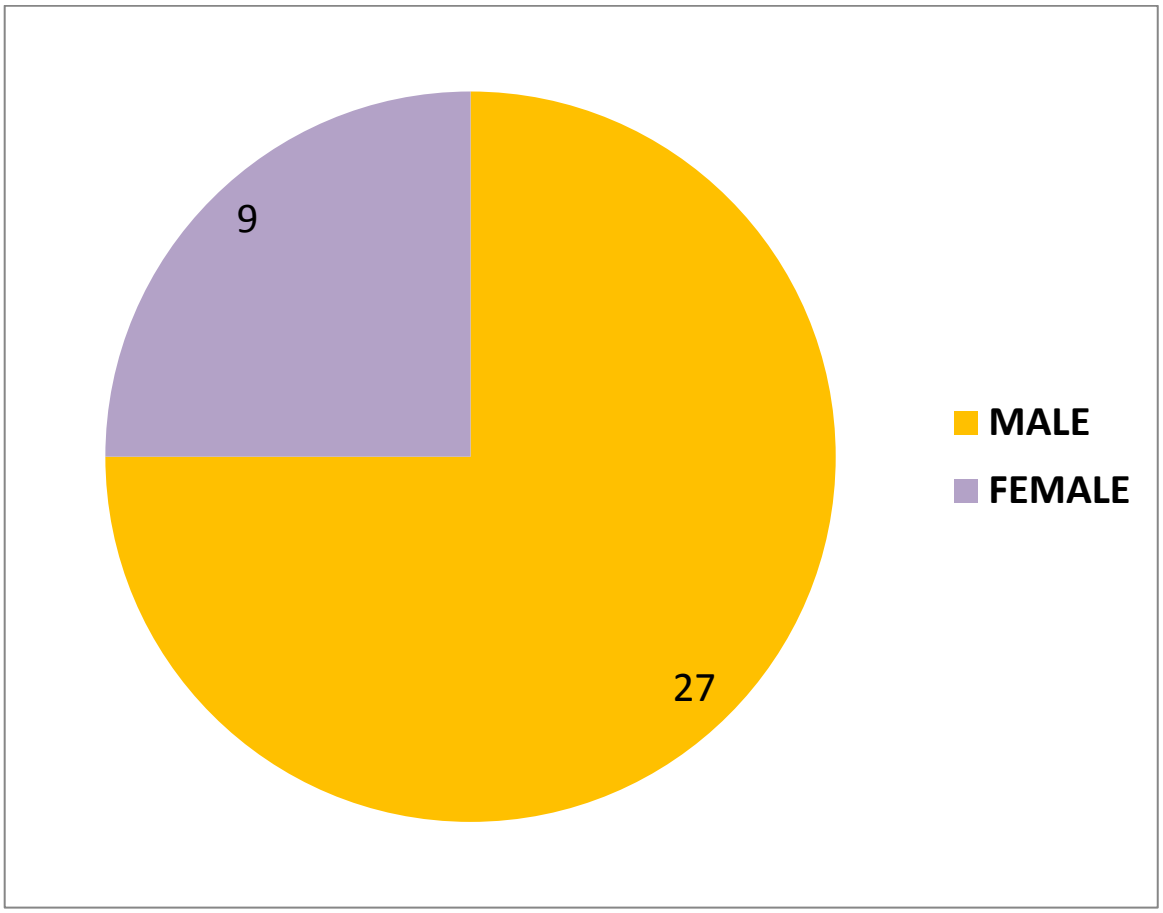


Fig 16 Laterality



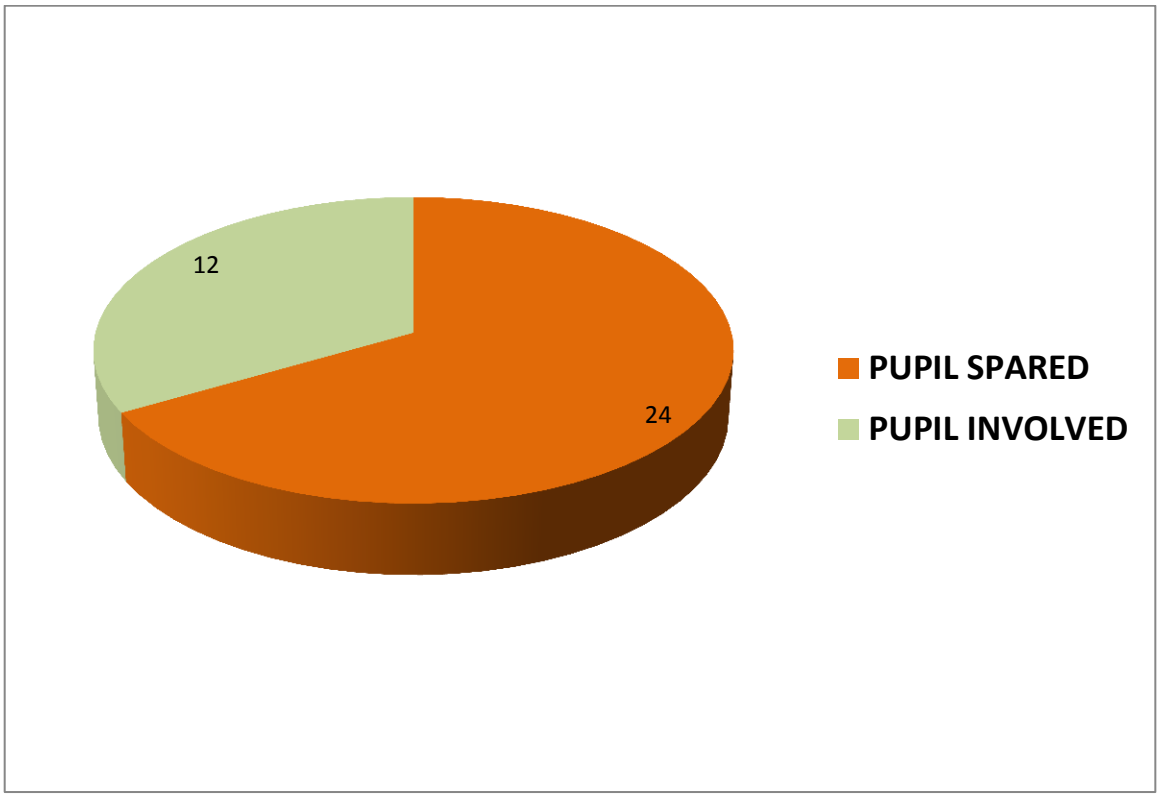
All 36 patients had unilateral oculomotor nerve palsy. Right eye was involved in 16 patients and left eye in 20 patients. No patients had Bilateral palsy.(Fig 16)

Fig 17 Gender Distribution



The Gender Distribution of isolated oculomotor palsy showed 27 males and 9 females.(Fig 17)

Fig 18 Distribution According To Pupillary Involvement



24 patients had pupil sparing oculomotor nerve palsy and 12 patients had pupil involving oculomotor nerve palsy . (Fig 18)

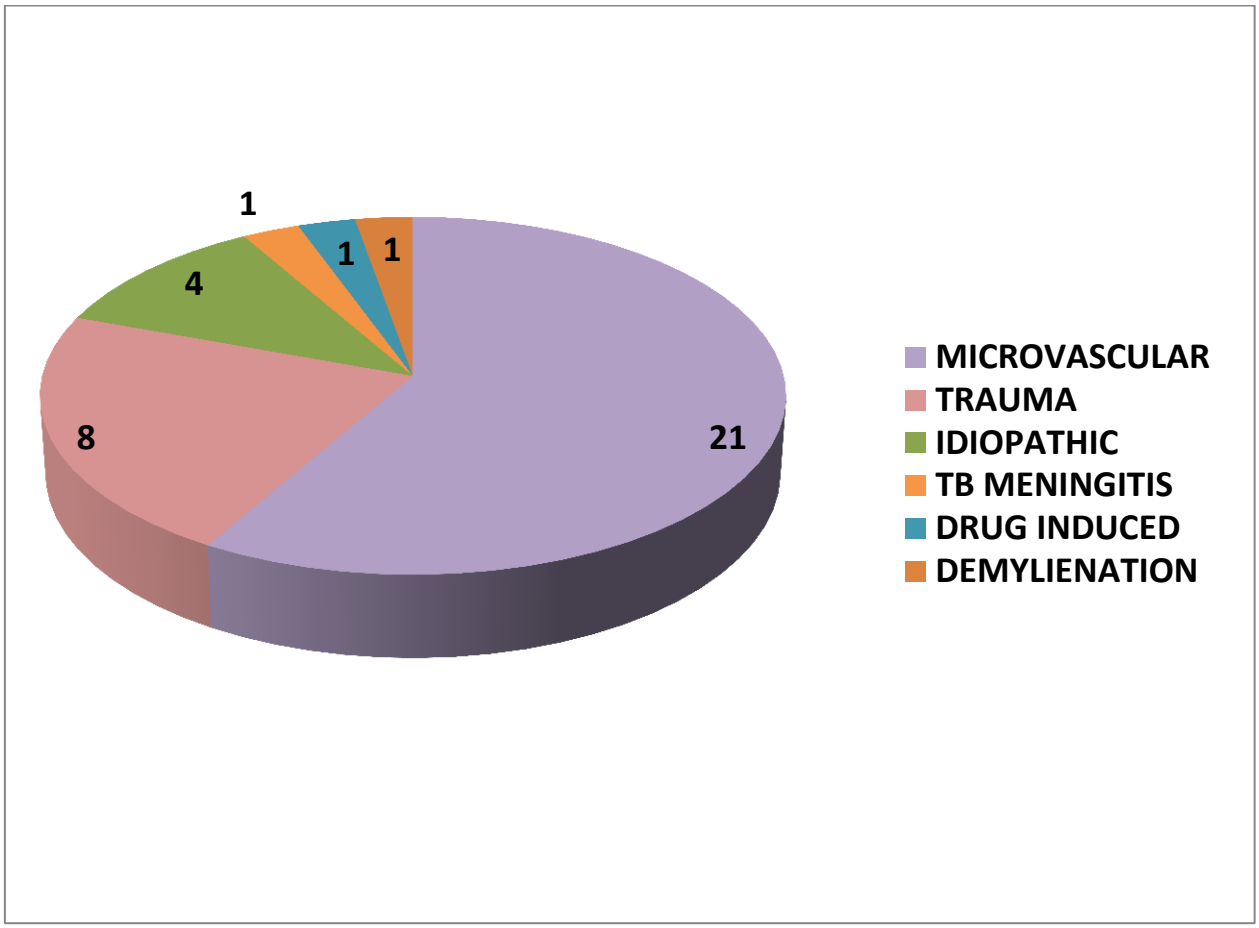
Tab 4 Distribution According To Etiology

Cause	Number	Percentage
Microvascular	**21**	**58.3%**
Trauma	**8**	**22.2%**
Idiopathic	**4**	**11.1%**
Miscellaneous	**3**	**8.3%**

Microvascular ischemia was the cause of isolated oculomotor nerve palsy in 21 patients .Out of 21 patients with Microvascular ischemia 13 had diabetes mellitus and 1 had systemic hypertension and 7 had both.

Trauma was the cause of isolated oculomotor nerve palsy in 8 patients. (Tab 4, Fig 19)

Fig 19 Distribution According To Etiology



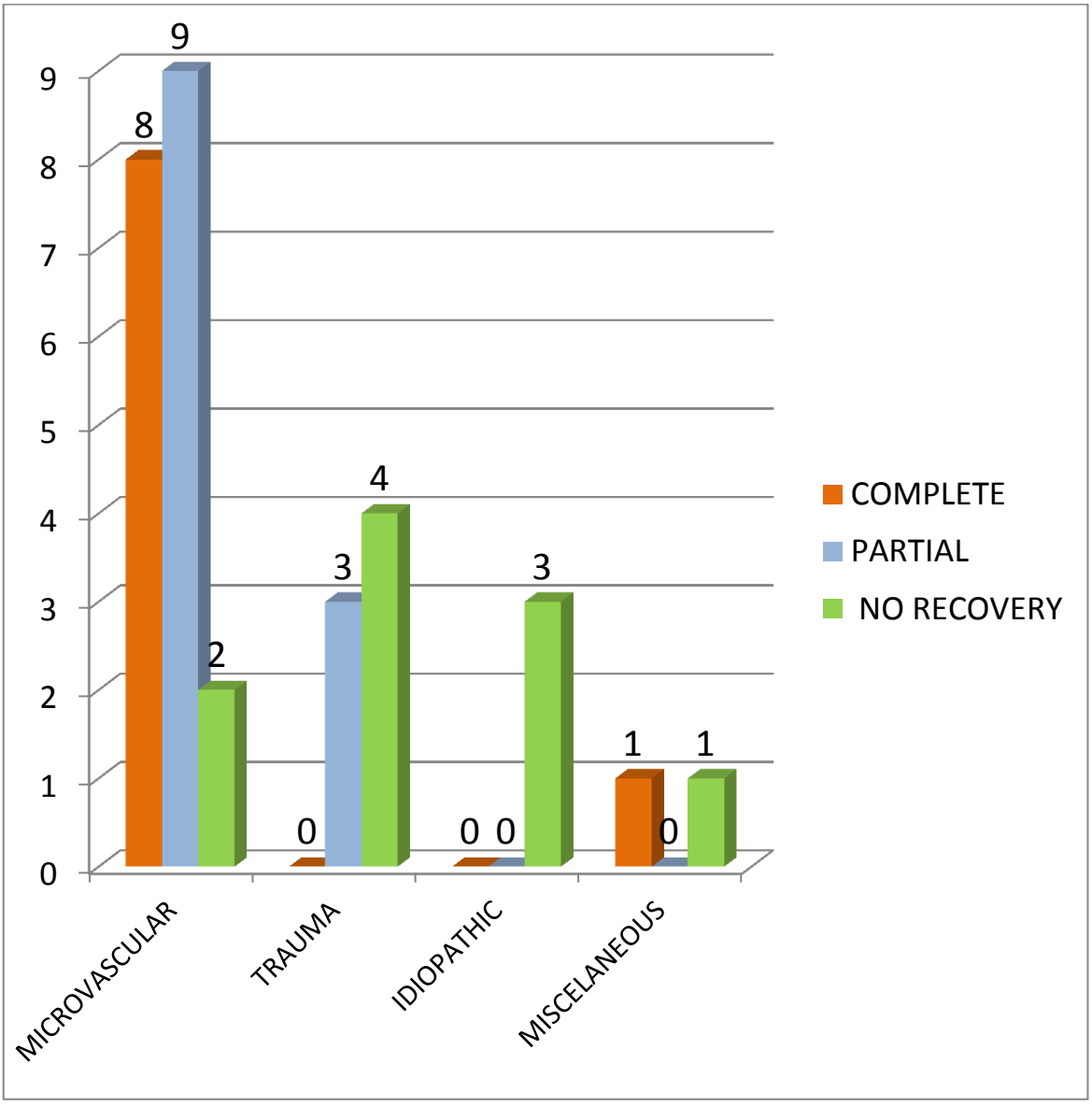
Undetermined cause for palsy was seen in 4 patients.3 other causes include TB meningitis, drug induced and demyelinating disease. (Fig 19)

Tab 5 Recovery at 6months in various Etiologies

	Complete recovery	Partial recovery	No recovery	Lost to followup	P valve 0.014
Microvascular	**8**	**9**	**2**	2	
Trauma	**0**	**3**	**4**	1	
Idiopathic	**0**	**0**	**3**	1	
Miscellaneous	**1**	**0**	**1**	1	

Among 21 patients with Microvascular etiology 8 patients completely recovered, 9 showed partial recovery, 2 patients did not recover and 2 patients were lost for follow up. Among 8 patients , 3 had partial recovery 4 showed no recovery and 1 patient was lost for follow up . (Tab 5, Fig 20)

Fig 20 Comparison Of Recovery With Etiology



Tab 6 Distribution of pupillary involvement in ischemic and non ischemic patients:

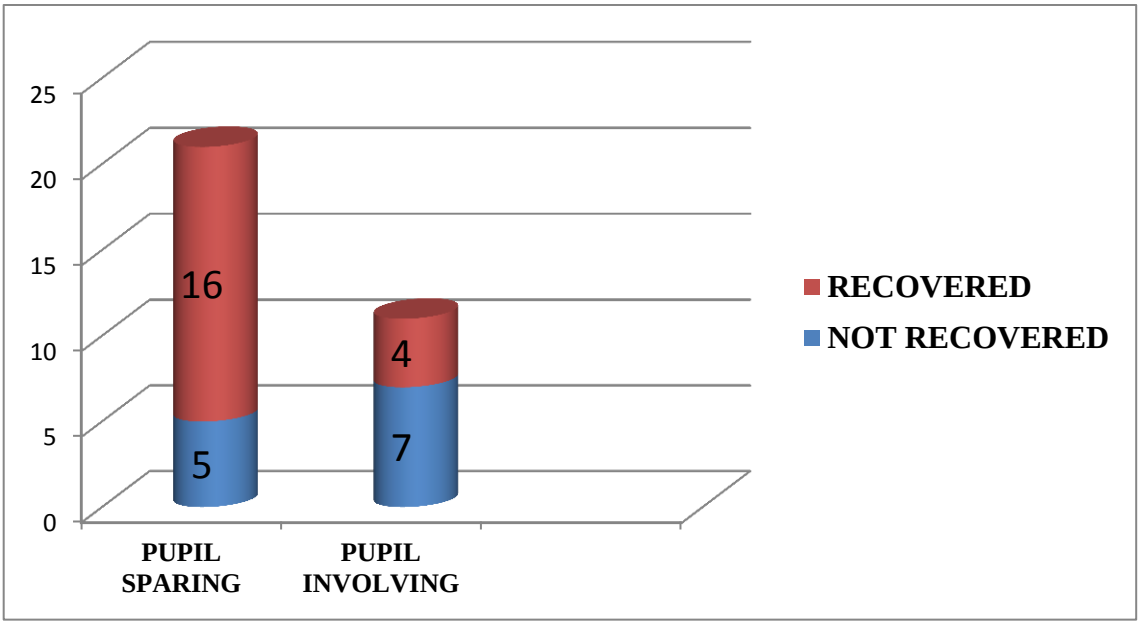
	Pupil Sparing	**Pupil Involving**
Ischemia	**21**	**0**
Non Ischemia	**2**	**13**

Among 21 patients with ischemic etiology ,all of them were pupil sparing nerve palsy. Out of 15 non ischemic patients, 2 were pupil sparing and 13 were pupil involving nerve palsy. (Tab 6)

Tab 7 Comparison Of Recovery With Pupillary Action

	Recovered	Not recovered	Chi - square P value 0.027
Pupil sparing	**16**	**5**	
Pupil involving	**4**	**7**	

Fig 21 Comparison Of Recovery With Pupillary Action

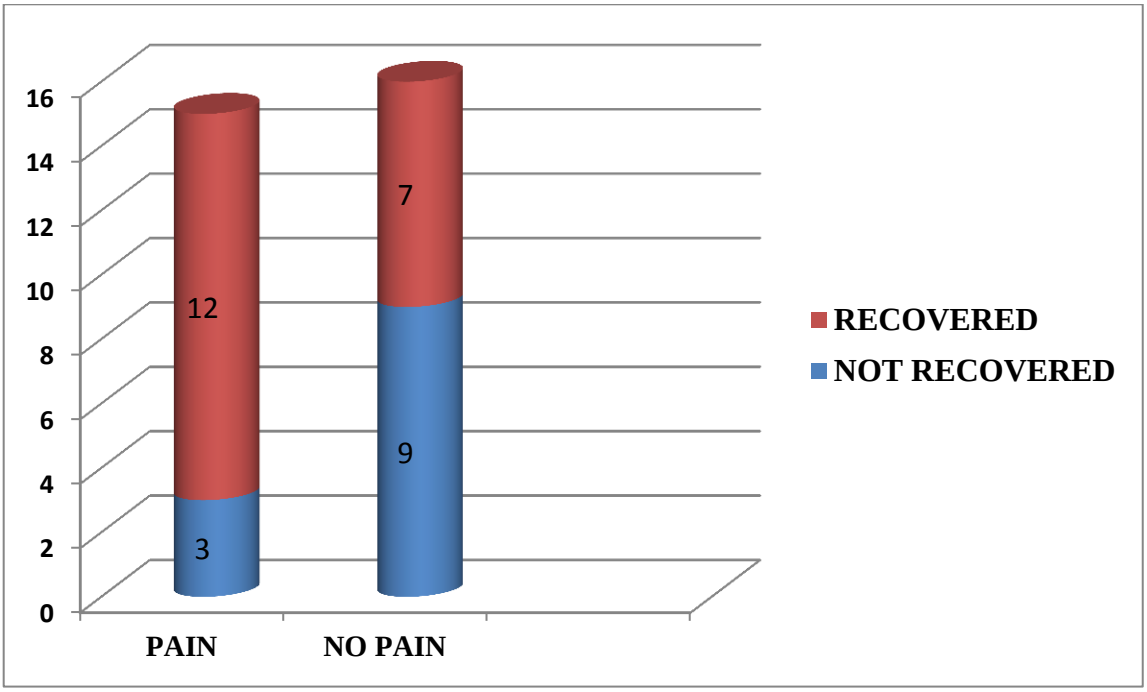


16 patients out of the 21 patients with pupil sparing palsy recovered and 4 out of the 11 patients with pupil involving nerve palsy recovered. (Tab 7, Fig 21)

Tab 8 Comparison Of Recovery With Pain

	Recovered	Not recovered	
Pain	**15**	**2**	**Fischer's Extact**
No pain	**8**	**7**	**P Value 0.049**

Fig 22 Comparison Of Recovery With Pain

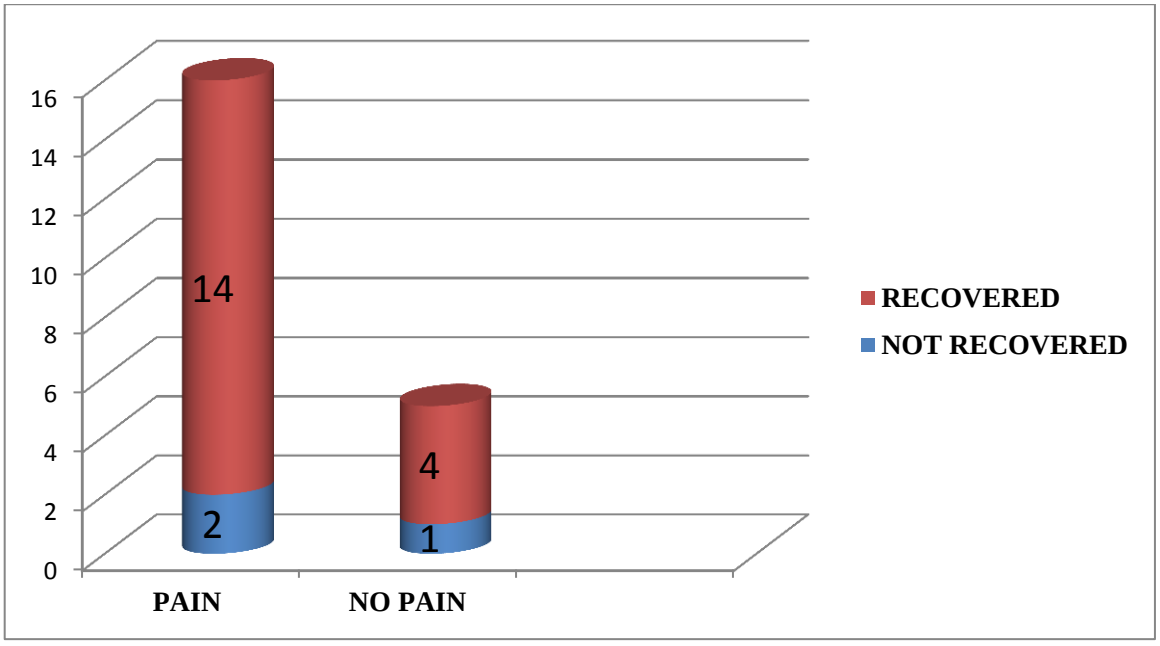


17 patients presented with pain and 15 with no pain. Out of the 17 patients with pain ,15 recovered and 2 had no recovery. (Tab 8, Fig 22)

Tab 9 Comparison Of Recovery With Pain in Palsy due to Ischemia:

	Ischemia		Fischer's exact P value 1
	Recovered	Not Recovered	
Pain	14	2	
No Pain	4	1	

Fig 23 Comparison Of Recovery With Pain in Palsy due to Ischemia:

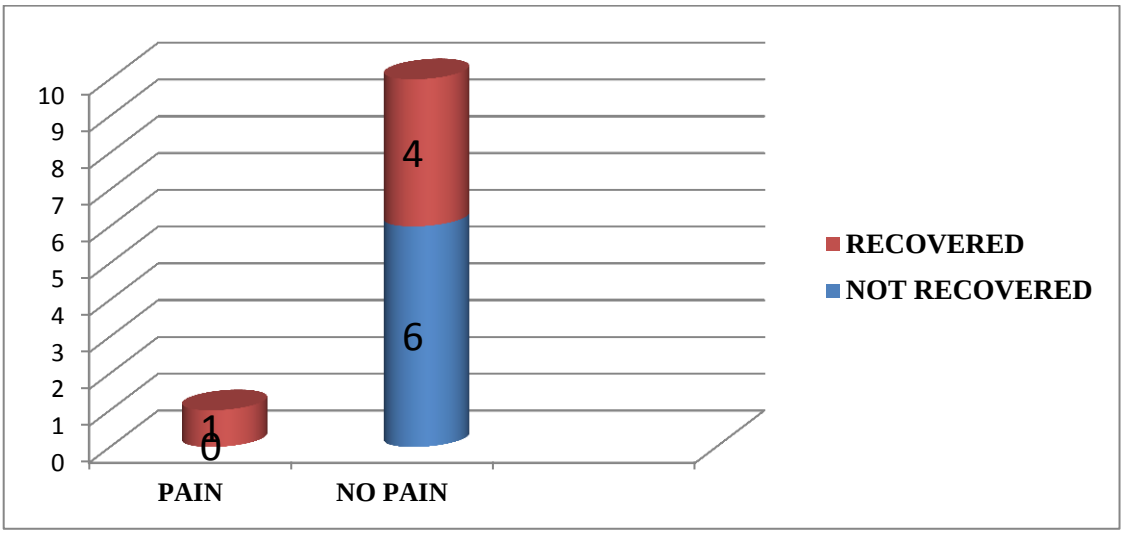


Among 21 patients with ischemia,16 had pain and 5 had no pain.Out of the 16 patients, 14 recovered and 2 did not recover. (Tab 9, Fig 23)

Tab 10 Comparison Of Recovery With Pain in Palsy due to non Ischemia:

	Non Ischemia		Fischer's exact P value 0.455
	Recovered	Not Recovered	
Pain	1	0	
No Pain	4	6	

Fig 24 Comparison Of Recovery With Pain in Palsy due to non Ischemia

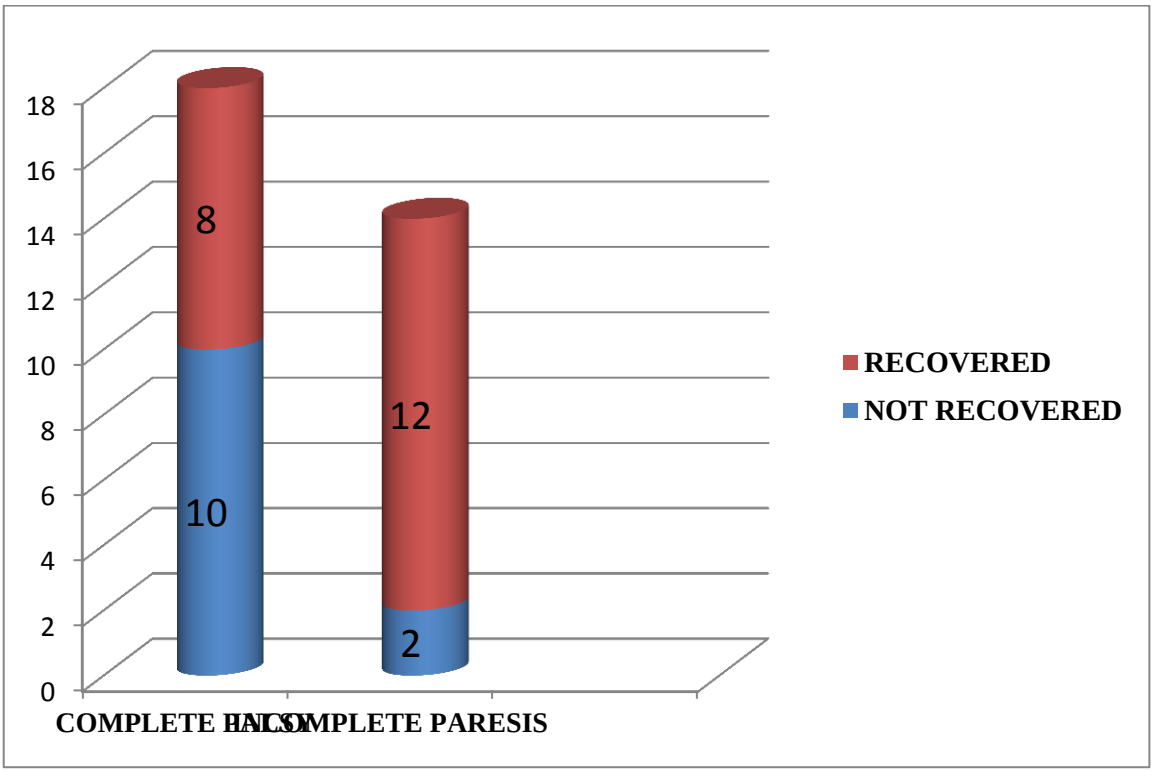


Among 11 patients with non ischemic palsy,1 had pain and 10 had no pain.the 1 patient with pain recovered and out of the 10 patients,4 recovered and 6 did not recover. (Tab 10, Fig 24)

Tab 11- Comparison Of Recovery With Type Of Palsy

	Recovered	Not Recovered	Chi - Square P Value 0.017
Complete Palsy	8	10	
Incomplete Paresis	12	2	

Fig 25- Comparison Of Recovery With Type Of Palsy

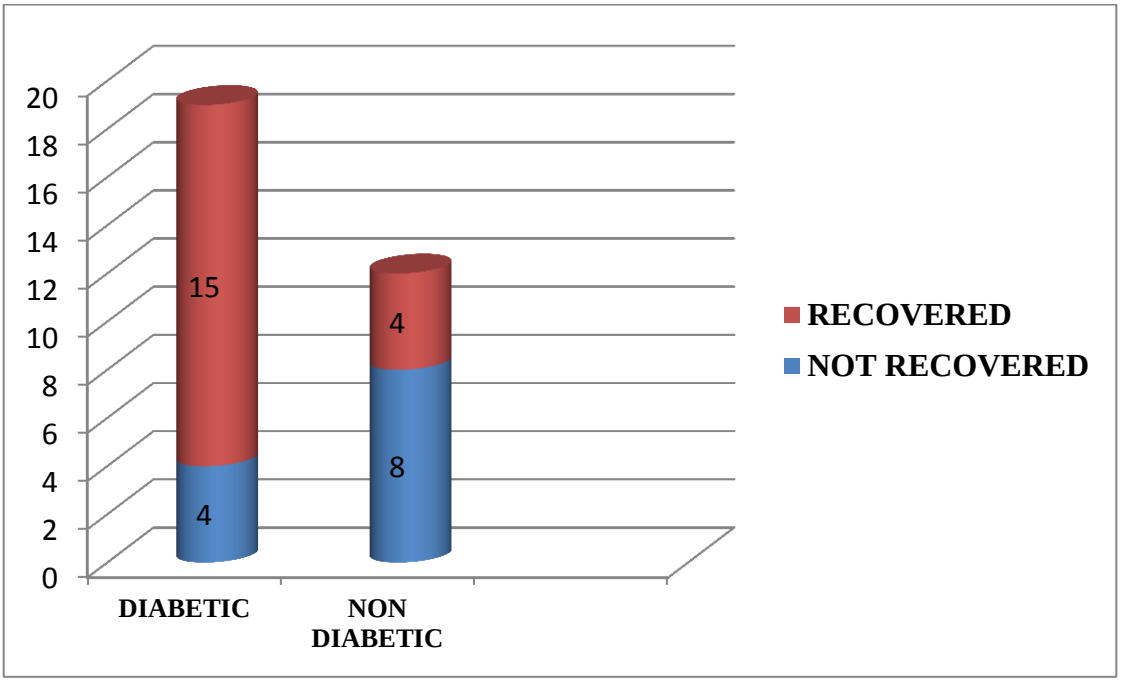


8 of the 18 patients with complete palsy recovered fully. 12 of the 14 patients with incomplete paresis recovered completely. (Tab 11, Fig 25)

Tab 1 2- Comparison Of Recovery With Diabetes:

	Recovered	Not Recovered	Fischer's Exact P Value 0.02
Diabetic	15	4	
Non Diabetic	5	8	

Fig 26 - Comparison Of Recovery With Diabetes

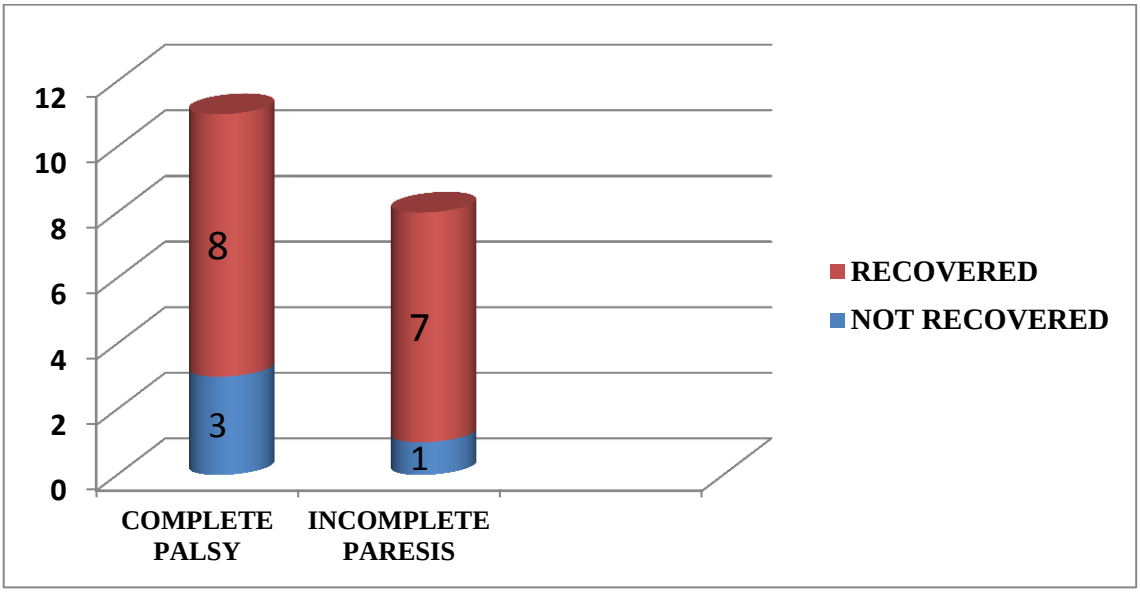


Out of 19 patients with diabetes ,15 patients showed recovery and 4 patients did not recover .Among 13 non –diabetic patients ,5 showed recovery and 8 patients did not recover. (Tab 12, Fig 26)

Tab 13- Comparison Of Recovery With Type Of Palsy In Diabetics

	Recovered	Not Recovered	Fischer's Exact P Value 0.603
Complete Palsy	8	3	
Incomplete Paresis	7	1	

Fig 27- Comparison Of Recovery With Type Of Palsy In Diabetics

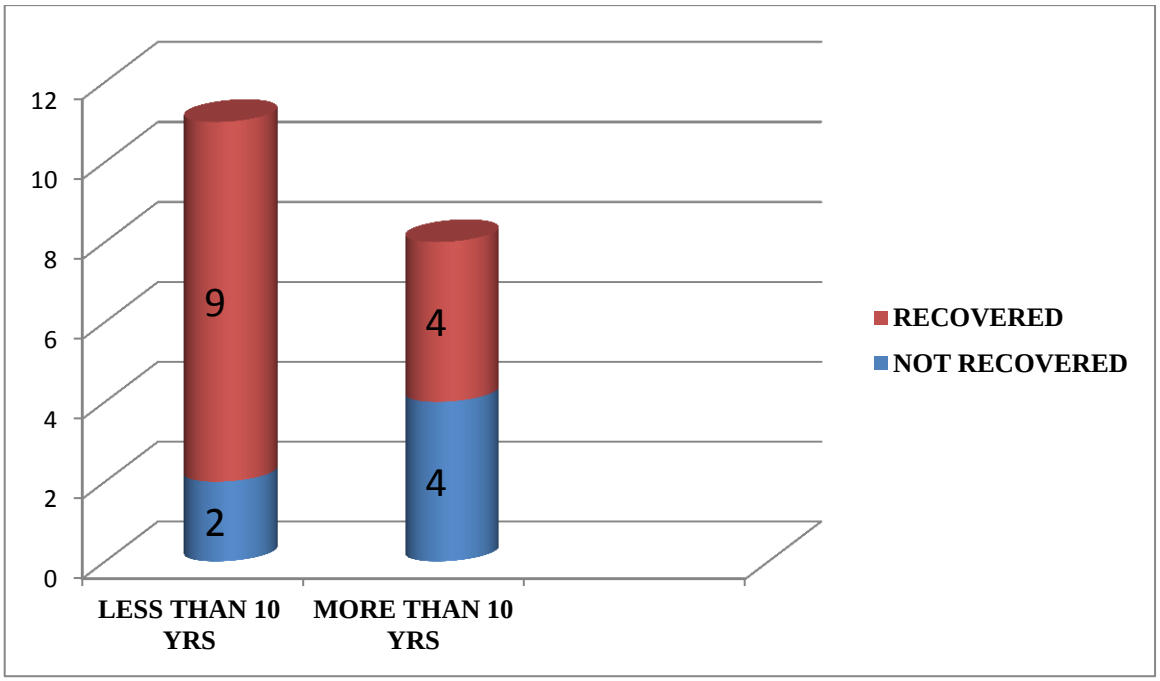


Among 19 diabetic patients, 11 had complete palsy and 8 had incomplete paresis. Out of the 11 patients with complete palsy ,8 patients recovered and 3 did not recover. In the 8 incomplete paresis patients, 7 showed recovery and 1 had no recovery. (Tab 13, Fig 27)

Tab 14- Comparison Of Recovery With Duration Of Diabetes

	Recovered	Not Recovered	Fischer's Exact P Value 0.319
Less than 10 years	9	2	
More than 10 years	4	4	

Fig 28- Comparison Of Recovery With Duration Of Diabetes

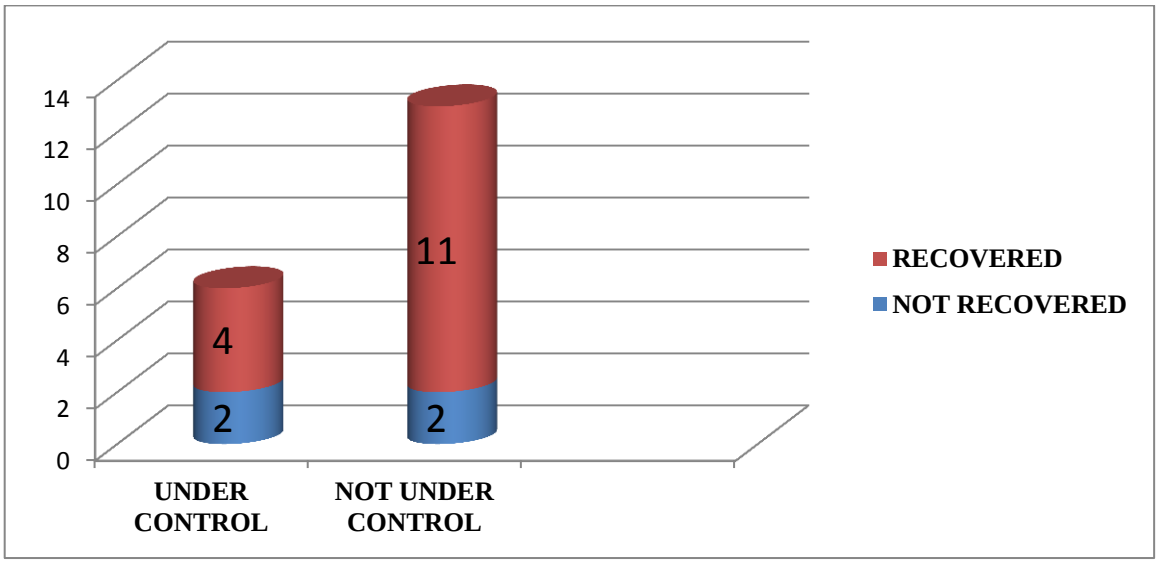


Out of 11 patients with diabetes less than 10 years,9 recovered and 2 did not recover. Among 8 patients with diabetes more than 10 years, 4 recovered and 4 did not recover. (Tab 14, Fig 28)

Tab 15- Comparison Of Recovery With Blood Sugar In Diabetics:

Random blood sugar	Recovered	Not Recovered	Fischer's Exact P Value 0.557
Under control	4	2	
Not under control	11	2	

Fig 29-Comparision Of Recovery With Blood Sugar In Diabetics

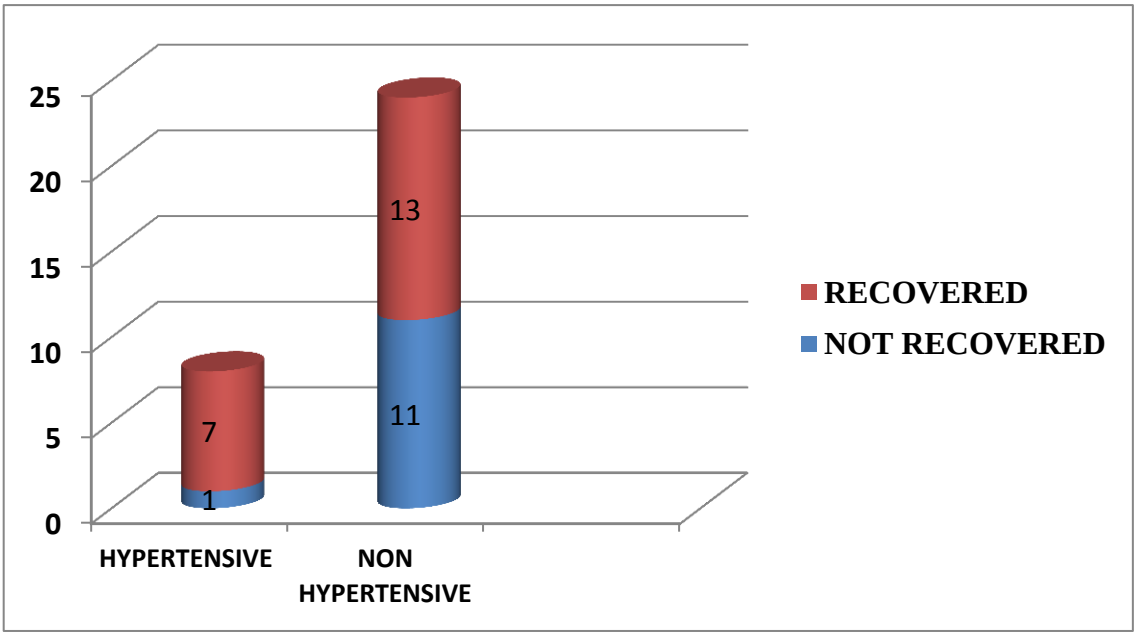


Out of 6 diabetic patients with blood sugar under control 4 patients recovered and 2 did not recover. Among 13 patients with blood sugar under control,11 recovered and 2 did not recover. (Tab 15, Fig 29)

Tab 16- Comparison Of Recovery With Hypertension:

	Recovered	Not Recovered	Fischer's Exact P Value 0.092
Hypertensive	7	1	
Non Hypertensive	13	11	

Fig 30- Comparison Of Recovery With Hypertension

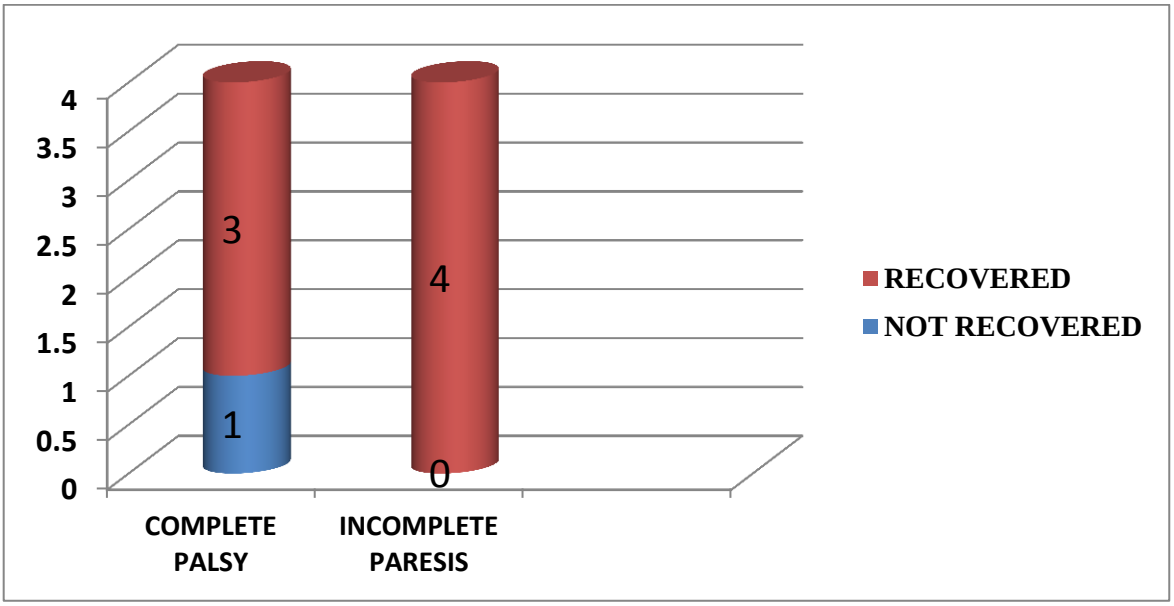


Out of 8 patients with hypertension 7 patients showed recovery from oculomotor nerve palsy and 1 patient did not recover. Among 24 non hypertensive patients ,13 recovered and 11 didnot recover. (Tab 16, Fig 30)

Tab 17 – Comparison Of Recovery With Type Of Palsy In Hypertension **:**

	Recovered	Not Recovered	Fischer's Exact P Value 1
Complete Palsy	**3**	**1**	
In Complete Paresis	**4**	**0**	

Fig31 - Comparison Of Recovery With Type Of Palsy In Hypertension

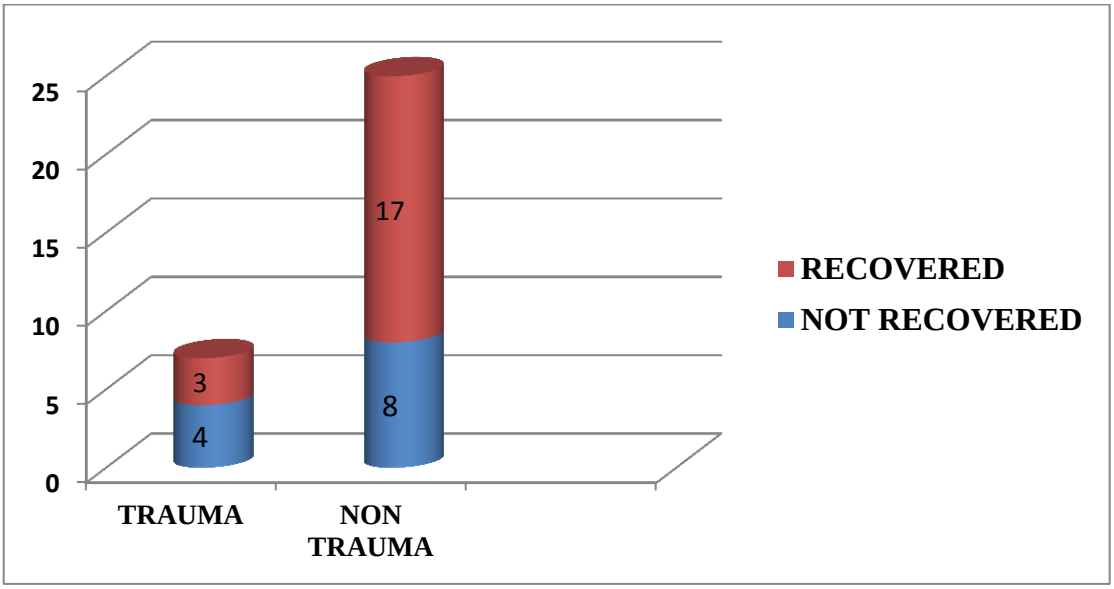


Among 4 hypertensive patients with complete palsy, 3 patients shows recovery and 1 did not recover. Out of hypertensive patients with incomplete paresis ,all of them recovered. (Tab 17, Fig 31)

Tab 18 - Comparison Of Recovery With Trauma:

	Recovered	Not recovered	Fischer's Exact P value 0.379
Trauma	**3**	**4**	
Non trauma	**17**	**8**	

Fig 32 - Comparison Of Recovery With Trauma:

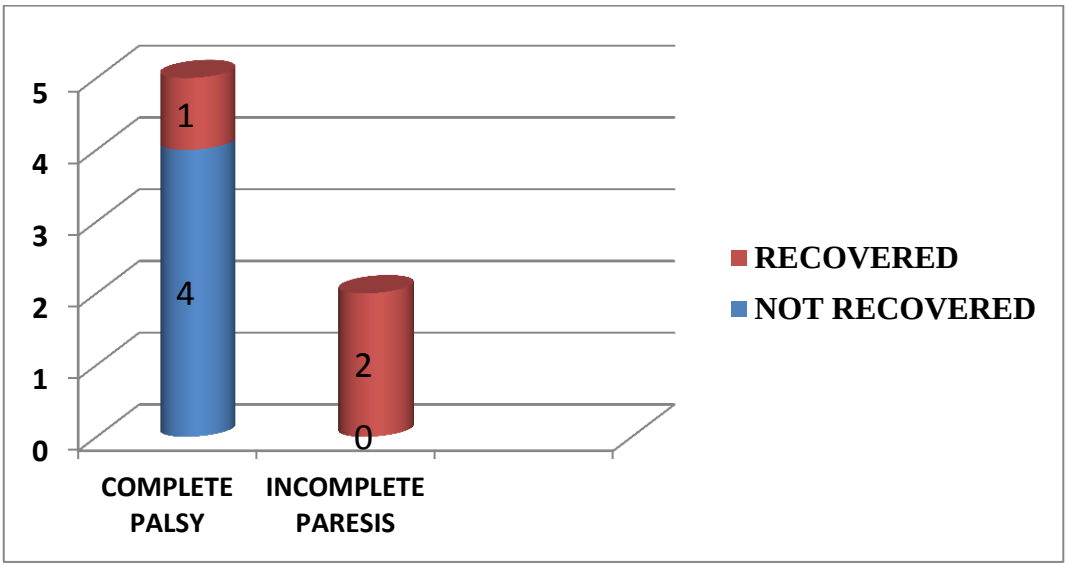


Out of 7 patients with traumatic oculomotor nerve palsy only 3 patients showed recovery and 4 patients had no recovery . Among 25 cases with no trauma 17 patients showed recovery and 8 patients had no recovery. (Tab 18, Fig 32)

Tab 19- Comparison Of Recovery With Type Of PalsyIn Trauma

	Recovered	Not Recovered	Fischer's Exact P Value 0.143
Complete Palsy	**1**	**4**	
In Complete paresis	**2**	**0**	

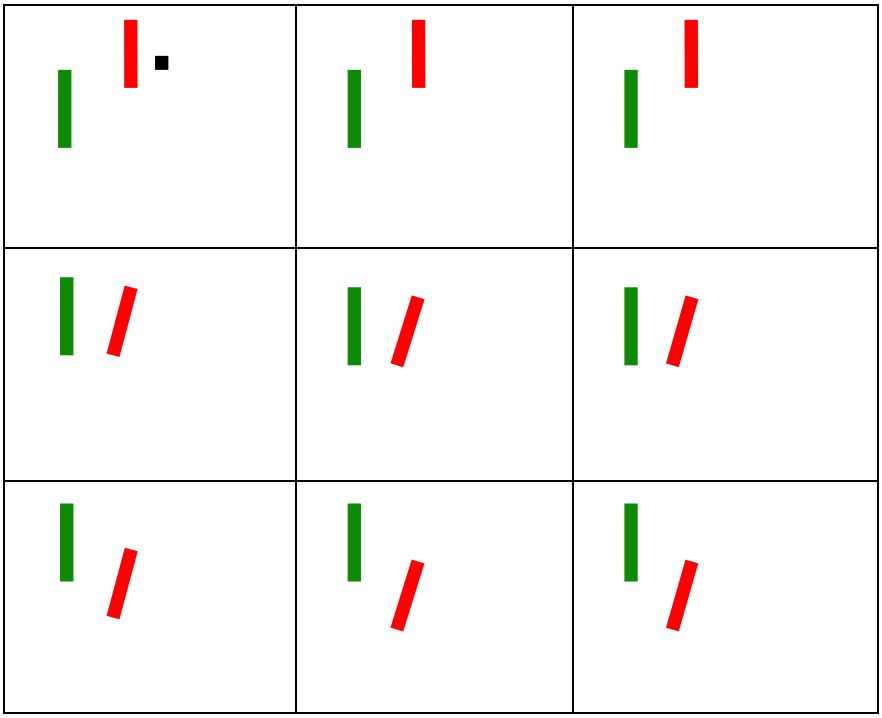
Fig 33 - Comparison Of Recovery With Type Of Palsy In Trauma



Among 5 trauma patients with complete palsy, 1 patient shows recovery and 4 had no recovery . Out of 2 trauma patient with incomplete paresis, both recovered. (Tab 19, Fig 33)

Fig 34 Diplopia Charting In Right Partial Third Nerve Palsy

L R



- **Crossed images**
- **Maximum separation in left gaze**
- **Right over left inelevation and left over right in depression(Fig 34)**

Among 36 patients, 31 patients underwent neuroimaging. Out of the 31 patients, 3 patients had radiological findings as subdural hematoma, haemorrhage in temparoparietal area and infarct in midbrain respectively.

DISCUSSION

Isolated oculomotor nerve palsy is the second most common ocular nerve palsy next to sixth nerve palsy.

In this study various aetiological pattern of was studied and factors influencing recovery are identified.

In this study maximum number of cases belong to fourth decade ie, from 41 to 50 years of age. while in Vimala Menon et al [29]study 11 to 40 years of age was the commonest age group.This may be because we have studid patients as 10 year age groups and Diabetes was the predominant cause in our study.

Maleto female involvement in this study was 3:1.while Greenet al[28] noted equal age distribution .

All cases in this study had unilateral oculomotor nerve palsy.No Bilatera l nerve involvement is noted. This is in contrast with Rush and Younge study[27] where 7.% bilateral nerve involvement is noted.

In this study Right nerve involvement was 55.5% and Left nerve involvement was 45.5% which is comparable with Rush and Younge[27] study in which there are 47.8% Right nerve and 48.1% are Left nerve involvement

Pain was present in 53%of patients and 44% presented with no pain. Among thepatients with pain,88% showed recovery which is

statisticaly significant(p value 0.049).Pain in ischemic cases (p value 1) and pain in non ischemic cases (p value 0.4) didnot show variable difference in recovery due to small sample size.

Internal ophthalmoplegia was noted in 33.3% of the total cases in our study which is comparable to Vimala Menon et al[29] study where Internal ophthalmoplegia was 36%..76% of patients with pupil sparing oculomotor nerve palsy recovered while only 36 % of patients with pupil involving palsy recovered.This is statisticaly significant as p value is 0.027. All patients (100%) with ischemic causes had Internal ophthalmoplegia and 13% of patients with non ischemic causes had Internal ophthalmoplegia..

88% of incomplete paresis patients recovered and only 43% of complete palsy patients showed recovery . This is statistically significant as p value is 0.017.

Microvascular ischemia (58.3%) was the most common etiology in this study which was more compared to Rama et al study 19.3% and Rush and Younge [30]study 20.7%..The higher incidence of microvascular etiology in our study is probably because we have studied only isolated oculomotor nerve palsies and the same is the reason why Trauma is less common cause.89% of patients with microvascular ischemia showed complete or partial recovery in our study. This is comparable with Rush and Younge [30]study which showed 71.2% recovery rate.

Out of microvascular ischemia ,diabetes was the most common cause. 78.9% of diabetic patients recovered while only 38 % of non diabetic patients showed recovery which is statisticaly significant(p value 0.02). Out of 21.1% diabetic patients did not recover,one had infarct in midbrain.In the diabetic patients there was no significant difference seenin recovery based on Type of palsy(p value-0.603).Duration of diabetes (p value 0.319) and blood sugar values (p value 0.557) at te time of presentation.

82% of hypertensive patients recovered while 50% of non hypertensive patients showed recovery .This is not statisticaly significant (p value 0.092) because non hypertensive group includes diabetic patients.Extent of palsy did not influence recovery in hypertensive patients as p value is 1.

Traumatic oculomotor nerve palsy accounts for 22.2% of cases which was comparable to Vimala Menon et al[29]study (22.2%) and Rucker et al study [27](21.2%).

In our study 42.8% of cases with Traumatic oculomotor nerve palsy showed recovery which is comparable to Rush and Younge[30]study which showed 38.9% recovery rate..In the patients with trauma, recovery was not influenced by extent of palsy (p 0.143).

Undetermined causes of oculomotor nerve palsy accounted for 11.1% of total cases which is comparable to Rama et al[32]study

(10.5%)..None of these patients recovered. A long follow up probably could have revealed a detectable cause.

TB Meningitis accounts for 2.7% of cases which is comparable to Vimala Menon et al study[29] (1.6%) and the patient showed complete recovery after ATT.

Demyelinating Poly Radiculopathy is reported in 1 patient (2.7%) . Drug induced palsy is reported in 1 patient(2.7%) and the agent implicated was chlorpromazine.

Overall 65.2% of cases showed complete or partial recovery in our study which was more than Rush and Younge study [30](44.6%) and V.P Singh study[34](50%).

SUMMARY

The following conclusions can be drawn from the present study

- Microvascular ischaemia appear to be the major aetiopathological factor producing oculomotor nerve paralysis which can be easily diagnosed with simple, laboratory investigations and has better chance for complete recovery.
- Diabetes is the most common etiology of isolated oculomotor nerve palsy.
- Most of the diabetic oculomotor nerve palsies recovered with in six months.
- Hypertension is the next common cause of isolated oculomotor nerve palsy and most of the patients recovered with in six months.
- Trauma is the third most common cause of isolated oculomotor nerve palsy and shows poor recovery over time.
- Most of the patients with pupil sparing oculomotor nerve palsy recovered with in 6 months.
- Inspite of neuroimaging, 11% patints were undiagnosed and they require longer follow-ups.

CONCLUSION

- Microvascular ischemia, Pupil sparing palsy and incomplete paresis are the significant indicators of complete recovery determined in my study.

BIBLIOGRAPHY

1. Eggers HM. Oculomotor (Third-Nerve Paralysis). Chapter 41. Part V. In:Master Techniques in Ophthalmic Surgery, Roy FH (ed). Baltimore: Williams and Wilkins 1995; 303-307.
2. Newman NJ. Third-Fourth- and Sixth-Nerve Lesions and Cavernous Sinus.Chapter 284. In: Principles and Practice of Ophthalmology, Albert DM,Jakobiec FA (eds). 2nd ed. Vol. 5. Philadelphia: WB Saunders Company2000; 3992-4028.
3. Duke Elder S. Ocular Motility and Strabismus. In: System of Ophthalmology,Duke Elder S (ed). London: Henry Kimpton; 1974.
4. Duke Elder S, Scott GI. The Motor System. Section III. In: Neuroophthalmology in System of Ophthalmology, Duke Elder S (ed).London: Henry Kimpton 1974; 713-793.
5. Miller NR. The ocular motor system: Embryology, Anatomy, Physiology andTopographic diagnosis. Section IV. In: Walsh and Hoyt's Clinicalneuro-ophthalmology. 4th ed. Vol. 2. Baltimore: Williams and Wilkins1985; 559-652.
6. Von Noorden GK. Binocular vision and ocular motility. 5th ed. St. LouisMosby; 1996
7. Optican LM. Extarocular muscles/eye movements. Section 14. In: Adler'sphysiology of the eye, Kaufman PL, Albert Alm (eds). 10th edition. St.Louis: Mosby 2003; 787-858
8. AAO,Neuroophthalmology,sec 7,2010-2011,chapter 1:55

9. Glaser JS, Siatkowski RM. Infranuclear disorders of eye movement. Chapter12. In: Neurophthalmology, Glaser JS (ed). Philadelphia: LippincottWilliams and Wilkins. 3rd ed. 1999; 405-460.
10. Sadun AA. Neuro-ophthalmology.Part 11. In: Ophthalmology, Yanoff M,Duker JS. 2nd ed. St. Louis: Mosby 2004; 1239-1410.
11. Bron AJ, Tripathi RC, Tripathi BJ. The extraocular muscles and ocularmovements.Chapter IV. In: Wolff's Anatomy of the eye and orbit.8th ed. London: Chapman and Hall Medical 1997; 107-177..
12. Khurana AK, Khurana I. Extraocular muscles and ocular motility. Chapter 8. In: Anatomy and physiology of the eye. 1st ed. New Delhi: C B SPublishers 1998; 253-293.
13. Michael and Brodsky,childhood oculomotor nerve palsy Paediatrics neurology**:**
14. Kestenbaum A. Clinical methods of neurophthalmologic examination. 2nd ed. Bombay: Kothari Book Depot 1956; 267-283.
15. Kanski JJ. Clinical ophthalmology. 5th ed. London: Butterworth Heinemann2003; 630
16. Walter KA, Newman NJ, Lessel S. Oculomotor palsy from minor headtrauma:Initial sign of intracranial aneurysm. Neurology 1994;44:148-150
17. Hopf HC, Gutmann L. Diabetic 3rd nerve palsy: Evidence for a mesencephalic lesion. Neurology 1990; 40:1041-1045
18. O'Conner PS, Tredici TJ, Green RP. Pupil sparing third-nerve palsy caused byaneurysm. AJO 1983; 95:395-397.

19. Keane JR. Cavernous sinus syndrome, analysis of 151 cases. Arch Neurol1996; 53:967-971.
20. Jacobson DM, McCanna TD, Layde PM. Risk factors for ischaemic ocularmotor nerve palsies. Arch Opthalmol 1994; 112:961-966.
21. Putterman AM. Basic oculoplastic surgery.Chapter 33. In: Principles andpractice of ophthalmology, Peyman GA, Sanders DR, Goldberg MF.New Delhi: JP Brothers 1987; 2246-2273
22. Lyle TK, Wybar KC. Lyle and Jackson's Practical orthoptics in the treatment of squint. 5th edition. New Delhi: JP Publishers; 1979.
23. Wright KW. Strabismus and Amblyopia. Section III. In: PaediatricOpthalmology and Strabismus, Wright KW (ed). St. Louis: Mosby1995; 89-250.
24. Trobe JD, Jacobson DM. The emerging role of magnetic resonanceangiography in the management of patients with third cranial nerve palsy. AJO 1999; 128:94-96.
25. Trobe JD. Third-nerve palsy and the pupil. Arch Ophthalmol 1988; 106:601-602
26. Simons BD. Surgical management of ocular motor cranial nerve palsies.Seminars in Opthalmol 1999; 14:81-94.
27. Rucker CW. The causes of paralysis of the third, fourth and sixth cranialnerves. AJO 1966; 61:353-358.
28. Green WR, Hackett ERF, Schlezenger NS. Neuro ophthalmologic evaluation of oculomotor nerve paralysis. Arch Ophthalmol 1964; 72:154-167.

29. Menon V, Singh J, Prakash P. Aetiological patterns of ocular motor nervepalsies. IJO 1984; 32:447-453.
30. Rush JA, Younge BR. Paralysis of cranial nerves III, IV and VI. ArchOphthalmol 1981; 99:76-79.
31. Abdul-Reza Tabassi MD,Etiology of Oculomotor Nerve Paralysis,Tehran medical centre:
32. Rama V, Vimala J, Chandrashekar M, Anjaneyulu C, Dinakar I. Ophthalmoplegia, a study of ninety cases. IJO 1980; 28:13-16
33. Younge BR, Jones FR Jr, Richards BW. Causes and prognosis in 4278 cases of paralysis of the oculomotor, trochlear and Abducen's cranial nerves. AJO 1992; 113:489-496.
34. Singh VP, Gupta M, Sarkar P, Nema N, Gulati R, Wadhva P. Causes andprognosis of paralysis of oculomotor, trochlear and Abducen's cranial prognosis of paralysis of oculomotor, trochlear and Abducen's cranialnerves. All India Ophthalmic Society Year Book; 2000

PROFORMA

Serial no:

Hospital no: MLC/NonMLC ;Date :

Name Age <20,20-40,40-60,>60 Sex- M/F

Occupation and income

Address

Complaints

HISTORY :

Duration of complaints-<1 week/1week-1month/1month-3month/>3months

Pain-yes/no, Drooping of lids-yes/no

Diplopia –yes/no,Defective vision-yes/no

Headache –throbbing,nausea,vomiting,convulsion

PAST HISTORY

DM/HTN/Previous episodes/injury/Tuberculosis

PERSONAL HISTORY;

Diet/smoking/alcohol/exposure to STD/Menstrual history

FAMILY HISTORY ;

TREATMENT HISTORY:

GENERAL EXAMINATION:

Pulse rate: Blood pressure:

Neurocutaneous marker-yes/no

OCULAR EXAMINATION:

Head posture-chin lift-yes/no,

face turn-yes/no,

head tilt-yes/no

Facial symmetry

Extent of strabismus-Hirschberg test

Exophthalmos/enophthalmos

RIGHT EYE **LEFT EYE**

Extraocular movements

Lid

Cornea

Pupils

Ptosis-measurement-MRD1- LPS action -

MRD2- Lid crease

Orbit-Trauma signs-yes/no

FUNDUS-Optic atrophy-yes/no

VISION

COLOUR VISION

FIELD OF VISION

DIPLOPIA CHARTING

OTHERS

CENTRAL NERVOUS SYSTEM:

Conscious/unconscious, Oriented/disoriented, Memory, Speech

Motor system

Sensory system

CRANIAL NERVE EXAMINATION:

SPECIALTY OPINION: Neurosurgery / Neuromedicine

INVESTIGATIONS: Blood Sugar

Xray Skull

CT Scan –Brain&Orbit

MRI Brain&Orbit

Others

FINAL DIAGNOSIS :

TREATMENT: MEDICAL/SURGICAL/OTHERS

FOLLOW UP VISITS:

MASTER CHART

S .No	Name	Age	Sex	PS/PI	Duration			RT/LT	Pain	Etiology					Investigations		Recovery
					<1 WK	1WK-1MNTH	>1MNTH			DM	HTN	TRAUMA	IDIOPATHIC	OTHERS	CT	MRI	COMPL/PART/NO
1	Jothilakshmi	45	F	PI		YES		LE	NO				YES			NORMAL	NO
2	Ramalaksman	60	M	PS		YES		LE	NO	YES							COMPLETE
3	Marikrishnan	32	M	PS	YES			LE	YES	YES							COMPLETE
4	Kandasamy	58	M	PS		YES		RE	YES	YES						NORMAL	PARTIAL
5	Arokiaselvi	49	F	PI	YES			RE	NO			YES			NORMAL		NO
6	Thirumalai	26	M	PI	YES			RE	NO			YES				HMGE TEMPEROPARIETAL LOBE	PARTIAL
7	Lakshmi	65	F	PS	YES			LE	YES	YES							COMPLETE
8	Ahamedsheik	21	M	PI			YES	RE	YES					DEMYELINATING			LOST
9	Thangalatha	37	F	PI		YES		RE	NO			YES			#MEDIAL WALL RT MAX SINUS		PARTIAL
10	Sundarajan	63	M	PS			YES	LE	NO	YES							NO
11	Joseph	69	M	PS		YES		RE	NO	YES	YES					NORMAL	COMPLETE
12	Balakrishnan	55	M	PS		YES		RE	YES	YES	YES					NORMAL	PARTIAL
13	Mahabuzan	72	M	PS			YES	RE	NO	YES						NORMAL	COMPLETE
14	Prabakar	45	M	PS			YES	LE	YES	YES						NORMAL	PARTIAL
15	Subhudabeevi	60	F	PS			YES	RE	NO					PSYCHOSIS	NORMAL		LOST
16	Antonycruz	40	M	PS	YES			LE	YES				YES			NORMAL	LOST
17	Janarthanan	65	M	PS		YES		RE	YES	YES						NORMAL	NO
18	selvaraj	70	M	PS		YES		RE	YES		YES				SDH		COMPLETE
19	Thomaswalter	59	M	PS			YES	RE	YES	YES						NORMAL	NO
20	Thilagarjebaraj	53	M	PS	YES			LE	NO	YES	YES					NORMAL	COMPLETE
21	Chandran	50	M	PS		YES		LE	YES	YES						NORMAL	PARTIAL
22	Sadhananthan	44	M	PI	YES			LE	NO			YES			NORMAL		NO
23	Subburaj	71	M	PS		YES		RE	YES	YES	YES				NORMAL		COMPLETE

24	Petchiammal	43	F	PI	YES			RE	NO				YES			NORMAL	NO
25	Kannan	29	M	PI	YES			LE	NO			YES			NORMAL		NO
26	Arociaraj	49	M	PS		YES		RE	YES	YES	YES					NORMAL	PARTIAL
27	Dhavamani	62	F	PS		YES		LE	YES	YES					NORMAL		PARTIAL
28	Eswari	35	F	PI	YES			RE	NO			YES			NORMAL		LOST
29	Muthupandi	52	M	PI		YES		LE	NO			YES			NORMAL		NO
30	Stephenraj	48	M	PS	YES			RE	YES	YES						NORMAL	COMPLETE
31	Madathi	64	F	PI			YES	RE	NO				YES		NORMAL		NO
32	Abdulkather	50	M	PS	YES			LE	YES	YES						NORMAL	PARTIAL
33	Karunakaran	36	M	PS		YES		LE	YES	YES	YES				NORMAL		PARTIAL
34	Palanisamy	50	M	PS		YES		LE	YES	YES	YES				INFARCT		NO
35	Mathavan	59	M	PI	YES			RE	NO			YES			NORMAL		PARTIAL
36	Jawahar	34	M	PI	YES			RE	YES					TB MENINGITIS	NORMAL		COMPLETE
	PS	-	Pupil Sparing														
	PI	-	Pupil Involving														
	RT	-	Right Eye														
	LT	-	Left Eye														
	DM	-	Diabetes Mellitis														
	HTN	-	Hypertension														
	CT	-	Computed Tomography														
	MRI	-	Magnetic Resonance Imaging														
	COMPL	-	Complete Recovery														
	PART	-	Partial Recovery														
	SDH	-	Sub Dural Hematoma														