Dissertation on

"A CLINICAL STUDY ON OCULAR MOTOR NERVE PALSIES FOLLOWING TRAUMA

Submitted in partial fulfillment of requirements of

M. S. OPHTHALMOLOGY

BRANCH III

of

REGIONAL INSTITUTE OF OPHTHALMOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003

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

CHENNAI-600 003

MAY 2020

CERTIFICATE

This is to certify that this dissertation entitled **"A CLINICAL STUDY ON OCULAR MOTOR NERVE PALSIES FOLLOWING TRAUMA"** is a bonafide record of the research work done by **Dr.A.SHAKILA**., Post graduate in Regional Institute of Ophthalmology, Madras Medical College and Research Institute, Government General Hospital,Chennai-03, in partial fulfillment of the regulations laid down by The Tamil Nadu Dr.M.G.R. Medical University for the award of M.S.Ophthalmology Branch III, under my guidance and supervision during the academic years 2017-2020.

Prof. Dr. M.V.S. PRAKASH.,MS.,DO.,
Head Of the Department,
Department of Orbit & Oculoplasty ,
Regional Institute of Ophthalmology
Madras Medical College & Research Institute,
Govt. General Hospital,
Chennai – 600 008

Prof.Dr. M.ANANDA BABU, M.S. D.O.,
Director and Superintendent,
Regional Institute of Ophthalmology
Madras Medical College & Research Institute,
Govt. General Hospital,
Chennai – 600 008

Prof. Dr.Jayanthi, M.D., FRCP (Glasgow).,

The Dean

Madras Medical College, Government General Hospital ,Chennai-03.

CERTIFICATE – II

This is to certify that this dissertation work titled **"A CLINICAL STUDY ON OCULAR MOTOR NERVE PALSIES FOLLOWING TRAUMA"** of the candidate **Dr.A.SHAKILA**., for the award of **M.S., DEGREE** BRANCH-III (OPHTHALMOLOGY). I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **3(three)** percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled, **"A CLINICAL STUDY ON OCULAR MOTOR NERVE PALSIES FOLLOWING TRAUMA"** is a bonafide and genuine research work conducted by me under the guidance of **Prof. Dr.M.V.S.PRAKASH., M.S., D.O.,** Head of Department of Orbit and Oculoplasty services, Regional institute of ophthalmology & Government Ophthalmic Hospital. Chennai-600008.

Dr. A. SHAKILA

Place: Chennai

Date

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.A.Shakila
I Year Post Graduate in MS Ophthalmology
Regional Institute of Ophthalmology & GOH/
Madras Medical College
Chennai

Dear Dr.A.Shakila,

The Institutional Ethics Committee has considered your request and approved your study titled **"A CLINICAL STUDY ON OCULAR MOTOR NERVE PALSIES FOLLOWING TRAUMA" - NO.04012018**

The following members of Ethics Committee were present in the meeting hold on **09.01.2018** conducted at Madras Medical College, Chennai 3

1. Prof.P.V.Jayashankar	:Chairperson
2. Prof.R.Narayana Babu,MD.,DCH., Dean,MMC,Ch-3	: Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4. Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch	: Member
5. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3	: Member
6. Prof.A.Pandiya Raj,Director, Inst. of Gen.Surgery,MMC	: Member
7. Prof.Shanthy Gunasingh, Director, Inst.of Social Obstetrics,KGH	: Member
8. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai	: Member
9. Prof. Susila, Director, Inst. of Pharmacology,MMC,Ch-3	: Member
10.Prof.K.Ramadevi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3	: Member
11.Prof.Bharathi Vidya Jayanthi,Director, Inst. of Pathology,MMC,Ch-3	: Member
12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist
14.Thiru K.Ranjith, Ch- 91	: Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

URKUND

Urkund Analysis Result

Analysed Document:	A CLINICAL STUDY ON OCULAR MOTOR NERVE PALSIES FOLLOWING TRAUMA.docx (D57534616)
Submitted:	23/10/2019 17:43:00
Submitted By:	shakila.prakasam25@gmail.com
Significance:	3 %

Sources included in the report:

dissertation (1).docx (D42807734)
Saudamini Nighute THESIS.docx (D44188215)
https://emj.bmj.com/content/18/4/310.full.pdf

Instances where selected sources appear:

12

ACKNOWLEDGEMENT

I express my sincere thanks to **Prof. Dr. R.JAYANTHI M.D., FRCP,** Dean Madras Medical College for permitting me to conduct this study.

I am very grateful to **Prof.Dr.M.ANANDA BABU M.S.,** Director and Superintendent, RIO & GOH, Chennai, for helping me conduct the study.

I express my gratitude to **Prof. Dr. M.V.S. PRAKASH., MS.,DO.,** Guide and Unit chief, Department of Orbit and Oculoplasty who with his vast knowledge and experience assigned me the topic of study and provided me all the necessary facilities and guidance and being the experienced single surgeon of all patients.

I am grateful to Assistant Professors **Dr.T. G. UMAMAHESWARI M.S., DR. P. GEETHA M.S.,D.O., DR.R. SUJATHA M.S.,** for rendering their valuable advice and guidance for the study.

I wish to express my sincere thanks to all the **Professors, Assistant professors** and all my **colleagues** who helped me in bringing out this study.

Finally I am indebted to all my **patients** for their sincere co-operation for completion of this study.

CONTENTS

INTRODUCTION

The term “Ocular motor system” refers to the entire somatic motor system which controls the position and movements of the eyes. It includes extraocular muscles, the cranial nerves and their nuclei that innervate them and the forces that stimulate and inhibit their actions.

“Ocular motor nerves” refers to third (Oculomotor), fourth (Trochlear) and sixth (Abducent) cranial nerves. Superior rectus, medial rectus, inferior rectus, inferior oblique, levator palpebrae superioris and the intrinsic muscles of the eye are supplied by the Oculomotor nerve. Superior oblique is supplied by the Trochlear nerve. Lateral rectus is supplied by the Abducent nerve[1]. Ocular motor nerves control the extra ocular movements of the eyes. Restricted ocular mobility can occur as the result of paralysis of the nerves supplying the extra ocular muscles or due to the pathology in the muscle itself or there may be the pathology at the myoneural junction.

Palsy is the term used to denote either paresis (partial) or paralysis (total) of that particular nerve. It could be either congenital or acquired. Most congenital palsies occur as isolated defects in an otherwise healthy individual, due to

developmental defects in the nucleus or the nerve pathway. Acquired palsies are more common and can result from various causes such as trauma, intracranial tumours, aneurysms or vascular diseases. Ophthalmoplegia can be caused by multiple ocular motor nerve palsies either single or multiple ocular motor nerve palsy. It becomes clinically important to identify and differentiate the type of palsy as treatment for these conditions varies according to the cause.

-

OCULOMOTOR NERVE

ANATOMY

The OCULOMOTOR NERVE is entirely motor in function. All the extraocular muscles of the eyeball except superior oblique and lateral rectus are supplied by the third nerve. The intraocular muscles - sphincter pupillae and ciliary muscle are also supplied by third nerve.

FUNCTIONAL COMPONENTS

1. SOMATIC EFFERENT –

Associated with movements of the eyeball.

2. GENERAL VISCERAL EFFERENT (parasympathetic)-

Associated with accommodation and contraction of the pupil.

3. GENERAL SOMATIC AFFERENT –

For carrying proprioceptive impulses from the extraocular muscles supplied by the third nerve.

THE OCULOMOTOR NUCLEAR COMPLEX

LOCATION

In the midbrain at the level of superior colliculus in the ventromedial part of central gray matter that surrounds the cerebral aqueduct.

It consists of about 10mm length longitudinal column extending above from the floor of the third ventricle and below it is related to the nucleus of the trochlear nerve.

The nucleus complex consists of 2 motor nuclei:

1. Main motor nucleus
2. Accessory Edinger westphal nucleus

MAIN MOTOR NUCLEUS contains the following sub nuclei:

NUCLEUS	**MUSCLE SUPPILED**
VENTROMEDIAN NUCLEUS	Ipsilateral medial rectus
DORSOLATERAL NUCLEUS	Ipsilateral inferior rectus
PARAMEDIAN NUCLEUS	Contralateral superior rectus
INTERMEDIATE NUCLEUS	Ipsilateral inferior oblique
CAUDAL CENTRAL NUCLEUS	Bilateral levator palpebrae superioris

EDINGER WESTPHAL NUCLEUS

It is situated posterior to the main oculomotor nuclear mass .

It constitutes of a median and two lateral parts. It gives rise to preganglionic parasympathetic fibres.

CONNECTIONS OF THE NUCLEUS

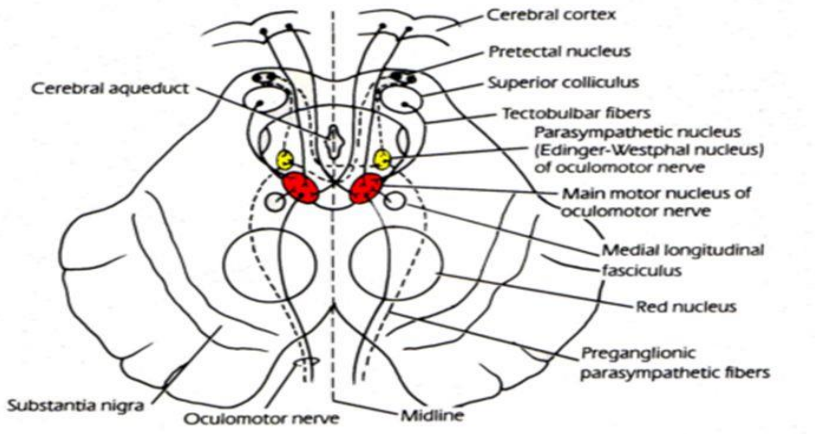
1. Cerebral cortex
    - The motor cortex
    - Visual cortex through the superior colliculus.
    - Frontal eye field

2. Nuclei of trochlear,abducent and vestibulocochlear nerve through the medial longitudinal bundle.

3. Torsional and vertical gaze centres

4. Pretectal nucleus of both sides

5. Cerebellum through the vestibular nuclei



Oculomotor nuclei and their central nervous connections

COURSE AND DISTRIBUTION

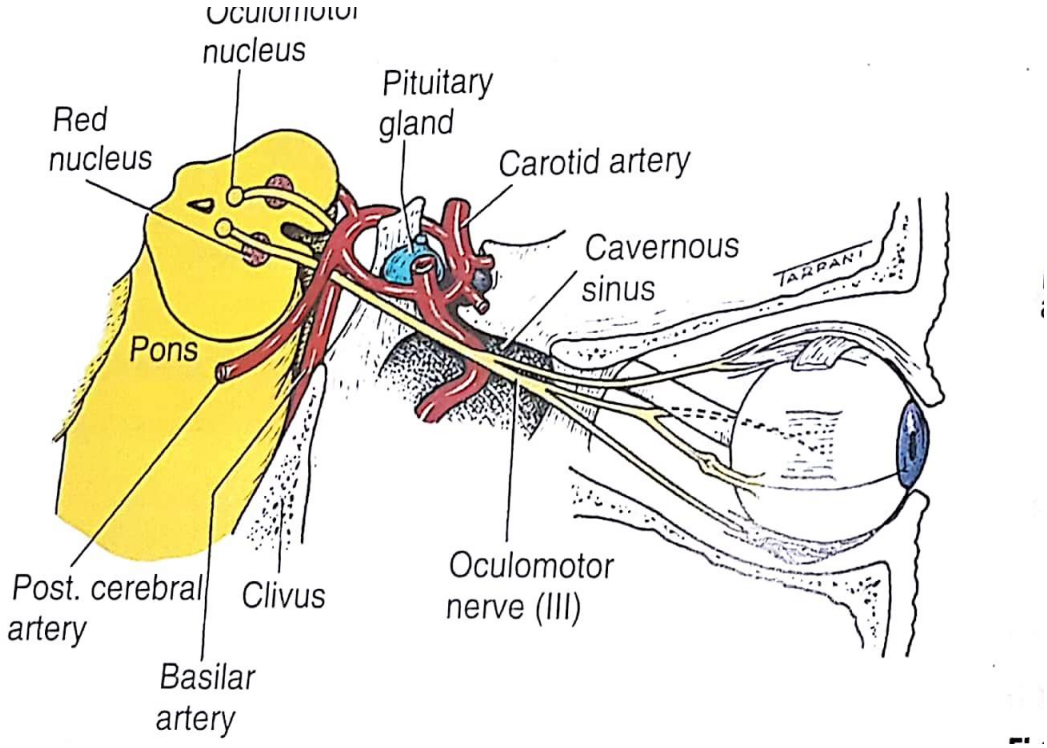
It can be divided into four parts

1. The Fascicular Part
2. The Basilar Part
3. The Intracavernous Part
4. The Intraorbital Part

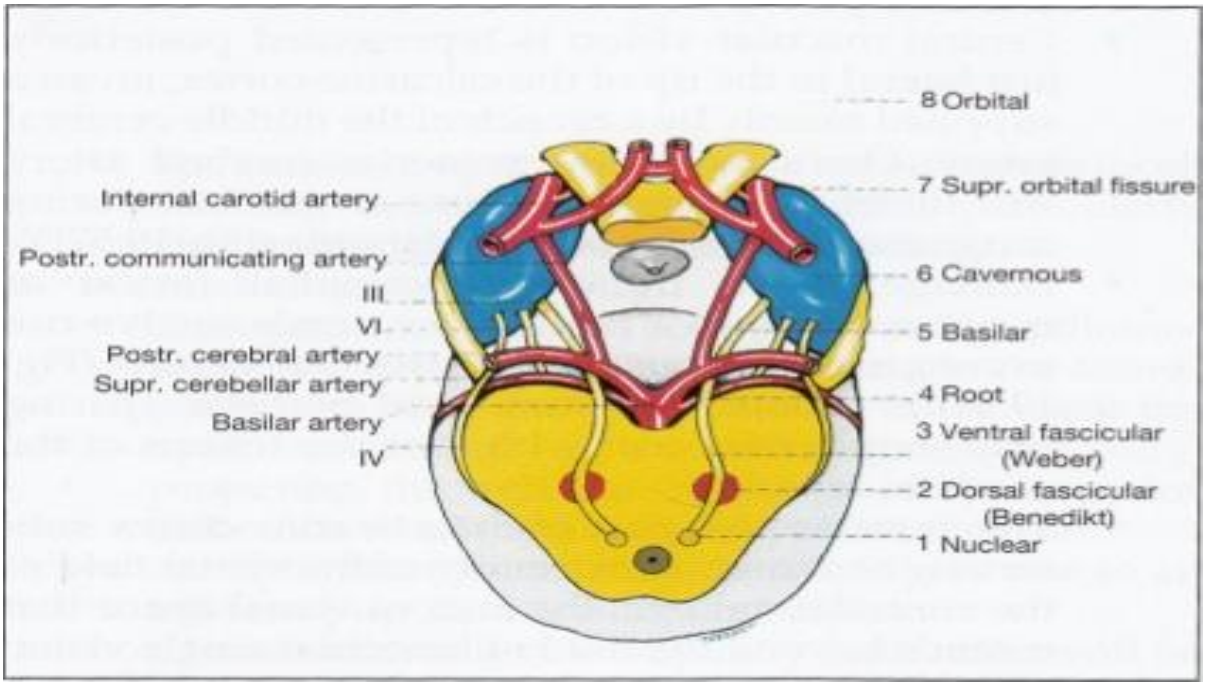
THE FASCICULAR PART

The third nerve nucleus contains efferent fibres that pass from the third nerve nucleus through the red nucleus and the medial aspect of cerebral peduncle which then emerge from the midbrain and pass into the interpeduncular space.

LATERAL VIEW OF THE COURSE OF THIRD NERVE



DORSAL VIEW OF THE THIRD CRANIAL NERVE



THE BASILAR PART

It consists of a series of 15 – 20 rootlets in the interpeduncular fossa. These rootlets coalesce to form a large medial and a small lateral root , which then unite to form a flattened nerve that gets twisted bringing the inferior fibres superiorly and vice versa .Subsequently the nerve becomes a rounded cord . The nerve then passes between the posterior cerebral artery and the superior cerebellar artery. Further it runs forward in the interpeduncular cistern to reach the cavernous sinus .

THE INTRACAVERNOUS PART :

The nerve enters the cavernous sinus by piercing the posterior part of its roof on the lateral side of the posterior clinoid process. It further descends on the lateral wall of the sinus , and here it lies above the trochlear nerve .

In the anterior part of the cavernous sinus , it divides into superior and inferior divisions which enter the orbit through the middle part of the superior orbital fissure within the annulus of zinn.

THE INTRAORBITAL PART :

In the orbit, the smaller superior division ascends on the lateral side of the optic nerve and supplies levator palpebrae superioris and superior rectus.

The larger inferior division divides into three branches :

i. Nerve to medial rectus which passes inferior to optic nerve

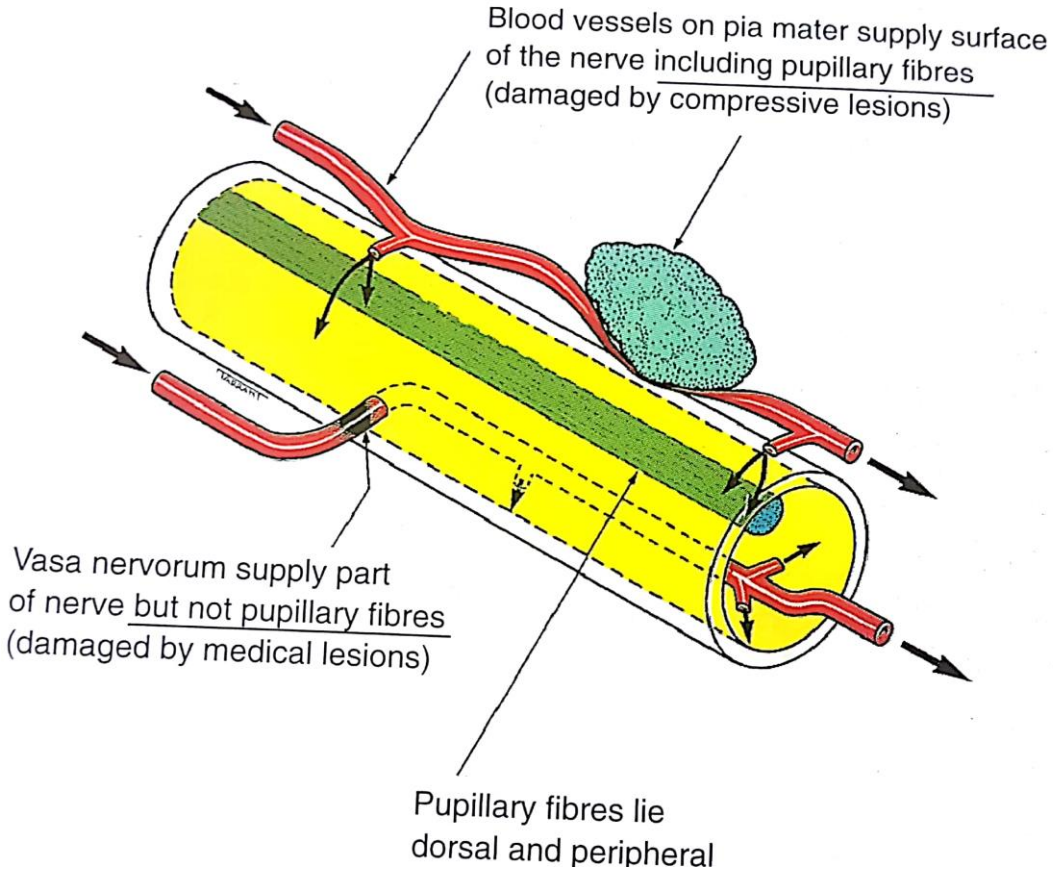
ii. Nerve to inferior oblique which passes (longest of the three branches) in between the inferior rectus and lateral rectus and supplies the inferior oblique from its posterior border .It also gives off the motor root to the ciliary ganglion .

iii. Nerve to inferior rectus which passes and enters the muscle on its upper aspect.

PUPILLOMOTOR FIBRES:

The pupillomotor fibres of the III nerve travel in the outer layers of the nerve and are closer to the nutrient blood supply enveloping the nerve.

The inner fibres are supplied by the vasa nervosum and the outer fibres are supplied by the pial plexus. Involvement or sparing of pupil is important as it helps to differentiate between medical and surgical lesion[2.]



LOCATION OF PUPILLOMOTOR FIBRES WITHIN THE TRUNK OF THE THIRD NERVE

TROCHLEAR NERVE

The trochlear nerve is entirely motor in function

It supplies only the superior oblique muscle of the eyeball.

PECULIARITIES

- The only cranial nerve to arise from the dorsal aspect of the brain [2].
- The only cranial nerve to cross completely on the other side
- The longest and thinnest of all cranial nerves.

FUNCTIONAL COMPONENTS

1. SOMATIC EFFERENT – Concerned with the primary,secondary and tertiary actions of superior oblique .

2. GENERAL SOMATIC AFFERENT – Carries proprioceptive impulses from the superior oblique . The impulses are relayed to the mesencephalic nucleus of the trigeminal nerve.

NUCLEUS

Situated in the ventromedial part of the central gray matter of the midbrain at the level of inferior colliculus. It is continuous with the third nerve nuclear complex . It belongs to the somatic efferent column of nuclei .

CONNECTIONS OF THE NUCLEUS

1 . Cerebral cortex

i. Motor cortex – of both sides through the corticonuclear tracts .

ii. Visual cortex - through the superior colliculus

iii . Frontal eye field .

2. Nuclei of 3, 6 and 8 cranial nerves through the medial longitudinal bundle .

3. Superior colliculi through the descending predorsal bundle.

4 . Vertical and torsional gaze centres.

5 . Cerebellum through the vestibular nuclei.

COURSE AND DISTRIBUTION

It is divided into

i) Fascicular part

ii) Precavernous part

iii)Intracavernous part

iv)Intraorbital part .

THE FASCICULAR PART

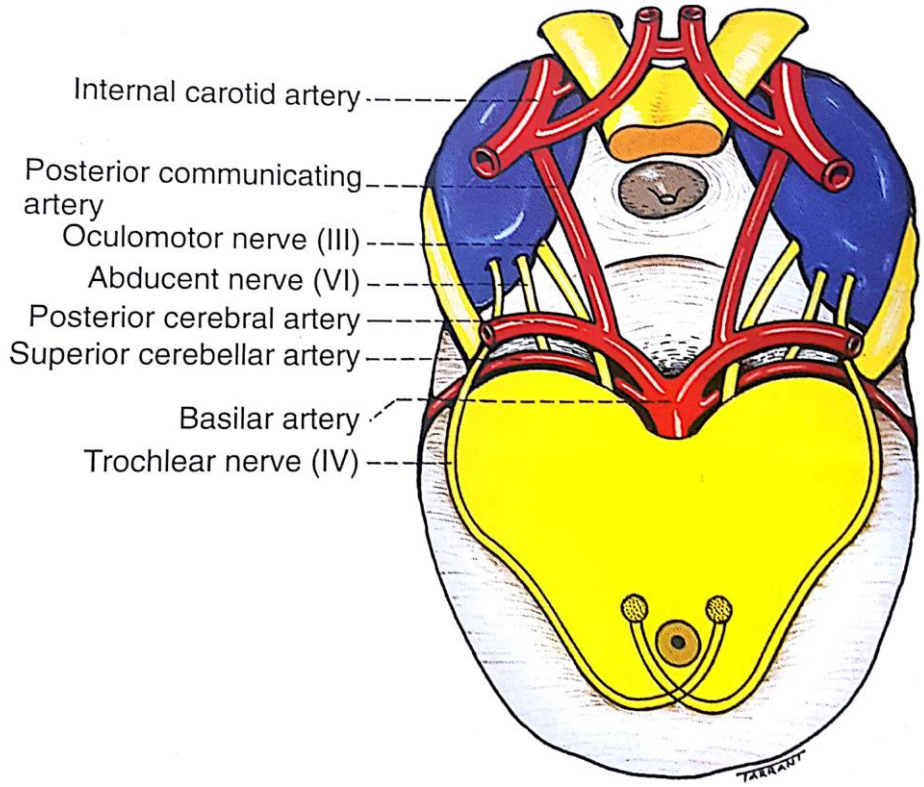
It consists of efferent fibres which after leaving the nucleus ,passes posteriorly around the aqueduct in the central gray matter and then decussate completely in the anterior medullary velum.

THE PRECAVERNOUS PART

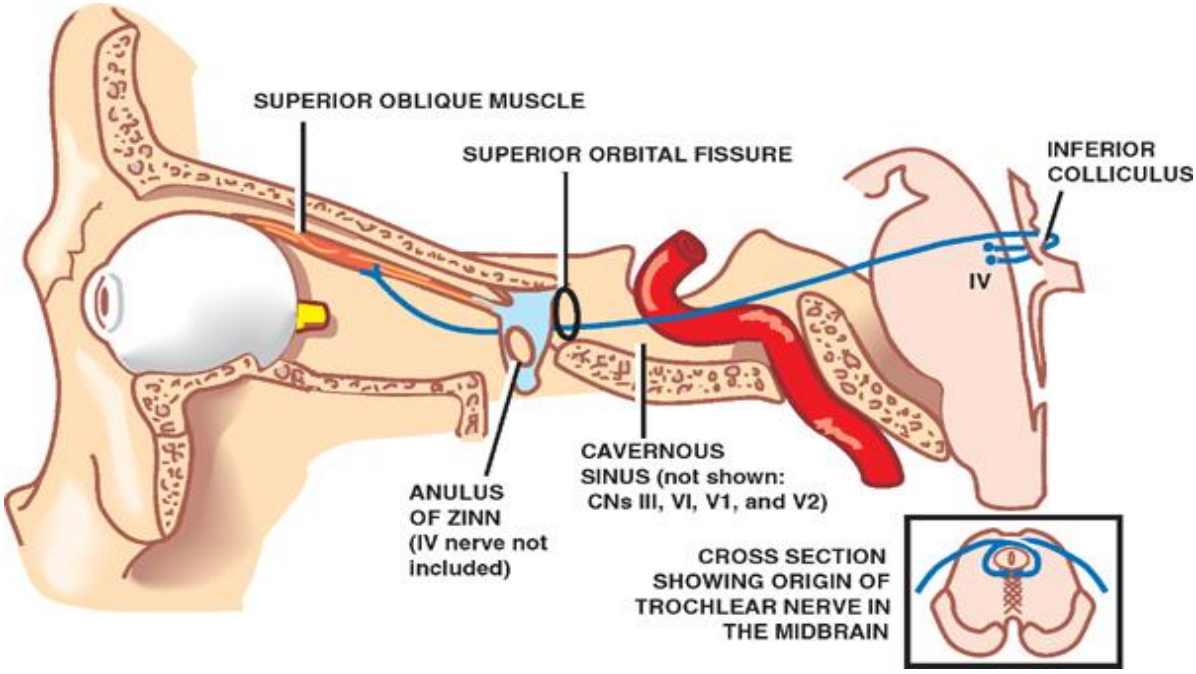
The trochlear nerve trunk emerges from the superior medullary velum just below the inferior colliculus on the dorsal aspect of midbrain. It winds around the superior cerebellar peduncle and the cerebral peduncle just above the pons. It runs beneath the free edge of the tentorium , and passes between the posterior cerebral and superior cerebellar arteries to appear ventrally lateral to cerebral peduncle .

Then it pierces the dura on the posterior corner of the roof of the cavernous sinus to enter into it .

DORSAL VIEW OF THE COUSRE OF TROCHLEAR NERVE



TROCHLEAR NERVE



THE INTRACAVERNOUS PART

In the cavernous sinus, the nerve runs forwards in its lateral wall. Which lies below the oculomotor nerve and above the first division of the trigeminal nerve . In the anterior part of the cavernous sinus, it rises, crosses over the oculomotor nerve and leaves the sinus to pass through the lateral part of the superior orbital fissure.

THE INTRAORBITAL PART.

After entering through the lateral part of the superior orbital fissure, the nerve passes medially above the origin of the LPS and later ends by supplying the superior oblique. The number of fibres in the intraorbital part of the trochlear nerve are greater than its intracranial part . These extra fibres carrying the proprioceptive impulses from the superior oblique leave the trochlear nerve to join the ophthalmic division of trigeminal nerve in the cavernous sinus.

ABDUCENT NERVE

It is an entirely motor nerve which supplies the lateral rectus muscle of the eyeball.

FUNCTIONAL COMPONENTS

i. SOMATIC EFFERENT - Lateral movements of the eye .

ii. GENERAL SOMATIC AFFERENT - For proprioceptive impulses from the lateral rectus muscle . These impulses ultimately reach the mesencephalic nucleus of the fifth nerve .

NUCLEUS

It is situated in the lower part of pons , ventral to the floor of the IV ventricle[2]. It is closely related to the fasciculus of the facial nerve . It contains two types of multipolar cells - large and small.

The large multipolar cells give rise to fibres of the abducent nerve and the fibres of the small multipolar cells relay in the oculomotor nucleus via the medial longitudinal fasciculus .

The small multipolar cells form the para-abducent nucleus.The abducent nucleus lies in line with the nuclei of III and IV nerves above and the hypoglossal nucleus below.

CONNECTIONS OF THE NUCLEUS

1 . Cerebral cortex –

i. Motor cortex -by the afferent corticonuclear fibres from both cerebral hemispheres.

ii) Visual cortex , through the superior colliculus.

iii) Frontal cortex

2. Nuclei of III , IV and VIII cranial nerves through the medial longitudinal bundle.

3. Pretectal nucleus of either sides .

4. Horizontal gaze centre through the medial longitudinal bundle.

5. Cerebellum through vestibular nuclei.

COURSE AND DISTRIBUTION

It is divided into

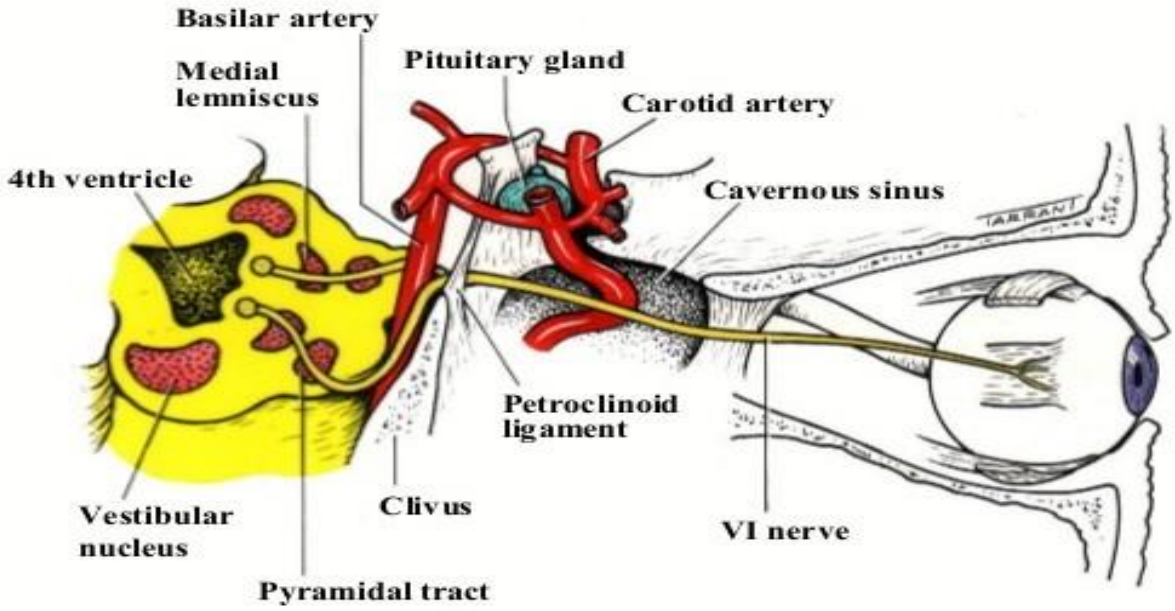
i. Fascicular part

ii. Basilar part

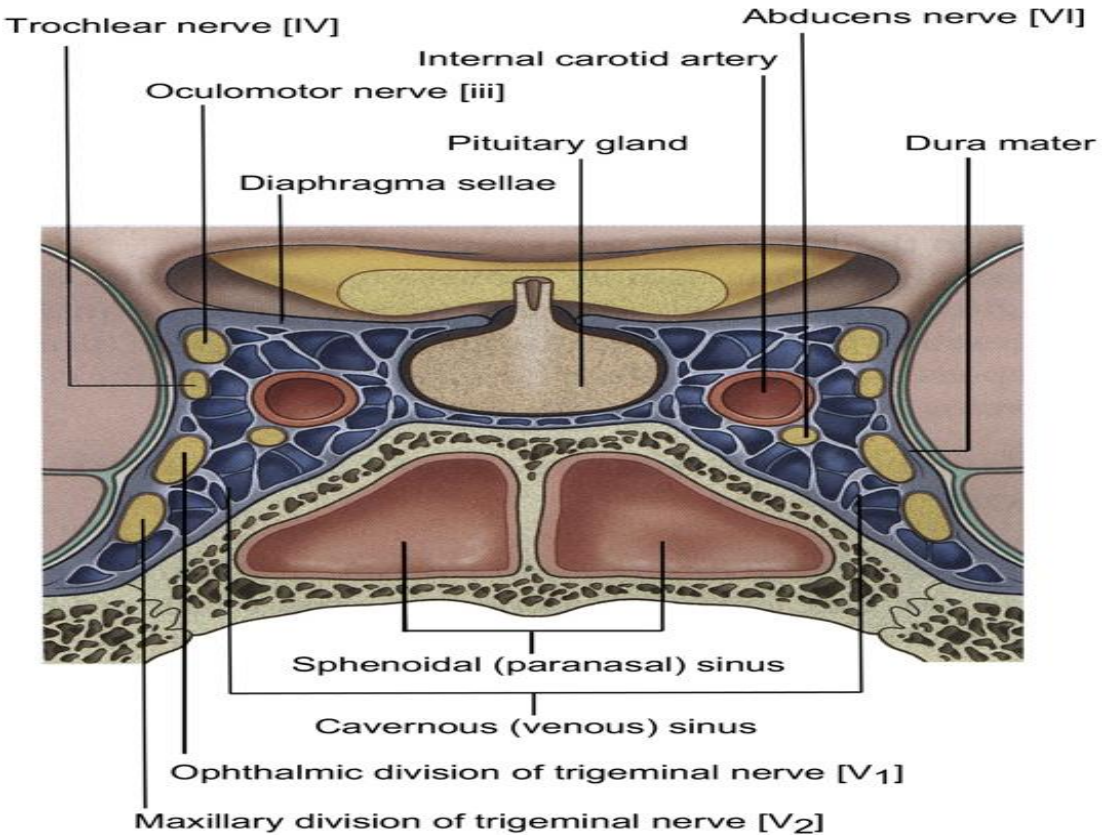
iii. Intracavernous part and

iv. Intraorbital part .

LATERAL VIEW OF THE COURSE OF THE SIXTH NERVE



LOCATION OF CRANIAL NERVES IN THE CAVERNOUS SINUS



THE FASCICULAR PART

It contains efferent fibres that start from the nucleus, pass forward traversing the medial lemniscus and pyramidal tract. These then emerge prominence . The rootlets join to form one nerve , at varying distances from the origin.

THE BASILAR PART

The nerve then runs forwards , upwards and slightly laterally through the cisterna pontis between the pons and occipital bone. The nerve then runs upwards on the back of petrous temporal bone near its apex .

At the upper border of the petrous bone, the nerve bends forward at right angles under petrosphenoidal ligament through the Dorello's canal and enters the cavernous sinus by piercing its posterior wall at a point lateral to dorsum sellae and superior to the apex of petrous temporal bone .

THE INTRACAVERNOUS PART

In the cavernous sinus , the nerve runs horizontally forward, occupying a position below and lateral to the internal carotid artery.

The nerve leaves the cavernous sinus and enters the orbit through the middle part of the superior orbital fissure through the annulus of zinn. In the

superior orbital fissure, the abducent nerve lies inferolateral to the oculomotor and nasociliary nerves .

THE INTRAORBITAL PART

In the orbit the nerve runs forwards and enters the ocular surface of the lateral rectus muscle just behind its middle portion after dividing into three or four branches .

OCULAR MOTOR NERVE PALSIES FOLLOWING TRAUMA

OCULOMOTOR NERVE

Head trauma accounts for 8-16% of all oculomotor nerve palsies[3]. Oculomotor nerve palsy is seen in 2.9% of patients with multiple cranial nerve involvement in head injuries[4]. Oculomotor nerve palsy in a patient with head injury imparts a sense of urgency in imaging and management owing to the possibility of an expanding intracranial haematoma in such a patient.

MECHANISM OF INJURY:

- Oculomotor nerve palsy occurs due to compression of the nerve at the tentorial hiatus by uncus in transtentorial herniation .
- Oculomotor nerve palsy is seen in midbrain haematoma in the tectal region.
- The oculomotor nerve can be injured in its course in the brainstem also by shearing injury.
- More often, oculomotor nerve palsy occurs with other ocular motor nerves when the cavernous sinus is involved in case of base of skull fracture.
- Isolated oculomotor nerve palsy can occur due to avulsion or stretching of the nerve at mesencephalo-pontine junction.

- Injury to superior or inferior divisions of the nerve can occur due to injury to superior orbital fissure, orbit or maxillofacial region.

DIAGNOSIS:

In case of head injury, unilateral mydriasis indicates ipsilateral supratentorial expanding haematoma .Mydriasis may be accompanied by ptosis and extraocular muscle weakness.

Rapid recovery of consciousness suggests an avulsion injury to the oculomotor nerve in a complete oculomotor nerve palsy.

The head injury can be relatively mild even in a complete oculomotor nerve palsy.

Palsy due to injury to the fascicles is likely to give rise to aberrant regeneration, where axons terminate in inappropriate structures. Such regeneration can result in pseudo von graefe sign or abnormal reaction of pupil such as light near dissociation.

TROCHLEAR NERVE

The incidence of trochlear nerve injury is 2.14% in head injuries[4], and the injury is often associated with injury to other ocular motor nerves.

MECHANISM OF INJURY:

- A sudden deceleration impact or blow to the head can cause coup and contrecoup injury to the brain which results in the brainstem to impact against the tentorium, resulting in trochlear nerve injury.
- The injury can occur in dorsal midbrain, or in the free edge of the tentorium.
- Trochlear nerve can be injured in its subarachnoid course and can present as isolated trochlear nerve palsy.
- Bilateral trochlear nerve injury is always due to trauma

DIAGNOSIS:

The patient complaints of diplopia which is compensated by adapting a characteristic head tilt. These patients will have difficulty in climbing down the stairs and in reading newspaper or a book. The head will be tilted away from the affected eye.

Examination reveals hypertropia that worsens on lateral gaze. Bilateral Trochlear nerve palsy is diagnosed by the presence of alternating hyperdeviation in various positions of upward gaze.

In a concomitant oculomotor nerve palsy, trochlear nerve palsy can be suspected from absence of intorsion.

ABDUCENT NERVE

Head injury accounts for nearly 3 –15% of abducent nerve palsies[5].

MECHANISM OF INJURY:

The long intradural course, its passage over the petrous ridge with its relative fixity under the petroclinoid ligament and to the cavernous sinus makes it vulnerable to stretch or tear.

Hyperextension trauma to cervical spine can also cause abducent nerve palsy, accompanied with lower cranial palsies.

DIAGNOSIS:

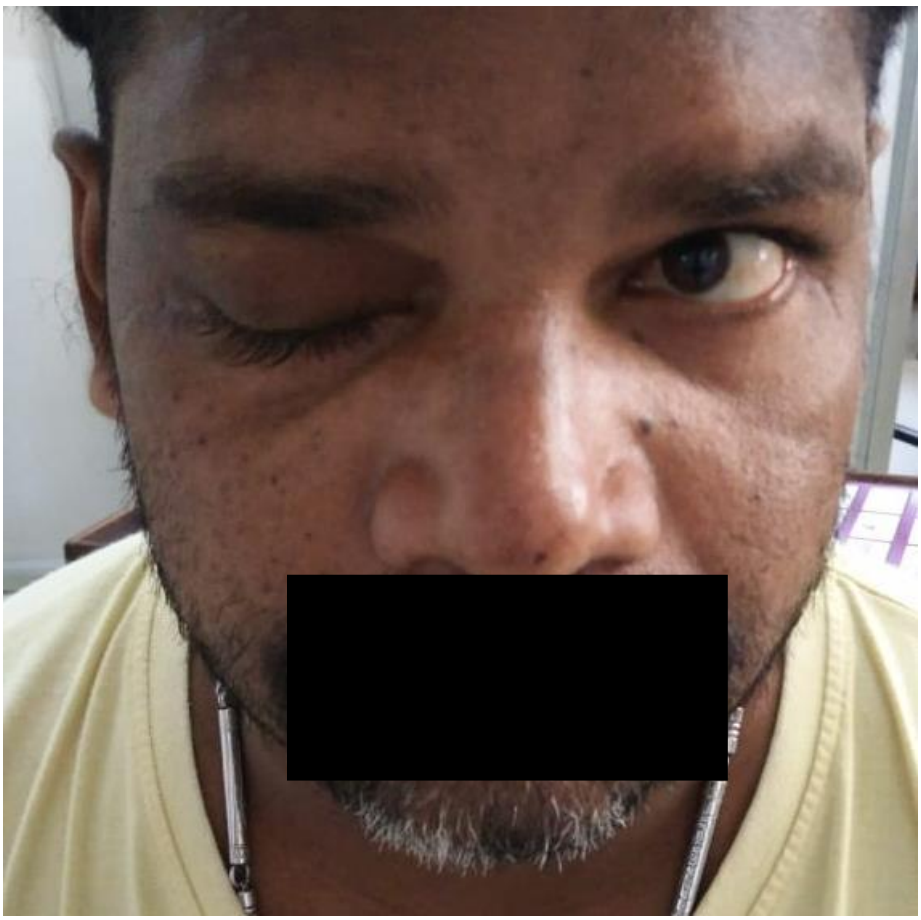
Patient complains of diplopia on lateral gaze, and examination of ocular movements reveals defective or absence of abduction indicating lateral rectus palsy.

In an unconscious patient, the eyeball can be seen adducted, with no abduction on oculocephalic response.

THE FEATURES OF THIRD NERVE PALSY :

It may be complete or incomplete and it may be congenital or acquired.

1. Ptosis - Due to paralysis of LPS

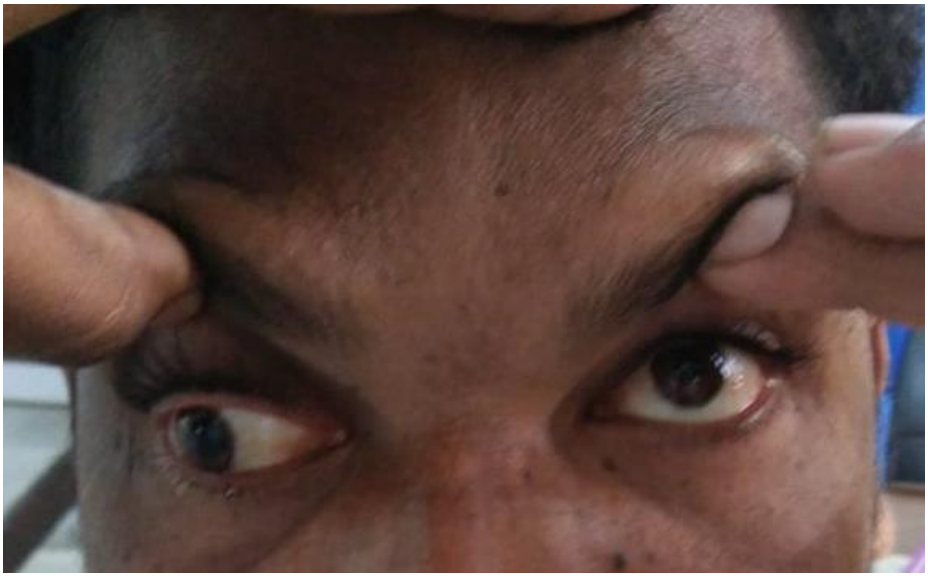


RE PTOSIS

2. Deviation – Eyeball is turned down , out and slightly intorted due to unopposed action of the lateral rectus and the superior oblique

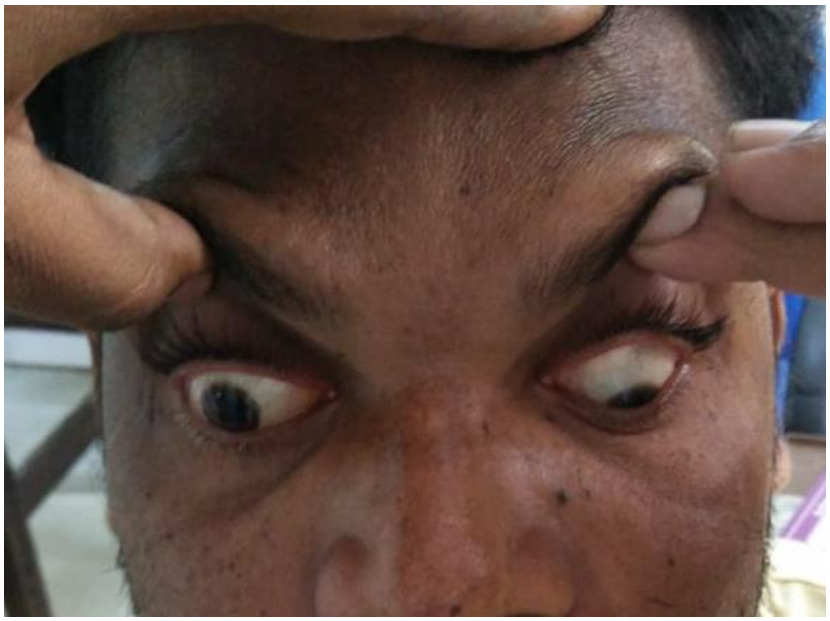
3. Ocular movements – Restriction of the following movements :

i.Elevation - Due to paralysis of superior rectus and inferior oblique,



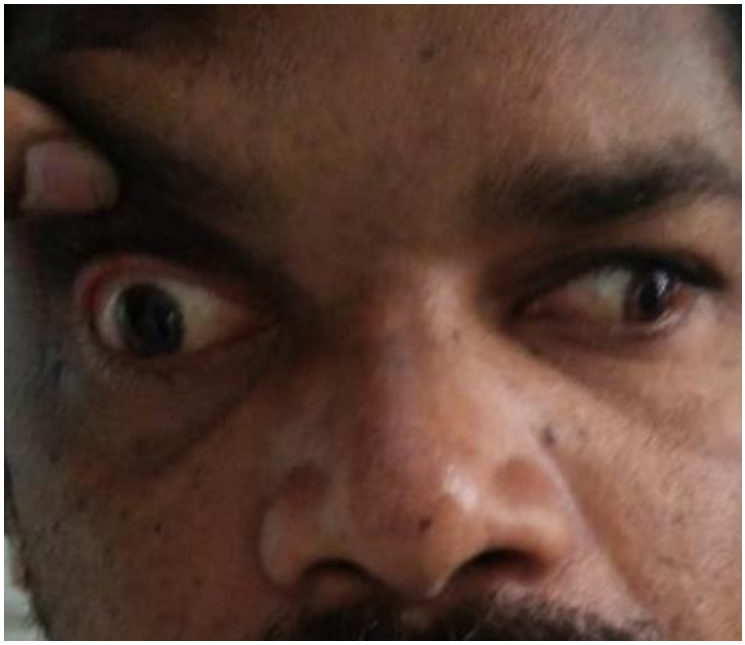
RE ELEVATION RESTRICTION

ii. Depression -Ddue to paralysis of inferior rectus



RE DEPRESSION RESTRICTION

iii. Adduction - Due to paralysis of medial rectus and



RE ADDUCTION RESTRICTION

iv. Extorsion - Due to paralysis of inferior rectus and inferior oblique.

4. Pupil - Fixed and dilated due to paralysis of sphincter papillae

5. Due to paralysis of ciliary muscle accommodation is completely lost

6. Crossed diplopia – Appears on manually raising the eyelid, that occurs due to paralytic divergent squint

7. Head posture - If the pupillary area is uncovered then head takes a posture consistent with the directions of actions of paralysed muscle

i.e head is turned towards the opposite side , tilted towards the same side and chin is slightly raised.

RECOVERY FROM III NERVE PALSY

1.Complete recovery:

In ischemic conditions recovery occurs within 6 months.

In traumatic nerve palsies recovery varies from 6 months to 1-2 years.

2. **No recovery or change in Palsy**:

Usually in cases where the nerve is transected by trauma or tumour infiltration or chronic compression , recovery is not noted

3**. Partial recovery**:

Partial recovery occurs in case of fascicular lesion.

4**. Partial recovery characterized by oculomotor nerve synkinesis**:

Usually becomes apparent 9 weeks after injury.

ABERRANT REGENERATION OF III NERVE

Aberrant regeneration of third nerve occurs after trauma and aneurysms, occasionally after tumour or syphilis;

TYPES

Primary –

It occurs without apparent III nerve palsy

It occurs in slow growing lesions of cavernous sinus such as meningiomas and aneurysm or birth trauma.

Secondary –

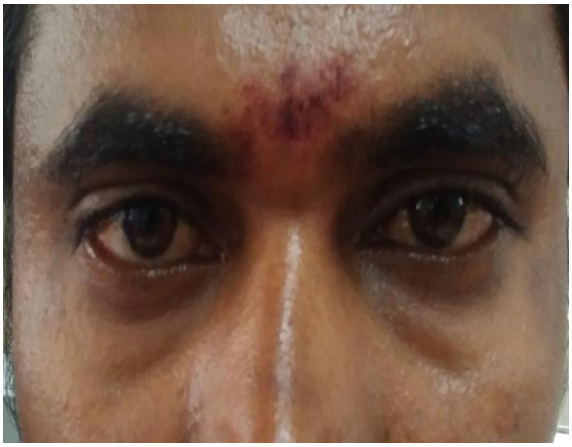
It occurs after apparent oculomotor nerve palsy when fibres regenerate following trauma the fibres get misdirected.

. If the patient is followed with a presumed diagnosis of ischaemic III nerve palsy and then develops signs of aberrant regeneration, then MR scanning and cerebral angiography are indicated.

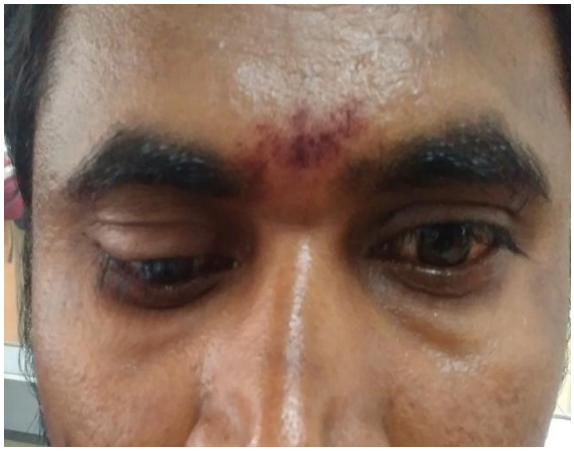
Misdirection can be identified by the following signs[6].

- **Pseudo von Graefe lid sign**

  When the eye attempts to move downwards the upper lid retracts, as some of the fibres originally supplying inferior rectus muscle are now misdirected to supply the levator palpebrae superioris.



LE PRIMARY GAZE



LE PSEUDO VON GRAEFE SIGN ON DOWNGAZE

Pseudo Argyll Robertson pupil:

Slow light reflex occurs and better papillary constriction with the near synkinesis. Fibres supplying the sphincter papillae are damaged and fibres to ciliary muscle for accommodation are misdirected to innervate the pupil.

Horizontal gaze lid dyskinesis:

The upper eyelid retracts as the eye is adducted and falls when the eye is abducted.

- Difficulty in vertical gaze
- Adduction on attempted vertical gaze
- Monocular optokinetic response

FEATURES OF IV NERVE PALSY

1. Ocular movements - depression is limited in adduction .

Intorsion is also limited.

2. Hyperdeviation occurs due to weakness of superior oblique.

It becomes more obvious when the head is tilted towards ipsilateral shoulder (Park Bielchowsky head tilt test).

3 . Vertical diplopia occurs on looking down.

4 . Abnormal head posture – To avoid diplopia head adopts a posture such that the action of superior oblique is less needed.i .e head is tilted towards the opposite side ,face is slightly turned to opposite side and the chin is depressed .

Recovery from IV nerve palsy

1. **Complete recovery** – Following ischemia or closed head injury or after relief of compression form tumor or aneurysms.

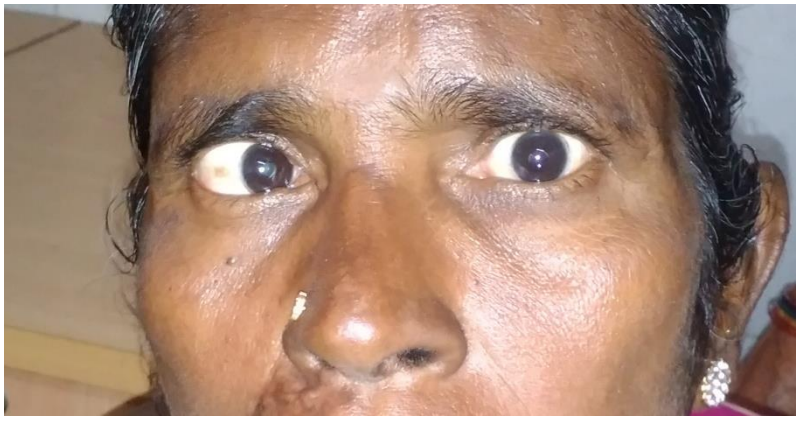
2. **Incomplete recovery** – Leaving the patient with mild persistent vertical and torsional diplopia.

3. **No recovery** – Primarily after mesencephalic injury or with transection of the trochlear nerve by trauma or compression.

CLINICAL FEATURES OF VI NERVE PALSY

1. Deviation -

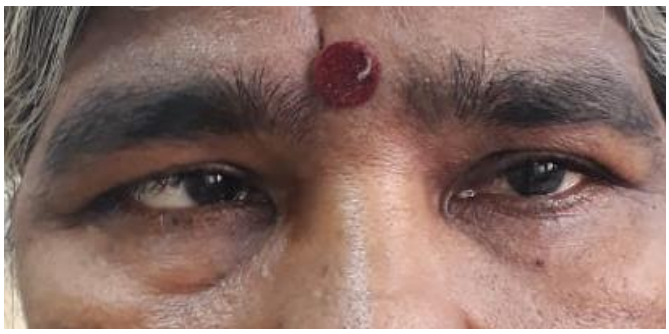
Eyeball is convergent due to unopposed action of the medial rectus muscle .



CONVERGENCE OF RE IN R SIXTH NERVE PALSY

2. Ocular movements –

Abduction is restricted .



LE ABDUCTION RESTRICTION

3. Diplopia –

Uncrossed horizontal diplopia occurs , that becomes worse towards the action of paralysed muscle .

4 . Head posture –

The face is turned towards the action of paralysed muscle

DIFFERENTIAL DIAGNOSIS OF VERTICAL DIPLOPIA

1.Ocular myasthenia

2. Thyroid eye disease

3. Orbital disease (tumour , trauma , inflammation , blow out fracture etc.)

4. III nerve palsy

5. Brown syndrome

6. Skew deviation

.

CLINICAL EVALUATION OF A CASE WITH OCULAR MOTOR NERVE PALSIES

After careful history including the medical and neurological conditions, the patient should be examined in general to rule out vascular diseases like diabetes, hypertension and arteriosclerosis. If there is history of cerebrovascular accident, complete neurological examination should be done.

Diplopia

If there is horizontal diplopia – Horizontal muscle palsy.

Vertical diplopia with a tilt, most likely to be superior oblique palsy.

Cover Test

Look for the type of deviation comparing the amount of movement for near and distance.

Ocular Movements

Both under action and over action are to be noted by doing alternate cover test in different positions of gaze. Maximum deviation is noted in the direction of the action of affected muscle. The examiner should remember that both the eyes may be affected, particularly after severe head injury.

Other signs like nystagmus especially in certain positions, or retraction of globe on horizontal or vertical gaze can occur in case of direct injury to muscles or their connection.

Diplopia Chart

1. Maximum separation of images in the direction of action of the affected muscle.

2. Abductors – lateral rectus, superior and inferior obliques produce uncrossed diplopia while others produce crossed diplopia.

3. Tilt though produced in vertical palsies is more common with superior oblique palsies.

Hess Chart

For diagnosis and also for follow up of a case of muscle palsy.

1. The smaller field always belongs to the paretic eye-inward displacement of dots indicates underaction of the paretic muscles and outward displacement indicate overaction of antagonist. Equal sized fields indicate muscle sequelae have developed denoting either congenital or longstanding palsy.

2. The outer field should be examined for small underaction and overaction which may not be apparent on inner fields. If the outer field is very close to

the inner field, a mechanical restriction of muscle movement rather than a neurogenic one is suspected.

Forced duction test and its applications

It is useful to differentiate a neurogenic palsy from restrictive palsy which may be due to contraction or fibrosis of a muscle, tightness of a muscle following excessive resection, scarring of conjunctiva, trapping of muscle fibres as in blow out fractures, symblepharon etc.

After application of local anaesthetic, the eye is grasped at the limbus with a forceps and is moved in the direction opposite to that in which mechanical restriction is suspected, taking care not to press the globe. When the test is done patient is asked to look at this hand which is held the direction in which the eye is moved.

This is to prevent the influence of the patient's innervation which may otherwise counteract the passive movement of the globe simulating a mechanical restriction.

Bielschowsky Head tilt test

To find out the vertical muscle involved, especially in cases of superior oblique palsies where on tilting, the eye becomes hypertropic on the affected

TREATMENT OF OCULAR MOTOR NERVE PALSIES

Follow up of cases of ocular motor nerve palsy that do not need urgent management , like the posterior communicating artery aneurysm must be at 6 weekly intervals till 6 months or two consecutive 6 weekly follow-ups reveal no change in motility.

Every time diplopia charting ,Hess charting , recording of deviations in nine gazes is done. During the meantime , patient is greatly disturbed by diplopia . So some nonsurgical modalities are practiced for symptomatic relief . If no resolution occurs after about 8 – 12 months then surgery is considered.

1**. Prisms**– are helpful in providing binocular vision as well as reducing the chances of development of contracture, but are useful only in small angle squints. Fresnel prisms are also used.

2. **Botulinum toxin** – the ipsilateral antagonist is paralysed by chemodenervation . The effect lasts for about 2 – 3 months . If necessary the injection can be repeated.

3. Occlusive prisms or **opaque contact lens**

4. **Surgery** – mainly to weaken the antagonist , usually ipsilateral and sometimes also the contralateral antagonist , in addition to strengthening the paralysed muscle . The amount of recession resection varies depending upon which eye habitually

fixates (secondary deviation or primary deviation needs to be corrected). Another principle is to restrain the contralateral antagonist by performing retroequatorial myopexy.

In the case of III nerve palsy, the aim is to achieve diplopia free ocular position in primary position and downgaze .The latter should never be compromised for the upgaze .Anyway it is difficult because the III nerve supplies most of the extraocular muscles except two . Moreover aberrant regenerations alter the clinical picture . Each case has to be considered on a individual basis .

In the case of IV nerve palsy, either strengthening of superior oblique or weakening ipsilateral inferior oblique or contralateral inferior rectus is done .The results of surgery for both congenital and acquired IV nerve palsy is excellent

REVIEW OF LITERATURE

1.**Rucker et al** [7-] Head trauma caused third nerve palsy , fourth nerve palsy, sixth nerve palsy in 14 cases (22.2%), 5 cases(41.6%), 9 cases (10.2%) respectively. Some of these patients with third nerve palsy had associated vision loss, internal ophthalmoplegia ,cortical blindness or homonymous hemianopia . About half of these cases did not show any recovery during the follow up period. Fractures of the skull also caused third nerev palsy. Head injury was found to be the most common of multiple nerve palsies in about 26.4% cases.

2. **Fang C, Leavitt JA et al**[8] reported that the most common causes of acquired third nerve palsy were microvascular (42%) followed by trauma (12%).

3.**Richards et al**[9] reported a much larger retrospective series of 4176 cases of acquired 3rd, 4th and 6th cranial nerve palsies seen over 40 years in the Mayo Clinic Department of Ophthalmology. They found a 15% occurrence of isolated 3rd nerve palsy attributable to head trauma.

4. Elston et al[10] ., studied 20 patients referred to Moorfields Eye Hospital, after traumatic third nerve palsy. All the patients sustained a closed head injury with prolonged loss of consciousness in a high speed deceleration accident.

5. Tokuno et al[11]., 71% had traumatic Subarachnoid hemorrhage and 57% had evidence of skull fractures, both of which seemed to be the cause for third nerve palsy.

6. Erenler et al[12] - Minor traumas may cause unilateral oculomotor nerve palsy without any additional injuries. In such patients, neuroimaging studies may also be normal.

7. Park UC et al[13] - Head trauma caused 18.7%, 30.4%, 19.4% of third, fourth and sixth nerve palsy respectively. Overall recovery rate was highest in patients with traumatic cause. Complete recovery rate was about 87.5% in traumatic ocular motor nerve palsies.

8. **Choi K D et al** [14] - Abducent nerve palsy was the most common (40%) of the acquired ocular motor nerve palsies, followed by oculomotor nerve palsy (27%) and trochlear nerve palsy (23%). The etiologies were microvascular ischemia (47%), inflammatory (21%) followed by trauma (5%).

9.**Jacobson et al** [15] - Fourth nerve palsy after mild head trauma has been observed in association with an underlying asymptomatic basal intracranial tumor in at least three reports.

10.**Baker et al**[16] **-** All aspects of head and face trauma that can lead to ocular motility disturbances.

11.**Hoya et al**[17]**-** Traumatic fourth nerve palsy can occur due to minor occipital impact with normal neuroimaging findings.

12..**Teller et al**[18] - Trochlear nerve injury was caused by slight trauma in 46% of cases.

13.**Buger et al**[19-] Bilateral trochlear nerve palsy occurred due to diffuse frontal impact.

14. **Valsa steven et al**[20] - The most common ocular motor nerves involved were abducent nerve (46.7%) followed by oculomotor nerve (23.3%), combined nerve involvement (20%), and finally by trochlear nerve (10%). The most common cause was trauma (36.7%).

15. **Berlit P et al**[21] - Manheim Neurological clinic, University of Heidelberg. It was a retrospective study based on the medical records of 412 patients. Palsies of the III nerve (n=172) and VI nerve (n=165) were more frequent than IV nerve (n=25). Combined nerve palsies (n=50) were generally combinations of the III and IV (n=21) or paresis of all 3 cranial nerves (n=17).

165 ocular nerve palsies were due to vascular causes – in 135 of them diabetes and or hypertension was present. In inflammatory diseases and brain tumors the abducent nerve was the most frequently affected.

AIM OF THE STUDY

1. To study the clinical profile of ocular motor nerve palsies following trauma

2. To study the mode of injury

3. To study the multiplicity of nerve involvement

4. To study the most commonly involved nerve palsy following trauma

5. To assess the recovery of post traumatic nerve palsy

MATERIALS & METHODS

The cases studied included those patients with neurogenic motor nerve palsies who presented to RIOGOH, Egmore, Chennai for a period of 1 year,March 2018-Feb 2019

Methodology:

Patient presenting to Neuro ophthalmology services, Regional Institute of Ophthalmology, Government Ophthalmic Hospital were registered, evaluated and followed up during the study period.

A detailed history and complete evaluation of the patient presenting with nerve palsies are reported.

INCLUSION CRITERIA:

Patients presenting with ocular motor nerve palsies with trauma either ocular or head injury whose general condition is stable.

EXCLUSION CRITERIA:

Patients presenting with ocular motor nerve palsies due to causes other than trauma and also patient with previous neurological disorders.

Patients with poor general condition requiring critical care.

REGISTRATION

Name

Age

Sex

Occupation

Address

HISTORY OF PRESENT ILLNESS

The common complaints were:

a. Drooping of eyelids-

   Unilateral/ bilateral

   Partial/total

b. Double vision-

   uniocular /binocular,

   constant/intermittent,

   more for near or distance,

in which gaze

whether images were horizontally or vertically separated,

c. Pain –

headache/periorbital pain, location, nature,

radiating/ not,

aggravating and relieving factors,

associated with nausea/vomiting.

d. Defective vision- apart from double vision, any blurring or inability to see due to drooping of lid.

e. Abnormal head posture

f. Deviation of eyeball-right/left, eye, duration

Any other significant medical/surgical history is also recorded.

PAST HISTORY

H/o systemic illness

H/o trauma- nature of injury, onset of nerve palsy

Treatment details

PERSONAL HISTORY

Smoking, alcoholism etc.

GENERAL EXAMINATION

General vital data like pulse, blood pressure, peripheral pulses are noted. It gives an idea of the health status of the patients.

OCULAR EXAMINATION

- Head posture, facial symmetry are noted.
- Any deviation of eyeball is noted.
- Extraocular movements are noted down-both ductions and versions. When checking for EOM, the aberrant innervation patterns are also looked for.

For all cases both right and left eyesare examined.

- Vision a. uncorrected (by Snellen's charts at 6 metres)

  b. best corrected.

- slit lamp examination done, details of the anterior segment from the lids to the lens are noted.

Lid

Conjunctiva

Cornea

Iris

Pupil

Anterior chamber

Lens

- A dilated fundus examination and refraction is done.
- Intra ocular pressureis recorded with goldmann applanation tonometer after topical anaesthesia.
- Measurement of deviation-primary & secondary deviation, cover uncover test in various gaze positions, for both near and distance is checked.
- Diplopia charting-

  Patient is asked to wear red and green goggles in such a way that red is in front of the right eye and green in front of the left eye. A torch light with the stenopaeic slit is used. The patient is asked to look at the torch light held 120cm away and then the torch light is moved to all 9 positions of gaze. The

false image is the fainter and farther one. Any tilt of the image and the distance between images at various position is asked for.

- Hess charting
- If superior oblique palsy is suspected, Parks Bielchowsky's 3 step head tilt test is done.

  The medial and lateral rectus muscles do not have a vertical action. The hypertropia of paretic etiology is due to weakness of one or more of the following vertically acting muscles

  a. Right inferior oblique (RIO) ; Left inferior oblique (LIO)

  b. Right superior oblique (RSO) ; Left superior oblique (LSO)

  c. Right inferior rectus (RIR) ; Right inferior rectus (LIR)

  d. Right superior rectus (RSR) ; Left superior rectus (LSR)

If the hypertropia is due to weakness of only one of these eight muscles, answering the following questions identifies the paretic muscle.

1. First step – which is the higher eye ?

a) If the patient has a Right hypertropia then the weak muscle is either a depressor of the RE (RSO/RIR) or an elevator of the LE(LSR / LIO).

b) If the patient has Left hypertropia , then the weak muscle is either an elevator of the RE (RSR / RIO) or depressor of the LE (LSO/LIR)

2. Second step – hypertropia worse on right or left gaze?

- The vertical rectus muscles (superior and inferior recti) have their greatest vertical action when the eye is abducted .

-The oblique muscles (superior and inferior obliques) have their greatest vertical action when the eye is adducted .

i. Right hypertropia worse on gaze right (RIR / LIO)

ii. Right hypertropia worse on gaze left (RSO / LSR)

iii. Left hypertropia worse on gaze right (LSO / RSR)

iv. Left hypertropia worse on gaze left (RIO / LIR)

3. Third step – Is the hypertropia worse on head tilt towards right or left?

a. The superior muscles (SR and SO) intort the eye ; the inferior muscles (IR and IO) extort the eye.

b. When the head is tilted to the right , right eye will be intorted by the contraction of the RSR and RSO ; these two muscles work together in effecting the intorsion and hence neutralize each others vertical action (RSR is an elevator and RSO is a depressor) .

c. If one of these muscles is the paretic muscle responsible for the hypertropia, then the vertical action will not be neutralized and the hypertropia becomes worse on tilting the head towards the right shoulder

- A forced duction test is done in doubtful cases to rule out restrictive etiology.

NEUROLOGIC EXAMINATION

Examination of other cranial nerves

Examination of motor, sensory and cerebellar system.

RADIOLOGY

X ray orbit

CT brain with orbit

MRI brain with orbital cuts

Specialist opinion (in indicated cases)

Neurophysician/Neurosurgeon

FOLLOW UP

Recording of patient's complaints-whether stable/improving/worsening.

- Vision
- Pupil assessment
- Extraocular movements
- Diplopia charting
- Fundus
- Examination for abnormal pupil or aberrant regeneration is done in case of third nerve palsy

RESULTS

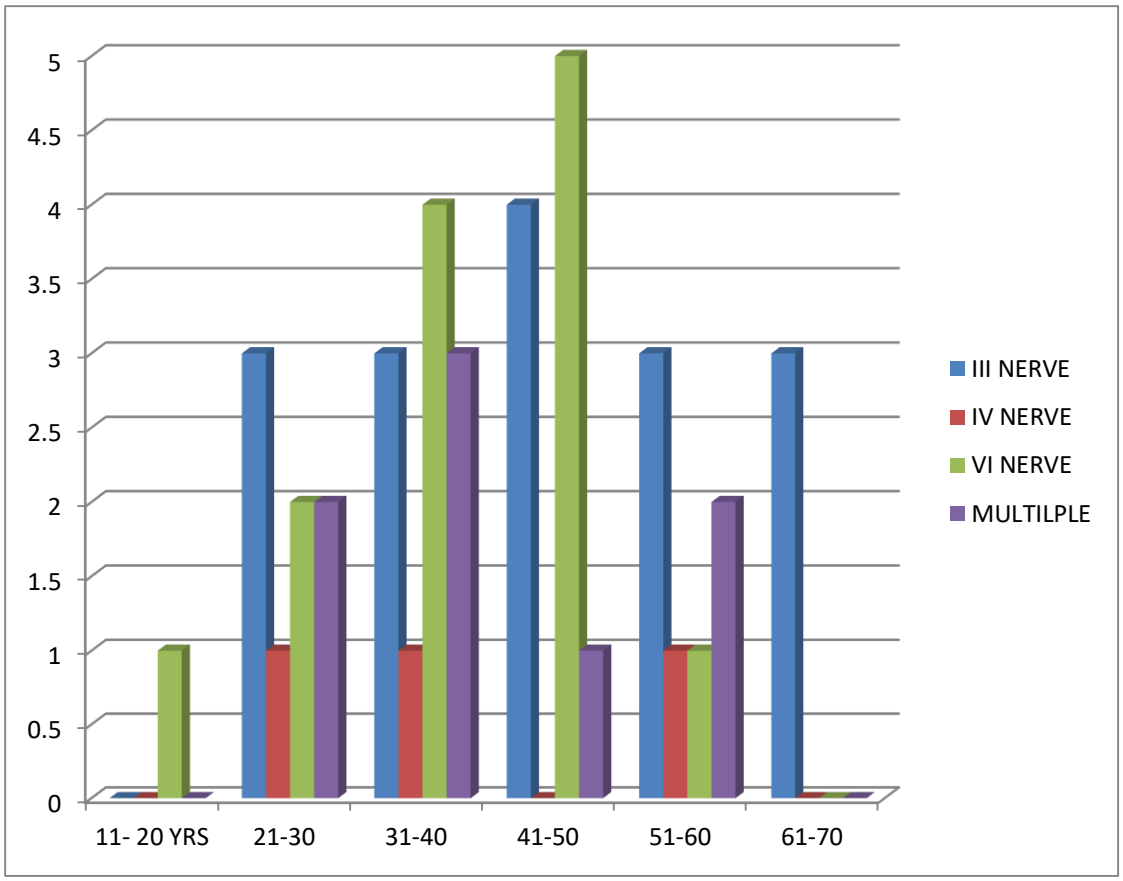
40 cases of ocular motor nerve palsy following trauma were examined. A prospective study was conducted.

1. AGE DISTRIBUTION:

The following table shows the age distribution in various ocular motor nerve palsies following trauma.

	III nerve	IV nerve	VI nerve	Multiple nerves	Total
10-20	0	0	1	0	1
21-30	3	1	2	2	8
31-40	3	1	4	3	11
41-50	4	0	5	1	10
51-60	3	1	1	2	7
61-70	3	0	0	0	3
					40

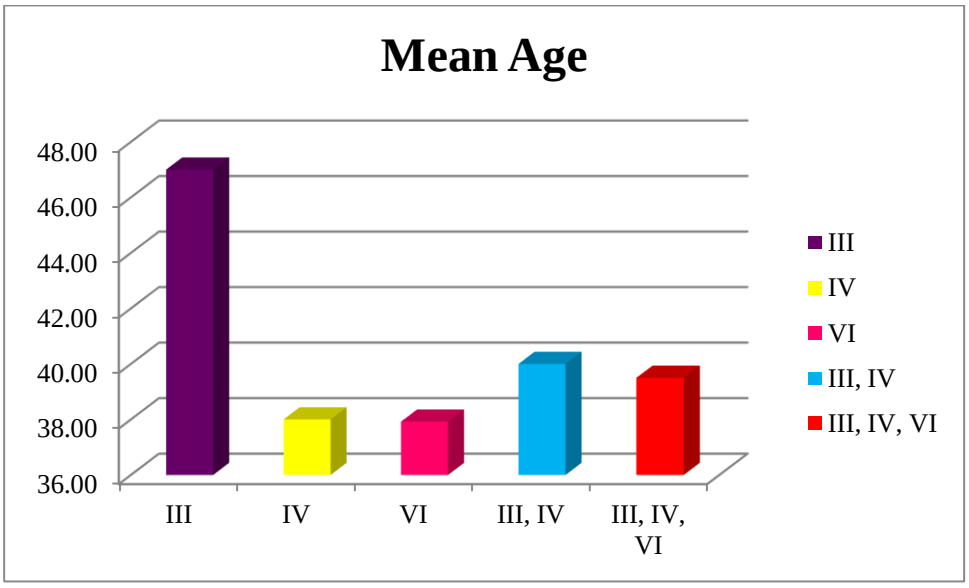
Regarding the age distribution, considering all the nerve palsies in total maximal number of patients belonged to 31- 40 years age group (27.5%), followed in frequency by 41-50 years age group with 25% of patients, 21-30 years age group with 20% patients, 51-60 years age group with 17.5% of patients, 61-70 years age group with 7.5% of the patients. The least number was seen in the age group of 10-20 years(2.5%).



AGE DISTRIBUTION

Nerves Involved	N	Mean Age	SD	Min	Max
III	16	47.00	14.36	25	68
IV	3	38.00	15.13	26	55
VI	13	37.92	11.20	17	55
III, IV	2	40.00	8.49	34	46
III, IV, VI	6	39.50	13.55	26	60

The mean age of presentation of third nerve palsy was 47 with standard deviation of 14.36. the mean age of presentation of IV nerve palsy, VI nerve palsy were 38 and 37.92 respectively. All the ocular motor nerves were affected in mean age of 39.50.



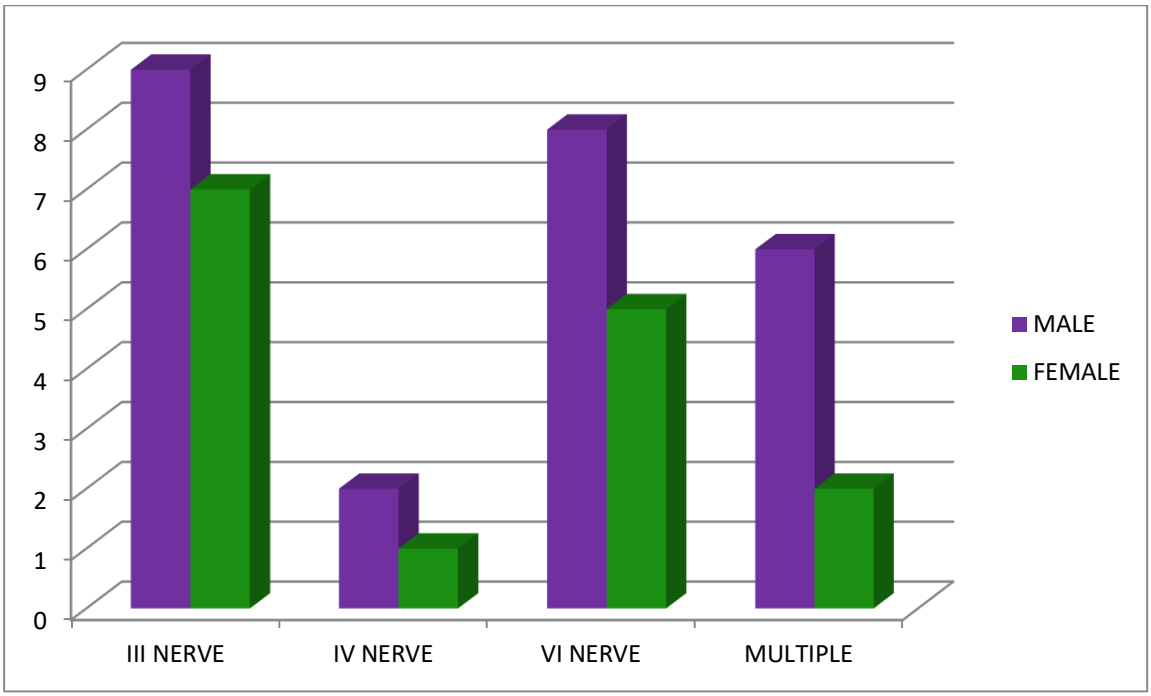
2.SEX DISTRIBUTION:

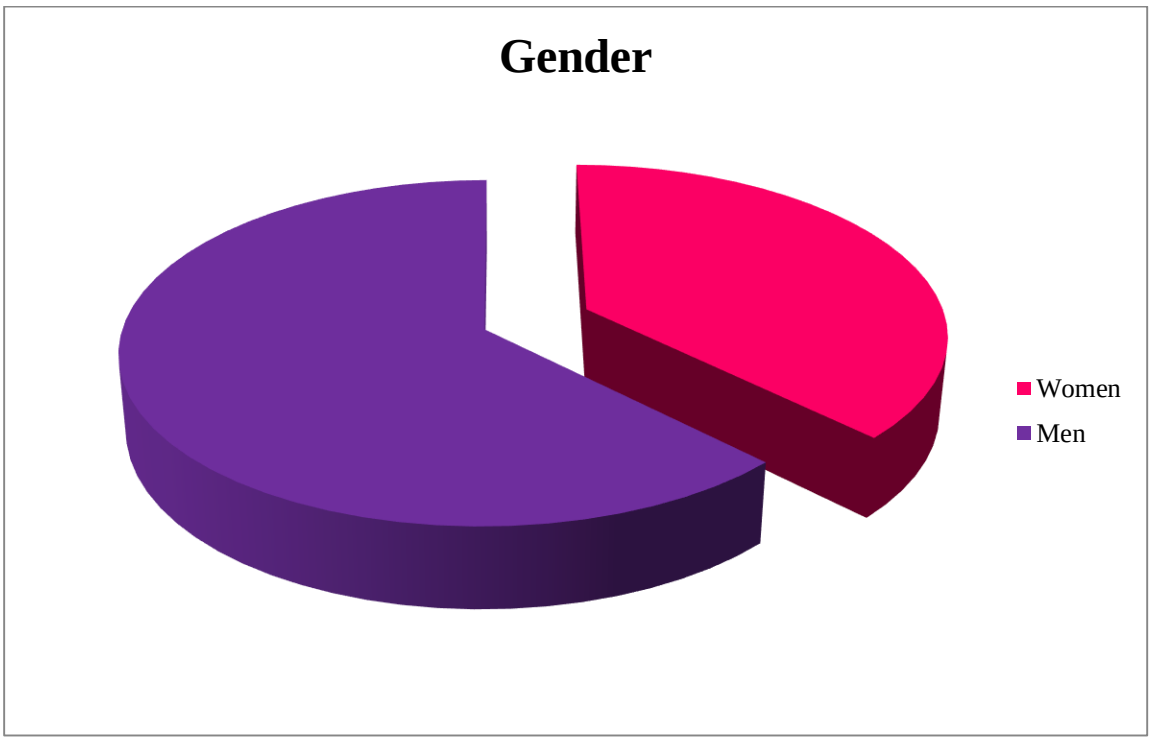
	MALE	FEMALE	TOTAL
III	9	7	16
IV	2	1	3
VI	8	5	13
MULTIPLE	6	2	8

Sex	Frequency.	Percent
Women	15	37.5
Men	25	62.5
Total	40	100

In the study , males were commonly affected (62.5%) when compared to females(37.5%). The incidence of III nerve palsy(22.5%) was higher in males followed by VI nerve palsy(20%), multiple nerve palsy(15%) and fourth nerve palsy in 5% of patients. In females,the incidence of III nerve palsy(17.5%) was higher, followed by VI nerve palsy(12.5%), multiple nerve palsy(5%) and fourth nerve palsy in 2.5% of patients.

SEX DISTRIBUTION

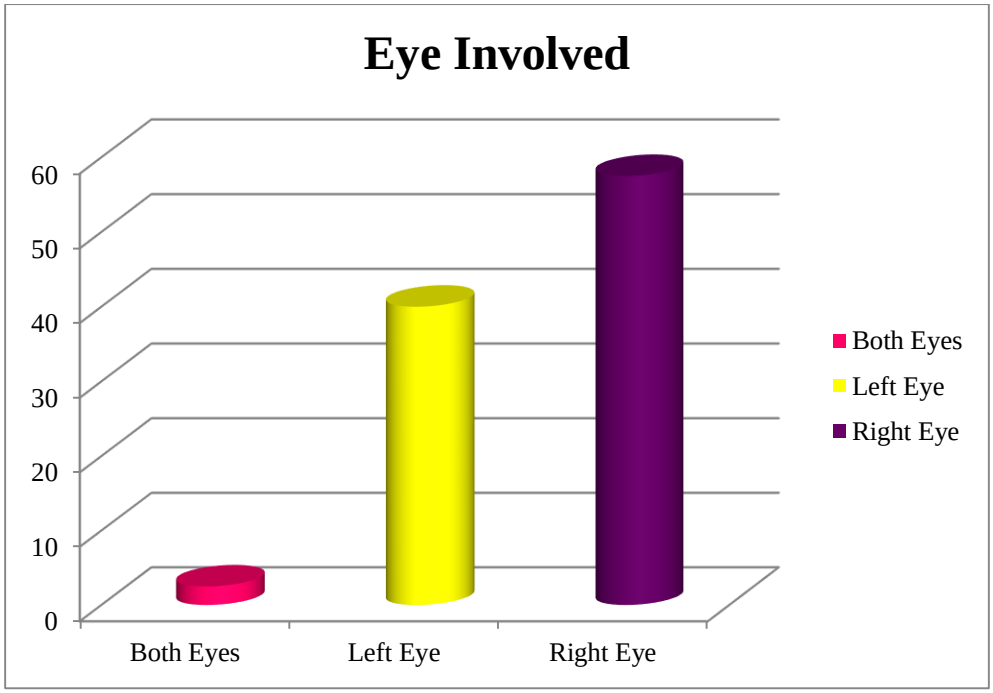




3.LATERALITY

Laterality	Frequency	Percentage
Both Eyes	1	2.5
Left Eye	16	40
Right Eye	23	57.5
Total	40	100

Right sided nerve involvement was common (57.5%) when compared to left sided nerve palsy(40%) following trauma. One patient had bilateral nerve palsy.

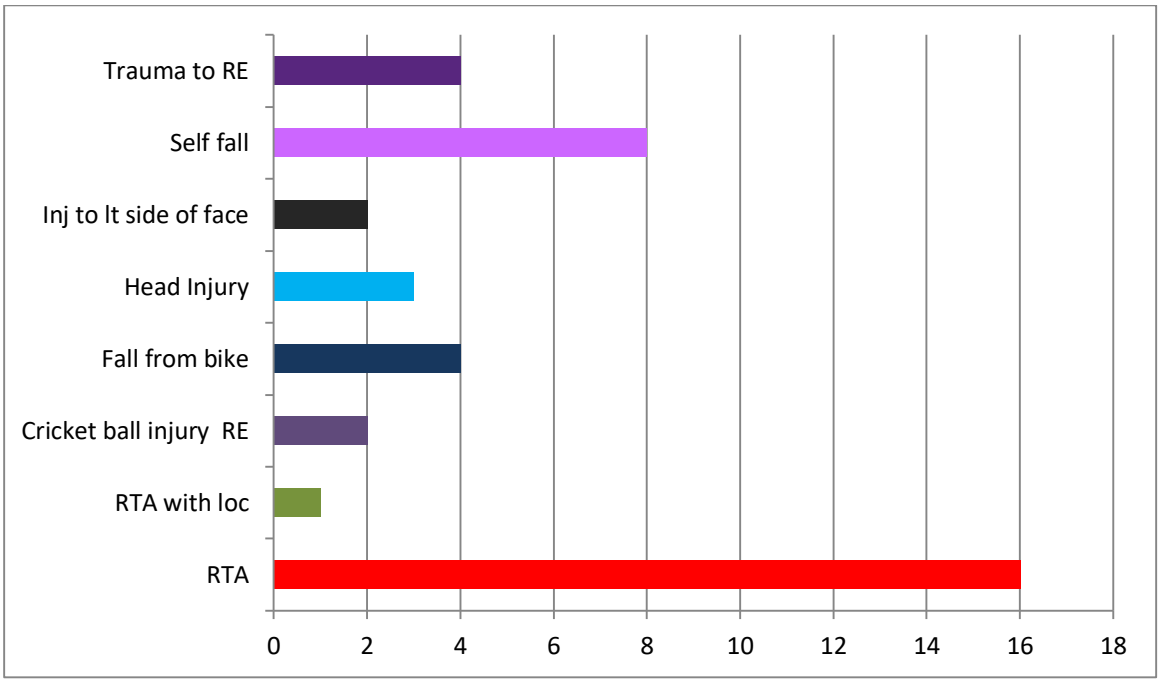


4.NATURE OF INJURY:

History	Freq.	Percent
RTA	16	40
RTA with loc	1	2.5
Cricket ball injury RE	2	5
Fall from bike	4	10
Head Injury	3	7.5
Inj to lt side of face	2	5
Self fall	8	20
Trauma to RE	4	10
Total	40	100

In the study RTA was most common cause of ocular motor nerve palsy in 40% of the patients. Ocular trauma caused about 15% cases of ocular motor nerve palsy. Trauma due to self fall and sustained injury caused ocular motor nerve palsy in 20% of the cases. Other causes like injury to the face also caused ocular motor nerve palsy in 5% of the cases.

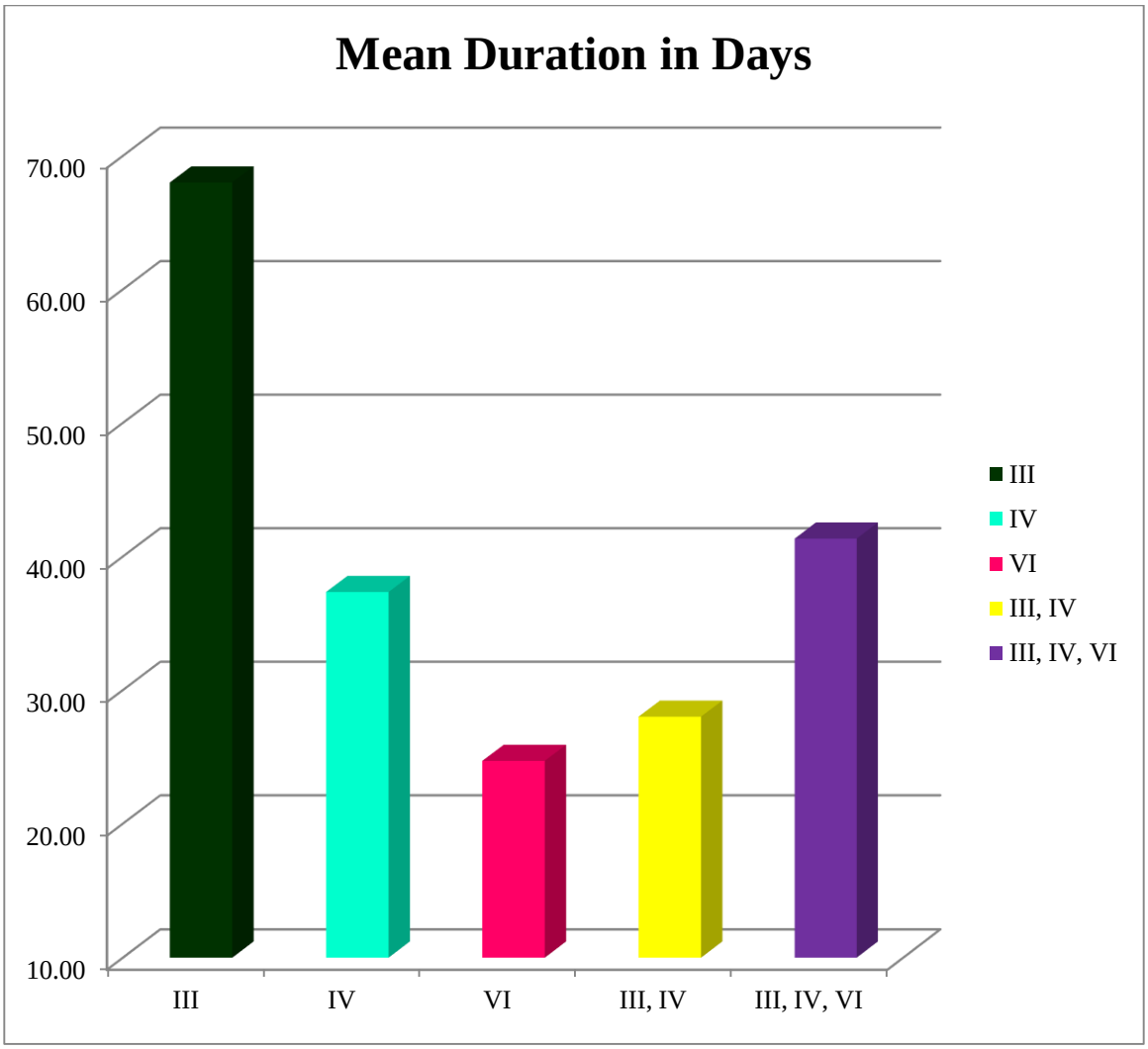
NATURE OF INJURY



5.DURATION OF PRESENTATION OF PATIENT

Nerves Involved	FREQUENCY	Mean Duration in Days	SD	Min	Max
III	16	68.00	116.72	7	364
IV	3	37.33	16.17	28	56
VI	13	24.69	16.33	7	56
III, IV	2	28.00	0.00	28	28
III, IV, VI	6	41.33	63.45	7	168

In the study the ocular motor nerve palsy the patients presented between 7 days and 1 year following injury with mean duration of 68 days.

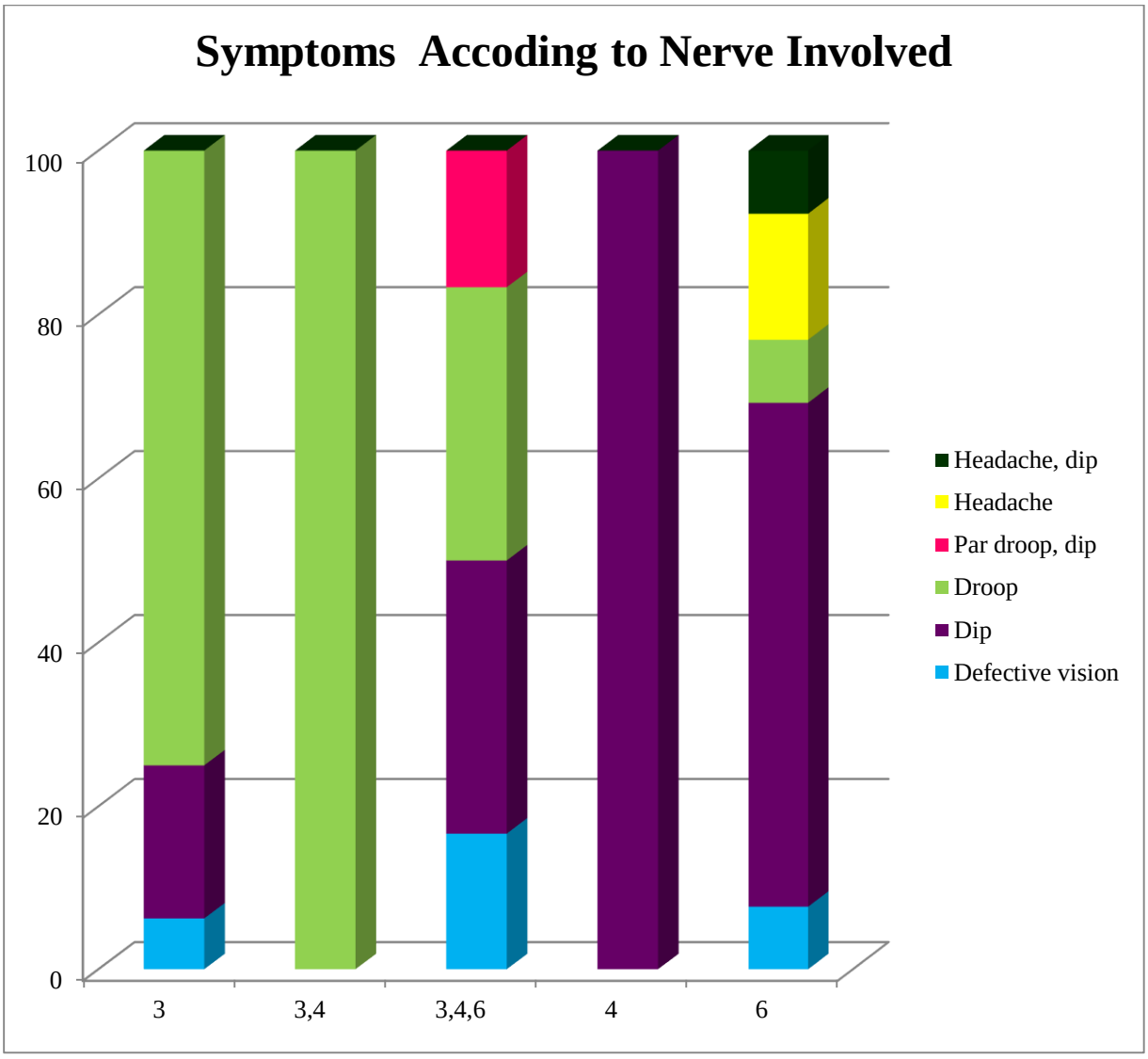

Mean Duration in Days
70.00
60.00
50.00
40.00
30.00
20.00
10.00
III
IV
VI
III, IV
III, IV, VI
III
IV
VI
III, IV
III, IV, VI

6..PRESENTING SYMPTOMS:

	3		3,4		3,4,6		4		6	
Pre Symp	Freq.	Percent	Freq.	Percent	Freq.	Percent	Freq.	Percent	Freq.	Percent
Defective vision	1	6.25	0	0	1	16.67	0	0	1	7.69
Diplopia	3	18.75	0	0	2	33.33	3	100	8	61.54
Drooping	12	75	2	100	2	33.33	0	0	1	7.69
Partial droop, diplopia	0	0	0	0	1	16.67	0	0	0	0
Headache	0	0	0	0	0	0	0	0	2	15.38
Headache, diplopia	0	0	0	0	0	0	0	0	1	7.69
Total	16	100	2	100	6	100	3	100	13	100

The symptoms varied according to the patients. The most common symptom in third nerve palsy was drooping of eyelid following trauma in 75% of patients. Mutltiple nerve palsy either presented with the symptom of diplopia or drooping in 33% of patients. All patients with fourth nerve palsy presented with diplopia.

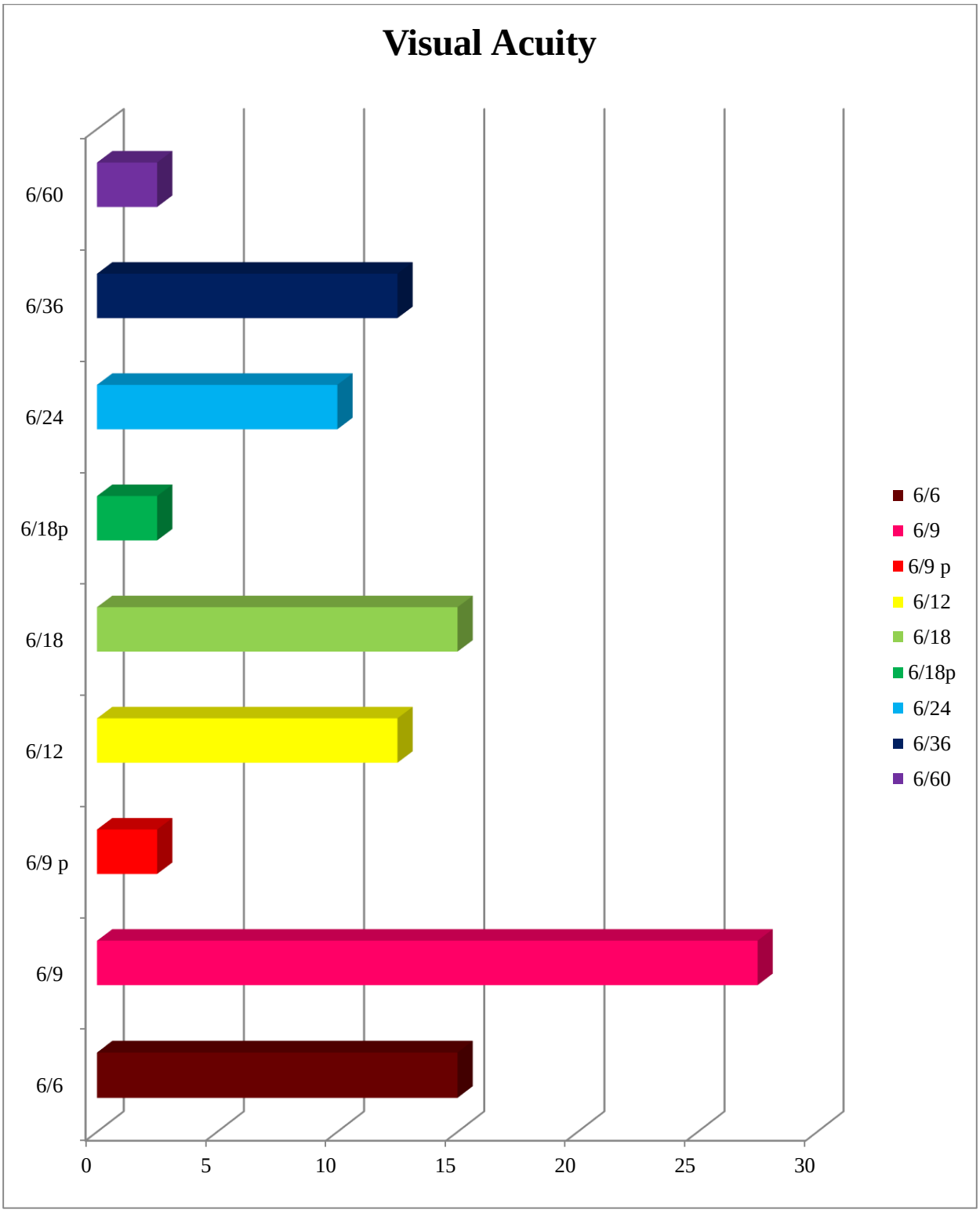
Sixth nerve palsy patients presented with diplopia in 61.54% of patients. Other patients with sixth nerve palsy had either headache(15.38%) or diplopia associated with headache(7.69%).


Symptoms Accoding to Nerve Involved
100
80
60
40
20
0
3
3,4
3,4,6
4
6
Headache, dip
Headache
Par droop, dip
Droop
Dip
Defective vision

7..VISUAL ACUITY:

VA	Frequency	Percentage
6/6	6	15
6/9	11	27.5
6/9 p	1	2.5
6/12	5	12.5
6/18	6	15
6/18p	1	2.5
6/24	4	10
6/36	5	12.5
6/60	1	2.5
Total	40	100

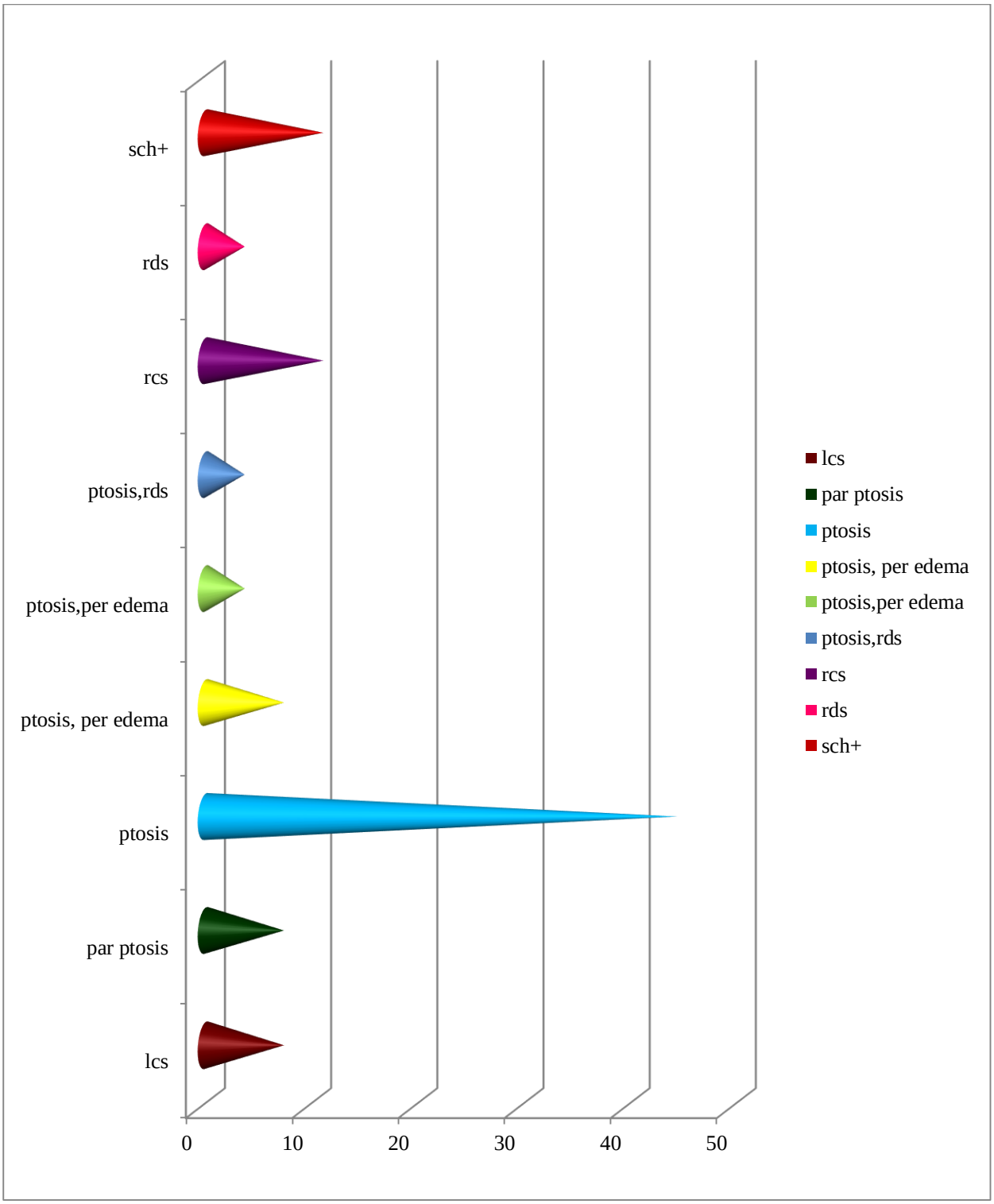
Visual acuity varied between 6/60 to 6/6. Many patients had visual acuity of 6/9(27.5), followed by 6/6 vision(15%).


Visual Acuity
6/60
6/36
6/24
6/18p
6/18
6/12
6/9 p
6/9
6/6
0
5
10
15
20
25
30
6/6
6/9
6/9 p
6/12
6/18
6/18p
6/24
6/36
6/60

8.ANTERIOR SEGMENT

ANT SEGMENT	Frequency	Percent
LCS	2	7.41
PARTIAL PTOSIS	2	7.41
PTOSIS	12	44.44
PTOSIS, PER EDEMA	3	11.1
PTOSIS,RDS	1	3.7
RCS	3	11.11
RDS	1	3.7
SCH+	3	11.11
TOTAL	27	100

Majority of the patients being III nerve palsy presented either with complete ptosis(44.4%) or partial ptosis(7.41%) or ptosis with RDS(3.7%). some patients had associated periorbital edema(11.1%). VI nerve palsy presented as convergent squint in 18.52% of patients.



ANTERIOR SEGMENT

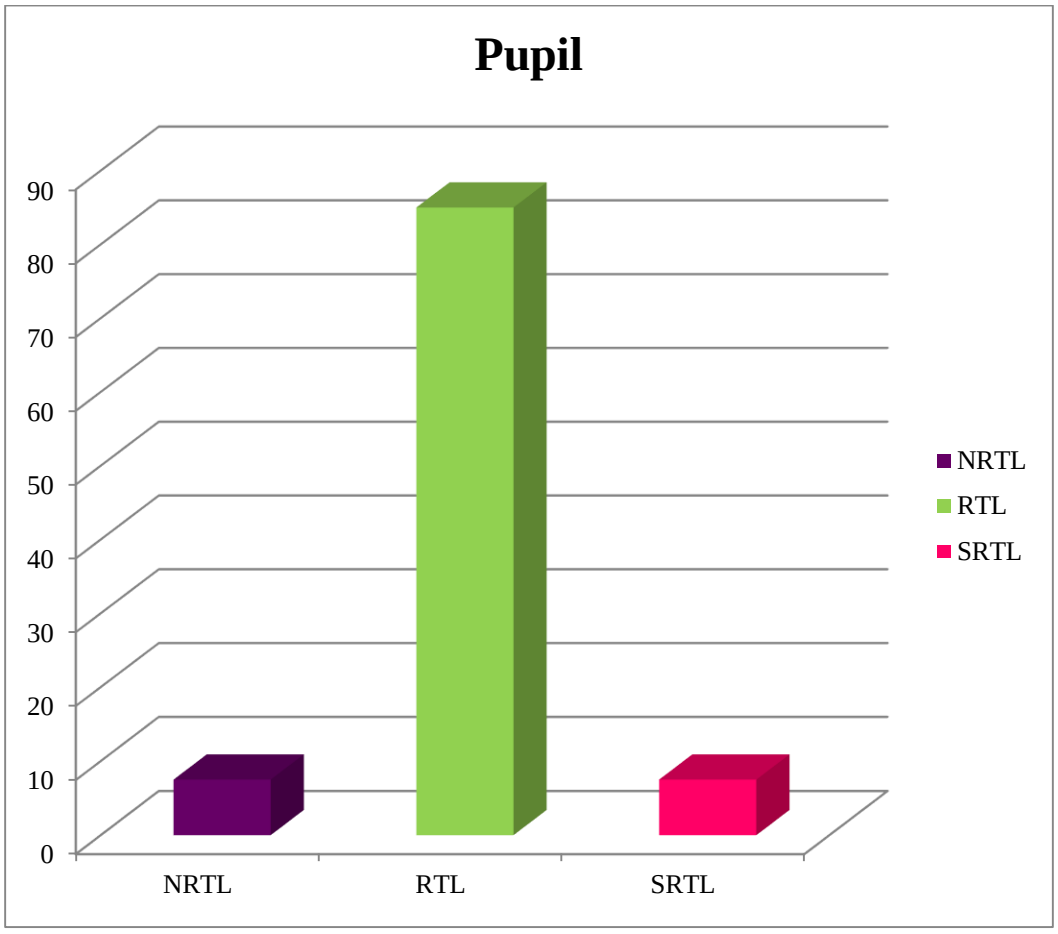
9.EXTRA OCULAR MOVEMENTS

EOM	Freq.	Percent
abd res	13	32.5
add,ele,dep res	16	40
add,ele,dep,abd ,int res	6	15
add,ele,dep,int res	3	7.5
int res	3	7.5
Total	40	100

Third nerve palsy patients had restriction of elevation, depression, adduction (4o%). When combined with fourth nerve presented along with intorsion restriction. When all the ocular motor nerves are involved all movements were restricted in 15% of patients.

10.PUPIL INVOLVEMENT

PUPIL	Freq.	Percent
NRTL	3	7.5
RTL	34	85
SRTL	3	7.5
Total	40	100



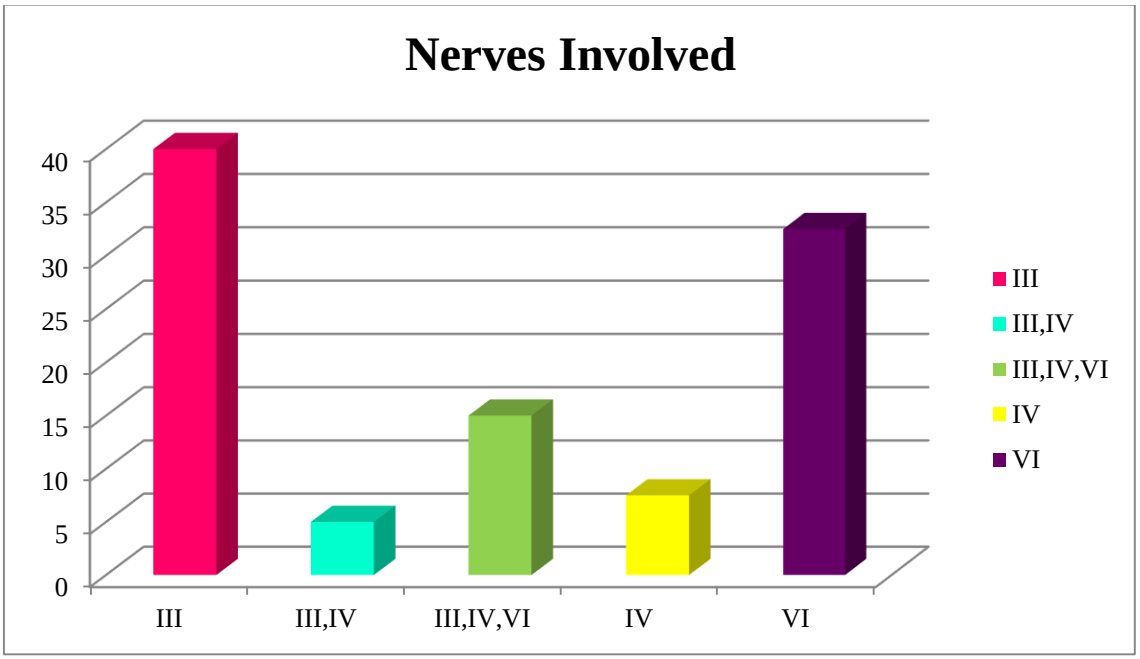
Majority of the patients had their pupils reacting to light(85%). 3 patients with third nerve palsy had pupil reaction sluggishly reacting to light, 2 patients had pupil not reacting to light. 1 multiple nerve palsy patient had pupil not reacting to light.

11.FUNDUS EXAMINATION was normal in all patients.

12.OCULOMOTOR NERVES INVOLVED

NERVES	Freq.	Percent
III	16	40
III,IV	2	5
III,IV,VI	6	15
IV	3	7.5
VI	13	32.5
Total	40	100

The most common nerve involved in trauma is the oculomotor nerve(40%), followed by VI nerve(32.5%), III,IV,VI nerves(15%), IV nerve(7.5%) respectively.



13. INVESTIGATIONS:

X RAY/CT/MRI	Freq.	Percent
# R parietal bone	1	2.5
# postero lateral wall L maxillary sinus	1	2.5
#L temporal bone	1	2.5
L tempero parietal contusion	1	2.5
L zygomatic complex #	1	2.5
NAD	22	55
R frontal lobe contusion	1	2.5
R lateral wall orbit#	1	2.5
R temporal bone #	1	2.5
R zygomatic complex#	1	2.5
Brain stem contusion,IVH in occipital horn	1	2.5
contusion R parietal lobe	1	2.5
Frontal contusion	1	2.5
Haemmorhagic contusion of R frontal lobe	1	2.5
Haemorrhage R caudate nucleus	1	2.5
Left lat wall of orbit #	1	2.5
Linear blooming foci of r temp lobe	1	2.5
Multiple facial bone injury with buttress plate	1	2.5
skull base#	1	2.5
Total	40	100

In 55% of patients neuroimaging was normal. Whereas in remaining 45% of patients had significant abnormalities in neuroimaging.

14.RECOVERY PATTERN

Recovery	Freq.	Percent
FR	23	57.5
NR	5	12.5
PR	10	25
Aberrant regenration	2	5
Total	40	100

Traumatic nerve palsies had full recovery in 57.5% of patients. Partial recovery in 25% of patients.12.5% were not recovered at the end of the study.

15. RECOVERY ACCORDING TO THE NERVE INVOLVED

	3		3,4		3,4,6		4		6	
Recovery	Freq.	Percent	Freq.	Percent	Freq.	Percent	Freq.	Percent	Freq.	Percent
FR	7	43.75	1	50	2	33.33	3	100	10	76.92
NR	2	12.5	0	0	1	16.67	0	0	1	7.69
PR	5	31.25	1	50	2	33.33	0	0	2	15.38
ab reg	2	6.25	0	0	0	0	0	0	0	0
Total	16	100	2	100	6	100	3	100	13	100

In this study, 43.75% of patients with third nerve palsy had full recovery , whereas 31.25% of patients had partial recovery and 12.5% of patients did not recover. 2 patients had aberrant regeneration. 50% of patient with combined third and fourth nerve palsy recovered and 50% had partial recovery. In patients with all ocular motor nerve palsy,33.33% of patients had full recovery, 33.33% had partial recovery, 1 patient did no recover. In patient with fourth nerve palsy all patients showed full recovery. In patients with sixth nerve palsy 76.92 % showed full recovery, 15.38% showed partial recovery respectively.1 patient did not recover.

DISCUSSION

AGE:

Majority of the ocular motor nerve palsies observed in our study affected the individuals in the 31- 40 years age group with a mean age of 41.90. Most of the patients with third nerve palsy were in the age group of 21- 70 years with the mean age of 47. In a study of 22 cases of III nerve palsy by Jack E Goldstein and David G. Cogan the average age was 62 years. The third nerve was the commonly affected nerve in this study whereas in literature the fourth nerve palsy is the most common nerve affected following trauma. Our study is similar to the studies of Patel et al in which there was a preponderance of male patients (87 males)

When compared to 13 female patients.

SEX:

In this study, there was male preponderance of about 62.5% when compared to females 37.5%.. This is slightly higher when compared with studies by Rush and Richards Jones[7] and Young where it was 52% and 54% respectively.

According to valsa et al[20] ocular motor nerve palsies due to traumatic causes were more seen in males compared to females (63.6% vs. 36.4%).This is similar to our study.

LATERALITY:

In the present study, right eye involvement (57.5%) was more when compared to left eye(40%). The laterality does not seem to have any significance comparing with the study of Goldstein & Cogan where right and left eye were equally affected.

NATURE OF INJURY:

Road traffic accident (40%) was the most common of ocular motor nerve palsies following trauma in our study. Head injury caused about 7.5% ocular motor nerve palsies but according to Patel et al the incidence of cranial nerve injury following head injury is 12.6% which is higher that our study.

Memon and Paine[22] reported 1.1% incidence of direct third nerve palsy in 1100 head injuries, studied at the University Hospital, Saskatoon, Canada over a 10 year period. But in our study 7.5% cases of ocular motor nerve palsy were due to head injury.

Dhaliwal et al[23] study reported that third cranial nerve palsy had the most severe head injury than those with fourth cranial nerve palsy which had an intermediate level of head injury; and those with cranial nerve 6 palsy had the lowest level of head injury.

According to Nagaseki et al [24] the trauma required to damage the oculomotor nerve is severe and associated with other neurologic deficits, basilar skull fracture, orbital injury or subarachnoid hemorrhage. But in our study study head injury and ocular injury caused third nerve palsy and associated neurological deficits were not noted.

According to Young et al[25] trauma accounts 44% of acquired trochlear nerve palsies with nearly 25% of cases being bilateral.But in our study 15% of cases of isolated trochlear nerve palsy were due to trauma.

OCULAR MOTOR NERVE INVOLVEMENT

According to Patel et al, cranial nerve palsies occurred either single or multiple. 12 cases with isolated third nerve palsy,7 cases with isolated abducent and trochlear nerve palsy. In this study 16 cases of isolated third nerve and 13 cases of isolated sixth nerve was seen which is higher that that of Patel et al study.

Solomons et al[26] reported 1.2% incidence of isolated direct third nerve palsy in 2100 patients with head injury treated in the neurosurgery department of a teaching hospital, Cape Town, South Africa over a 12 year period. But in our study the incidence of third nerve palsy is about 40%.

Minor head injury may precipitate third nerve palsy in patients with occult intracranial mass lesion because of the mechanical stress[27,28]. Our patients, however did not have any of the above mentioned features,

According to Kwartz et al [29] 4th nerve palsy can occur after apparently trivial head trauma. But in our study 2 cases of fourth nerve palsy occurred due to head injury and one case occurred due to head injury.

According to Valsa et al traumatic nerve palsies more commonly involved are 3rd and 6th nerves (36.4% each) followed by 4th nerve followed by combined nerve palsies (9%). This is in contrast to our study where the common nerve involved was ocular motor nerve(40%) followed by sixth nerve(32.5%).

According to Lee et al [30] study of 237 patients with presumed isolated sixth nerve palsy,31 cases were due to traumatic origin.

RECOVERY PATTERN

According to Colpak et al31 study of 112 patients, 79 patients had full recovery, 8 patients of third nerve palsy, 18 patients of fourth nerve palsy and 6 patients of sixth nerve palsy did not recover. Fourth nerve was the least to recover. In our study, 20 patients had full recovery(7 patients with third nerve palsy, 3 patients with fourth nerve palsy, 10 patients with sixth nerve palsy).2 patients with third nerve palsy and 1 patient with sixth nerve palsy did not show signs of recovery.

The rate of recovery of oculomotor nerve palsy caused by trauma was slow and prolonged according to the study conducted by Walter et al[32.]. But in our study the 43% patients with oculomotor nerve palsy had full recovery.

Follow-up of the patients showed that there was partial recovery of the Ocular motor nerve palsy in 31.2% of patients , which was similarly reported in a few case series[33,34]

ABERRANT REGENERATION

Aberrant regeneration may be seen after third nerve palsy due to congenital causes, trauma, aneurysm,migraine[35,36]. In our study 2 cases of third nerve palsy presented with aberrant regeneration

SUMMARY

1. Of the 40 cases studied, the age group ranged from 17-68 years. 52.5% of cases were in the 31-50 years group. Third nerve palsies occurred in a wide range of age group.

2. There was a preponderance in males (62.5%) than in females(37.5%).

3. The right eye was involved in (57.5%) , left eye in (40%) cases

4. In the study RTA was most common cause of ocular motor nerve palsy in 40% of the patients. Ocular trauma caused about 15% cases of ocular motor nerve palsy.Trauma due to self fall and sustained injury caused ocular motor nerve palsy in 20% of the cases

5. In the study the ocular motor nerve palsy prsented between 7 days and 1 year after injury

6. The symptoms varied according to the patients. The most common symptom in third nerve palsy was drooping of eyelid following trauma in 75% of patients. Mutltiple nerve palsy either presented with the symptom of diplopia or drooping in 33% of patients. All patients with fourth nerve palsy presented with diplopia.

7. Majority of the patients being III nerve palsy presented with complete ptosis (44.4%). VI nerve palsy presented as convergent squint in 18.52% of patients. Patients with IV nerve palsy presented with head tilt.

8. Majority of the patients had their pupils reacting to light (85%).

9 .Fundus examination was normal in all patients

10. The most common nerve involved in trauma is the oculomotor nerve(40%), followed by VI nerve(32.5%), III,IV,VI nerves(15%), IV nerve(7.5%) respectively

11. In 55% of patients neuroimaging was normal. Whereas in remaining 45% of patients had significant abnormalities in neuroimaging.

12. Traumatic nerve palsies had full recovery in 57.5% of patients. Partial recovery in 25% of patients.12.5% were not recovered at the end of the study.

In this study, 43.75% of patients with third nerve palsy had full recovery , whereas 31.25% of patients had partial recovery and 12.5% of patients did not recover. 2 patients had aberrant regeneration.

CONCLUSION

Cranial nerves can be injured before, during or after their passage through the skull. In addition to and following the immediate effect of injury, some of the cranial nerves may be indirectly damaged by complications such as the tissue reaction at a fracture site and increased intra cranial pressure.

Closed head injury even trivial is associated with trochlear nerve palsies while forcible head trauma as in cases of RTA can lead to III & VI cranial nerve palsies.

It is important to ask for a history of trauma and perform a detailed neurological evaluation as even a trivial trauma can predispose to intracranial bleed especially in those with vascular disease.

Careful examination and history taking of the patient is necessary. Radiological investigations should be carried out.,The imaging abnormalities may not correlate with a particular cranial nerve injury. Long term follow up of the patient is necessary.

BIBLIOGRAPHY

1. Parsons' diseases of the eye. 22nd edition. Elseiver publications.2015 page 408-409,446

2. Jack J.Kanski,'s Clinical Ophthalmology – A systematic Approach . Eighth Edition 2016, Page 821-830.

3. Rucker C Wilbur. "Paralysis of the third, fourth and sixth cranial nerves". American Journal of Ophthalmology 46.6 (1958): 787-794.

4.Patel P, Kalyanaraman S, Reginald J, et al. Post-traumatic cranial nerve injury. Ind J Neurotrauma 2005; 2:27-32.

5. Rush JA, Younge BR. Paralysis of cranial nerve III, IV and VI.Cause and prognosis in 1000 cases.Arch Ophthalmol 1981; 99: 76-80.

6.Walsch And Hoyt's clinical neuro ophthalmology The Essentials. Second edition. page 377-406

7. Rucker C Wilbur. "The causes of paralysis of the third, fourth and sixth cranial nerves". American Journal of Ophthalmology 61.5 (1966): 1293-1298.

8. Fang C, Leavitt JA, Hodge DO et al. Incidence and Etiologies of Acquired Third Nerve Palsy Using a Population-Based Method. JAMA Ophthalmol. 2017;135(1):23–28.

9.Richards BW, Jones FR, Young BR. Causes and prognosis in 4278 cases of paralysis of the oculomotor, trochlear and abducens cranial nerves. Am J Ophthalmol 1992;113:489– 96

10. Elston JS. Traumatic third nerve palsy. Br J Ophthalmol 1984;68:538–43.

11.Tokuno T, Nakazawa K, Yoshida S, Matsumto S, Shingu T, Sato S, et al. Primary oculomotor nerve palsy due to head injury: analysis of 10 cases. No Shinkei Geka. 1995; 23: 497 – 501.

12. Erenler AK, Yalçın A, Baydin A. Isolated unilateral oculomotor nerve palsy due to head trauma. Asian J Neurosurg. 2015;10(3):265–267. doi:10.4103/1793-5482.161169.

13.Park UC, Kim SJ, Hwang JM, Yu YS. Clinical features and natural history of acquired third, fourth, and sixth cranial nerve palsy. Eye(Lond) 2008;22:691–696.

14.Choi KD, Choi SY, Kim JS, et al. Acquired Ocular Motor Nerve Palsy in Neurology Clinics: A Prospective Multicenter Study. J Clin Neurol. 2019;15(2):221–227. doi:10.3988/jcn.2019.15.2.221

15.Jacobson DM, Warner JJ, Choucair AK, et al. Trochlear nerve palsy following minor head trauma: a sign of structural disorder. J Clin Neuroophthalmol 1988;8:263–268

16. Baker RS, Epstein AD. Ocular motor abnormalities from head trauma. Surv Ophthalmol 1991;35:245–267

17. Hoya K, Kirino T. Traumatic trochlear nerve palsy following minor occipital impact – four case reports. Neurol Med Chir (Tokyo) 2000;40:358–360

18.Teller j Carmon G savir. Long term follow up of traumatic unilateral superior oblique palsy.Ann Ophthalmol 20: 424-425,1988.

19. Lewis j. Burger, Nicholas H. Kalvin, j. Lawton smith, Acquired lesions of the fourth cranial nerve , Brain, Volume 93, Issue 3, 1970, Pages 567–574,

20. Valsa T Stephen, Susan Philip, K C Sreelatha. Clinical Profile of Third, Fourth, and Sixth Cranial Nerve Palsies Presenting to a Tertiary Care Ophthalmic Center. Int J Sci Stud 2017;5(3):93-97.

21.Berlit, P, Reinhardt Eckstein J, 'Krause KH. – isolated abduces paralysis-a retrospective study of 165 patients – Forschr. Neurol. Psychiatr. 1989 Jan. 57

22. Memon MY, Paine KWE. Direct injury of the oculomotor nerve in craniocerebral trauma. J Neurosurg 1971;35:461–4.

23.Dhaliwal A[1], West AL, Trobe JD, Musch DC.Third, fourth, and sixth cranial nerve palsies following closed head injury.

24. Nagaseki Y, Shimizu T, Kakizawa T, Fukamachi A, Nukui H. Primary internal ophthalmoplegia due to head injury. Acta Neurochir (Wien) 1989;97:117–122.

25. Younge BR, Sutula F. Analysis of trochlear nerve palsies. Diagnosis, etiology, and treatment. Mayo Clin Proc. 1977; 52(1):11-18.

26. Solomons NB, Solomon DJ, DeVilliers JC. Direct traumatic third nerve palsy. S AfrMed J 1980;58:109–11.

27. Eyster EF, Hoyt WF, Wilson CB. Oculomotor palsy from minor head trauma. JAMA 1972;220:1083–6.

28. Walter KA, Newman NJ, Lessell S. Oculomotor palsy from minor head trauma: initial sign of intracranial aneurysm.Neurology 1994;44:148-150.

29. Kwartz J, Leatherbarrow B, Davis H. Diplopia following head injury. Injury 1990;21:351–2.

30. Lee AG, Brazis PW. Clinical Pathways in Neuro- Ophthalmology: An Evidence-Based Approach. 2nd ed. New York: Thieme Medical Publishing; 2003:217–233

31.Çolpak and Batur Çağlayan; Isolated Third, Fourth Sixth Nerve Palsies. Turk J Neurol 2019;25:32-35

32.Walter KA, Newman NJ, Lessell S. Oculomotor palsy from minor head trauma : initial sign of intracranial aneurysm. Neurology. 1994;44:148–150.

33. Janssen K, Wojciechowski M, Poot S, De Keyser K, Ceulemans B. Isolated abducens nerve palsy after closed head trauma : a pediatric case report. Pediatr Emerg Care. 2008;24:621–623

34. Liu YT, Lee YC, Liu HC. Isolated oculomotor nerve palsy due to head injury. J Chin Med Assoc. 2004;67:149–151.

35. Barr D, Kupersmith M, Turbin R, et al. Synkinesis following diabetic third nerve palsy. Arch Ophthalmol 2000;118:132–134

36. Custer PL. Lagophthalmos: an unusual manifestation of oculomotor nerve aberrant regeneration. Ophthal Plast Reconstr Surg 2000;16:50–51.

PROFORMA

CASE NO:

NAME :

AGE/SEX:

OCCUPATION:

IP NO:

DATE:

CHIEF COMPLAINTS:

HISTORY:

1.VISUAL COMPLAINTS

2.DROOPING OF EYELIDS

3.DIPLOPIA

4.HEADACHE

MODE OF INJURY:

TIME OF INJURY:

PREVIOUS MEDICAL HISTORY:

PERSONAL HISTORY :

VISUAL ACUITY AT THE TIME OF EXAMINATION :

EXAMINATION: **RE** **LE**

HEAD POSTURE: Head tilt/ Face turn / chin lift

LIDS

CONJUNCTIVA :

CORNEA :.

IRIS :

ANTERIOR CHAMBER :

PUPIL :

LENS :

EXTRA OCULAR MOVEMENTS:

FUNDUS :

INTRA OCULAR PRESSURE :

FORCED DUCTION TEST:

OTHER CRANIAL NERVE EXAMINATION:

CNS EXAMINATION:- HIGHER FUNCTIONS

HESS SCREEN:

DIPLOPIA CHARTING:

INVESTIGATIONS :

X-RAY/ CT SCAN/MRI SCAN:

FOLLOW UP

KEY TO MASTER CHART

Re – right eye

Le- left eye

Dip- diplopia

Droop- drooping

Def vn- defective vision

RTA- Road traffic accident

Inj- injury

Add- adduction

Ele-elevation

Dep-depression

Int-intorsion

Abd- abduction

Rtl- reacting to light

Srtl- sluggish reaction to light

#- fracture

FR/PR/NR- full/partial/no recovery

Ab reg- aberrant regeneration

LOC- loss of consciousness

Pre symp- presenting symptom

Lat- lateral

IVH- intraventricular haemmorhage

Occ- occipital

m- male

f-female

s.no	Name	age	pre symp	duration in wk	history	sex	later	VA	A.S	EOM	PUPIL	FUNDU	NERVES	CNS	CT/MRI	FOLLOW UI	
1	Gnanasekar	60	droop	1 1/2	RTA	M	re	6/12	ptosis	add,ele,dep res	rtl	normal	III	NAD	NAD	4 mon	NR
2	mohan	52	dip	1	self fall	M	re	6/18	ptosis, per	add,ele,dep,abd, i	rtl	normal	III,IV,VI	NAD	NAD	3 mon	FR
3	deivanai	65	droop	1	cricket ball inj Re	F	re	6/18p	ptosis, per	add,ele,dep res	rtl	normal	III	NAD	NAD	4 mon	FR
4	murugammal	42	dip	1	inj to lt side of face	F	le	6/9	lcs	abd res	rtl	normal	VI	NAD	NAD	6 mon	FR
5	Shobini	28	headache	4	RTA	f	re	6/6	lcs	abd res	rtl	normal	VI	NAD	NAD	6 mon	FR
6	ganesh kumar	17	dip	4	self fall	m	le	6/9 p		abd res	rtl	normal	VI	NAD	NAD	4 mon	FR
7	mohana priya	26	par droop	6	head inj	f	re	6/9	par ptosis	add,ele,dep,abd ,i	rtl	normal	III,IV,VI	NAD	skull base#	6 mon	PR
8	kanchana	36	droop	7	self fall	f	le	6/18	ptosis	add,ele,dep res	rtl	normal	III	NAD	NAD	5 mon	PR
9	anjalai	50	dip	1	self fall	f	re	6/36	rds	add,ele,dep res	rtl	normal	III	NAD	NAD	5 mon	PR
10	murugesan	46	droop	4	RTA	m	le	6/18	ptosis	add,ele,dep,int res	rtl	normal	III,IV	NAD	L tempero parietal contusion	5 mon	PR
11	sherif	26	dip	4	RTA	m	be	6/9		int res	rtl	normal	IV	NAD	NAD	3 mon	FR
12	vasanthakumar	60	droop	1	RTA	f	le	6/18	ptosis	add,ele,dep res	srtl	normal	III	NAD	NAD	6 mon	PR
13	padmavathy	50	dip	8	self fall	f	le	6/36	par ptosis	add,ele,dep res	srtl	normal	III	NAD	NAD	4 mon	PR
14	adhikesavan	47	dip	1	cricket ball inj Re	m	re	6/6	rcs	abd res	rtl	normal	VI	NAD	R lat wall orbit#	6 mon	FR
15	devaraj	50	dip	1 1/2	inj to lt side of face	m	le	6/12	rcs	abd res	rtl	normal	VI	NAD	left lat wall of orbit #	5 mon	FR
16	pancharam	32	dip	2	RTA	m	le	6/12	rcs	abd res	rtl	normal	VI	NAD	L zygomatic complex #	4 mon	FR
17	anand	34	droop	4	RTA	m	re	6/9	ptosis	add,ele,dep res	rtl	normal	III,IV	NAD	NAD	4 mon	FR
18	somasundaram	28	droop	52	fall from bike	m	le	6/9	ptosis	add,ele,dep res	nrtl	normal	III	NAD	brain stem contusion,IVH in occ horn of lat ventricle	6 mon	ab re
19	kavitha	55	headache	8	RTA	f	re	6/24		abd res	rtl	normal	VI	NAD	haemorhag R cauda nuc	5 mon	FR
20	palani	60	droop	8	fall from bike	m	re	6/36	ptosis	add,ele,dep res	nrtl	normal	III	NAD	NAD	6 mon	PR
21	kamalakannan	68	droop	4	trauma to RE	m	re	6/24	ptosis,rds	add,ele,dep res	rtl	normal	III	NAD	NAD	6 mon	FR
22	girijamary	46	droop	3	self fall	f	re	6/9		abd res	rtl	normal	VI	NAD	haem cont of r frontal lobe	5 mon	FR
23	kumar	55	dip	4	head inj	m	le	6/9		int res	rtl	normal	IV	NAD	NAD	4 mon	FR
24	durairaj	35	droop	4	fall from bike	m	re	6/18	ptosis	add,ele,dep res	nrtl	normal	III,IV,VI	NAD	R zyg compl #	5 mon	NR
25	rukmani	36	def vn	4	RTA	f	re	6/24		abd res	rtl	normal	VI	NAD	R temp bone #	3 mon	FR
26	jack samuel	33	droop	24	RTA	m	re	6/12	ptosis	add,ele,dep res	rtl	normal	III	NAD	linear blooming foci of r temp lobe	6 mon	ab re
27	shanthi	47	def vn	4	trauma to RE	f	re	6/36		add,ele,dep res	rtl	normal	III	NAD	NAD	3 mon	FR
28	bose	60	def vn	2	RTA	m	re	6/60	sch+	add,ele,dep,abd,in	rtl	normal	III,IV,VI	NAD	NAD	6 mon	PR
29	sankari	45	droop	4	RTA	f	re	6/9	ptosis	add,ele,dep res	rtl	normal	III	NAD	NAD	4 mon	FR
30	rajkumar	25	droop	3	trauma to RE	m	re	6/9	ptosis	add,ele,dep res	rtl	normal	III	NAD	NAD	5 mon	FR
31	varadharajan	37	dip	1 1/2	fall from bike	m	le	6/9	sch+	add,ele,dep,int,abd	rtl	normal	III,IV,VI	NAD	# post lat wall L max sinus	5 mon	NR
32	sanjay	35	headache	4	RTA	m	le	6/18		abd res	rtl	normal	VI	NAD	#Ltemporal bone	4 mon	PR
33	vishwanath	48	dip	8	head inj	m	re	6/6		abd res	rtl	normal	VI	NAD	cont R parietal lobe	3 mon	PR
34	lakshmi	33	dip	8	RTA	f	re	6/6		int res	rtl	normal	IV	NAD	R frontal lobe contusion	3 mon	FR
35	saran	27	dip	52	RTA with loc	m	re	6/6		add,ele,dep res	rtl	normal	III	NAD	# R parietal bone	5 mon	NR
36	vijayalakshmi	29	droop	1	RTA	f	le	6/6	ptosis,per	add,ele,dep,abd,in	rtl	normal	III,IV,VI	NAD	NAD	6 mon	FR
37	arun munusamy	36	droop	1	self fall	m	le	6/12	ptosis	add,ele,dep res	srtl	normal	III	NAD	NAD	5 mon	FR
38	mahadevan	24	dip	4	RTA	m	le	6/9		abd res	rtl	normal	VI	NAD	frontal contusion	4 mon	FR
39	rajasekar	33	dip	1 1/2	inj RE	m	re	6/24	sch+	abd res	rtl	normal	VI	NAD	multiple facial bone injury with butress plating repair	5 mon	NR
40	arumugam	60	droop	4	self fall	m	le	6/36		add,ele,dep res	rtl	normal	III	NAD	NAD	4 mon	FR