ANALYSIS OF NEURO OPHTHALMIC FEATURES OF HEAD TRAUMA

A Dissertation submitted for Master of surgery degree

Branch III – Ophthalmology

March – 2010

The Tamil Nadu Dr.M.G.R. Medical University

Chennai

Tamil Nadu
CERTIFICATE

This is to certify that this dissertation entitled “Analysis of Neuro Ophthalmic Features of Head Trauma” has been done by DR.J.A.Durga under my guidance in the department of ophthalmology, Madurai Medical College, Madurai.

I certify regarding the authenticity of the work done to prepare this dissertation.

Dr.A.SULAIMAN., M.S., D.O.,
Professor & HOD
Department of ophthalmology,
Govt. Rajaji Hospital
Madurai Medical College,
Madurai
DECLARATION

I, Dr.J.A.Durga solemnly declare that the dissertation titled “Analysis of Neuro Ophthalmic Features of Head Trauma” has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of master of ophthalmology, Branch III degree Examination to be held in March 2010.

Place : Madurai

Date :

DR. J.A.DURGA
ACKNOWLEDGEMENT

I am highly obliged to the Dean, Madurai Medical College, for enabling me to utilize the clinical material available in the hospital.

I express my deep gratitude to my respected teacher, professor and Head of the Department of Ophthalmology, Dr. A. Sulaiman, M.S., D.O., for his valuable guidance throughout this work.

I evenly express my deep gratitude to Dr. P. Thiagarajan M.S. D.O., Professor, Department of Ophthalmology for his valuable suggestions and proper guidance.

I am deeply thankful to my guide Dr. T. Badri Narayanan M.S. D.O., Senior Assistant Professor, incharge of Neuro-Ophthalmology clinic, for his constant encouragement and valuable comments to shapeup this dissertation.

I extent my heartfelt thanks to all my Assistant professors and my dear colleagues who extended their support at every step of my study.

I am grateful to my family for their invaluable mental support at every point of time.

Finally, I express my heartfelt gratitude to all my patients for their kind co-operation and regular follow-up without which this dissertation could not have been possible.
INDEX
PART I

CONTENTS PAGE NO

1. INTRODUCTION 1
2. ANATOMICAL CONSIDERATIONS 2
3. PATHOPHYSIOLOGY OF HEAD TRAUMA 11
4. BIOMECHANICS OF BRAIN INJURY 13
5. NEURO OPHTHALMIC SIGNS IN HEAD TRAUMA 19
6. MANAGEMENT OF HEAD TRAUMA 39
7. LITERATURE REVIEW 42

PART II

1. AIM OF THE STUDY 45
2. MATERIALS AND METHODS 46
3. OBSERVATIONS AND ANALYSIS 48
4. RESULTS 60
5. DISCUSSION 62
6. CONCLUSION 72

BIBLIOGRAPHY
PROFORMA
MASTER CHART
INTRODUCTION

The Darwin’s theory of “survival of the fittest” has made man, endeavour to attain higher and still higher speeds in travel and to search for more and still more effective techniques of destruction in war. These have all combined to heighten the incidence of head injuries to an extent unknown to the previous generation.

India has only 1% of the world’s automobiles but 6% of road accidents. The death rate per 1000 vehicles is 2.5, which is the highest in the world, even without the recently burgeoning vehicular traffic.

Falls, assault and domestic accidents also account for a significant proportion of head injuries. They affect the active and productive age group in the prime of life.

We often wonder how nature is so resourceful to confine the entire visual system within the brain, but to retain the globe alone to the exterior. This play gifted by nature has, made any insult to the brain in the form of trauma, tumour etc., to be reflected outside by the globe like a mirror. Thus, neuro-opthalmologists and neurosurgeons play essential roles to pick up these neuroophthalmics signs in head injuries, not only to localise the lesion, but also to save the life of the patient and to predict the prognosis.
ANATOMICAL CONSIDERATIONS

Anatomy of visual pathway, ocular motor system and brain relevant to head injury are discussed below.

THE AFFERENT SYSTEM

Anatomy of the optic nerve

The optic nerve is a fibre tract of the brain consisting of a compact bundle of ganglion cell axons, oligodentrocytes and glial cells ensheathed in three meningeal layers. The cranial subarachnoid space and the perineural space directly communicate. The subdural space is a potential space which may be occupied by haematoma. The dura which lines the optic canal as periosteum merges with the periosteum of the sphenoidal bone intracranially and splits into two layers, one becoming orbital periosteum and the other remaining as the outer sheath of the nerve.
The average length of the optic nerve is 47-50mm.

It is divided into four parts.

1. **Intraocular part**

   The average length of the intra ocular part is 0.7-1mm

   It has 4 zones,

   1. pars retinalis,
   2. Pars choroidalis,
   3. Pars scleralis (lamina cribrosa)
   4. retrolaminar portion.

   The prelaminar part is non-myelinated normally.

2. **Intra orbital part**

   It is 30-35 mm long. It extends from the back of the eye ball to the optic foramen

   At the optic foramen it is surrounded by the extraocular muscles originating at the annulus of Zinn. The superior and medial recti origins are adherent to it. The upper and lower divisions of oculomotor nerve, nasociliary nerve, abducent nerve, ophthalmic vein, and ciliary ganglio lie between it and the lateral rectus. Therefore trauma to this region can damage the optic nerve as well as more than one ocular motor nerve and sensory nerves producing the orbital apex syndrome. Orbital fat, the long and short ciliary nerves and arteries surround it. The central retinal artery which enters the nerve 12mm behind its
insertion into the globe is the point of demarcation for anterior and posterior indirect optic nerve injury.

3. **Intracanalicular part**

   It is 6-10 mm long.

   It lies between the struts at the base of the lesser sphenoidal wing. The optic canal is an average of 9.22mm in length and contains the optic nerve, meninges, ophthalmic artery and the post ganglionic sympathetic fibres. The Dura of the nerve sheath is fixed to the periosteum and bone of the canal.

   The medial wall of the canal is on average 0.21 mm thick and is absent in 4% of normal persons, the dural sheath being in contact with the sphenoidal sinus mucosa.

   The distal portion of the canal, closest to the orbit has the thickest wall and narrowest cross section.

4. **Intracranial part (10mm):**

   This part of the optic nerve is about 10mm in length and lies above the cavernous sinus and converges with its fellow (over the diaphragma sellae) to form the optic chiasma.

   **Relations**

<table>
<thead>
<tr>
<th>Superior</th>
<th>Anterior perforated substance, olfactory tract, Anterior cerebral artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior</td>
<td>Diaphragma sellae, cavernous sinus</td>
</tr>
</tbody>
</table>
Lateral : Internal carotid artery
Medial : Ophthalmic artery

**Optic Chiasma**

The two optic nerves end in optic chiasma. The nasal fibres decussate. The temporal fibres do not decussate.

**Optic Tract**

The optic chiasma continues as optic tracts each optic tract carrying the temporal fibres of the same eye and the nasal fibres of the opposite eye.

**Lateral Geniculate Body (LGB)**

The optic tracts end in LGB from where the III order neuron starts. The pupillary fibres from the optic tract go to the pretectal nuclei without going to the LGB.

The optic radiations from the LGB terminate in the visual cortex in the occipital lobe (Striate cortex) (area 17) and visual association areas are (Area 18 and Area 19) parastriate and peristrate cortex respectively.
THE EFFERENT SYSTEM

Neural basis of eye movements

Types of eye movement

Fast Eye Movements
(velocity:300°-700°/sec)
1. Saccades Spontaneous
2. Nystagmus quick phase
3. REM in sleep (Rapid eye movement)

Slow Eye Movements
(velocity:20°-50°/sec)
1. Voluntary smooth pursuit
2. Vergence
3. Optokinetic reflex
4. Vestibulo-optic reflex

Premotor Command

Horizontal Movements : Paramedian Pontine
Reticular formation

Vertical Movements : Rostral mesencephalic reticular formation
These nuclei project to specific ocular motor nuclear subtroups and receive afferents from the cortex, vestibular system, cerebellum, superior colliculi etc.

**Cortical areas**

1. Area striata of occipital cortex (area 17) has various visuomotor connections, concerned with fixation reflexes.

2. Frontal eye field
   - It is situated at the posterior middle frontal gyrus, area 8
   - It is concerned with contralateral conjugate gaze
   - Damage may cause ipsilateral gaze deviation (or) poor contralateral gaze but normal reflex movements.

3. Parietal cortex [area 7]
   - It is related to attention to visual targets and smooth pursuit. Bipareital lesions produce ocular motor apraxia.
Cerebellum

It is the major coordinator for movements including eye movements. It receives afferents from visual and vestibular apparatus, neck and proprioceptors. It projects to supranuclear zones concerned with ocular movements, both smooth pursuit and saccades.

Disorders cause nystagmus, ocular dysmetria, opsoclonus or ocular flutter.

Medial longitudinal bundle

This white matter tract extends from the rostral midbrain to the spinal cord. It is in close connection to ocular motor and other cranial nerve nuclei.

It mediates correlation between all three ocular motor nuclei and all four vestibular nuclei. Lesions produce internuclear ophthalmoplegia.
OCULAR MOTOR NERVES

Oculomotor nerve (The III cranial nerve):

**Nucleus**: It is situated in the midbrain at the level of superior colliculus.

**Courses**: It passes forwards between posterior cerebral and superior cerebellar arteries and laterally parallel to posterior communicating artery. On lateral side of posterior clinoid process, it pierces the dura and enters lateral wall of cavernous sinus, above the IV cranial nerve. It divides into superior and inferior divisions which enter the orbit through superior orbital fissure within the tendinous ring.

**Muscles supplied**: Superior rectus, inferior rectus, medial rectus, inferior oblique and levator palpebrae superioris.

Trochlear Nerve (The IV cranial nerve)

**Peculiarities**

This is the only cranial nerve to arise from the dorsal aspect of the brain.

This is the longest and thinnest of all cranial nerves

**Nucleus**: It is situated in the midbrain at the level of inferior colliculus.

**Course**: It emerges on posterior surface of brain stem, decussates and passes forward around the cerebral peduncle. It enters the lateral wall of the cavernous sinus and lies below the III Cranial Nerve and above the first
division of V Cranial nerve. It passes through the upper part of superior orbital fissure, outside the tendinous ring.

**Muscle supplied**: Superior oblique muscle.

---

**Abducens Nerve (The VI Cranial Nerve)**

**Nucleus**: It lies in upper part of the floor of the fourth ventricle.

**Course**: It emerges between the pons and medulla oblongata, runs upward, forward and laterally, makes an acute bend across the sharp border of petrous part of the temporal bone and run within the cavernous sinus inferolateral to the Internal carotid artery. It enters the orbit through the superior orbital fissure between the two divisions of the III Cranial nerve.

**Muscle supplied**: Lateral rectus muscle.
THE PATHOPHYSIOLOGY OF HEAD TRAUMA

Occurrence of ophthalmological complications of head injury depends on

1. Brain trauma itself [concussion contusion, laceration] with visual complications

2. Skull fracture in the occiput or base with damage to the visual cortex or the nerves subserving the eyes.

3. Cerebral compression due to meningeal haemorrhage [acute or chronic, extradural, subdural, subarachnoid or intracerebral]

Progressive cerebral compression due to intracranial bleed goes through four stages:

Harvery Cushing proposed the following stages:

1. Stage of physical compensation: CSF displaced, BP rises, and circulation maintained–symptomless.


4. Irreversible stage of perivascular haemorrhages and progressive brain edema.
Consequences of a space occupying lesion (SOL)

A) Herniation of the cingulated gyrus under the falx

B) Tentorial herniation of the parahippocampal gyrus

C) Herniation of cerebellar tonsil through the foramen magnum

D) Impingement of the crus against the tentorium

E) Haemorrhage into the midbrain
BIOMECHANICS OF BRAIN INJURY

By definition head trauma is an anatomical damage or physiological disturbance caused by application of mechanical force to the head.

Therefore the nature, site and magnitude of the mechanical force are the main determinants of head injury. The modification or elimination of this force is the major preventive strategy.

Primary brain injury

1. Contact Injuries
2. Inertial Injuries

Contact injuries

A direct impact causes complex mechanical events both near and distant to the point of contact. They typically cause focal injuries and do not cause diffuse brain injury. Contact forces produce injury in the immediate vicinity of skull injury. Since most impacts set the skull in motion, there are associated acceleration injuries also.
Local contact Effects

1. Depressed fractures
2. Linear fractures
3. Basilar skull fractures
4. Extradural haematoma
5. Coup contusions

Remote contact effects

Remote contact effects are caused by skull deformation and stress waves transmitted at high velocity

1. Remote linear fractures (vault or base)
2. Remote brain contusions and haematomas
3. Brain herniations caused by transient distortion, classically in infant skulls.

Small high velocity objects may shutter the skull and drive bone fragments deeply.

When the surface of contact is larger or the skull hits a fixed surface the result depends on the nature of mechanical loading.
Types of Mechanical Loading

<table>
<thead>
<tr>
<th>Static loading</th>
<th>Dynamic Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>Common</td>
</tr>
<tr>
<td>Force duration</td>
<td>Input force applied in less than 50ms</td>
</tr>
<tr>
<td>Examples</td>
<td>Most RTAs, falls from height</td>
</tr>
</tbody>
</table>

**Dynamic Loading**

In dynamic loading there may be contact forces or inertial forces. Accordingly there are two subtypes of dynamic loading.

1. Impulsive loading
2. Impact loading

**Impulsive loading**

- Only inertial forces
- The head is accelerated or decelerated without directly being struck
- For example, blow to face or thorax, or fall from height

**Impact loading**

- Contact forces + inertial forces
- A short impact to head accelerates or decelerates it.
- Local skull injury in common
**Inertial injuries**

Head acceleration causes structural or functional damage to neural and vascular structure by the relative movement of the brain in relation to the dura and skull. They cause concussion, contusion and diffuse axonal injuries in the brain tissue.

The types of acceleration are

1. Translational
2. Rotational
3. Angular i.e. translational and rotational

Angular acceleration is most commonly encountered. The center of angulation is usually the midcervical spine.

Angular acceleration is the most injurious as it causes shear strain deformation because of differential motion of one portion of the brain with respect to another.

**Secondary cerebral injury**

In contrast to primary brain injury which occurs at the movement of impact, secondary cerebral injury is the result of intracranial space occupying lesion, either by oedema or haematoma.
Many of the effects of secondary damage are preventable by active management of head injuries in the early stages and by a close observation of clinical signs.

<table>
<thead>
<tr>
<th>Type of primary lesion</th>
<th>Degree of tissue damage</th>
<th>Resulting space occupying lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Diffuse axonal injury &amp; contusion</td>
<td>+++</td>
<td>Oedema</td>
</tr>
<tr>
<td>2) Subdural</td>
<td>+</td>
<td>Haematoma</td>
</tr>
<tr>
<td>3) Extradural</td>
<td>+</td>
<td>Haematoma</td>
</tr>
<tr>
<td>4) Intracerebral</td>
<td>++</td>
<td>Haematoma</td>
</tr>
</tbody>
</table>

**Important Note:**

Senile cerebral atrophy favours increased shear deformation and also subdudal haemorrhage.
## Correlation of brain lesions and injury mechanism

<table>
<thead>
<tr>
<th>Brain Lesion</th>
<th>Mechanism of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Skull fractures, epidural haematoma coup contusion</td>
<td>Contact forces</td>
</tr>
<tr>
<td>2) Contrecoup contusions</td>
<td>Inertial effects (impact or impulsive)</td>
</tr>
<tr>
<td>3) Intra cerebral haematoma</td>
<td>Stress waves and acceleration induced strains</td>
</tr>
<tr>
<td>4) Subdural Haematoma</td>
<td>Bridging vein disruption</td>
</tr>
<tr>
<td>5) Cerebral concussion</td>
<td>Angular accelerations with electrophysiologic dysfunction</td>
</tr>
<tr>
<td>6) Diffuse axonal injury</td>
<td>Long duration angular acceleration esp. in coronal plane</td>
</tr>
</tbody>
</table>
THE NEURO OPHTHALMIC SIGNS IN

HEAD TRAUMA

Neuro ophthalmic signs following head trauma

It is divided into those that occur during

1. Acute phase
2. Chronic phase

Acute phase of head injury involves

1. Eye position
2. Eye Movements
3. Pupillary signs
4. Cranial nerve injuries
5. Cortical blindness

Chronic phase of head injury involves

1. Delayed III & VI nerve palsies
2. Post-traumatic papilledema / Optic atrophy
3. Post-traumatic field defects
4. Aberrant regeneration of the Cranial nerves
5. Vascular complications
6. Infections
EYE POSITION IN UNCONSCIOUS PATIENTS

Abnormal position of the eyes in an unconscious patient is indicative of structural damage to the brain and the brainstem.

- Unilaterally abducted eye indicates unilateral tentorial herniation.
- Bilaterally abducted eyes are suggestive of central herniation.
- Skew deviation of the eyes is produced by posterior fossa lesion.
- Persistent gaze deviation is caused by
  - Ipsilateral frontal eye field damage or
  - Contralateral pontine gaze centre damage.
- Persistent down gaze deviation with upward gaze palsy occurs when there is posterior transtentorial herniation with tectal compression. It is called Parinaud syndrome / dorsal midbrain syndrome.

EYE MOVEMENTS

Intact or absent eye movements in unconscious head injured patients reflect the integrity of the brainstem function. Absence of eye movements indicates that there is primary or secondary (due to herniations) injury to the brainstem and therefore is a poor prognostic sign.

Injury to the cervical spines should be ruled out before eliciting the following reflex eye movements.
Oculocephalic reflex: (“Doll’s Eye Movements”)  

In an unconscious patient, when the head is turned to one side the eyes will deviate to the opposite side. There will not be any movements if the brainstem is damaged.

Oculo-vestibular reflex  

This test is performed by syringing the ears (with intact drums) with cold water (30°C) or warm water (44°C). When cold water is used, the eyes will deviate to the same side and nystagmus occurs in the opposite direction. With warm water, the eyes will deviate to the opposite side and the direction of the nystagmus would be to the side of syringing.( cold – opposite, warm – same COWS)
PUPILLARY SIGNS FOLLOWING HEAD TRAUMA

Examination of the pupils is a simple and the most reliable test in head trauma. Involvement of pupils rules out a metabolic cause of coma.

I. Bilaterally Dilated pupils

In a semiconscious irritable patient, dilated but reacting pupils may be caused by anoxia. This is usually temporary. The pupils become normal on oxygen administration.

In a deeply comatose patient, bilateral dilated and fixed pupils indicate severe injury to the brainstem.

Primary injury to the midbrain may produce mid dilated sluggishly reacting pupils due to lesions of both sympathetic and parasympathetic tracts.

II. Unilaterally dilated pupil

This may occur due to

1. Transtentorial herniation [coning]
2. 3rd nerve palsy
3. Traumatic mydriasis
1. A widely dilated, immobile, Hutchinsonian pupil / cook’s pupil

   There are 3 stages in its evolution.

   1. An initial miosis from ipsilateral nerve irritation

   2. A dilatation of ipsilateral pupil which still reacts to light and convergence while the patient may be drowsy.

   3. A true Hutchinsonian pupil, a unilateral dilated fixed pupil, patient shows increasing drowsiness to produce coma.

**Mechanism**

   A space occupying hematoma / expanding brain edema may lead to herniation of temporal lobe into the tentorial hiatus to impinge directly on the III nerve. It is associated with contralateral motor signs. It needs urgent decompression to prevent damage to the brainstem which may have grave prognosis.

**Kernohan notch syndrome**

   Occasionally pupillary and motor signs are ipsilateral due to cross compression of opposite cerebral peduncle.

2. III Nerve palsy

   Patient has dilation of pupil, ptosis, paralysis of adduction, elevation and depression. Patient is usually conscious.
3. Traumatic mydriasis

There will be evidence of injury in and around the orbit. This may be due to injury to

1. iris
2. ciliary ganglion
3. short ciliary nerve

III. Bilateral constricted pupils

Intrinsic pontine lesions produced by contusion or haematoma cause bilateral small constricted pupils [pin-point pupils or “pontine” pupils’. This is due to the injury to bilateral sympathetic nerves. Pupillary reaction to light can be observed if magnifying lens is used.

IV. Unilateral constricted pupils

It is caused by oculosympathetic paralysis that is Horner’s syndrome. Other sign are, partial ptosis enophthalmos and anhidrosis.

Marcus-Gunn pupil [Relative afferent pupillary defect]

It is seen in optic neuropathy. It is elicited by swinging flash light test. On throwing the torch light to normal eye both the pupils will constrict. Upon swinging the light to the abnormal eye, both the pupils will paradoxically dilate.
TRAUMATIC OPTIC NEUROPATHY [TON]

Traumatic optic neuropathy is divided into 3 categories

1. Avulsion
2. Direct injury
3. Indirect injury

Avulsion

Avulsion is the rarest form of optic neuropathy. Total avulsion implies complete separation of the lamina cribrosa from its attachment to the sclera. The choroids, retina and vitreous are completely separated from the optic disc. The retinal blood vessels are partly or totally interrupted.

Partial avulsion involves a localized segment of the optic nerve. Optic nerve avulsion may be caused by penetrating or non-penetrating injuries. Severe orbital fracture due to blunt trauma is the commonest cause.

3 mechanisms postulated

1. Increased intraocular pressure due to the globe being compressed against the bony orbit and the optic nerve being pushed out of the scleral canal.
2. Increased intraorbital pressure forcing the globe forward, stretching and tearing the optic nerve.
3. Extreme rotation and displacement of the globe within the orbit.
Visual prognosis depends on the extent of avulsion and total blindness occurs in complete avulsion.

Immediately after injury the optic disc is obscured by an overlying vitreous hemorrhage. Associated ocular findings are subconjunctival haemorrhage, limitation of extraocular movements, proptosis and a dilated, fixed pupil.

When the fundus view improves, the disc contour is seen distorted or the scleral canal devoid of the disc tissue is seen. Subsequently the defect is closed by a gliotic scar which may extend into the vitreous.

**Direct Optic Nerve Injury Mechanisms**

<table>
<thead>
<tr>
<th>Lacerations</th>
<th>Vascular Insufficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incomplete</td>
<td>Ischaemia</td>
</tr>
<tr>
<td>Complete</td>
<td>Infarction</td>
</tr>
</tbody>
</table>

**Bone fracture or deformation**

<table>
<thead>
<tr>
<th>Optic canal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orbital walls</td>
</tr>
<tr>
<td>Anterior clinoid process</td>
</tr>
<tr>
<td>Haemorrhage</td>
</tr>
<tr>
<td>Nerve sheath</td>
</tr>
<tr>
<td>Intra neural</td>
</tr>
</tbody>
</table>
Direct optic nerve injury is due to impingement of the nerve by either

(1) A foreign body that has penetrated the globe, orbit or cranium or

(2) Injury from a displaced fracture or spicule of bone in the region of the optic canal.

**Types of direct injury**

**Anterior type**

If the damage is anterior to the entrance of the central retinal artery the ophthalmoscopic picture is of central retinal artery occlusion, and lose of vision is instantaneous and complete.

**Posterior type**

More posterior injury may demonstrate a normal fundus or mid retinal oedema due to axoplasmic statsis. Features of primary optic atrophy appear in 3 to 4 weeks.

**Indirect optic nerve injury**

It is the commonest form of traumatic optic neuropathy.

Indirect trauma most commonly affects the intracanicular portion of the optic nerve.
### Pathophysiology

Three mechanisms are postulated

Vascular insufficiency is the common factor:

1. Shear causes rupture, spasm or thrombosis of small arterioles which leads to haemorrhage, infarction and oedema compressing the nerve in the canal causing further ischaemia.

2. Shear causes oedema, raised intracanalicular pressure, compression and ischemia.

3. Nerve haematoma: Subdural / intradural / subarachnoid

Holographic interferometry shows stress, for surface loads on frontal and malar eminence are transmitted to optic foramen even without fracture.

In closed head trauma, the intracanalicular portion of the optic nerve, being fixed is commonly damaged.

The intraocular segment is occasionally damaged.

The intraorbital and intracranial portions due to relative mobility and laxity are usually spared.

### Types of indirect injury

1. Anterior type - Fundus abnormalities are present

2. Posterior type - Fundus normal is initially
Anterior type

The intraorbital segment containing the central retinal artery is affected and the fundus picture resembles central retinal artery occlusion. Disc edema with normal retinal circulation and fluorescein angiographic findings of impaired posterior ciliary artery circulation is another form.

Lastly small anterior marginal tears of the disc with small haemorrhages less than one-third disc circumference, which resolves in two weeks with a heavily pigmented scar, followed 1 month later by mild disc pallor may occur.

Posterior type

When the optic nerve dysfunction is associated with a normal fundus initially and no evidence of chiasmal lesion then the lesion is presumed to lie in between the central retinal artery entrance and the chiasm. Disc pallor retinal nerve fibre layer defects become apparent 4 to 8 after head injury.

Clinical investigations in indirect injury

1. Every effort should be taken to evaluate visual function
2. Snellen, rosenbaum near card should be used
3. If the patient does not have perception of light, projection should be checked in all the four quadrants.
4. Pupillary responses are critical. Grading of RAPD should be done with neutral density filters.
Pupillary response to light is the most reliable sign of the extent of optic nerve injury.

5. Field: Central scotoma or NFL defects
6. Direct / Indirect ophthalmoscopy: Initially it may be normal
7. VER – Adjunctive Test. There is the good correlation between initial VER and ultimate acuity especially in comatose patients.

**Chiasmal lesions**

The chiasm bears a close relationship to the III ventricle and hypothalamus. It is damaged relatively rarely in severe frontal trauma due to acute traumatic necrosis, ruptured pial vessels or direct damage to nerve fibres. This causes bitemporal hemianopia with hypothalamic symptoms.

**Optic tract lesions**

Usually the severity of injury precludes the survival.

- Theoretically a retrochiasmatic, tract lesion causes contralateral homonymous heminopia, which is incongruous if incomplete with Wericke’s hemianopic pupil.

**Lateral geniculate body lesion**

Field defect, as for tract or a relatively congruous sectoranopia. It is extremely unlikely to be damaged in isolation in head injury.
Optic radiation lesion

It causes quadrantanopia to hemianopia – usually congruous

Visual cortex lesion

It causes congruous hemianopia.

Concussion and edema may cause reversible cortical blindness. The posterior occipital pole containing the macular area is susceptible to occipital trauma. It is also the watershed zone between the posterior cerebral artery and middle cerebral artery territories and so susceptible to ischaemia caused by hypovolemic shock and generalized hypoperfusion state. A central homonymous hemianopic scotoma results.

OCULAR MOTOR DISTURBANCES

Ocular motor disturbances following head injury apart from those caused by direct damage to muscles and nerves in the orbit account for 15% of all ocular motor palsy, either occurring due to birth trauma or accidents later in life. They complicate 1% of accidental trauma to the head.

Clinical presentation

A complete spectrum of clinical presentations are reported from partial paresis of individual nerves to bilateral total ophthalmoplegia.
**Supranuclear palsy**

Supranuclear ocular motor palsies are rare. They cause conjugate gaze deviations where there is retention of ocular parallelism and absence of diplopia.

- Pontine lesion: It causes horizontal gaze palsy
- Midbrain lesion: It causes vertical gaze palsy

**Cerebral lesions**

Cerebral lesions may cause

1. Conjugate palsies, often temporary with exaggerated fixation reflexes.
2. Spasm of conjugate gaze to the side opposite the lesion.

The psycho-optical reflexes – fixation, convergence, blinking etc. may be disoriented. Nystagmus may occur due to vestibular or cerebellar involvement.

Defects of convergence and accommodation may occur which frequently represent unmasking of a previous phoria and often perpetuated as a neurosis.
CRANIAL NERVE INJURIES

The exact incidence of damage to each cranial nerve varies with patient selection and length of follow up. Olfactory, facial and auditory vestibular nerves are damaged most often by blunt head trauma.

III Nerve injury

Trauma is one of the most common causes of third nerve palsy and accounts for 20% of ocular motor injury. Anisocoria due to orbital trauma has to be distinguished.

The lesions may be in the nucleus or root which is associated with brainstem damage or in the trunk due to basal fractures or haematoma compression. Stretching and concussion may cause temporary lesions, and laceration or ischaemia cause permanent damage.

Spontaneous recovery takes 4-6 months. Aberrant regeneration is well documented in the following frequency:

1. On down gaze upper lid retracts – it is called Pseudo Von Graefe Sign
2. Constriction of the pupil associated with lid retraction.
3. Widening of the palpebral fissure on adduction, narrowing on abduction
   – It is called Pseudo Duane Syndrome
4. Dilated pupil unreactive to light but constricts with convergence / adduction – It is called Pseudo Argyll Robertson Pupil.
IV Nerve injury

Traumatic IV nerve injuries are usually diagnosed when the patient recovers consciousness and experiences diplopia. The usual head posture is tilt to the opposite side, with hypertropia of the affected eye.

Park’s Bielschowsky three step test:

This test is useful in the diagnosis of fourth nerve palsy.

Step 1: In primary position in the affected side is hypertropic

Step 2: On lateral gaze, hypertropia worsens on opposite gaze. (WOOG)

Step 3: On head tilt, hypertropia becomes better on opposite tilt. (BOOT)

Trauma accounts for 29% percent of all IV nerve injuries.

Bilateral fourth nerve palsy is nearly always post-traumatic.

VI Nerve injuries

Minor degrees of abduction deficit are often ambiguous after head injury. Bilateral VI nerve palsies are relatively common.

Head trauma is the cause of 15% of VI nerve palsy.

Facial nerve injury

The facial nerve may be implicated in cranial trauma due to

1. Fracture of the petrous temporal bone frequently associated with ear bleed, deafness and vestibular disturbance (nausea, tinnitus, nystagmus,

2. Direct trauma near the stylo-mastoid foramen,

3. Displaced fracture of the mandible, or

4. Penetrating trauma or contusion of the nerve branches.

Epiphora, lagophthalmos and paralytic ectropion result with exposure keratitis if unrecognized.

Localizing signs

Facial nerve injury between the 6th nerve nucleus and the geniculate ganglion involves fibres destined for the greater superficial petrosal nerve and impaires lacrimation, whereas distal lesions do not.

The intermediate nerve of Wrisberg which synapses in the geniculate ganglion and leaves the facial nerve 3 to 4mm above the stylomastoid foramen innervates salivary secretion and taste to the anterior 2/3 of the tongue. Involvement of the stapedius nerve in a fracture may also cause hyperacusis.

Healing may be associated with a number of aberrant regeneration syndromes, the classic one being a paradoxical gustatory lacrimal reflex [Crocodile tears].
Trigeminal nerve involvement

This may occur intracerebrally or at the skull base causing anesthesia over the area subserved (including cornea) by the affected branch. It is usually associated with some ocular motor palsy.

Vascular complications

Terson syndrome

Rarely, subarachnoid hemorrhage will lead to papilledema, retinal hemorrhage and ophthalmoplegia.

Carotid cavernous fistula

It rarely occurs due to direct damage to the vessel wall. It is a late complication of head trauma. It is characterized by abrupt onset of pain, a noise felt in the head by the patient and pulsating proptosis and hypoxic eye ball syndrome.

Chronic phase of head injury

Delayed 6th and 3rd nerve palsies

Delayed VI nerve palsy often results from increased intracranial pressure or haemorrhagic meningitis. Delayed III nerve damage, even without pupillary involvement is a more ominous sign, indicating trastentorial herniation.
Post-compression blindness

A few patients who survive intracranial haematomas and brain oedema, complain of defective vision. This type of visual failure results from two mechanisms.

(i) When there is diffuse oedema of the brain, the medial and inferior portions of the frontal lobes (gyrus rectus) swell and compress the optic nerves and the chiasma, resulting in optic atrophy.

(ii) During transtentorial herniation the medial portion of the temporal lobe compresses the 3rd nerve as well as the posterior cerebral artery. This results in ischaemia of the occipital lobe and a homonymous field defect.

Post-traumatic optic atrophy

The causes are:

1. Traumatic optic neuropathy
2. Injury to the optic chiasm
3. Optico-chiasmal arachnoiditis
4. Post-papilloedemic optic atrophy
Post-traumatic papilloedema

Any post-traumatic lesion which causes a sustained increase in intracranial pressure for 2 to 3 weeks can produce papilloedema. The usual causes are:

1. Chronic extradural haematoma
2. Chronic subdural haematoma
3. Communicating hydrocephalus caused by adhesions following subarachnoid haemorrhage.
4. Post-traumatic brain abscess
5. Incomplete occlusion of major venous sinuses.

Post-traumatic field defects

The optic radiations in the temporal and parietal lobes may get injured by deep contusions or intracerebral haematomas. The occipital poles may themselves get contused in coup and contre-coup injuries. These type of injuries will produce homonymous field defects (when one of the occipital lobes is involved) Incomplete injuries to the optic chiasma cause bitemporal field defects. Post-traumatic optico-chiasmal arachnoiditis produces irregular field defects.
MANAGEMENT OF HEAD TRAUMA

Initial Assessment and resuscitation forces on airway, breathing and circulations. Initial blood loss is replaced by crystalloid till the site of bleeding is located.

Neurological Assessment

Accurate history regarding the mode of injury, loss of consciousness is obtained. Assessment should include a gauge of level of consciousness as well as focal neurological deficit. The Glasgow Coma Scale grades eye opening, best motor response and verbal response on a 15 point scale. Blood pressure, respiratory rate should be recorded. Pupillary response should be noted. It has a high degree of interobserver consistency.

Skull Radiology

77% of adults and 62% children who develop haematoma have skull fractures. CT should be performed if

(1) There is a depressed skull fracture
(2) Focal neurological signs
(3) Deterioration of consciousness or coma
(4) Not regained consciousness despite adequate resuscitation
(5) Post traumatic seizure.
Radiological findings following head injury

Skull fracture if close to vascular markings should alert the observer to the risk of intracranial haematoma. Opacity of air sinuses or pneumocephalus indicate a compound fracture. CT gives an accurate picture of the nature and extent of any bone injury and it is an accurate method of detecting an intracranial bleed. Fresh blood may be of mixed intensity but haematoma appears hyperdense. Extradural haematoma is convex, subdural haematoma is concave. Subdural haematoma is usually associated with brain contusion, if present alone, a primary vascular cause for injury should be suspected. MRI is superior to CT to detect soft tissue injuries.

Medical management

The aim is to prevent secondary changes and optimize recovery from the primary injury. When a patient needs ventilation the aim is to control ICP by preventing fluctuations in oxygenation and circulating volume, preferably with continuous ICP monitory. Raised ICP is controlled using IV mannitol, frusemide and hyperventilation.

Surgical Management of hematoma

In the absence of CT, when patient is rapidly deteriorating the site of a skull fracture is the first guide for burr hole placement particularly if it is close to a vascular marking, otherwise CT guides the location.
**Management of traumatic optic neuropathy**

In the initial stage, pulse steroid therapy in the form of IV methyl prednisolone 1g in 100ml of NS infused over 15-20 min given OD for 3 days followed by oral predinisolone 1mg/kg/day for 11 days followed by rapid tapering over the next 3 days.

If there is no response, transethmoiodal decompression of the canalicular portion of the optic nerve should be attempted.

**Deviation of eye**

Diplopia secondary to ocular motor nerve palsies are treated with prisms and muscle surgeries based on the severity and duration of paralysis.

**Management of complications**

Infections like meningitis/ cerebral abscess should be treated with IV broad spectrum antibiotics.

Carotid Cavernous Fistula ➔ Detachable flow guided balloon, to close the CCF.
REVIEW OF LITERATURE


Sabales et al in 1991 conducted a study with 181 cases and found that loss of consciousness and ocular motor nerve palsy are significantly correlated statistically. Lack of seat belt and neuro ophthalmic abnormalities were also correlated significantly.

2. Jacobi et al in 1986 examined 741 cases of head trauma and inferred that the most common cranial nerve affected was oculomotor nerve.


3. Gjerris in 1976 quoted that approximately 5% of all patients with head trauma. Manifest injury to some portion of visual pathway.


Mariak et al found positive correlation between avulsion of III cranial nerve and basilar skull fracture in 1997.

Keane et al examined 96 patients in his study in 1984. He revealed that the most common mode of injury was motor vehicle accident [97%].


Moster et al in 1998 examined 46 head trauma patients in rehabilitation and found the III nerve as the most common efferent nerve injured.


Sabales et al found that the mean age of patient with head trauma was 31 years and functional visual field defect in the most common afferent defect.


Lepore in 1995 studied 60 head trauma patients and inferred that the most common efferent deficit was IV nerve palsy.


Van Stavern et al in his study in 2000 concluded that retrochiasmal visual field defect was the commonest afferent visual defect.

Kowal et al in 1992 in his study with 161 patients found optic neuropathy as the most common afferent defect among head injured patients.


Van Stavern et al found abnormal neuro imaging studies only in 1/3rd of patients with significant neuroophthalmic deficit.

12. **Lepore F. Disorders of ocular motility following head trauma. Arch Neurol 1995;52:924-6.**

Lapore et al in 1995 found statistically significant correlation between bilateral cranial nerve palsies and corticospinal tract damage.
AIM OF THE STUDY

1. To determine the mode of injury in head injury patients.
2. To document the incidence and nature of neuroophthalmic deficits in head injury patients.
3. To correlate the initial level of consciousness and the incidence of neuro-ophthalmic deficits.
4. To correlate the documented neuro-ophthalmic deficits with the neuroimaging studies.
5. To analyse the recovery pattern of the head injured patients.
MATERIALS AND METHODS

A clinical study was carried out in the Department of Ophthalmology. Government Rajaji Hospital, Madurai, during the period from May 2008 to October 2009.

Out of 1086 patients admitted in head injury ward, 182 patients with the history of head injury were referred from the department of neurosurgery who had ophthalmic complaints. Of them 100 patients were found to have abnormal neuroophthalmic deficits.

Inclusion criteria

1. Patients with definite history of head trauma.
2. Patients with ophthalmic symptoms.
3. Patients whose level of consciousness is good with or without history of loss of consciousness in the immediate post-head trauma period.

Exclusion Criteria

1. Unconscious patients who did not subsequently recover adequate consciousness.
2. Patients whose neuro-ophthalmic deficits were not confirmed or follow up was not possible due to default [Against Medical Advice/Absconded]

The cases were examined in the Department of Neurosurgery and in the Neuro-ophthalmology Clinic in the Department of Ophthalmology subsequently.

All the data were collected or a standardized proforma and wherever appropriate slit lamp examination, indirect ophthalmoscopy, diplopia charting, forced duction tests visual field analysis etc were done.

In cases requiring ophthalmic medical or surgical intervention, they were instituted appropriate work-up in the ophthalmology Dept., All the cases were essentially managed in the Department of Neurosurgery with routine follow up.

All the patients were followed up for a minimum period of 3 months. They were reexamined and the results were recorded regarding the original problem and any new findings.
OBSERVATIONS AND ANALYSIS

I. Age distribution

A total of 182 patients were examined. The age range was 2-70 years with a median of 32 years. Maximum number of head injury patients were in the age group of 20-40 years. Least number of patients were in the extremes of age.

<table>
<thead>
<tr>
<th>Age range (in years)</th>
<th>Number of patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-9</td>
<td>5</td>
<td>2.74</td>
</tr>
<tr>
<td>10-19</td>
<td>20</td>
<td>10.99</td>
</tr>
<tr>
<td>20-29</td>
<td>53</td>
<td>29.12</td>
</tr>
<tr>
<td>30-39</td>
<td>58</td>
<td>31.87</td>
</tr>
<tr>
<td>40-49</td>
<td>20</td>
<td>10.99</td>
</tr>
<tr>
<td>50-59</td>
<td>15</td>
<td>8.24</td>
</tr>
<tr>
<td>60-69</td>
<td>11</td>
<td>6.04</td>
</tr>
</tbody>
</table>
II. Sex Ratio

Of 182 patients, 112 patients were males and 70 were females. M:F ratio was 1.6:1.

<table>
<thead>
<tr>
<th>Gender</th>
<th>Number of patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>112</td>
<td>61.54</td>
</tr>
<tr>
<td>Female</td>
<td>70</td>
<td>38.46</td>
</tr>
</tbody>
</table>

![Sex Ratio Chart]

- Male: 112
- Female: 70
III. Mode of Injury

112 patients out of 182 gave a history of road traffic accident. Of them, 84 were passengers / drivers, 28 were pedestrians being hit by some vehicles. History of helmet wear was given only in 8 out of 84 patients. 31 Patients gave a history of accidental fall and 35 patients gave a history of assault. 4 gave history of injury at work place.

<table>
<thead>
<tr>
<th>Mode of Injury</th>
<th>Number of patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RTA</td>
<td>112</td>
<td>61.54</td>
</tr>
<tr>
<td>Passenger /driver</td>
<td>84</td>
<td>75</td>
</tr>
<tr>
<td>Pedestrian</td>
<td>28</td>
<td>25</td>
</tr>
<tr>
<td>Accidental fall</td>
<td>31</td>
<td>17.03</td>
</tr>
<tr>
<td>Assault</td>
<td>35</td>
<td>19.23</td>
</tr>
<tr>
<td>Others</td>
<td>4</td>
<td>2.20</td>
</tr>
</tbody>
</table>
IV. History of loss of consciousness [LOC]

Out of 182 patients, only 69 patients gave a history of loss of consciousness.

<table>
<thead>
<tr>
<th>History of LOC</th>
<th>Number of patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>69</td>
<td>37.94</td>
</tr>
<tr>
<td>Absent</td>
<td>113</td>
<td>62.09</td>
</tr>
</tbody>
</table>

![History of Loss of Consciousness](image)
IV. Neuroimaging studies

83 out of 182 patients had some finding in CT brain.

<table>
<thead>
<tr>
<th>CT finding</th>
<th>Number of patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any significant present</td>
<td>83</td>
<td>45.6%</td>
</tr>
<tr>
<td>1. ICH</td>
<td>49</td>
<td>59.03</td>
</tr>
<tr>
<td>2. Skull fracture</td>
<td>22</td>
<td>26.50</td>
</tr>
<tr>
<td>3. Contusion</td>
<td>30</td>
<td>36.14</td>
</tr>
</tbody>
</table>
VI. Neuro ophthalmic deficits

Of 182 patients, only 100 patients had neuroophthalmic deficits on detailed examination. Other had non-neuro-ophthalmic deficits like lid laceration, refractive error minor anterior segment injury. They were treated symptomatically and given reassurance.

<table>
<thead>
<tr>
<th>Ophthalmic deficit</th>
<th>Number of patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuro-ophthalmic</td>
<td>100</td>
<td>54.94</td>
</tr>
<tr>
<td>Non-neuro ophthalmic</td>
<td>82</td>
<td>45.05</td>
</tr>
</tbody>
</table>
VII. Type of Neuro ophthalmic deficits

Out of 100 patients with neuro ophthalmic deficits, 43 patients had afferent deficits and 57 patients had efferent deficits.

<table>
<thead>
<tr>
<th>Type of Neuroophthalmic Deficits</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Afferent Neuro ophthalmic deficit</td>
<td>43</td>
</tr>
<tr>
<td>Efferent neuro ophthalmic deficit</td>
<td>57</td>
</tr>
</tbody>
</table>

![Type of Neuroophthalmic Deficits](image-url)
VIII. Afferent neuroophthalmic deficits

30 out of 43 patients had traumatic optic neuropathy. Majority were of indirect type. They had unilateral visual defect with RAPD. 7 patients had findings suggestive of retro chiasmal lesion. 6 patients had cortical blindness.

<table>
<thead>
<tr>
<th>Location of lesion</th>
<th>Number of patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TON</td>
<td>30</td>
<td>69.76</td>
</tr>
<tr>
<td>Retro chiasmal</td>
<td>7</td>
<td>16.28</td>
</tr>
<tr>
<td>Cortical blindness</td>
<td>6</td>
<td>13.95</td>
</tr>
</tbody>
</table>
IX. Ocular motor nerve palsies

51 patients out of 57 had some ocular motor nerve palsies. The most common cranial nerve injured was IV nerve followed by III nerve. VI nerve was least commonly affected. Maximum number of bilateral cases were seen in IV nerve injury. Multiple cranial nerve palsies were also seen whose incidence nearly equals VI cranial nerve palsy.

<table>
<thead>
<tr>
<th>Ocular motor palsy</th>
<th>Number of patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>III Nerve (B/U)</td>
<td>23 (2/23)</td>
<td>45</td>
</tr>
<tr>
<td>IV Nerve (B/U)</td>
<td>27 (10/27)</td>
<td>52.1</td>
</tr>
<tr>
<td>VI Nerve (B/U)</td>
<td>15 (4/15)</td>
<td>29.40</td>
</tr>
<tr>
<td>Multiple</td>
<td>14</td>
<td>27.45</td>
</tr>
</tbody>
</table>

![INCIDENCE OF OCULAR MOTOR NERVE PALSIES](image)
X. Other efferent neurological deficits

One patient had convergence insufficiency. Two patients had supranuclear gaze palsies and three patients had horner’s syndrome.

<table>
<thead>
<tr>
<th>Other efferent deficits</th>
<th>Number of patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Convergence insufficiency</td>
<td>1</td>
<td>1.75</td>
</tr>
<tr>
<td>Horner’s syndrome</td>
<td>3</td>
<td>5.20</td>
</tr>
<tr>
<td>Supranuclear gaze palsy</td>
<td>2</td>
<td>3.50</td>
</tr>
</tbody>
</table>

XI. Recovery pattern after 3 months of follow up

Recovery pattern was poor in patients with TON and retrochiasmal injury. Patients with cortical blindness usually showed good recovery.

Recovery pattern was very poor in multiple cranial nerve palsies.

Interestingly, 4 cases of III nerve injury showed features of aberrant degeneration.
<table>
<thead>
<tr>
<th>Deficit</th>
<th>FR</th>
<th>PR</th>
<th>NR</th>
</tr>
</thead>
<tbody>
<tr>
<td>TON</td>
<td>2</td>
<td>7</td>
<td>21</td>
</tr>
<tr>
<td>Retrochiasmal</td>
<td>-</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Cortical blindness</td>
<td>4</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>III</td>
<td>6</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>IV</td>
<td>8</td>
<td>12</td>
<td>7</td>
</tr>
<tr>
<td>VI</td>
<td>7</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Multiple</td>
<td>0</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Convergence in sufficiency</td>
<td>-</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Horners</td>
<td>2</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Supra nuclear gaze palsy</td>
<td>-</td>
<td>-</td>
<td>2</td>
</tr>
</tbody>
</table>
RESULTS

1. Of the 182 cases of head injury, the age range was 2-70 years, with the maximum number of patients in the age group of 20-40 yrs constituting more than 60% of the total.

2. Males were more commonly affected than female with a M:F ratio of 1.6:1.

3. Road traffic accidents was the commonest mode of injury in our study. This constitutes 61% of the cases.

4. Only 38% of head injury patients gave a history of loss of consciousness.

5. Of the 182 patients, 83 patients had abnormal finding in CT brain. Majority of the patient with CT abnormality had intracranial hemorrhage (59%). They had III or IV nerve palsies. Basilar skull fracture was associated with bilateral VI nerve palsies.

6. Abnormal neuro ophthalmic findings were seen in 100 out of 182 patients. The rest of the patients had non-neuroophthalmic problems.

7. Efferent pathway deficit constituted 57% and more common than afferent pathway deficit (43%).

8. Traumatic optic neuropathy was the commonest afferent pathway deficit constituting 70%.
9. Of the ocular motor nerve palsy, IV nerve injury was the commonest injury (52.1%) followed by III nerve injury.

10. IV nerve palsy was quite often bilateral.

11. Only 29 patients showed full recovery 4 out of 6 patients with cortical blindness recovered fully. Aberrant regeneration was seen in 4 patients of III nerve injury in 3 months of follow up.
DISCUSSION

The incidence of motor vehicle accidents has dramatically increased in the past three decades. This is due to the mischievous behaviour of the adolescents and young adults in whom awareness of helmet wear while riding is lacking. Drunken driving is also one of the major causes of head injury in India.

MODE OF INJURY:

Motor vehicle accident was the commonest mode of injury in the present series constituting about 61%. Only 9.5% of cases gave history of helmet wear. This is comparable to various studies conducted in the past.

COMPARISION OF MOST COMMON MODE OF INJURY

<table>
<thead>
<tr>
<th>STUDIES</th>
<th>MOST COMMON MODE OF INJURY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jacobi et al</td>
<td>45.6%</td>
</tr>
<tr>
<td>Keane</td>
<td>97.9%</td>
</tr>
<tr>
<td>Sabates et al</td>
<td>57%</td>
</tr>
<tr>
<td>Lepore</td>
<td>88.3%</td>
</tr>
<tr>
<td>Mariak et al</td>
<td>91.7%</td>
</tr>
<tr>
<td>Van Stavern et al</td>
<td>59.8%</td>
</tr>
<tr>
<td>Present series</td>
<td>61%</td>
</tr>
</tbody>
</table>
AGE GROUP:

Age range studied was 2 – 70 years. Maximum number of cases were in the age group of 20 – 40 years.

COMPARISION OF AGE GROUP STUDIED

<table>
<thead>
<tr>
<th>STUDIES</th>
<th>AGE RANGE(Years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sabates et al</td>
<td>5 - 74</td>
</tr>
<tr>
<td>Lepore</td>
<td>8 - 66</td>
</tr>
<tr>
<td>Mariak et al</td>
<td>28 - 85</td>
</tr>
<tr>
<td>Keane</td>
<td>15 - 52</td>
</tr>
<tr>
<td>Van Stavern et al</td>
<td>2 - 86</td>
</tr>
<tr>
<td>Kowal</td>
<td>13 - 38</td>
</tr>
<tr>
<td>Present series</td>
<td>2 - 70</td>
</tr>
</tbody>
</table>
SEX DISTRIBUTION:

Men were more commonly affected than women. In the present series, out of 182 patients, 112 patients were males and 70 were females.

COMPARISION OF SEX DISTRIBUTION

<table>
<thead>
<tr>
<th>STUDIES</th>
<th>SEX DISTRIBUTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jacobi et al</td>
<td>M = 471; F = 270</td>
</tr>
<tr>
<td>Sabates et al</td>
<td>M = 125; F = 56</td>
</tr>
<tr>
<td>Lepore</td>
<td>M = 28; F = 32</td>
</tr>
<tr>
<td>Mariak et al</td>
<td>M = 7; F = 5</td>
</tr>
<tr>
<td>Van Stavern et al</td>
<td>M = 203; F = 123</td>
</tr>
<tr>
<td>Present series</td>
<td>M = 112; F = 70</td>
</tr>
</tbody>
</table>

HISTORY OF LOSS OF CONSCIOUSNESS:

Loss of consciousness is not associated with any of the neuroophthalmic deficits in the present series. A similar finding was seen by Van Stavern et al in his study.

COMPARISION OF HISTORY OF LOSS OF CONSCIOUSNESS

<table>
<thead>
<tr>
<th>HISTORY OF LOC</th>
<th>Van Stavern et al</th>
<th>Present series</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>39.9%</td>
<td>37.94%</td>
</tr>
<tr>
<td>Absent</td>
<td>60.1%</td>
<td>62.09%</td>
</tr>
</tbody>
</table>
MOST COMMON AFFERENT DEFICIT:

Traumatic optic neuropathy (TON) was the most common afferent deficit in the present series. Of them indirect optic nerve trauma constituted majority of cases (74.5%). This is comparable with many of the studies. But it contradicts the study by Van Stavern et al which shows the retro chiasmal defects as the most common afferent deficit.

COMPARISION OF THE MOST COMMON AFFERENT DEFICIT

<table>
<thead>
<tr>
<th>STUDIES</th>
<th>MOST COMMON AFFERENT DEFICIT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Keane</td>
<td>TON</td>
</tr>
<tr>
<td>Kowal</td>
<td>TON</td>
</tr>
<tr>
<td>Moster et al</td>
<td>TON</td>
</tr>
<tr>
<td>Van Stavern et al</td>
<td>Retro chiasmal defects</td>
</tr>
<tr>
<td>Present series</td>
<td>TON</td>
</tr>
</tbody>
</table>
MOST COMMON EFFERENT DEFICIT:

Commonest cause of efferent pathway deficit was IV cranial nerve injury in the present series.

### COMPARISON OF THE MOST COMMON EFFERENT DEFICIT

<table>
<thead>
<tr>
<th>STUDIES</th>
<th>MOST COMMON EFFERENT DEFICIT</th>
<th>INCIDENCE (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jacobi et al</td>
<td>III Cr. N</td>
<td>5.1</td>
</tr>
<tr>
<td>Keane</td>
<td>III Cr. N</td>
<td>28.1</td>
</tr>
<tr>
<td>Sabates et al</td>
<td>IV Cr..N</td>
<td>40</td>
</tr>
<tr>
<td>Kowal</td>
<td>IV Cr..N</td>
<td>24</td>
</tr>
<tr>
<td>Lepore</td>
<td>IV Cr..N</td>
<td>33.3</td>
</tr>
<tr>
<td>Mariak et al</td>
<td>III Cr. N</td>
<td>50</td>
</tr>
<tr>
<td>Moster et al</td>
<td>III Cr. N</td>
<td>33</td>
</tr>
<tr>
<td>Van Stavern et al</td>
<td>IV Cr..N</td>
<td>51.2</td>
</tr>
<tr>
<td>Present series</td>
<td>IV Cr..N</td>
<td>52.1</td>
</tr>
</tbody>
</table>
INCIDENCE OF III CRANIAL NERVE PALSY:

The incidence of third cranial nerve palsy in the present series was 45%. Of them 8.69% of cases were bilateral.

COMPARISON OF THE INCIDENCE OF III CRANIAL NERVE PALSY

<table>
<thead>
<tr>
<th>STUDIES</th>
<th>INCIDENCE OF III CRANIAL NERVE PALSY(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jacobi et al</td>
<td>15.9</td>
</tr>
<tr>
<td>Keane</td>
<td>28.1</td>
</tr>
<tr>
<td>Sabates et al</td>
<td>33</td>
</tr>
<tr>
<td>Kowal</td>
<td>10</td>
</tr>
<tr>
<td>Lepore</td>
<td>28.3</td>
</tr>
<tr>
<td>Mariak et al</td>
<td>50</td>
</tr>
<tr>
<td>Moster et al</td>
<td>33</td>
</tr>
<tr>
<td>Van Stavern et al</td>
<td>46.4</td>
</tr>
<tr>
<td>Present series</td>
<td>45</td>
</tr>
</tbody>
</table>
INCIDENCE OF IV CRANIAL NERVE PALSY:

The incidence of fourth cranial nerve palsy in the present series was 52.1%. About 37% of cases were bilateral.

COMPARISON OF THE INCIDENCE OF IV CRANIAL NERVE PALSY

<table>
<thead>
<tr>
<th>STUDIES</th>
<th>INCIDENCE OF IV CRANIAL NERVE PALSY(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Keane</td>
<td>19.8</td>
</tr>
<tr>
<td>Sabates et al</td>
<td>40</td>
</tr>
<tr>
<td>Kowal</td>
<td>24</td>
</tr>
<tr>
<td>Lepore</td>
<td>33.3</td>
</tr>
<tr>
<td>Mariak et al</td>
<td>17</td>
</tr>
<tr>
<td>Moster et al</td>
<td>28</td>
</tr>
<tr>
<td>Van Stavern et al</td>
<td>51.2</td>
</tr>
<tr>
<td>Present series</td>
<td>52.1</td>
</tr>
</tbody>
</table>
INCIDENCE OF VI CRANIAL NERVE PALSY:

The incidence of sixth cranial nerve palsy in the present series was 29.40%. Bilateral sixth nerve palsy was seen in 26.67%.

COMPARISON OF THE INCIDENCE OF VI CRANIAL NERVE PALSY

<table>
<thead>
<tr>
<th>STUDIES</th>
<th>INCIDENCE OF VI CRANIAL NERVE PALSY(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Keane</td>
<td>26</td>
</tr>
<tr>
<td>Sabates et al</td>
<td>27</td>
</tr>
<tr>
<td>Kowal</td>
<td>10</td>
</tr>
<tr>
<td>Lepore</td>
<td>11.7</td>
</tr>
<tr>
<td>Mariak et al</td>
<td>17</td>
</tr>
<tr>
<td>Moster et al</td>
<td>20</td>
</tr>
<tr>
<td>Van Stavern et al</td>
<td>25</td>
</tr>
<tr>
<td>Present series</td>
<td>26.67</td>
</tr>
</tbody>
</table>
INCIDENCE OF MULTIPLE CRANIAL NERVE PALSY:

Multiple cranial nerve palsy was seen in 27.45% patients. This is comparable to study by Van Stavern et al.

COMPARISION OF THE INCIDENCE OF MULTIPLE CRANIAL NERVE PALSY

<table>
<thead>
<tr>
<th>STUDIES</th>
<th>INCIDENCE OF MULTIPLE CRANIAL NERVE PALSY(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Van Stavern et al</td>
<td>22.6</td>
</tr>
<tr>
<td>Present series</td>
<td>27.45</td>
</tr>
</tbody>
</table>
SIGNIFICANT NEURO IMAGING ABNORMALITY:

In the present series, only 45.6% of patients had evidence of injury in CT. Van Stavern et al found significant abnormality in 47.3% of head injury patients. There was a correlation between intra cranial haemorrhage and third and fourth cranial nerve palsies. Basillar skull fracture was associated with sixth cranial nerve palsies. A similar inference was given by Van Stavern et al in his study.

**COMPARISION OF SIGNIFICANT NEURO IMAGING ABNORMALITY**

<table>
<thead>
<tr>
<th>NEURO IMAGING ABNORMALITY</th>
<th>Van Stavern et al</th>
<th>Present series</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>52.7%</td>
<td>54.4%</td>
</tr>
<tr>
<td>Present</td>
<td>47.3%</td>
<td>45.6%</td>
</tr>
<tr>
<td>ICH</td>
<td>62.1%</td>
<td>59.03%</td>
</tr>
<tr>
<td>Skull fracture</td>
<td>24.2%</td>
<td>26.50%</td>
</tr>
<tr>
<td>Contusion</td>
<td>28.7%</td>
<td>36.14%</td>
</tr>
</tbody>
</table>
CONCLUSION

The following conclusions are made from the above observations:

1. Head injury is more common in the age group of 20-40 years.
2. Males are more commonly affected than females.
3. The most common mode of head injury is road traffic accident.
4. Loss of consciousness is not associated with any neuroophthalmic deficits.
5. Efferent visual pathway deficit is more common than afferent visual pathway deficit.
6. Commonest efferent visual pathway deficit is IV nerve injury.
7. Commonest afferent visual pathway deficit is indirect traumatic optic neuropathy.
8. Bilateral injury is more common with IV nerve injury.
9. Recovery pattern of visual pathway deficits are not very good inspite of timely management.
10. The presence of significant neuroimaging abnormality, particularly intracranial haemorrhage is significantly associated with III and IV nerve injuries.
11. Even in the absence of any neuroimaging abnormality, the prevalence of neuro-ophthalmic deficits is high.

12. Meticulous awareness of traffic rules, helmet wear, giving up of drunken drive are the safety measures to avoid road traffic accidents and to prevent head injury for, “prevention is always better than cure”.
PROFORMA

Neuro ophthalmic manifestations of head injuries

Date:

1. Name
2. Age/sex
3. Occupation & address
4. I.P. No.
   Head injury No.                                  Unit: N.S. I/II/III
   Eye OP NO.
   Neuro-opthal clinic No.
5. Date of Admission:   Discharge:
6. Ocular Complaints/duration:
7. Information related to injury:
   1. Mode of injury:
      RTA: pedestrian/non motorized/two wheeler / closed vehicle
      Fall: Home / workplace <6ft/6ft
      Domestic accident
      Industrial /blast/falling object
   2. Assault : Blunt/sharp weapon
   3. Under alcohol
      Associated injuries: (specify)
      Chest/abdomen / faced ENT / Spine / orthopaedic
8. Post-traumatic   :   LOC duration
   Amnesia / vomiting / giddiness
   Fits: type & number
   ENT bleed/rhinorrhoea / otorrhoea
   Vision / diplopia in immediate period.
   (if symptomatic at examination)
9. Glasgow coma scale:
   On admission:   On exam:   trend:


11. History of Ocular problems:   Onset/ Course / duration
   1)
   2)
   3)

12. Detailed description of salient complaint:

   EXAMINATION :
   Right Eye   Left Eye

13. Proptosis / enophthalmos

14. Orbital margins

15. Lids / periocular area

16. Conjunctiva / cornea

17. AC/ins

18. Pupil size / shape
   - Reflex
   Direct   Consensual
   - RAPD    Grade
   - Near synkinesis

19. Lens

20. Sensation
   - Corneal
   - Lids and face

   OPTIC NERVE AND VISUAL PATHWAY

21. Vision (Bedside)
    [FC and near card]

22. Refr. Error and BCVA

23. Colour perception
- Simple red target
- Ishihara’s

24. Brightness sense
   (Compare eyes)

25. Fields
   - Confrontation
   - Tangent screen / perimeter (target size)

26. **EXTRA OCULAR MOVEMENTS**

   Tropia:
   - ET/XT/HT
   - 1° deviation
   - Sec. Deviation

27. Nystagmus

28. Diplopia charting

29. Conjugate movements
   (Suranuclear)

30. Oculocephalic / Oculorestibular reflex
    [only if gaze affected]

31. Oculokinetic nystagmus

32. Fundus examination

   Media
   Disc margins
   Size / Shape / colour
   Swelling
   Peripapillary & background
   Vessels
   Macula

33. Slit lamp examination

34. Hertel’s exophthalmometry
35. Tension

36. General examination

37. CNS: Higher functions
   Cranial nerves
   Motor system
   Sensory system
   DTR and plantar reflex
   Gait and coordination

INVESTIGATIONS

38. Skull x-ray

39. CT scan Brain
   Skull bone # location / type
   (1) Haemorrhage EDH / SDH/SAH/ICh
   (2) Parenchyma
      • Oedema
      • Contusion
   (3) Compression
      • Ventricule
      • Midline shift
   (4) Pneumocephalus

   Review CT Brain :

40. CT Scan orbit
   Orbital wall #:
   Optic nerve:
   Intra canalicular
   Intra orbital
   Soft tissue; haematoma / emphysema
   Globe:

41. Provisional Diagnosis:
   1. Ophthalmic / Neuro ophthalmic
   2. Neurosurgical

42. Coincident surgical problems
Chest
Abdomen
Spine
Orthopaedic

43. Medical Problems : Diabetes / hypertension / uremia
44. Progress & Result :
45. Follow up : in Ophthalmology OPD
<table>
<thead>
<tr>
<th>S.No.</th>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>IP No.</th>
<th>Mode of Injury</th>
<th>H/o LOC</th>
<th>Pre Symptoms</th>
<th>Laterality</th>
<th>VA</th>
<th>Ant Segm</th>
<th>Pupil</th>
<th>Fundus</th>
<th>Clinical Diagnosis</th>
<th>Neuro imaging</th>
<th>Follow up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Raju</td>
<td>30</td>
<td>M</td>
<td>012467</td>
<td>RTA</td>
<td>-</td>
<td>Eye pain diplopia</td>
<td>LE</td>
<td>6/6</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>6th N palsy</td>
<td>SDH</td>
<td>FR</td>
</tr>
<tr>
<td>2</td>
<td>Karpagam</td>
<td>27</td>
<td>F</td>
<td>011354</td>
<td>RTA</td>
<td>-</td>
<td>Z</td>
<td>BE</td>
<td>6/9</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>Retrochoroidal lesion</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>3</td>
<td>Ponni</td>
<td>29</td>
<td>F</td>
<td>019765</td>
<td>RTA</td>
<td>+</td>
<td>Diplopia</td>
<td>BE</td>
<td>6/6</td>
<td>NAD Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>Basilar #</td>
<td>NR</td>
</tr>
<tr>
<td>4</td>
<td>Palsamy</td>
<td>60</td>
<td>M</td>
<td>018564</td>
<td>Fall</td>
<td>+</td>
<td>Def. Vn</td>
<td>RE</td>
<td>HM</td>
<td>SCH</td>
<td>RAPD</td>
<td>NAD II</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>5</td>
<td>Nattraj</td>
<td>32</td>
<td>M</td>
<td>013856</td>
<td>Assault</td>
<td>-</td>
<td>Def. Vn</td>
<td>RE</td>
<td>No PL</td>
<td>SCH</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>ICH (Rt) FL</td>
<td>NR</td>
</tr>
<tr>
<td>6</td>
<td>Hemesh</td>
<td>16</td>
<td>M</td>
<td>017621</td>
<td>RTA</td>
<td>-</td>
<td>Pain, HA</td>
<td>RE</td>
<td>6/6</td>
<td>NAD Diplopia</td>
<td>NAD</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>7</td>
<td>Ramesh</td>
<td>30</td>
<td>M</td>
<td>017629</td>
<td>RTA</td>
<td>+</td>
<td>Def. Vn</td>
<td>LE</td>
<td>6/36</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>8</td>
<td>Eswaran</td>
<td>53</td>
<td>M</td>
<td>018719</td>
<td>Fall</td>
<td>+</td>
<td>Diplopia</td>
<td>LE</td>
<td>6/24</td>
<td>NAD</td>
<td>DIL. NRTL</td>
<td>NAD</td>
<td>III N Palsy</td>
<td>Contusion</td>
<td>MB</td>
</tr>
<tr>
<td>9</td>
<td>Manoharan</td>
<td>35</td>
<td>M</td>
<td>017257</td>
<td>RTA</td>
<td>+</td>
<td>Diplopia</td>
<td>BE</td>
<td>6/6</td>
<td>SCH</td>
<td>DIL. NRTL</td>
<td>NAD</td>
<td>III N Palsy</td>
<td>Hge MB</td>
<td>PR</td>
</tr>
<tr>
<td>10</td>
<td>Muthukumar</td>
<td>36</td>
<td>M</td>
<td>016999</td>
<td>Assault</td>
<td>-</td>
<td>Def. Vn</td>
<td>LE</td>
<td>6/60</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>FR</td>
</tr>
<tr>
<td>11</td>
<td>George</td>
<td>17</td>
<td>M</td>
<td>019274</td>
<td>Assault</td>
<td>-</td>
<td>Eye pain</td>
<td>(BE)</td>
<td>6/9</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>Basilar #</td>
<td>PR</td>
</tr>
<tr>
<td>12</td>
<td>Palpandi</td>
<td>27</td>
<td>M</td>
<td>023595</td>
<td>RTA</td>
<td>-</td>
<td>Ptosis, HA</td>
<td>RE</td>
<td>6/6</td>
<td>SCH</td>
<td>DIL. NRTL</td>
<td>NAD</td>
<td>III, IV N Palsy</td>
<td>Hge MB</td>
<td>PF</td>
</tr>
<tr>
<td>13</td>
<td>Suresh</td>
<td>8</td>
<td>M</td>
<td>025496</td>
<td>Fall</td>
<td>-</td>
<td>Def. Vn</td>
<td>(BE)</td>
<td>6/60</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>Cortic. Blindness</td>
<td>NAD</td>
<td>FR</td>
</tr>
<tr>
<td>14</td>
<td>Paramasivam</td>
<td>47</td>
<td>M</td>
<td>025235</td>
<td>RTA</td>
<td>+</td>
<td>Diplopia</td>
<td>LE</td>
<td>6/24</td>
<td>NAD Diplopia</td>
<td>DIL. NRTL</td>
<td>Papilledema</td>
<td>III, IV, VI, palsy</td>
<td>Hge MB</td>
<td>NR</td>
</tr>
<tr>
<td>15</td>
<td>Pandi</td>
<td>29</td>
<td>M</td>
<td>024431</td>
<td>RTA</td>
<td>+</td>
<td>Def. Vn</td>
<td>RE</td>
<td>PL</td>
<td>SCH</td>
<td>RAPD</td>
<td>NAD</td>
<td>Disc edema</td>
<td>II N Palsy</td>
<td>NAD</td>
</tr>
<tr>
<td>16</td>
<td>Kannan</td>
<td>19</td>
<td>M</td>
<td>024371</td>
<td>RTA</td>
<td>-</td>
<td>Ptosis, HA</td>
<td>RE</td>
<td>6/12</td>
<td>NAD</td>
<td>DIL. NRTL</td>
<td>NAD</td>
<td>III, IV N Palsy</td>
<td>Hge MB</td>
<td>NR</td>
</tr>
<tr>
<td>17</td>
<td>Abdul</td>
<td>30</td>
<td>M</td>
<td>025146</td>
<td>RTA</td>
<td>-</td>
<td>Def. Vn</td>
<td>BE</td>
<td>6/6</td>
<td>SCH</td>
<td>RTL</td>
<td>NAD</td>
<td>Supra. Nu. gaze palsy</td>
<td>Contusion (Rt) FL</td>
<td>NR</td>
</tr>
<tr>
<td>18</td>
<td>Gurusamy</td>
<td>54</td>
<td>M</td>
<td>026714</td>
<td>Work place</td>
<td>+</td>
<td>Diplopia</td>
<td>RE</td>
<td>6/24</td>
<td>NAD Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>NAD</td>
<td>FR</td>
</tr>
<tr>
<td>19</td>
<td>Geetha</td>
<td>37</td>
<td>F</td>
<td>027738</td>
<td>RTA</td>
<td>-</td>
<td>Diplopia</td>
<td>RE</td>
<td>6/6</td>
<td>NAD Diplopia</td>
<td>RTL</td>
<td>Papilledema</td>
<td>VI N Palsy</td>
<td>Basilar #</td>
<td>PR</td>
</tr>
<tr>
<td>20</td>
<td>Madhivanan</td>
<td>48</td>
<td>M</td>
<td>023998</td>
<td>Fall</td>
<td>+</td>
<td>Def. Vn</td>
<td>(BE)</td>
<td>3/60</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>Cortic. Blindness</td>
<td>Contusion</td>
<td>occi. Lobe</td>
</tr>
<tr>
<td>21</td>
<td>Anbunathan</td>
<td>47</td>
<td>M</td>
<td>026620</td>
<td>Assault</td>
<td>-</td>
<td>Def. Vn</td>
<td>LE</td>
<td>No PL</td>
<td>SCH</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td># O.Canal</td>
<td>NR</td>
</tr>
<tr>
<td>22</td>
<td>Chellammal</td>
<td>32</td>
<td>F</td>
<td>029995</td>
<td>RTA</td>
<td>+</td>
<td>Def. Vn</td>
<td>BE</td>
<td>6/12</td>
<td>NAD (L) Homo Hemianopia</td>
<td>RTL</td>
<td>NAD</td>
<td>Contusion Rt. PL</td>
<td>PR</td>
<td></td>
</tr>
<tr>
<td>S.No.</td>
<td>Name</td>
<td>Age</td>
<td>Sex</td>
<td>IP No.</td>
<td>Mode of Injury</td>
<td>H/o LOC</td>
<td>Pre Symptoms</td>
<td>Laterality</td>
<td>VA</td>
<td>Ant Segm</td>
<td>Pupil</td>
<td>Fundus</td>
<td>Clinical Diagnosis</td>
<td>Neuro imaging</td>
<td>Follow up</td>
</tr>
<tr>
<td>-------</td>
<td>----------</td>
<td>-----</td>
<td>-----</td>
<td>--------</td>
<td>---------------</td>
<td>---------</td>
<td>--------------</td>
<td>------------</td>
<td>----</td>
<td>----------</td>
<td>-------</td>
<td>--------</td>
<td>-------------------</td>
<td>---------------</td>
<td>-----------</td>
</tr>
<tr>
<td>23</td>
<td>Mary</td>
<td>67</td>
<td>F</td>
<td>029998</td>
<td>RTA</td>
<td></td>
<td>Diplopia</td>
<td>BE</td>
<td>6/24</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>Basilar #</td>
<td>PR</td>
</tr>
<tr>
<td>24</td>
<td>Mari Raj</td>
<td>34</td>
<td>M</td>
<td>028718</td>
<td>Fall</td>
<td></td>
<td></td>
<td>(LE)</td>
<td>6/6</td>
<td>NAD, Diplopia</td>
<td>DIL, NRTL</td>
<td>NAD</td>
<td>III N Palsy</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>25</td>
<td>Gokul</td>
<td>18</td>
<td>M</td>
<td>030019</td>
<td>RTA</td>
<td>+</td>
<td>Def. Vn</td>
<td>RE</td>
<td>2/60</td>
<td>NAD</td>
<td>RAPD</td>
<td>Disc edema</td>
<td>II N Palsy</td>
<td>RIH</td>
<td>FR</td>
</tr>
<tr>
<td>26</td>
<td>Rajesh</td>
<td>28</td>
<td>M</td>
<td>031286</td>
<td>RTA</td>
<td>-</td>
<td>Diplopia</td>
<td>RE</td>
<td>6/6</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>NAD</td>
<td>NR</td>
</tr>
<tr>
<td>27</td>
<td>Surya</td>
<td>26</td>
<td>F</td>
<td>031481</td>
<td>RTA</td>
<td>+</td>
<td>Diplopia</td>
<td>RE</td>
<td>6/6</td>
<td>SCH</td>
<td>RTL</td>
<td>NAD</td>
<td>VI N Palsy</td>
<td>NAD</td>
<td>FR</td>
</tr>
<tr>
<td>28</td>
<td>Pitchai</td>
<td>57</td>
<td>M</td>
<td>032167</td>
<td>Fall</td>
<td>-</td>
<td>Pain</td>
<td>RE</td>
<td>6/12</td>
<td>Ptosis</td>
<td>DIL, NRTL</td>
<td>NAD</td>
<td>IV, III N Palsy</td>
<td>SDH</td>
<td>NR</td>
</tr>
<tr>
<td>29</td>
<td>Amsavalli</td>
<td>28</td>
<td>F</td>
<td>032817</td>
<td>RTA</td>
<td>-</td>
<td>Posis</td>
<td>LE</td>
<td>6/6</td>
<td>SCH, Ptosis</td>
<td>DIL, NRTL</td>
<td>NAD</td>
<td>III N Palsy</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>30</td>
<td>Angayarkanni</td>
<td>31</td>
<td>F</td>
<td>032264</td>
<td>RTA</td>
<td>+</td>
<td>Def. Vn</td>
<td>RE</td>
<td>6/60</td>
<td>NAD</td>
<td>RAPD</td>
<td>Disc edema</td>
<td>II N Palsy</td>
<td>Ons. Haematoma</td>
<td>PR</td>
</tr>
<tr>
<td>31</td>
<td>Govindan</td>
<td>26</td>
<td>M</td>
<td>038192</td>
<td>RTA</td>
<td>-</td>
<td>Def. Vn</td>
<td>LE</td>
<td>HM</td>
<td>NAD</td>
<td>RAPD</td>
<td>Disc edema</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>32</td>
<td>Mookayee</td>
<td>38</td>
<td>F</td>
<td>039937</td>
<td>Assault</td>
<td>-</td>
<td>Diplopia</td>
<td>RE</td>
<td>6/6</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>33</td>
<td>Rohini</td>
<td>27</td>
<td>F</td>
<td>038252</td>
<td>Assault</td>
<td>+</td>
<td>Posis</td>
<td>LE</td>
<td>6/6</td>
<td>SCH, Ptosis</td>
<td>DIL, NRTL</td>
<td>NAD</td>
<td>III N Palsy</td>
<td>EDH</td>
<td>PR</td>
</tr>
<tr>
<td>34</td>
<td>Sundaram</td>
<td>30</td>
<td>M</td>
<td>039733</td>
<td>RTA</td>
<td>-</td>
<td>Diplopia</td>
<td>(RE)</td>
<td>6/9</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>35</td>
<td>Kanthammal</td>
<td>46</td>
<td>F</td>
<td>039898</td>
<td>Fall</td>
<td>-</td>
<td>Def. Vn</td>
<td>RE</td>
<td>6/60</td>
<td>SCH</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>Ons. Haematoma</td>
<td>PR</td>
</tr>
<tr>
<td>36</td>
<td>Saira banu</td>
<td>32</td>
<td>F</td>
<td>039921</td>
<td>Assault</td>
<td>+</td>
<td>Def. Vn</td>
<td>LE</td>
<td>No PL</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>O.Canal</td>
<td>NR</td>
</tr>
<tr>
<td>37</td>
<td>Hari haran</td>
<td>32</td>
<td>M</td>
<td>045395</td>
<td>RTA</td>
<td>+</td>
<td>plosis</td>
<td>LE</td>
<td>6/12</td>
<td>SCH, Ptosis</td>
<td>DIL, NRTL</td>
<td>NAD</td>
<td>III N Palsy</td>
<td>Basilar #</td>
<td>PR</td>
</tr>
<tr>
<td>38</td>
<td>Anjali</td>
<td>26</td>
<td>F</td>
<td>046954</td>
<td>RTA</td>
<td>-</td>
<td>Headache</td>
<td>Def.vn</td>
<td>LE</td>
<td>6/60</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>EDH</td>
</tr>
<tr>
<td>39</td>
<td>Roseline</td>
<td>30</td>
<td>F</td>
<td>046817</td>
<td>RTA</td>
<td>-</td>
<td>Diplopia</td>
<td>BE</td>
<td>6/6</td>
<td>SCH, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>Basilar #</td>
<td>PR</td>
</tr>
<tr>
<td>40</td>
<td>Kanthimathi</td>
<td>52</td>
<td>F</td>
<td>041110</td>
<td>Fall</td>
<td>-</td>
<td>plosis</td>
<td>RE</td>
<td>6/24</td>
<td>NAD</td>
<td>DIL, NRTL</td>
<td>NAD</td>
<td>III N Palsy</td>
<td>NAD</td>
<td>FR</td>
</tr>
<tr>
<td>41</td>
<td>Thangam</td>
<td>29</td>
<td>M</td>
<td>047421</td>
<td>Assault</td>
<td>+</td>
<td>Def. Vn</td>
<td>BE</td>
<td>6/18</td>
<td>NAD, (RT) homo lhemianopia</td>
<td>RTL</td>
<td>NAD</td>
<td>Retrochias mal injury</td>
<td>ICH (RT) PL</td>
<td>PR</td>
</tr>
<tr>
<td>42</td>
<td>Mariappan</td>
<td>44</td>
<td>M</td>
<td>045753</td>
<td>Assault</td>
<td>-</td>
<td>Def. Vn</td>
<td>RE</td>
<td>6/60</td>
<td>SCH</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>Contusion (RT) FL</td>
<td>NR</td>
</tr>
<tr>
<td>43</td>
<td>Abirami</td>
<td>28</td>
<td>F</td>
<td>045579</td>
<td>RTA</td>
<td>-</td>
<td>HA, eye pain</td>
<td>RE</td>
<td>6/6</td>
<td>SCH, enophthal</td>
<td>SRTL</td>
<td>Papiledema</td>
<td>Horner's</td>
<td>ICH MB</td>
<td>FR</td>
</tr>
<tr>
<td>S.No.</td>
<td>Name</td>
<td>Age</td>
<td>Sex</td>
<td>IP No.</td>
<td>Mode of Injury</td>
<td>H/o LOC</td>
<td>Pre Symptoms</td>
<td>Laterality</td>
<td>VA</td>
<td>Ant Segm</td>
<td>Pupil</td>
<td>Fundus</td>
<td>Clinical Diagnosis</td>
<td>Neuro imaging</td>
<td>Follow up</td>
</tr>
<tr>
<td>-------</td>
<td>------------</td>
<td>-----</td>
<td>-----</td>
<td>--------</td>
<td>---------------</td>
<td>---------</td>
<td>-------------------</td>
<td>------------</td>
<td>----</td>
<td>----------</td>
<td>-------</td>
<td>--------</td>
<td>-------------------</td>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>44</td>
<td>Alagar</td>
<td>28</td>
<td>M</td>
<td>048596</td>
<td>RTA</td>
<td></td>
<td>Diplopia</td>
<td>RE</td>
<td>6/6</td>
<td>NAD, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>ICH MB</td>
<td>NR</td>
</tr>
<tr>
<td>45</td>
<td>Velammal</td>
<td>62</td>
<td>F</td>
<td>031001</td>
<td>RTA</td>
<td></td>
<td>Def.vn, HA</td>
<td>(RE)</td>
<td>No PL</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>SDH</td>
<td>NR</td>
</tr>
<tr>
<td>46</td>
<td>Suresh</td>
<td>26</td>
<td>M</td>
<td>051214</td>
<td>RTA</td>
<td></td>
<td>Ptosis, eye pain</td>
<td>(LE)</td>
<td>6/6</td>
<td>SCH, Ptosis</td>
<td>DIL,</td>
<td>NRTL</td>
<td>III IV N Palsy</td>
<td>ICH MB</td>
<td>NR</td>
</tr>
<tr>
<td>47</td>
<td>Amsuvalli</td>
<td>32</td>
<td>F</td>
<td>051875</td>
<td>Fall</td>
<td></td>
<td>Posis</td>
<td>RE</td>
<td>6/9</td>
<td>SCH</td>
<td>DIL,</td>
<td>NRTL</td>
<td>Papilledema</td>
<td>III, IV N Palsy</td>
<td>SDH</td>
</tr>
<tr>
<td>48</td>
<td>Balaji</td>
<td>15</td>
<td>M</td>
<td>051815</td>
<td>RTA</td>
<td></td>
<td>Def.Vn</td>
<td>BE</td>
<td>6/60</td>
<td>NAD</td>
<td>NAD</td>
<td>NAD</td>
<td>Cortical blindness</td>
<td>Contusion OL</td>
<td>FR</td>
</tr>
<tr>
<td>49</td>
<td>Mani</td>
<td>34</td>
<td>M</td>
<td>053976</td>
<td>RTA</td>
<td></td>
<td>HA, Ptosis</td>
<td>RE</td>
<td>6/9</td>
<td>Ptosis, Diplopia</td>
<td>DIL,</td>
<td>NRTL</td>
<td>III, IV N Palsy</td>
<td>SDH</td>
<td>NR</td>
</tr>
<tr>
<td>50</td>
<td>Amaravathy</td>
<td>35</td>
<td>F</td>
<td>058807</td>
<td>Fall</td>
<td></td>
<td>HA</td>
<td>LE</td>
<td>6/9</td>
<td>Ptosis, Enophthal</td>
<td>Miotic</td>
<td>SRXL</td>
<td>NAD</td>
<td>Horneis</td>
<td>PR</td>
</tr>
<tr>
<td>51</td>
<td>Karuppayee</td>
<td>50</td>
<td>F</td>
<td>059555</td>
<td>Assault</td>
<td></td>
<td>Def.Vn</td>
<td>LE</td>
<td>6/60</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy # O.Canal</td>
<td></td>
<td>NR</td>
</tr>
<tr>
<td>52</td>
<td>Saravanan</td>
<td>26</td>
<td>M</td>
<td>059785</td>
<td>RTA</td>
<td></td>
<td>HA, Diplopia</td>
<td>LE</td>
<td>6/12</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>EDH</td>
<td>PR</td>
</tr>
<tr>
<td>53</td>
<td>Gomathy</td>
<td>45</td>
<td>F</td>
<td>061110</td>
<td>Assault</td>
<td></td>
<td>Def.Vn</td>
<td>RE</td>
<td>No PL</td>
<td>SCH</td>
<td>RAPD</td>
<td>Retin.he</td>
<td>II N Palsy</td>
<td>SAH</td>
<td>NR</td>
</tr>
<tr>
<td>54</td>
<td>Vijay</td>
<td>15</td>
<td>M</td>
<td>061094</td>
<td>RTA</td>
<td></td>
<td>HA</td>
<td>BE</td>
<td>6/6</td>
<td>NAD Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>EDH</td>
<td>FR</td>
</tr>
<tr>
<td>55</td>
<td>Raju</td>
<td>26</td>
<td>M</td>
<td>062137</td>
<td>Fall</td>
<td></td>
<td>Diplopia</td>
<td>RTA</td>
<td>NAD</td>
<td>Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>VI N Palsy</td>
<td>NAD</td>
<td>FR</td>
</tr>
<tr>
<td>56</td>
<td>Manikandan</td>
<td>29</td>
<td>M</td>
<td>063936</td>
<td>RTA</td>
<td></td>
<td>Def.Vn</td>
<td>(RE)</td>
<td>6/60</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>EDH</td>
<td>NR</td>
</tr>
<tr>
<td>57</td>
<td>Chellam</td>
<td>28</td>
<td>F</td>
<td>063381</td>
<td>RTA</td>
<td></td>
<td>Headache</td>
<td>(BE)</td>
<td>6/6</td>
<td>NAD, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>Basilar #</td>
<td>PR</td>
</tr>
<tr>
<td>58</td>
<td>Abdul</td>
<td>9</td>
<td>M</td>
<td>063993</td>
<td>RTA</td>
<td></td>
<td>Def.Vn</td>
<td>BE</td>
<td>3/60</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>Cortical Blindness</td>
<td>NAD</td>
<td>FR</td>
</tr>
<tr>
<td>59</td>
<td>Gururaj</td>
<td>31</td>
<td>M</td>
<td>068235</td>
<td>RTA</td>
<td></td>
<td>Diplopia</td>
<td>LE</td>
<td>6/6</td>
<td>NAD, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>Hge MB</td>
<td>NR</td>
</tr>
<tr>
<td>60</td>
<td>Thangam</td>
<td>22</td>
<td>F</td>
<td>069595</td>
<td>RTA</td>
<td></td>
<td>Def.Vn</td>
<td>LE</td>
<td>6/36</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>NR</td>
</tr>
<tr>
<td>61</td>
<td>Rajapandi</td>
<td>63</td>
<td>M</td>
<td>069641</td>
<td>RTA</td>
<td></td>
<td>Def.Vn</td>
<td>LE</td>
<td>6/60</td>
<td>SCH</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>NR</td>
</tr>
<tr>
<td>62</td>
<td>Arunkumar</td>
<td>24</td>
<td>M</td>
<td>067728</td>
<td>RTA</td>
<td></td>
<td>Headache, Diplopia</td>
<td>RE</td>
<td>6/12</td>
<td>NAD, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>63</td>
<td>Siddique</td>
<td>49</td>
<td>M</td>
<td>068975</td>
<td>Work place</td>
<td></td>
<td>Posis, HA</td>
<td>RE</td>
<td>6/12</td>
<td>Posis, Diplopia</td>
<td>DIL</td>
<td>NRTL</td>
<td>III N Palsy</td>
<td></td>
<td>FR</td>
</tr>
<tr>
<td>64</td>
<td>Kanaga</td>
<td>24</td>
<td>F</td>
<td>069143</td>
<td>RTA</td>
<td></td>
<td>Def. Vn (LE) 1/2 of VF</td>
<td>LE</td>
<td>6/12</td>
<td>NAD, (Lt) homo.hemianopia</td>
<td>RTL</td>
<td>NAD</td>
<td>Retrochiasmal injury</td>
<td>Contusion Rt. PL</td>
<td>NR</td>
</tr>
<tr>
<td>65</td>
<td>Banu</td>
<td>17</td>
<td>F</td>
<td>067149</td>
<td>RTA</td>
<td></td>
<td>HA, Posis</td>
<td>RE</td>
<td>6/6</td>
<td>SCH, Diplopia</td>
<td>DIL</td>
<td>NRTL</td>
<td>III IV N Palsy</td>
<td>Basilar #</td>
<td>NR</td>
</tr>
<tr>
<td>66</td>
<td>Eswaran</td>
<td>25</td>
<td>M</td>
<td>068789</td>
<td>RTA</td>
<td></td>
<td>Headache</td>
<td>LE</td>
<td>6/6</td>
<td>NAD, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>VI N Palsy</td>
<td></td>
<td>NR</td>
</tr>
<tr>
<td>S.No.</td>
<td>Name</td>
<td>Age</td>
<td>Sex</td>
<td>IP No.</td>
<td>Mode of Injury</td>
<td>H/o LOC</td>
<td>Pre Symptoms</td>
<td>Laterality</td>
<td>VA</td>
<td>Ant Segm</td>
<td>Pupil</td>
<td>Fundus</td>
<td>Clinical Diagnosis</td>
<td>Neuro imaging</td>
<td>Follow up</td>
</tr>
<tr>
<td>-------</td>
<td>--------------</td>
<td>-----</td>
<td>-----</td>
<td>----------</td>
<td>----------------</td>
<td>---------</td>
<td>--------------</td>
<td>------------</td>
<td>------</td>
<td>----------------</td>
<td>-------</td>
<td>--------</td>
<td>------------------------</td>
<td>---------------</td>
<td>-----------</td>
</tr>
<tr>
<td>67</td>
<td>Ammaponnun</td>
<td>32</td>
<td>F</td>
<td>062134</td>
<td>RTA</td>
<td></td>
<td>Diplopia</td>
<td>BE</td>
<td>6/6</td>
<td>SCH, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>Basilar #</td>
<td>NR</td>
</tr>
<tr>
<td>68</td>
<td>Anbu</td>
<td>37</td>
<td>F</td>
<td>059891</td>
<td>Assault</td>
<td></td>
<td>Def. Vn</td>
<td>RE</td>
<td>6/6</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>NR</td>
</tr>
<tr>
<td>69</td>
<td>Saravanan</td>
<td>30</td>
<td>M</td>
<td>058735</td>
<td>RTA</td>
<td></td>
<td>HA</td>
<td>RE</td>
<td>6/6</td>
<td>Homo.hemianopia, with macular sparing</td>
<td>RTL</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>ICH (RT) OL</td>
<td>NR</td>
</tr>
<tr>
<td>70</td>
<td>Periakaruppan</td>
<td>56</td>
<td>M</td>
<td>069494</td>
<td>Fall</td>
<td></td>
<td>+ Def. Vn</td>
<td>BE</td>
<td>6/36</td>
<td>NAD</td>
<td>RALD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>Contusion OL</td>
<td>FR</td>
</tr>
<tr>
<td>71</td>
<td>Palaniammal</td>
<td>45</td>
<td>F</td>
<td>068172</td>
<td>Assault</td>
<td></td>
<td>- Def. Vn</td>
<td>(LE)</td>
<td>3/60</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>NR</td>
</tr>
<tr>
<td>72</td>
<td>Paul</td>
<td>25</td>
<td>M</td>
<td>068986</td>
<td>RTA</td>
<td></td>
<td>+ Def. Vn</td>
<td>RE</td>
<td>No PL</td>
<td>Ecchymosis, SCH</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>Contusion (Rt) FL</td>
<td>PR</td>
</tr>
<tr>
<td>73</td>
<td>Pitchai</td>
<td>25</td>
<td>M</td>
<td>068198</td>
<td>RTA</td>
<td></td>
<td>Ptosis</td>
<td>BE</td>
<td>6/9</td>
<td>NAD, Diplopia</td>
<td>DIL, NRTL</td>
<td>NAD</td>
<td>III N Palsy</td>
<td>Hge MB</td>
<td>FR</td>
</tr>
<tr>
<td>74</td>
<td>Priya</td>
<td>15</td>
<td>F</td>
<td>068279</td>
<td>RTA</td>
<td></td>
<td>Def. Vn</td>
<td>LE</td>
<td>6/6</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>NR</td>
</tr>
<tr>
<td>75</td>
<td>Shankar</td>
<td>49</td>
<td>M</td>
<td>071012</td>
<td>Fall</td>
<td></td>
<td>Diplopia</td>
<td>LE</td>
<td>6/6</td>
<td>NAD, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>VI N Palsy</td>
<td>NAD</td>
<td>FR</td>
</tr>
<tr>
<td>76</td>
<td>Selvamahesh</td>
<td>33</td>
<td>M</td>
<td>071921</td>
<td>Fall</td>
<td></td>
<td>HA, Diplopia</td>
<td>RE</td>
<td>6/6</td>
<td>SCH, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>VI N Palsy Basilar #</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>77</td>
<td>Sharada</td>
<td>54</td>
<td>F</td>
<td>072026</td>
<td>RTA</td>
<td></td>
<td>+ Def. Vn</td>
<td>LE</td>
<td>No PL</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>NR</td>
</tr>
<tr>
<td>78</td>
<td>Madhusudhan</td>
<td>36</td>
<td>M</td>
<td>072148</td>
<td>Assault</td>
<td></td>
<td>- Headache</td>
<td>RE</td>
<td>6/9</td>
<td>SCH, Diplopia, Ptosis</td>
<td>DIL, NRTL</td>
<td>NAD</td>
<td>III N Palsy Continus MB</td>
<td>Basilar #</td>
<td>PR</td>
</tr>
<tr>
<td>79</td>
<td>Kathik</td>
<td>33</td>
<td>M</td>
<td>073763</td>
<td>Assault</td>
<td></td>
<td>Headache</td>
<td>RE</td>
<td>6/12</td>
<td>Enophth, Ptosis</td>
<td>Miotic</td>
<td>SRTL</td>
<td>NAD</td>
<td>Horner's Basilar #</td>
<td>NR</td>
</tr>
<tr>
<td>80</td>
<td>Natarajan</td>
<td>27</td>
<td>M</td>
<td>074040</td>
<td>RTA</td>
<td></td>
<td>Diplopia</td>
<td>RE</td>
<td>6/6</td>
<td>NAD, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>NAD</td>
<td>FR</td>
</tr>
<tr>
<td>81</td>
<td>Pooja</td>
<td>9</td>
<td>F</td>
<td>074168</td>
<td>RTA</td>
<td></td>
<td>+ Def. Vn</td>
<td>BE</td>
<td>1/60</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>Cortic. Blindness</td>
<td>OL Hge</td>
<td>NR</td>
</tr>
<tr>
<td>82</td>
<td>Kokila</td>
<td>29</td>
<td>F</td>
<td>074327</td>
<td>RTA</td>
<td></td>
<td>Def. Vn</td>
<td>RE</td>
<td>6/6</td>
<td>NAD, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>VI N Palsy</td>
<td>Contusion Rt. TL</td>
<td>FR</td>
</tr>
<tr>
<td>83</td>
<td>Sundar</td>
<td>16</td>
<td>M</td>
<td>074565</td>
<td>Fall</td>
<td></td>
<td>HA, Diplopia</td>
<td>BE</td>
<td>6/6</td>
<td>SCH, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>VI N Palsy Basilar #</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>84</td>
<td>Ramesh</td>
<td>16</td>
<td>M</td>
<td>078807</td>
<td>RTA</td>
<td></td>
<td>Posis, HA</td>
<td>RE</td>
<td>6/6</td>
<td>NAD</td>
<td>DIL, NRTL</td>
<td>NAD</td>
<td>III N Palsy Hge MB</td>
<td>Basilar #</td>
<td>PR</td>
</tr>
<tr>
<td>85</td>
<td>Ayyavoo</td>
<td>67</td>
<td>M</td>
<td>071015</td>
<td>RTA</td>
<td></td>
<td>Def. Vn</td>
<td>RE</td>
<td>PL</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td># O.Canal PR</td>
<td>PR</td>
</tr>
<tr>
<td>86</td>
<td>Pothom Ponnu</td>
<td>28</td>
<td>F</td>
<td>073381</td>
<td>Assault</td>
<td></td>
<td>Def. Vn</td>
<td>RE</td>
<td>6/6</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>FR</td>
</tr>
<tr>
<td>87</td>
<td>Ranjani</td>
<td>28</td>
<td>F</td>
<td>076286</td>
<td>RTA</td>
<td></td>
<td>Def. Vn, HA</td>
<td>RE</td>
<td>3/60</td>
<td>SCH</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy SAH</td>
<td>NAD</td>
<td>NR</td>
</tr>
<tr>
<td>88</td>
<td>Kumarasamy</td>
<td>42</td>
<td>M</td>
<td>079413</td>
<td>Assault</td>
<td></td>
<td>HA, Posis</td>
<td>LE</td>
<td>6/6</td>
<td>SCH</td>
<td>DIL, NRTL</td>
<td>NAD</td>
<td>III N Palsy Basilar #</td>
<td>PR</td>
<td>PR</td>
</tr>
<tr>
<td>S.No.</td>
<td>Name</td>
<td>Age</td>
<td>Sex</td>
<td>IP No.</td>
<td>Mode of Injury</td>
<td>H/o LOC</td>
<td>Pre Symptoms</td>
<td>Laterality</td>
<td>VA</td>
<td>Ant Segm</td>
<td>Pupil</td>
<td>Fundus</td>
<td>Clinical Diagnosis</td>
<td>Neuro imaging</td>
<td>Follow up</td>
</tr>
<tr>
<td>-------</td>
<td>--------</td>
<td>-----</td>
<td>-----</td>
<td>-------</td>
<td>---------------</td>
<td>---------</td>
<td>--------------</td>
<td>------------</td>
<td>--------</td>
<td>------------</td>
<td>-------</td>
<td>--------</td>
<td>-------------------</td>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>89</td>
<td>Muthu</td>
<td>26</td>
<td>M</td>
<td>079942</td>
<td>RTA</td>
<td>-</td>
<td>Def .Vn</td>
<td>BE</td>
<td>6/12</td>
<td>NAD, Rt Homohemianopia</td>
<td>RTL</td>
<td>NAD</td>
<td>Retrochiasmal injury</td>
<td>Contusion (Lt) PL</td>
<td>PR</td>
</tr>
<tr>
<td>90</td>
<td>Kuppusamy</td>
<td>55</td>
<td>M</td>
<td>079987</td>
<td>Fall</td>
<td>+</td>
<td>HA, Def. Vn</td>
<td>BE</td>
<td>6/18</td>
<td>NAD</td>
<td>RTL</td>
<td>Papilledema</td>
<td>Conv. Insufficiency</td>
<td>SDH</td>
<td>PR</td>
</tr>
<tr>
<td>91</td>
<td>Mariammal</td>
<td>25</td>
<td>F</td>
<td>079093</td>
<td>RTA</td>
<td>-</td>
<td>Diplopia</td>
<td>RE</td>
<td>6/6</td>
<td>NAD, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>Basilar #</td>
<td>PR</td>
</tr>
<tr>
<td>92</td>
<td>Renuka</td>
<td>25</td>
<td>F</td>
<td>078194</td>
<td>RTA</td>
<td>-</td>
<td>Def. Vn</td>
<td>LE</td>
<td>No PL</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>EDH</td>
<td>NR</td>
</tr>
<tr>
<td>93</td>
<td>Rajan</td>
<td>23</td>
<td>M</td>
<td>079211</td>
<td>RTA</td>
<td>+</td>
<td>Diplopia</td>
<td>RE</td>
<td>6/6</td>
<td>NAD, Diplopia</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>Contusion MB</td>
<td>FR</td>
</tr>
<tr>
<td>94</td>
<td>Babu</td>
<td>29</td>
<td>M</td>
<td>080463</td>
<td>RTA</td>
<td>-</td>
<td>Eye Pain, Ptoxis</td>
<td>RE</td>
<td>6/12</td>
<td>SCH, Ptoxis, Diplopia</td>
<td>DIL,</td>
<td>NRTL</td>
<td>III N Palsy</td>
<td>Basilar #</td>
<td>PR</td>
</tr>
<tr>
<td>95</td>
<td>Velu</td>
<td>17</td>
<td>M</td>
<td>080781</td>
<td>RTA</td>
<td>-</td>
<td>HA, Diplopia</td>
<td>LE</td>
<td>6/6</td>
<td>NAD</td>
<td>RTL</td>
<td>NAD</td>
<td>IV N Palsy</td>
<td>EDH</td>
<td>FR</td>
</tr>
<tr>
<td>96</td>
<td>Mohamed Ali</td>
<td>69</td>
<td>M</td>
<td>080948</td>
<td>Fall</td>
<td>-</td>
<td>Headache</td>
<td>(BE)</td>
<td>6/36</td>
<td>SCH</td>
<td>miotic</td>
<td>NAD</td>
<td>Supra.Nuclear palsy</td>
<td>Hge Pons</td>
<td>NR</td>
</tr>
<tr>
<td>97</td>
<td>Ganesan</td>
<td>32</td>
<td>M</td>
<td>082888</td>
<td>RTA</td>
<td>-</td>
<td>Def. Vn</td>
<td>(BE)</td>
<td>6/12</td>
<td>NAD (Lt) Homohemianopia</td>
<td>RTL</td>
<td>NAD</td>
<td>Retrochiasmal injury</td>
<td>ICH (RT) TL</td>
<td>NR</td>
</tr>
<tr>
<td>98</td>
<td>Murugavel</td>
<td>47</td>
<td>M</td>
<td>083892</td>
<td>Assault</td>
<td>+</td>
<td>Def. Vn</td>
<td>(RE)</td>
<td>1/60</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>PR</td>
</tr>
<tr>
<td>99</td>
<td>Alagar</td>
<td>30</td>
<td>M</td>
<td>084144</td>
<td>RTA</td>
<td>+</td>
<td>Ptoxis</td>
<td>(LE)</td>
<td>6/12</td>
<td>NAD, Ptoxis, Diplopia</td>
<td>DIL,</td>
<td>NRTL</td>
<td>III, IV N Palsy</td>
<td>ICH MB</td>
<td>NR</td>
</tr>
<tr>
<td>100</td>
<td>Lakshmanan</td>
<td>35</td>
<td>M</td>
<td>085163</td>
<td>Assault</td>
<td>-</td>
<td>Def. Vn</td>
<td>RE</td>
<td>HM</td>
<td>NAD</td>
<td>RAPD</td>
<td>NAD</td>
<td>II N Palsy</td>
<td>NAD</td>
<td>NR</td>
</tr>
</tbody>
</table>
LESIONS AT VARIOUS LEVELS OF VISUAL PATHWAY AND CORRESPONDING VISUAL FIELD DEFECTS
VISUAL PATHWAY

- Retina
- Optic nerve
- Optic chiasma
- Optic tract
- Lateral geniculate body
- Optic radiations
- Visual cortex
THE PATHWAY OF PUPILLARY LIGHT REFLEX
DORSAL VIEW OF THE COURSE OF THE THIRD NERVE

LATERAL VIEW OF THE COURSE OF THE THIRD NERVE
LOCATION OF THE CRANIAL NERVES IN THE Cavernous Sinus Viewed From Behind

MECHANISM OF THIRD NERVE PALSY BY EXTRA DURAL HAEMATOMA
LOCATION OF PUPILLOMOTOR FIBRES WITHIN THE TRUNK OF THE THIRD NERVE

- Blood vessels on pia mater supply surface of the nerve including pupillary fibres (damaged by compressive lesions)
- Vasa nervorum supply part of nerve but not pupillary fibres (damaged by medical lesions)
- Pupillary fibres lie dorsal and peripheral
DORSAL VIEW OF THE COURSE OF THE FOURTH NERVE
THE SIXTH NERVE NUCLEUS AT THE LEVEL OF THE PONS

LATERAL VIEW OF THE COURSE OF THE SIXTH NERVE
ANATOMICAL PATHWAYS FOR HORIZONTAL EYE MOVEMENTS

PPRF : Pontine Paramedian Reticular Formation
MLF : Medial Longitudinal Fasciculus
MR : Medial Rectus
LR : Lateral Rectus
OCULOMOTOR NERVE PALSY – LEFT EYE
BILATERAL ABDUCENS NERVE PALSY
MULTIPLE CRANIAL NERVE PALSY – LEFT EYE
TROCHLEAR NERVE PALSY – LEFT EYE