Dissertation on

STUDY OF THE CLINICAL COURSE OF ABDUCENS NERVE PALSY AND OUTCOME OF ITS SURGICAL INTERVENTION

Submitted in partial fulfillment of requirements of

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CERTIFICATE

This is to certify that this dissertation entitled **"STUDY OF THE CLINICAL COURSE OF ABDUCENS NERVE (LATERAL RECTUS) PALSY AND OUTCOME OF ITS SURGICAL INTERVENTION"** is a bonafide record of the research work done by **Dr. ROSHANI DESAI,** post graduate in Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Madras Medical College and 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 2009-2012.

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CONTENTS

ABBREVIATIONS

RE	:	RIGHT EYE
LE	:	LEFT EYE
NAD	:	NO ABNORMALITY DETECTED
DR	:	DIABETIC RETINOPATHY
DM	:	DIABETES MELLITUS
HTN	:	HYPERTENSION
FR	:	FIXING WITH RIGHT EYE
FL	:	FIXING WITH LEFT EYE
PD	:	PRISM DIOPTERS
FDT	:	FORCED DUCTION TEST
FGT	:	FORCED GENERATION TEST
MR	:	MEDIAL RECTUS
LR	:	LATERAL RECTUS
IOP	:	INTRAOCULAR PRESSURE
BP	:	BLOOD PRESSURE

FBS : FASTING BLOOD SUGAR

PP : POST PRANDIAL

EMG : ELECTROMYOGRAPHY

PNS : PARA NASAL SINUSES

MRI : MAGNETIC RESONANCE IMAGING

MRA : MAGNETIC RESONANCE ANGIOGRAPHY

EOM : EXTRA OCULAR MOVEMENT

VDRL: VENEREAL DISEASE RESEARCH LABORATORY

ENT : EAR NOSE THROAT

PART I

INTRODUCTION

AND

HISTORY

INTRODUCTION AND HISTORY

The **abducens nerve** or **abducent nerve** (the **sixth cranial nerve**, also called the **sixth nerve** or **VI**) is a somatic efferent nerve that controls the movement of a single muscle, the lateral rectus muscle of the eye, in humans. In most other mammals it also innervates the musculus retractor bulbi, which can retract the eye for protection. Homologous abducens nerves are found in all vertebrates except lampreys and hagfishes.

The human sixth cranial nerve is derived from the basal plate of the embryonic pons.

The Latin name for the sixth cranial nerve is **nervus abducens**. The Terminologia Anatomica officially recognizes two different English translations: abducent nerve and abducens nerve. Either term is correct.

ANATOMY AND COURSE OF THE ABDUCENS NERVE

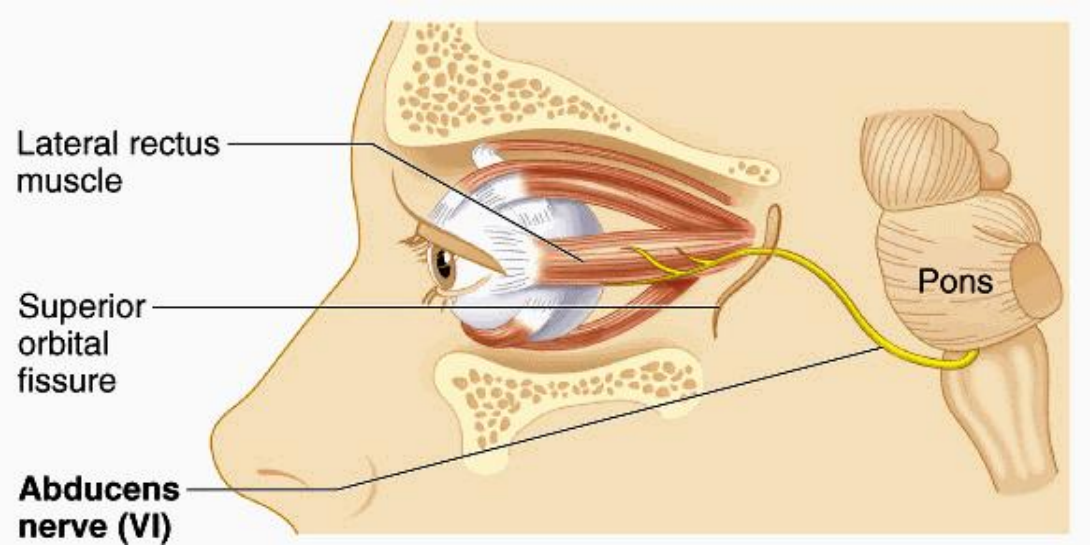


Fig 1: Picture depicting the anatomical course of the Abducens nerve

ANATOMY AND COURSE OF THE ABDUCENS NERVE

NUCLEUS

It consists of two types of multipolar cells – large and small. The large multipolar cells give rise to fibres of the abducent nerve, while the fibres of the small multipolar cells relay in the oculomotor nucleus via the medial longitudinal fasciculus.

SUPERFICIAL EMERGENCE

It emerges between the lower border of pons and lateral part of pyramid as seven or eight rootlets. They join at varying and greater distances from their emergence and some may separate until the nerve pierces the duramater.

COURSE

The nerve passes upwards and anterolaterally in the subarachnoid space of the posterior cranial fossa to pierce the arachnoid and duramater lateral to the dorsum sellae. It ascends between the layers of the duramater on the posterior surface of the petrous bone near its apex turning anteriorly to traverse the cavernous sinus. It enters the orbit through the superior orbital fissure within the annular tendon to supply the lateral rectus muscles

RELATIONS

At Nuclear level

The motor nucleus is small and situated beneath the floor of the upper part of the fourth ventricle. It lies close to the midline and beneath the facial colliculus. Since it belongs to the group of somatic efferent nuclei, it lies in line with nuclei of fourth and third nerves above and with the nucleus of hypoglossal nerve below.

At emergence

The abducent nerves are about 1cm apart, and between them is the basilar artery at its formation from the two vertebrals. Lateral to each abducent is the emergence of the facial nerve at the lateral side of the olive.

In the posterior cranial fossa

The flat, fasciculated nerve becomes rounder and firmer and sleeved by pia matter it ascends anterolaterally into the cistern pontis of the subarachnoid space between the pons and the occipital bone. A course of 15 mm takes it to its entry into the dura mater covering the basoccipital bone about 2cm inferolateral to the posterior clinoid process, and posteromedial to the inferior petrosal sinus in the petro basilar suture. The nerve pierces the dura opposite the occipital bone. At its emergence, it is crossed by the anterior inferior cerebellar artery which lies ventral usually but can be dorsal or pass between the rootlets. The oculomotor, trochlear and trigeminal nerves

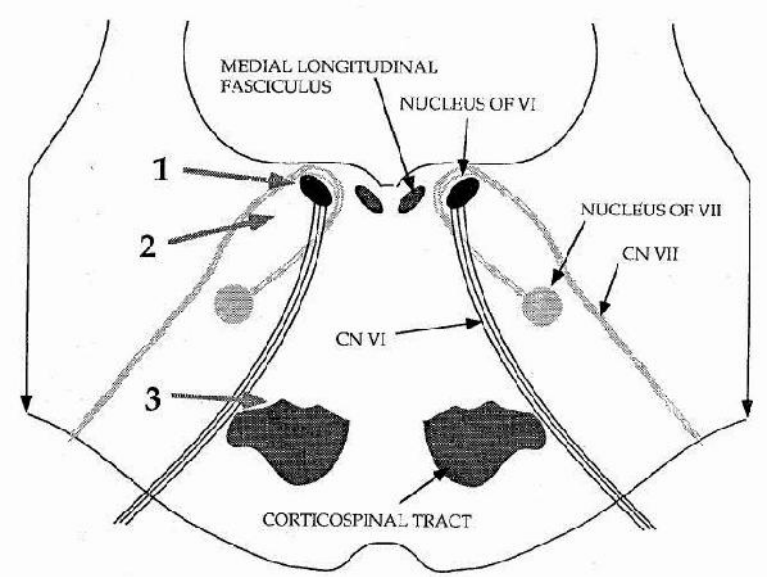


Fig 2: Picture representing the various relations of the Abducens nerve nucleus at the level of pons

1- Nucleus of Sixth nerve

2- Seventh nerve

3 – Pyramidal tract

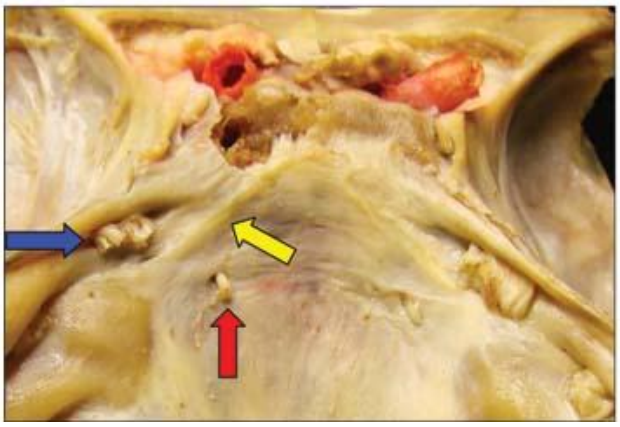


Fig 3: Picture of the posterior view of the petro – clival region. The yellow arrow indicates the Gruber's ligament. The blue arrow shows the trigeminal nerve and the red arrow points at the abducens nerve leading itself through the Dorello's channel to the cavernous sinus.

gradually approach the abducent as they pass towards the middle cranial fossa. The nerve passes inferior to the inferior petrosal sinus in an anterolateral direction, and ascends the petrous temporal near its apex. Here it lies in the Dorello's canal. At the upper border of the bone, it turns forward at right angle under the petrosphenoid ligament and superior petrosal sinus to enter the cavernous sinus with the inferior petrosal sinus by a common opening.

In the cavernous sinus

Here the nerve runs forwards. Posteriorly it spirals round the lateral aspect of the ascending part of the internal carotid artery. Beyond this the nerve lies inferotemporal to the horizontal portion of the artery with its sympathetic plexus which may communicate with the nerve.

In the superior orbital fissure

The abducens nerve traverses the fissure within the annulus of Zinn, at first below the divisions of the oculomotor nerve, then between them and lateral to the nasociliary nerve.

In the orbit

The nerve divides into three or four filaments which enter the ocular surface of the lateral rectus muscle behind its midpoint.

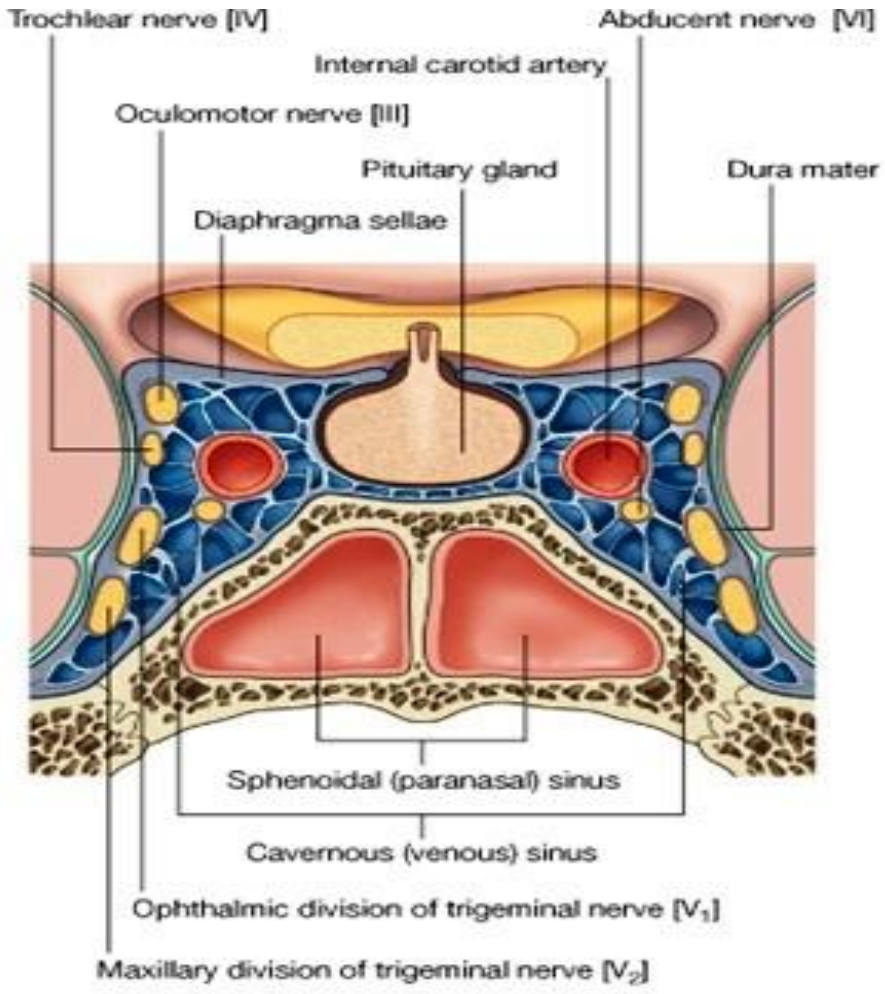


Fig 4: Diagrammatic representation of the relations of the sixth nerve in the Cavernous sinus

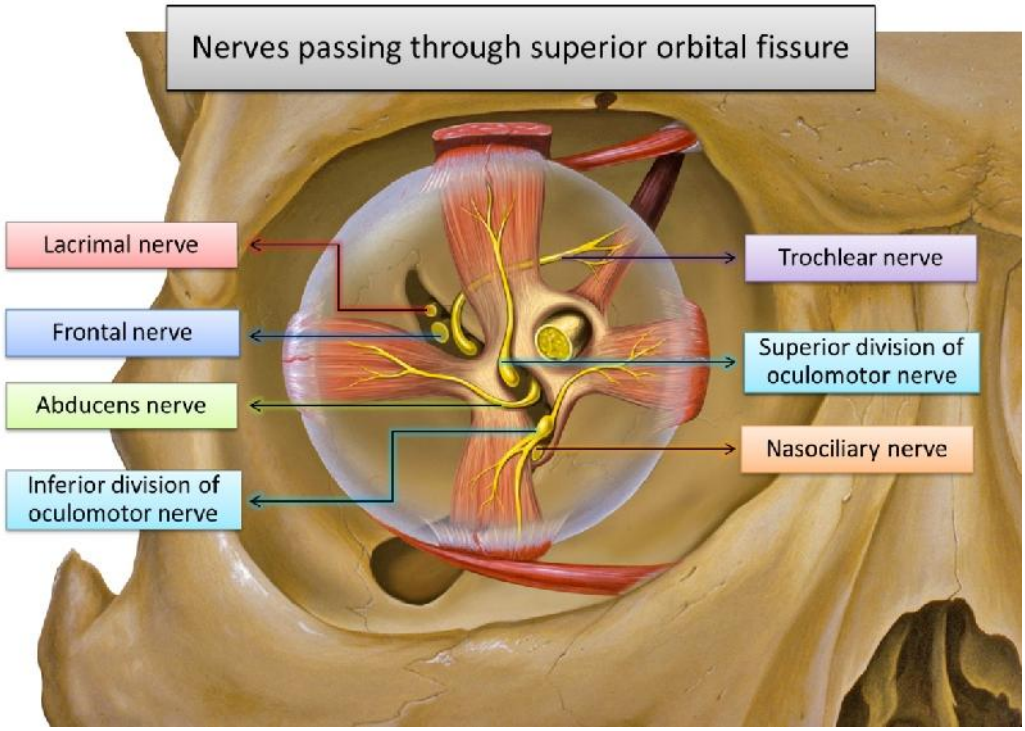


Fig 5: Picture showing the relations of the sixth nerve in the Superior orbital fissure

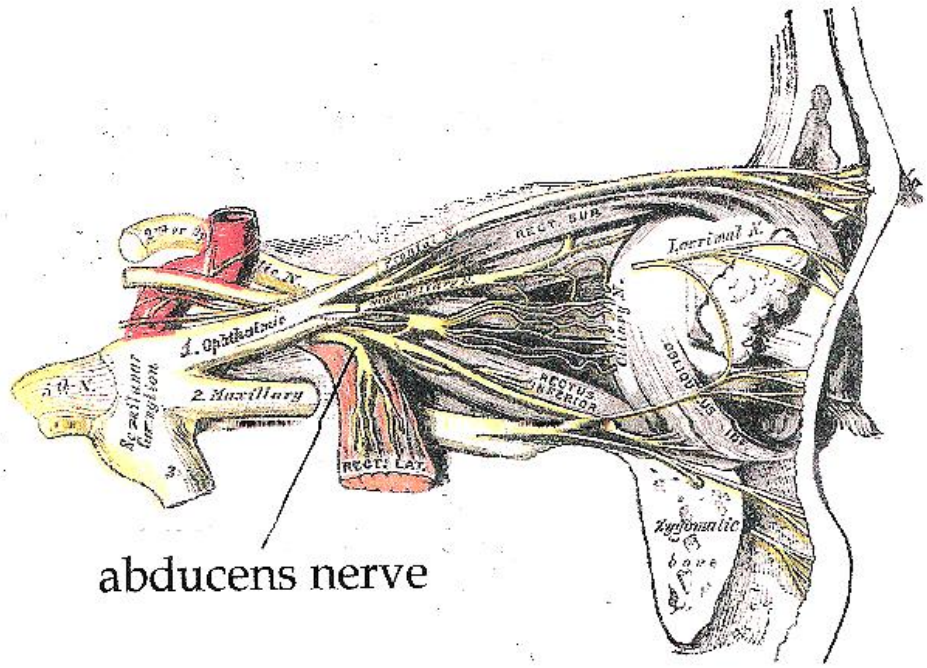


Fig 6: Picture showing the relations of the sixth nerve in the orbit

LESIONS OF THE ABDUCENT NERVE

APPLIED ANATOMY AND LESIONS AT VARIOUS LEVELS OF THE ABDUCENS NERVE

CONGENITAL

~ The abducens nerve can be affected due to birth trauma.

~ Mobius syndrome

~ Duane's Retraction Syndrome (Stilling Turk Duane Syndrome)[1,2]

ACQUIRED

Lesions of the Abducent Nerve Nucleus

The abducens nucleus contains motor neurons of the lateral rectus along with cell bodies of internuclear neurons that cross the midline and ascend to the contralateral medial longitudinal fasciculus to synapse in the ipsilateral medial rectus subnucleus. Thus lesions of the abducent nucleus cause ipsilateral **gaze palsy.** In most cases it is associated with ipsilateral facial nerve palsy, because the facial nerve fascicle loops around the abducent nucleus before exiting the brain stem. [1,2,3,4,7,8]

CAUSES:

~ Ischaemia

~ Infiltration

~ Trauma

~ Inflammation

~ Compression

~ Wernicke Korsakoff syndrome

Lesions of the Abducent Nerve Fascicle

1. Foville's Syndrome or Anterior inferior artery syndrome

    - Ipsilateral paralysis of abduction
    - Ipsilateral facial palsy
    - Loss of taste from anterior two – thirds of the tongue
    - Ipsilateral central Horner's Syndrome
    - Ipsilateral analgesia of the face
    - Ipsilateral peripheral deafness

2. Millard – Gubler Syndrome

    - Ipsilateral abducens palsy
    - Ipsilateral peripheral facial paralysis
    - Contralateral hemiplegia

3. Raymond – Cestan syndrome

    - Ipsilateral abducens palsy
    - Contralateral hemiplegia

CAUSES:

~ Ischaemia
~ Infiltration
~ Trauma
~ Inflammation – multiple sclerosis (most common)
~ Tumour Compression

Abducens palsy in association with Diabetes Mellitus is usually assumed to occur from involvement of the subarachnoid or cavernous sinus portion of the nerve.

Lesions of the Abducent Nerve in the subarachnoid space

The location and the long course of the nerve in the subarachnoid space explain its frequent involvement in raised intracranial pressure producing a false localizing sign.

1. Along the ventral surface of the pons, the nerve can be compressed by the anterior inferior cerebellar artery.
2. Compression by the posterior inferior cerebellar artery or the basilar artery when they are atherosclerotic or aneurysms.
3. After its exit from the pons, it overlies the clivus. Here it can be affected by posterior fossa lesions, like descent of brainstem associated with vertex blows, space occupying masses above the tentorium and structural abnormalities (e.g., Chiari malformation)
4. Trauma either direct neurosurgical or indirect from blunt closed globe injury can cause unilateral or bilateral abducens palsy.
5. Meningitis.
6. Basal tumours – meningioma, chordoma, acoustic neuroma, glioma, medulloblastoma, ependymoma, cavernous angioma, etc.

Lesions of the Extradural portion of Abducent Nerve at Petrous Apex.

1. In this region the nerve lies adjacent to mastoid air cells. In severe mastoiditis the inflammation may extend to the tip of petrous apex producing localized meningitis producing **Gradenigo's Syndrome** characterized by – paresis of abducens nerve, Gasserian ganglion inflammation – severe pain on ipsilateral side of face and facial paralysis.
2. Tumours and aneurysms of the intrapetrosal segment of internal carotid artery.
3. Lateral sinus thrombosis extending to inferior petrosal sinus.

Lesions of the Abducent Nerve in the Cavernous Sinus and Superior Orbital Fissure

Here abducens palsy occurs isolated or in combination with other cranial neuropathies and postganglionic Horner's syndrome.

CAUSES:

~ Tumours – meningioma, metastatic carcinoma, nasophyrangeal carcinoma, Burkitt's lymphoma, pituitary adenoma with/out apoplexy, craniopharyngioma, etc. Large tumours cause bilateral palsy.

~ Ischaemia – hypertension, diabetes mellitus, giant cell arteritis, systemic lupus erythematosus and migrane.

~ Granulomatous and non granulomatous inflammation.

~ Herpes Zoster infection

~ Primary and traumatic intracavernous aneurysms.

Lesions of the Abducent Nerve within the Orbit

The nerve has a very short course in the orbit, so isolated involvement in this region is rare like primary orbital schwannoma or accidental injection of anesthetic agent.

STAGES OF PARALYTIC LATERAL RECTUS MUSCLE

1. Paresis of ipsilateral lateral rectus.
2. Overaction of ipsilateral medial rectus.
3. Underaction of contralateral lateral rectus.

These stages do not follow a definite rule and may take weeks, to months, to several years.

In the first stage, the maximal deviation is in abduction of the affected eye.

In the second stage, as the medial rectus overacts, the deviation increases in the opposite field also.

In the third stage, the contralateral lateral rectus develops inhibitional palsy and the spread of incomitance is complete, with the deviations being same in all gazes and primary and secondary deviations also becoming equal.[4]

DIFFERENTIAL DIAGNOSIS

1. Thyroid eye disease – perform Orbital B SCAN and Thyroid Function Tests
2. Myasthenia gravis – serum assay for antiserum antibodies and single fiber electromyography.[4]

HISTORY AND EVALUATION

PATIENTS WITH ABDUCENS NERVE PALSY

Fig 7

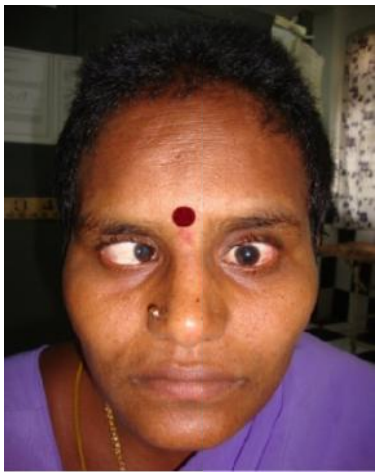


Fig 8

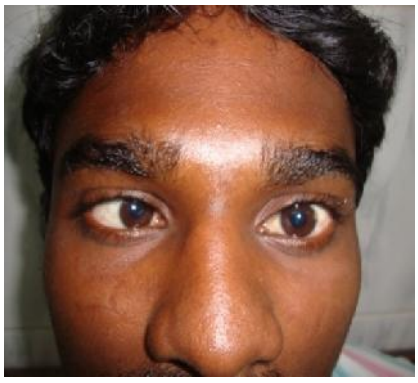


Fig 9

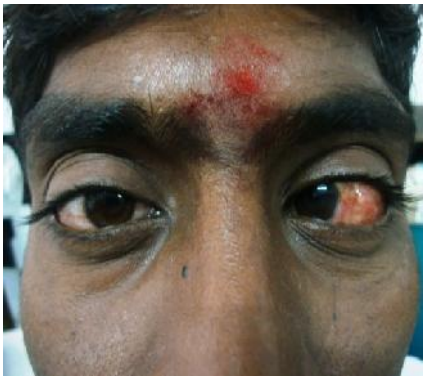


Fig 10

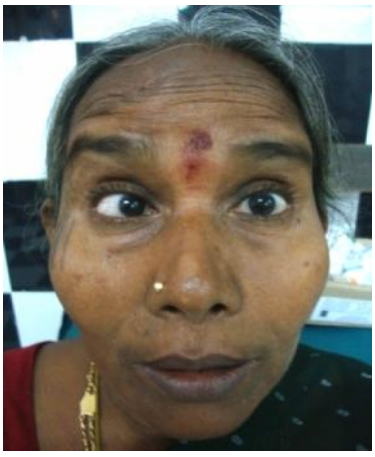


Fig 11

Fig 12

Fig 7 – 12: Clinical photographs of few patients with isolated lateral rectus palsy.

HISTORY AND EVALUATION

1. Symptoms:

~ Diplopia and lateral gaze i.e side of the paretic muscle, Face turn, Past pointing

2. History

~ Trauma, Diabetes Mellitus, Hypertension, Neurosurgical procedure, Viral illness, Headache and vomiting (signs of raised intracranial pressure), Previous ocular surgery, Similar episodes in the past

3. Clinical Features

~ Esotropia: measured objectively by Cover Test and subjectively by Maddox rod or any other method to dissociate the two eyes.

~ Limitation of abduction in the eye.

~ Secondary deviation > primary deviation (since the paretic eye requires more effort to come to primary position to take up fixation, this extra effort is passed on to the contralateral synergist, which is normal, increasing the ocular deviation.

~ **Diplopia charting**

Subjective deviation is recorded by asking the patient to quantify the separation between images which are dissociated with red and green goggles. It is done in a dark room. Patient is asked Armstrong goggles with red in front of right eye and green in front of left eye. A torch light with a

Fig 13: Clinical photograph of a patient with Red – Green goggles

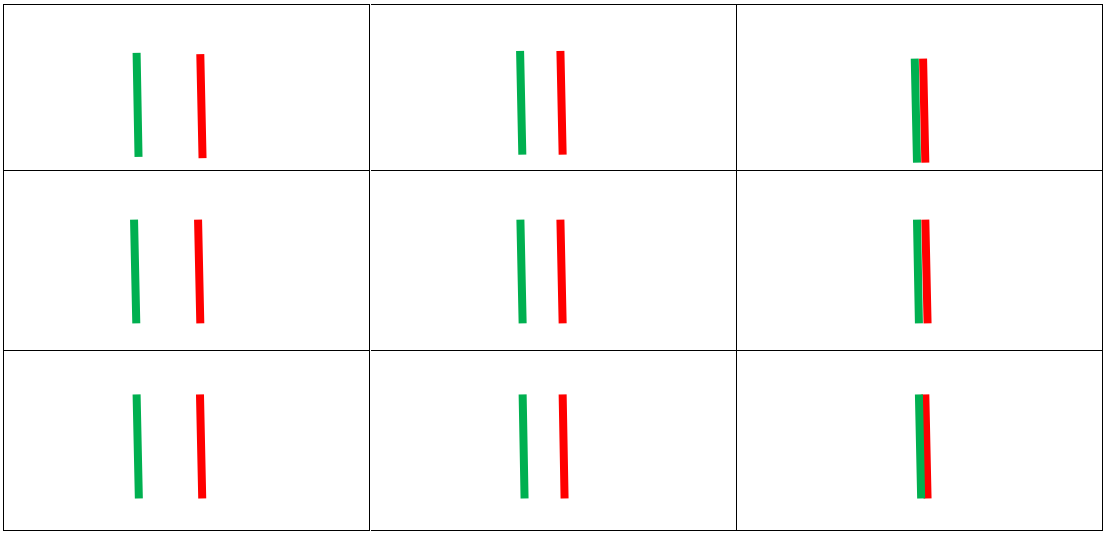


Fig 14: Diplopia chart of a patient with Left Lateral Rectus palsy.

stenopic slit is used. The patient is asked to look at a torch held 120 cm away and then the torch is moved to nine cardinal gaze positions. Patient is asked to describe the position of the images. The false image is usually the fainter and farther one. Any tilt of images and variation in the distance between images at various positions is asked for. In sixth nerve palsy the separation is maximal in abduction.

~ **Hess/ Lees' charting**

This uses red- green color dissociation, green in front of the testing eye. With the eye with red filter fixating, the projection of the other eye with the green filter is charted. This test charts the excursion of the other eye with the normal excursion of the "fixating eye" red filter. The fixating eye determines the innervational input and the excursion of the other eye if underacting would move less or more in case of over actions.

~ **Forced duction test** – to rule out restrictive component. A paralytic squint can develop restrictive element due to contracture of the ipsilateral antagonist (medial rectus in lateral rectus palsy) in long standing paresis. It is a passive test.

Active forced generation test – to determine and quantify the ability of the lateral rectus to contract. It also helps in distinguishing restrictive squint from a paralytic squint in which secondary restriction has developed. It is an active test.

Fig 15: Clinical photograph of a patient performing Hess charting

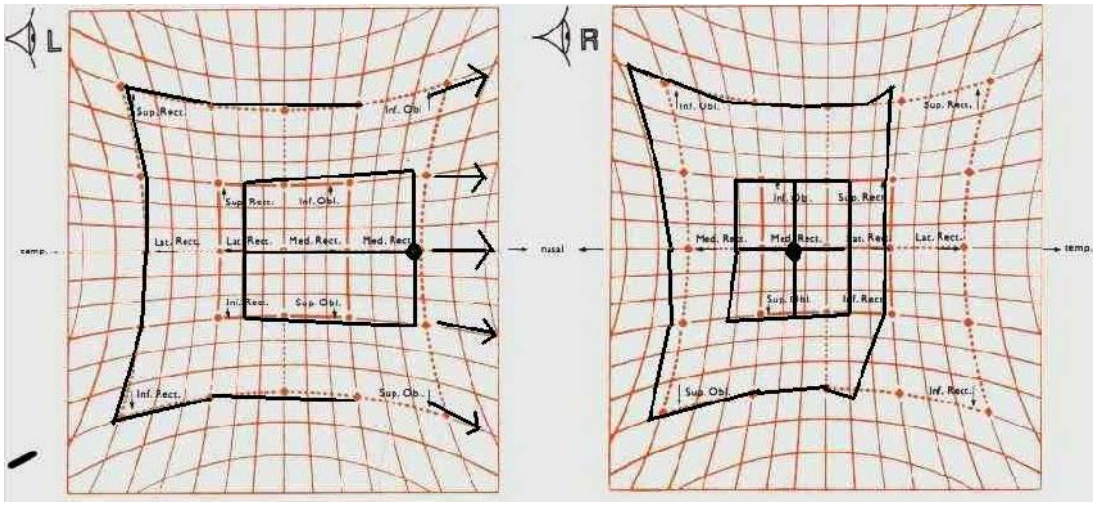
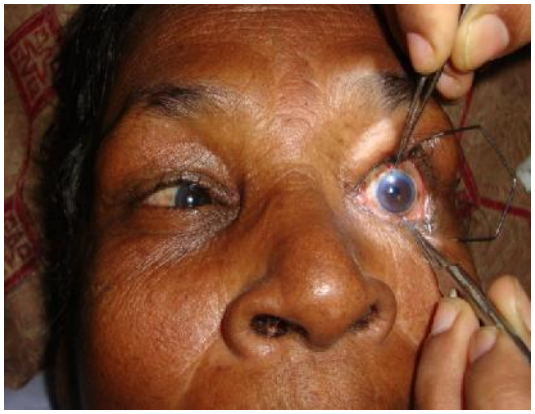


Fig 16: Photograph of the Hess Chart plotted by a patient with

right sided sixth nerve palsy



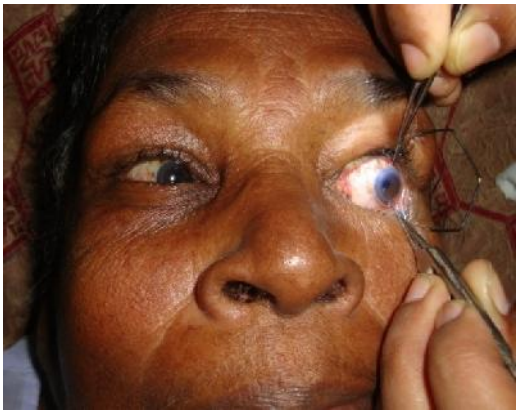


Fig 17 **Fig 18**

Fig 17- 18: Clinical photograph of a Forced Duction Test being performed for a patient with left sided lateral rectus palsy

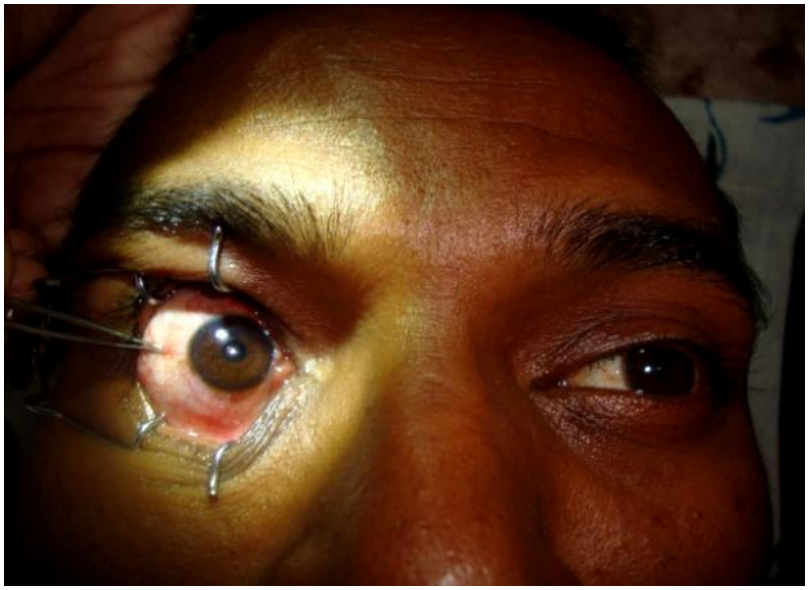


Fig 19: Clinical photograph of a Forced Generation Test being performed for a patient with right sided lateral rectus palsy

~ Measurement of intraocular pressure in various positions of gaze – when the muscle is acting against resistance more force has to be generated; also the restrictive (tight) muscle presses on the globe increasing the intraocular pressure.

~ Saccadic velocity recording

~ Electro – myography (EMG) – to distinguish myasthenia and myopathy from neurogenic palsy, and while injecting botulinum toxin.

OTHER INVESTIGATIONS

~ Dilated fundus examination – to look for papilledema and signs of diabetic or hypertensive changes.

~ Examination of other cranial nerves, integrity of oculosympathetic pathway, long tract and cerebellar signs.

~ Blood investigation - Complete haemogram

- Blood sugar levels

~ Neuroimaging - X-Ray orbit (to look for fractures in cases of trauma)

- CT Scan and MRI Brain (to rule out intracranial cause)

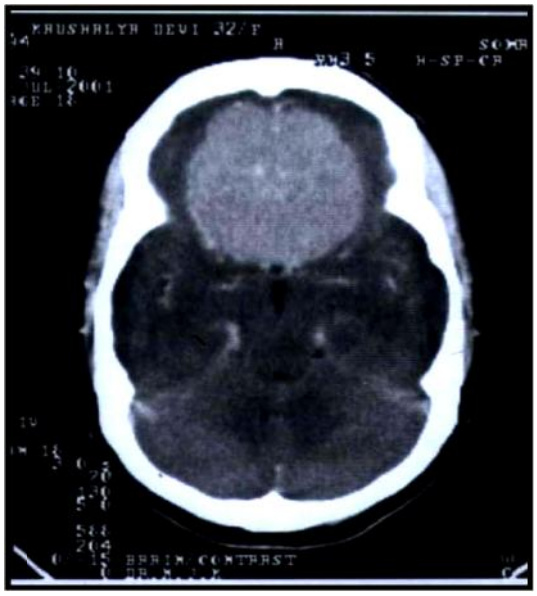


Fig 20: Radiographic photo of CT Scan - Brain of a patient with large space occupying lesion causing bilateral abducens palsy

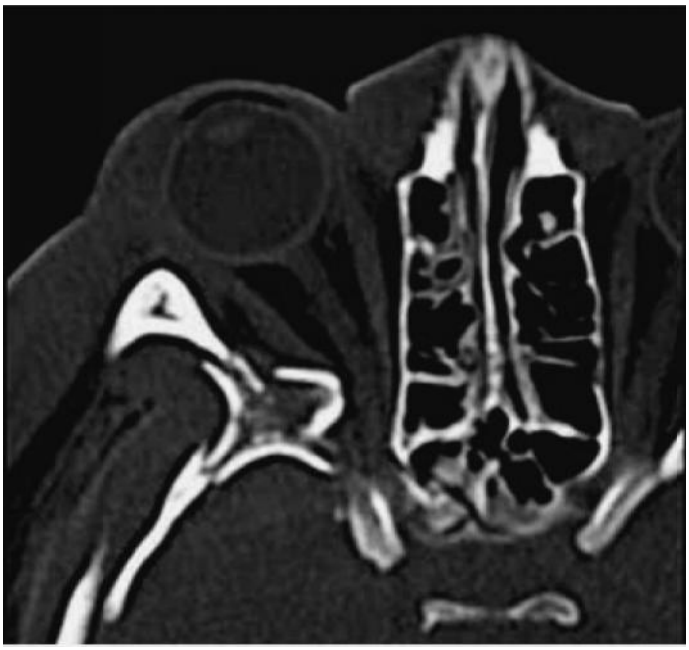
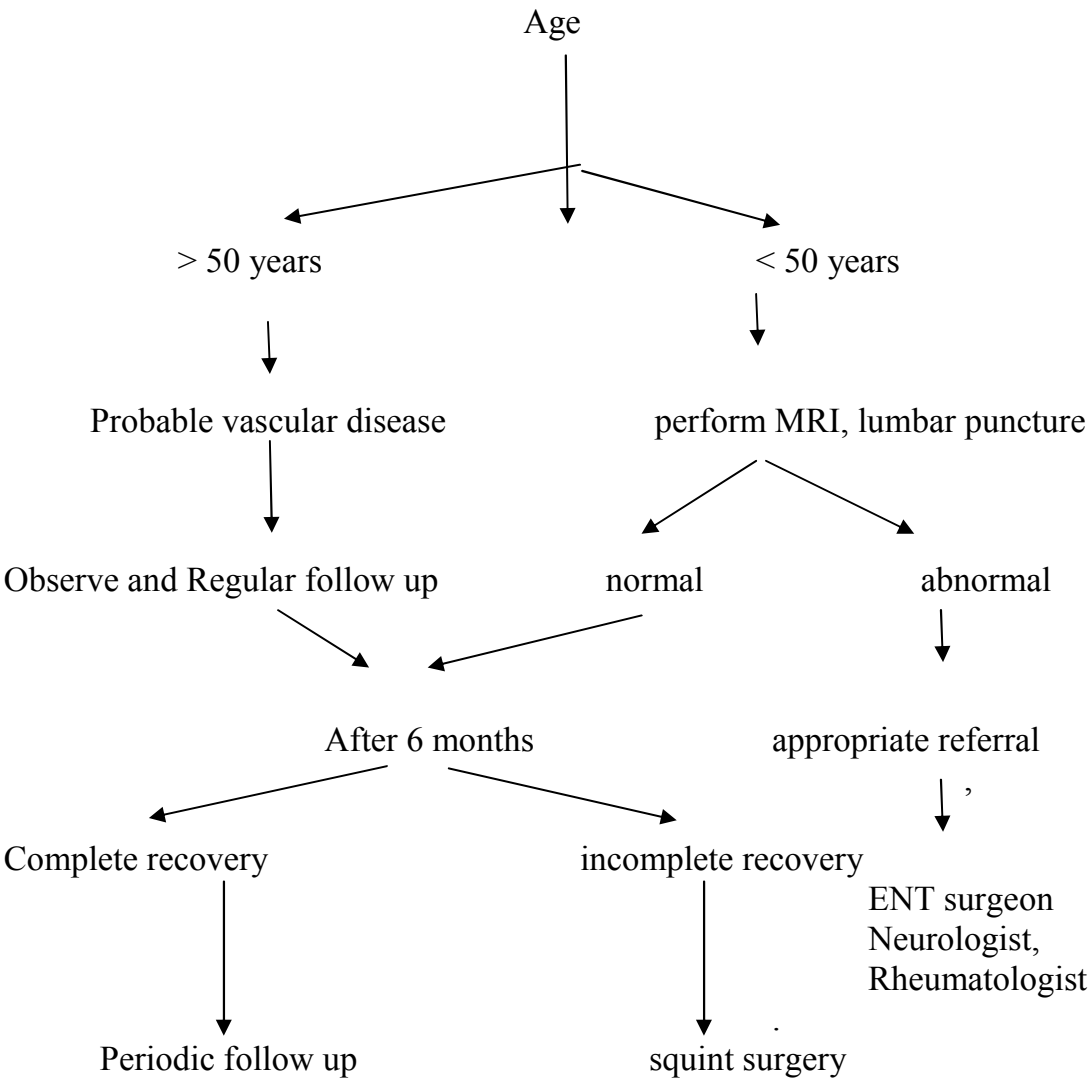


Fig 21: Radiographic photo of CT Scan – Orbit of a patient with right sided lateral wall of orbit fracture causing ipsilateral traumatic lateral rectus palsy

MANAGEMENT

MANAGEMENT

PROTOCOL FOR MANAGEMENT OF ISOLATED ABDUCENS PALSY[4,6]



While the patient is awaiting strabismus surgery,

~ Diplopia can be overcome either by occlusion of the affected eye with patch, or neutral density filters or opaque contact lenses or occlusive intraocular lenses larger than the pupil size.

~ Prisms can be tried in stable deviations.

~ Some patients are able to ignore their visual confusion by motor or sensory adaptation.

~ Patients under 8 years of age should undergo alternate occlusion of either eye to prevent amblyopia.

~ Chemodenervation of the antagonist muscle with botulinum toxin can be done in both acute and chronic nerve palsies.[6,11,14]

ROLE OF BOTULINUM TOXIN

1. Diagnostic: To assess the function of a palsied or a previously recessed muscle by weakening its ipsilateral antagonist, to know whether the reduced movement is due to over weakened muscle or contracture of contralateral synergist.
2. Therapeutic:
   ~ Recurrent injections of botulinum toxin improve the ocular deviation in patients for whom no further surgery is deemed possible, usually because of excessive scarring or general systemic contraindications for surgery. Injections are repeated every 4 – 6 months.

~ In association with muscle transposition surgery in order to weaken the ipsilateral medial rectus without interfering with its blood supply..

~ To reduce symptoms of an overacting ipsilateral medial rectus while awaiting spontaneous recovery from the nerve palsy, when the cause is known, as the toxin might mask the development of fresh neurological signs.[6,12,13,15,16,17,18]

METHOD OF INJECTION

Dilution of the botulinum toxin: Reconstitute the 100 I.U. vial of Botox with 4ml of non preserved saline. Do not shake the bottle after reconstitution. 0.1 ml containing 2.5 units of toxin is injected per muscle.

Technique:

~ After asking the patient to abduct the eye, the needle is inserted subconjunctivally over the approximate area of insertion of medial rectus.

~ The needle is directed posteriorly, in a direction parallel to the medial wall of the orbit towards the apex of the orbit.

~ If EMG is not available, injections can be given directly into the muscle belly after peritomy or by holding the muscle with Dastoor's forceps.

~ 0.1 ml botulinum toxin is injected at this point.

Complications:

Subconjunctival hemorrhage, headache, transient ptosis, unwanted deviation and perforation of the globe.

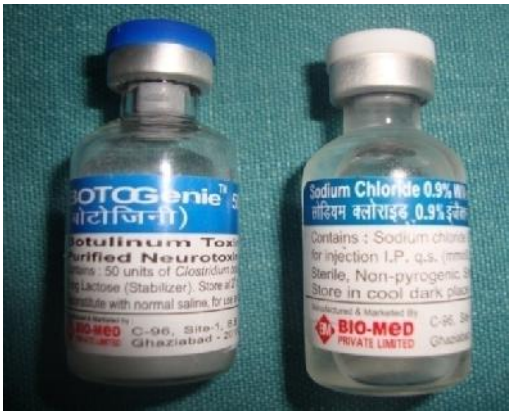


Fig 22: Photograph of a vial of Botulinum toxin (left) and diluent – Sodium chloride (right)

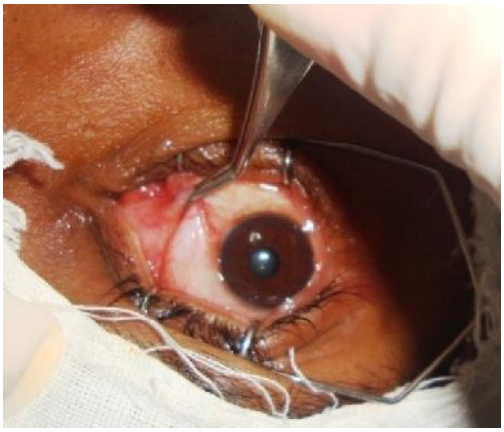


Fig 23:
Clinical photograph taken while holding the medial rectus with Dastoor's forceps

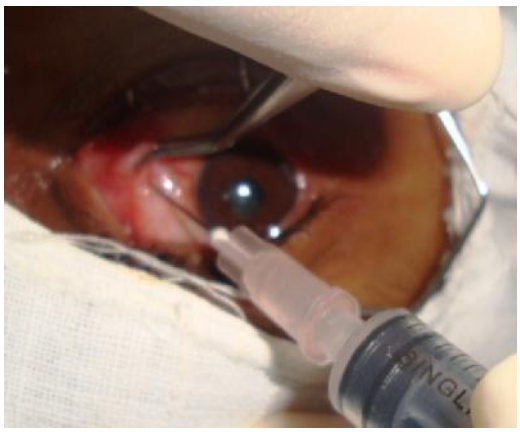


Fig 24:
Clinical photograph taken while injecting 3 – 5 I.U Botulinum toxin into the medial rectus with 26 G needle.

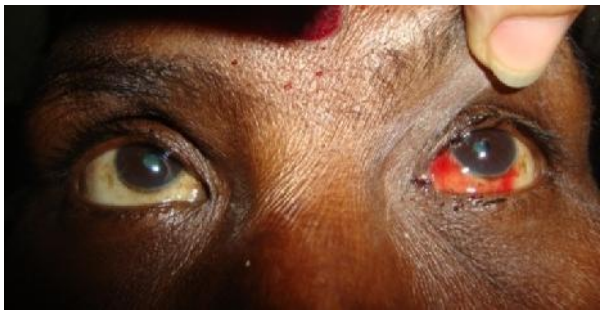


Fig 25: Clinical photograph of a complication of Botulinum toxin injection – sub conjunctival hemorrhage.

SURGERY ON THE RECTUS MUSCLE

Standard medial rectus recession

The medial rectus is detached from the globe and replaced further from the limbus. This shortens the distance between the origin and insertion of the muscle and therefore has a weakening effect.

METHOD:

1. The eye is draped, speculum inserted. Two traction sutures are placed through the conjunctiva and episclera at the 12 and 6 o' clock positions at the limbus.
2. A conjunctival peritomy is made around the limbus in the region of the muscle for 110 deg (3 clock hours). From the edge of this a radial incision is made into the conjunctiva – superiomedially. The interpalpebral conjunctiva is avoided while making the radial incision.
3. The subconjunctival space is dissected down on either side of the muscle using Wescott scissors in a spreading fashion. Dissection is not done directly over the muscle as this tends to bleed.
4. A squint hook is passed into this area and the muscle is hooked.
5. The Tenon's capsule is cleaned from the muscle, using blunt dissection if possible.
6. The squint hook is replaced with a Chavasse hook, which spreads the muscle.

RECESSION OF MEDIAL RECTUS

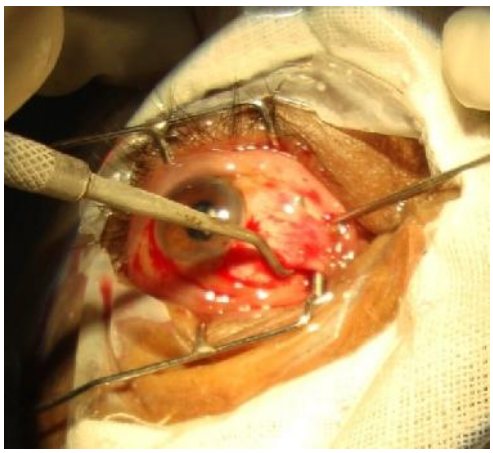


Fig 26: Per operative photograph showing isolation of Medial Rectus using two muscle hooks

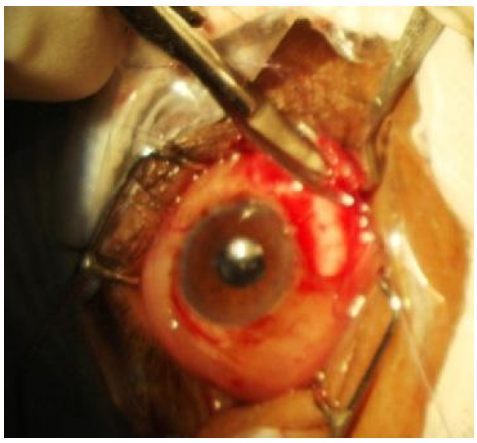


Fig 27: Per operative photograph showing application of stay sutures with 6 – 0 vicryl on either ends of the muscle width, the MR is then disinserted from its insertion

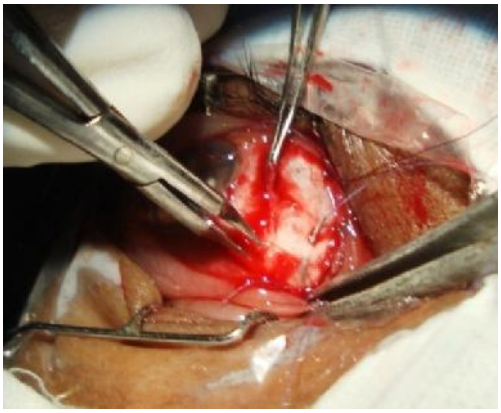


Fig 28: Per operative photograph while securing the insertion of MR few millimeters behind its original insertion – RECESSION

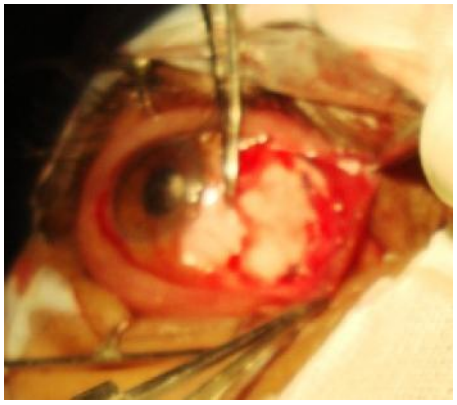


Fig 29: Per operative photograph while confirming attachment of the muscle and absence of any adhesions

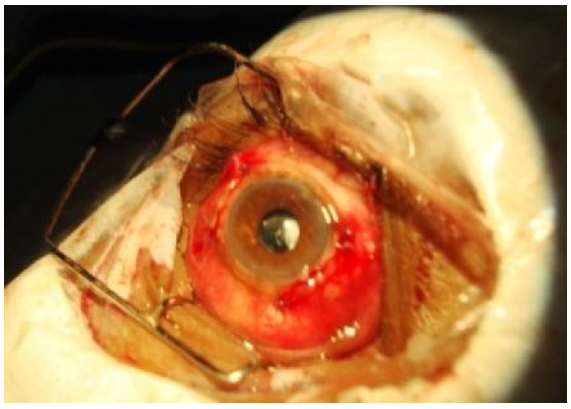


Fig 30: Per operative photograph showing closure of peritomy.

7. A 6/0 absorbable suture is applied into each outer third of the muscle at its insertion, by passing two throws of the suture – one partial thickness and one full thickness.
8. The muscle is disinserted from the sclera using Wescott scissors, carefully preserving the suture.
9. The distance that the muscle has to be recessed is measured using calipers.
10. The suture is then anchored to the sclera at the desired distance from the insertion.
11. When tying the suture, the end of the suture is held with the needle on it and is kept taut. The other end of the suture is run down this taut end. This prevents snagging of the knot and holds the eye in optimum position to prevent adhesion of the Tenon's capsule and the conjunctiva.
12. The conjunctiva is closed using 8/0 absorbable sutures and an injection of local anesthetic (Bupivicaine 0.1 ml) is given subconjunctivally over the muscle.[6,11,19,20,22,23,24]

Resection of lateral rectus

The calculated length of the muscle is excised; which shortens it thereby strengthening the lateral rectus muscle.

METHOD:

1. Steps 1 to 6 are same as that for medial rectus.
2. The length of the lateral rectus muscle to be resected is measured using calipers. 6/0 absorbable suture is placed into each outer third of the muscle at its insertion, by passing two throws of the suture – one partial thickness and one full thickness.
3. A straight artery forceps is applied across the muscle, proximal to the sutures and the muscle is gently crushed. Gentle diathermy is applied across the crushed area.
4. The muscle is cut across at this site using Wescott scissors, taking small bites at least 1mm proximal to the sutures.
5. The muscle is held by the sutures, pulled towards the insertion and inspected to ensure that it is not twisted.
6. The suture on the lower third of the muscle is passed through the lower end of the previous muscle insertion. The same is done with the upper suture so ensuring that the muscle is spread in full width.
7. The muscle is inspected. If the centre is hanging back from the insertion, an extra suture is applied through this and tied up to the insertion.
8. The conjunctiva is closed using 8/0 absorbable sutures and an injection of local anesthetic (Bupivicaine 0.1 ml) is given subconjunctivally over the muscle. [6,11,19,20,22,23,24]

9. RESECTION OF LATERAL RECTUS

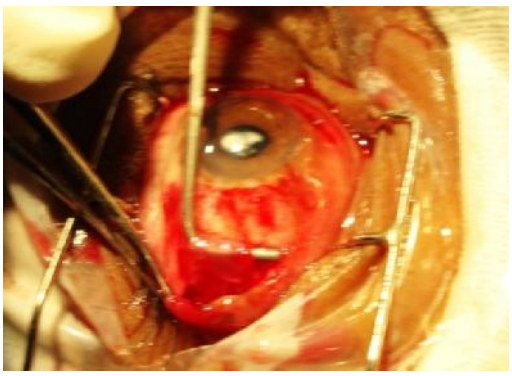


Fig 31: Per operative photograph showing isolation of LR using muscle hook.

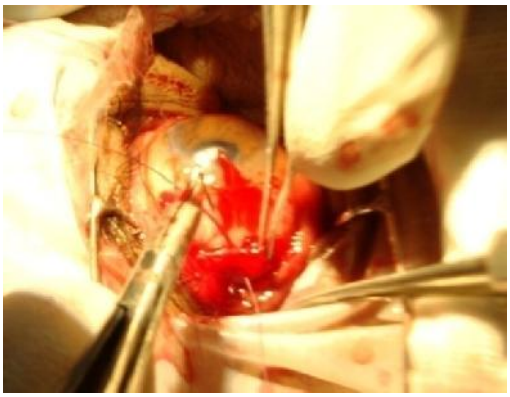


Fig 32: Per operative photograph while applying stay sutures with 6 – 0 vicryl few millimeters behind the insertion.

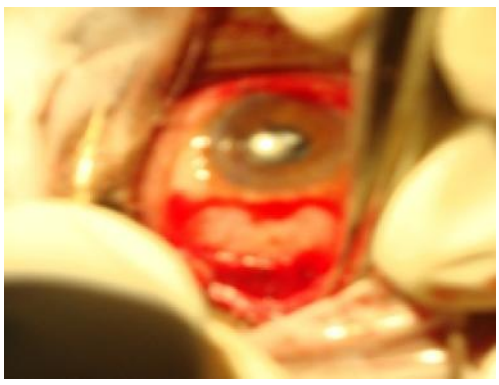


Fig 33: Per operative photograph after disinsertion of LR. The stay sutures are used to attach the muscle at the original insertion, and the excess muscle is cut off.

Transposition surgery

Superior and inferior rectus muscles are transposed away from their usual anatomic position to allow them to take up some action of the paralysed lateral rectus muscle. At least three months should be allowed between previous squint surgery and a transposition surgery to allow the posterior ciliary arteries to take up their function.

METHOD:

1. After applying stay sutures on the medial side, two radial relieving incisions are made on each side of lateral rectus and generous limbal peritomies are performed so as to gain access to the superior and inferior rectus.
2. The two vertical rectii are dissected out and hooked in turn, split vertically into equal halves and sutures are applied to the insertion as in recession surgery.
3. In the Hummelsheim procedure, each half of the muscle is moved towards the lateral rectus. The edges of the muscle are sutured to the side of lateral rectus muscle insertion. The remaining half the muscle is retained at its original insertion.
4. In the Jensen procedure, the bellies of the superior and inferior rectus muscles are split along their length and attached to the belly of the lateral rectus muscle – which has also been split lengthwise – to produce some abduction.

5. The conjunctiva is closed with buried 8/0 absorbable sutures and local anesthetic is injected subconjunctivally. [22,23,24]

Postoperative complications

1. Anterior segment ischemia.
2. Development of a secondary deviation. Unwanted vertical squint may develop.
3. Poor globe movement due to scarring – especially in cases who have had previous surgeries.

Botulinum Toxin + Vertical transposition

The medial rectus is weakened by injection of botulinum toxin and a full transposition of the superior and inferior recti laterally is carried out to replace the function of the paralysed lateral rectus. The anterior ciliary arteries of the lateral and medial recti are preserved, thereby reducing the risk of anterior segment ischaemia. It is indicated in totally unrecovered lateral rectus palsy.

VERTICAL MUSCLE TRANSPOSITION

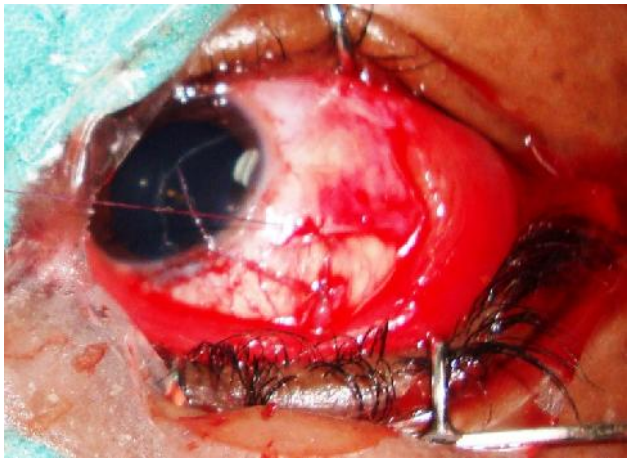


Fig 34: Per operative photograph showing attachment of Superior rectus to Lateral rectus

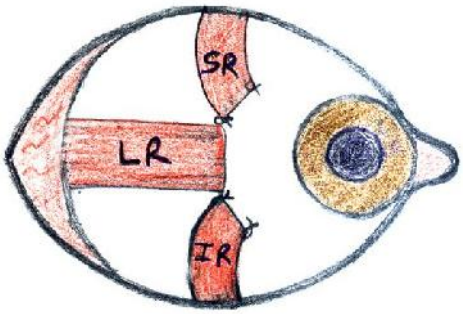


Fig 34: Diagrammatic representation showing attachment of Superior and Inferior rectus to Lateral rectus

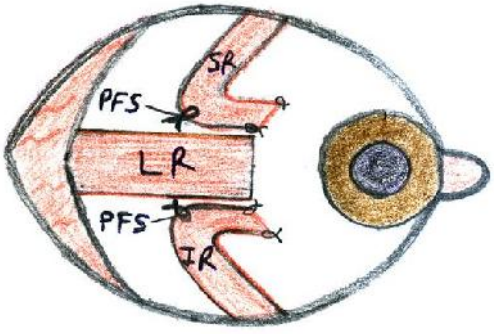
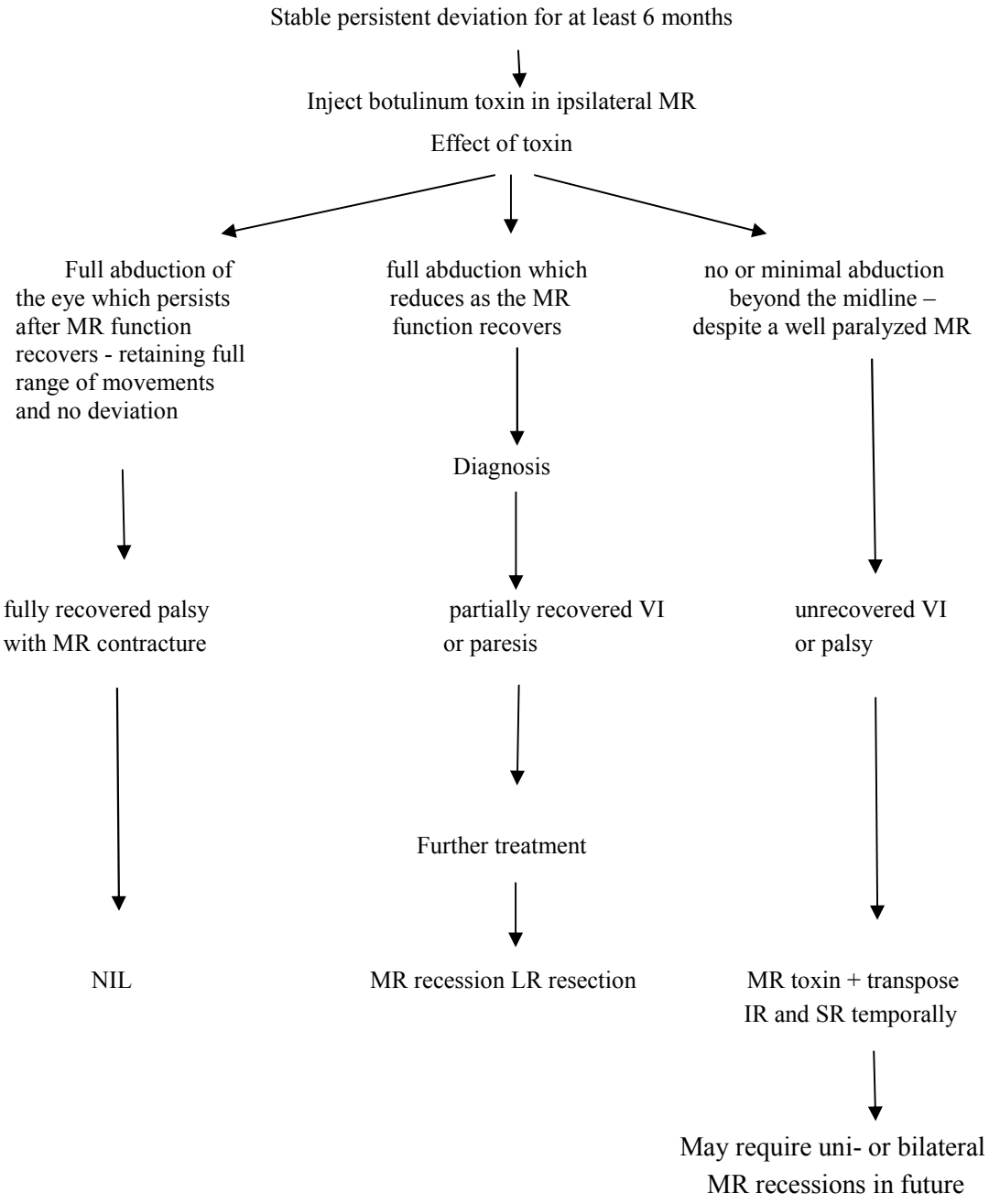


Fig 35: Diagrammatic representation showing vertical muscle transposition with augmentation

Algorithm for using Botulinum toxin in lateral rectus palsy



PRE OPERATIVE

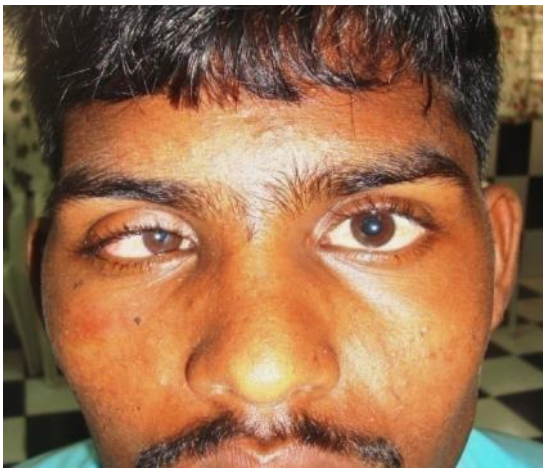


Fig 36: Clinical photograph of a patient with Right sided traumatic lateral rectus palsy

RE VERTICAL TRANSPOSITION WITH BOTULINUM INJECTION POST OPERATIVE

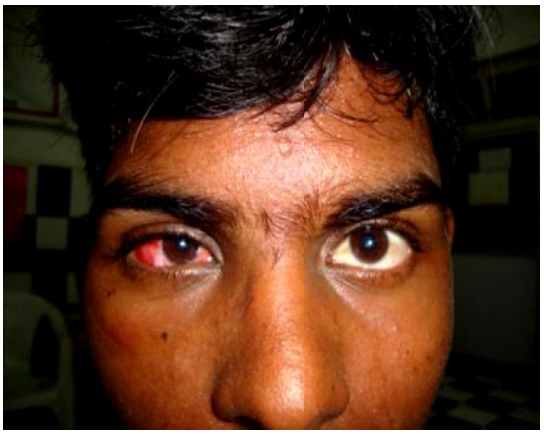


Fig 37: Clinical photograph of the same patient the same patient on the first post operative day.

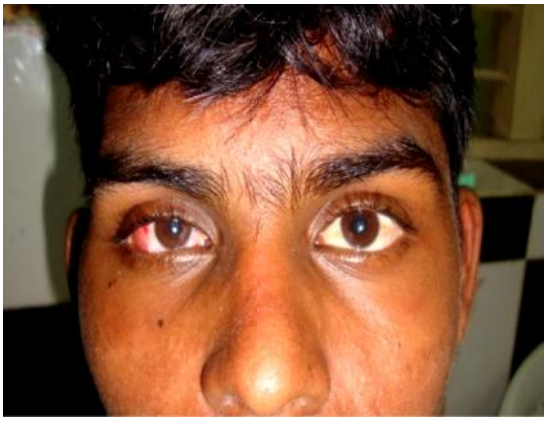


Fig 38: Clinical photograph of four weeks later.

PRE OPERATIVE

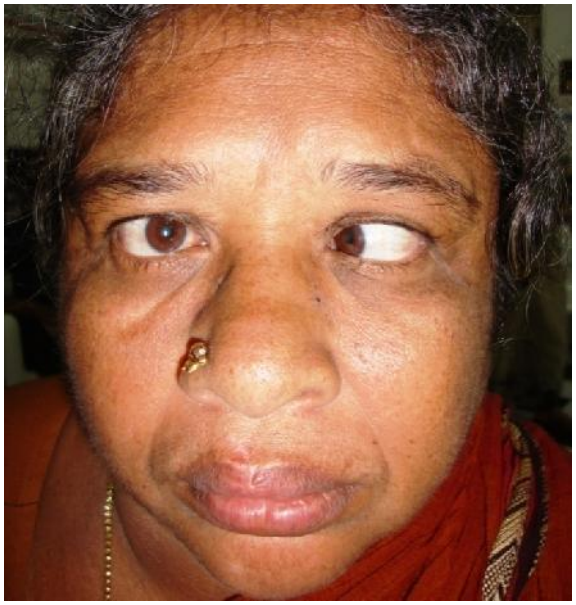


Fig 39: Clinical photograph of a patient with bilateral ischemic lateral rectus palsy

BILATERAL LATERAL RECTUS RESECTION AND MEDIAL RECTUS RECESSION POST OPERATIVE

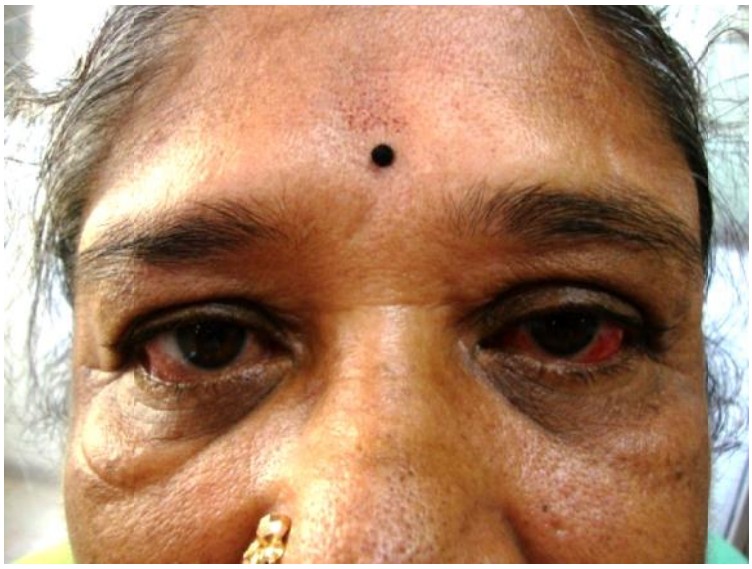


Fig 40: Clinical photograph of the same patient 4 weeks after surgery

PART - II

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

Primary Objectives:

1. To determine the etiology of sixth cranial nerve palsy.

2. To analyze the clinical course of the disease.

3. To alleviate diplopia.

4. To regain full range of binocular extra ocular movements.

5. To align eyes in primary gaze surgically.

Secondary Objective:

To improve the cosmetic appearance of the patients.

MATERIALS AND METHODS

MATERIALS AND METHODS

Subject Selection : 57 eyes of 50 patients attending Ophthalmology OPD diagnosed with isolated 6^{th} cranial nerve palsy during two year period. (June 2009 and June 2011).

Inclusion Criteria :

1. All patients with diplopia due to lateral rectus palsy.
2. Patients of all age groups

Exclusion Criteria :

1. Associated neurological manifestations
2. Duane's retraction syndrome
3. Restrictive causes of esodeviation
4. Sensory esotropia

REGISTRATION

NAME: AGE: SEX: M/F

OCCUPATION: ADDRESS:

EYE INVOLVED: RE/ LE

HISTORY OF THE PRESENTING ILLNESS

The common complaints were,:

1. Double vision – whether uniocular/binocular, constant intermittent, fluctuating or not, more for near or distance, whether images were horizontally or vertically separated, whether it is increased on any particular direction.

2. Pain – headache/ periorbital pain, location, nature, any radiation, aggravating and relieving factors any association with nausea / vomiting.

3. Defective vision – apart from double vision, any blurring or inability to see.

4. Deviation of eyeball – right/left eye, duration

5. Abnormal head posture.

Details of the progress from onset, the treatment undergone to the present state were noted.

PAST HISTORY

H/o systemic diseases like diabetes mellitus, hypertension, connective tissue disorder, tuberculosis, syphilis, AIDS, malignancy in the present or past, H/o migraine or neurologic disease, exanthematous fever and vaccination.

PERSONAL HISTORY

Smoking, alcoholism, type of diet.

GENERAL EXAMINATION

General vital data like pulse, blood pressure, peripheral pulses were noted

OCULAR EXAMINATION

1. Head position – head tilt, face turn, chin elevation/depression were noted
2. Any skull abnormalities
3. Eyelids – ptosis / retraction / lid lag / Marcus Gunn jaw winking phenomenon / Duane's retraction syndrome were noted

4. Extra ocular movements were noted both ductios and versions.

5. Pupil size, shape and reaction noted

6. Anterior segment examined in detail with slit lamp.

7. A dilated fundus and refraction was done.

8. Colour vision, visual field testing and intra ocular pressure measurement were done for all patients

9. Diplopia charting, Hess charting and orthoptic evaluation

10. Forced duction and forced generation test were done for all patients.

NEUROLOGIC EXAMINATION

Examination of other cranial nerves. Motor, sensory and cerebellar symptoms and signs noted.

INVESTIGATIONS:

BP, COMPLETE HAEMOGRAM, FBS/ PP

URINE ROUTINE, MANTOUX, VDRL

RADIOLOGICAL STUDY:

X-RAY ORBIT, SKULL – AP/ LATERAL and PNS,

CT SCAN and MRI (in indicated cases)

REFERRAL TO OTHER SPECIALITIES (in indicated cases)

DIABETOLOGIST, NEUROLOGIST, ENT SURGEON, etc.

FOLLOW UP

Recording the patients complaints whether stable/ improving / worsening

- ~ Vision with pin hole
- ~ Extra Ocular Movements
- ~ Cosmetic appearance
- ~ Diplopia charting

RESULTS

RESULTS

57 eyes of 50 patients with isolated sixth cranial nerve palsy were studied. A Prospective, Interventional study was conducted.

1. AGE DISTRIBUTION

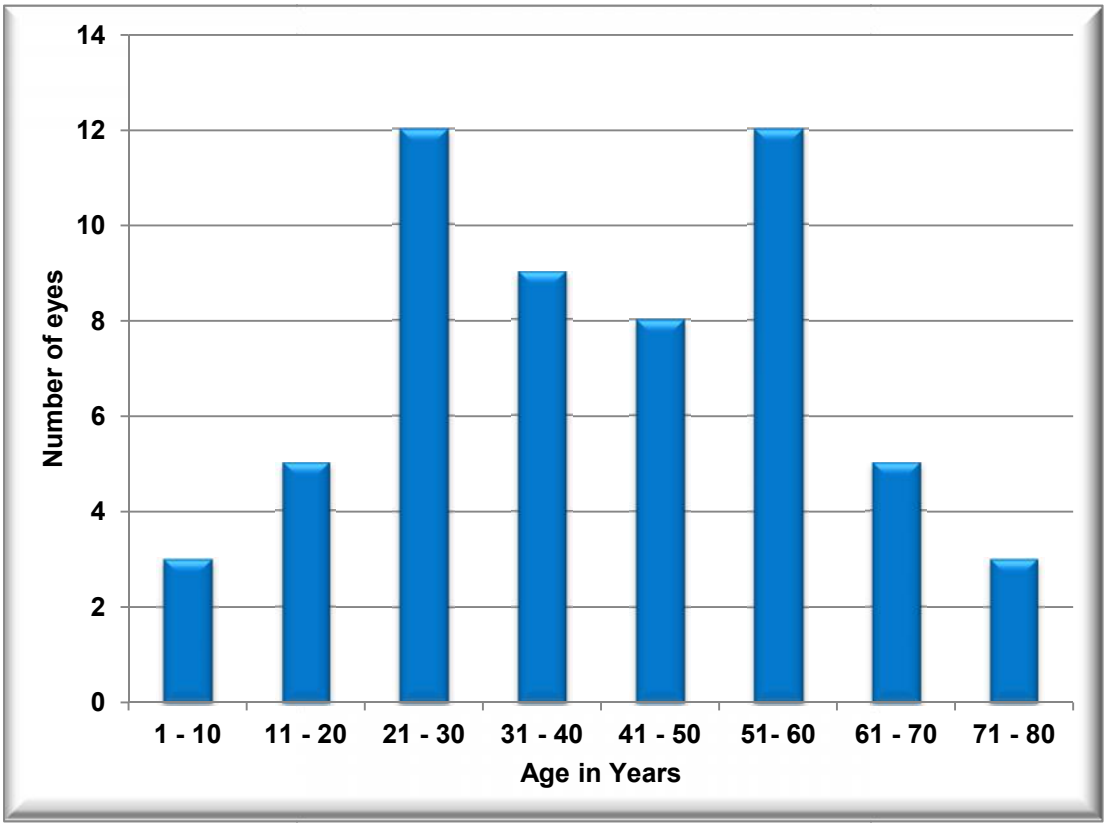
The following table shows the age distribution in the patients with sixth cranial nerve palsy.

TABLE – 1

Age group	Patients	%
1 – 10	3	5.3
11 – 20	5	8.8
21 – 30	**12**	**21.1**
31 – 40	9	15.8
41 – 50	8	14.0
51 – 60	**12**	**21.1**
61 – 70	5	8.8
71 – 80	3	5.3
TOTAL	**57**	**100.0**

GRAPH -1

AGE DISTRIBUTION



In our study of 57 eyes with isolated sixth cranial nerve palsy, the maximum number of eyes (12) belonged to the age group of 51 – 60 and 21 – 30 years (21.1%) each. 9 eyes were from the age group 31- 40 years forming 15.8 % of the total eyes , followed by 8 eyes in the age group 41 – 50 years (14%). 5 eyes belonged to age groups from 11 – 20 years and 61 – 70 years each contributing to 8.8 %. 3 eyes were from the age groups 1 – 10 years and 71 – 80 years (5.3 %).

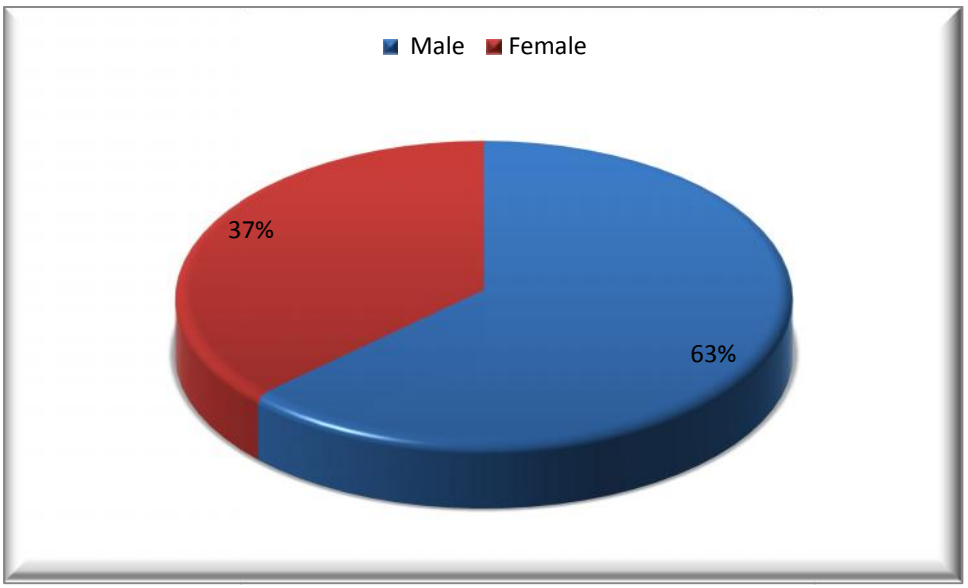
2. SEX DISTRIBUTION

TABLE – II

	Frequency	%
Male	36	63.2
Female	21	36.8
Total	**57**	**100.0**

In our study there was a significant gender difference, with 36 eyes from males out numbering 21 eyes from females – 63.2 % males against 36.8 % females.

GRAPH – 2: SEX DISTRIBURION



3. LATERALITY

TABLE – III

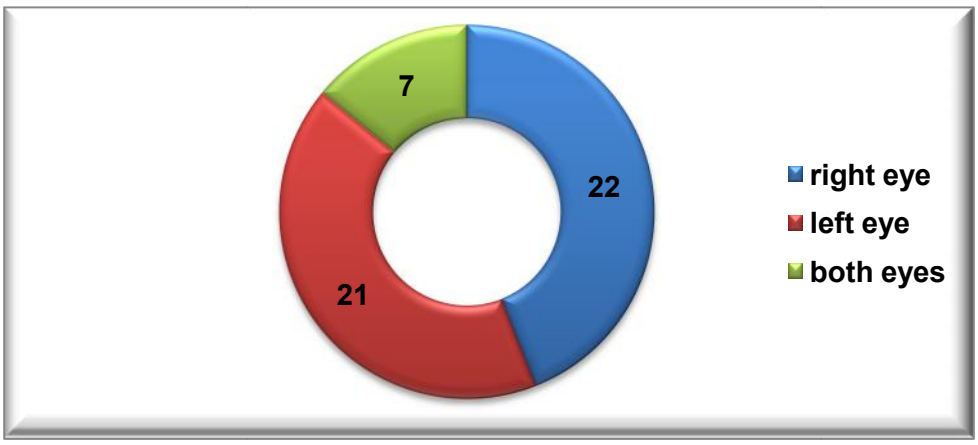
Right eye	Left eye	Both eyes	Total
22	21	7	**50**

TABLE – IV

	Frequency	%
LE	28	49.1
RE	29	50.9
Total	**57**	**100.0**

Both eyes were almost equally affected with a slight predominance of RE, 22 patients over LE, 21 patients by 2 %. In 7 patients, which to contributed 14 % of all individuals, both eyes were affected.

GRAPH – 3: LATERALITY



4. ETIOLOGY

TABLE – V

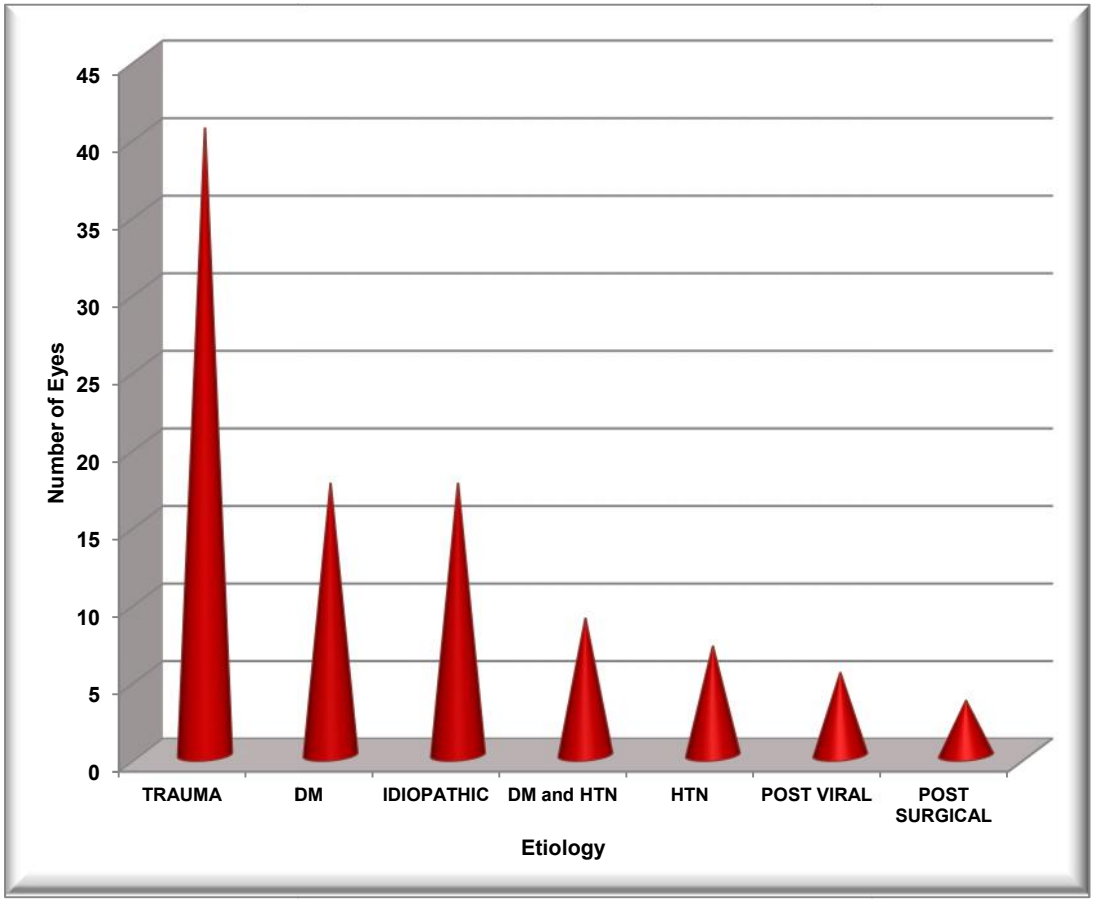
Etiology	Frequency	%
Trauma	**23**	**40.4**
DM	10	17.5
Idiopathic	10	17.5
DM and HTN	5	8.8
HTN	4	7.0
Post Viral	3	5.3
Post Surgical	2	3.5
Total	**57**	**100.0**

In our study, 23 (40.4 %) eyes presented with isolated sixth nerve palsy in patients with a history of head trauma or closed globe injury. 19 eyes (35.3 %) were diagnosed with isolated sixth nerve palsy due to ischemic pathology. Of these diabetes mellitus was found in 10 (17.5 %), hypertension in 4 (7 %) and both DM and HTN in 5 (8.8 %). 3 eyes (5.3%) were from patients with history of fever in the week preceding the symptoms of diplopia. 2 eyes (3.5 %) were from patients who had undergone mastoidectomy, following which they developed acute sixth nerve palsy (post surgical).

For 10 eyes (17.5 %) the cause of isolated sixth nerve palsy could not be determined and was presumed to be due to several causes like; ear infections, migraine, post ictal and idiopathic.

The duration of disease in the diabetic group was less than 5 years in 50 % (7 of 14) of the patients, between 5 – 10 years in 28.57 % of patients (4 of 14) and more than 20 years in 21.43 % of all patients (3 of 14).75 % of all hypertensives (6 out of 8) were diagnosed in the last 5 years only.

GRAPH – 4: ETIOLOGY



28.57 % of all diabetics (4 patients) were diagnosed for the first time with the disease only after investigating at our institution after presentation with sixth nerve palsy. 42.86 % of the Diabetics (6 patients) had uncontrolled blood sugar levels i.e post prandial Blood Sugar levels more than 200 mg/dl at the time of their presentation. 21.43 % of the diabetics (3 patients) were under treatment with insulin therapy.

28.57 % of all diabetics (4 patients) had concurrent diabetic retinopathy. All the patients belonged to the ETDRS classification of Non Proliferative Diabetic Retinopathy. The remaining 71.43 % of the diabetics (10 patients) had normal fundus picture.

26.32 % of the patients (5 out of 19) with history of trauma had concurrent bony fractures, 4 of which had lateral wall of orbit involvement and one had zygomatic bone fracture.

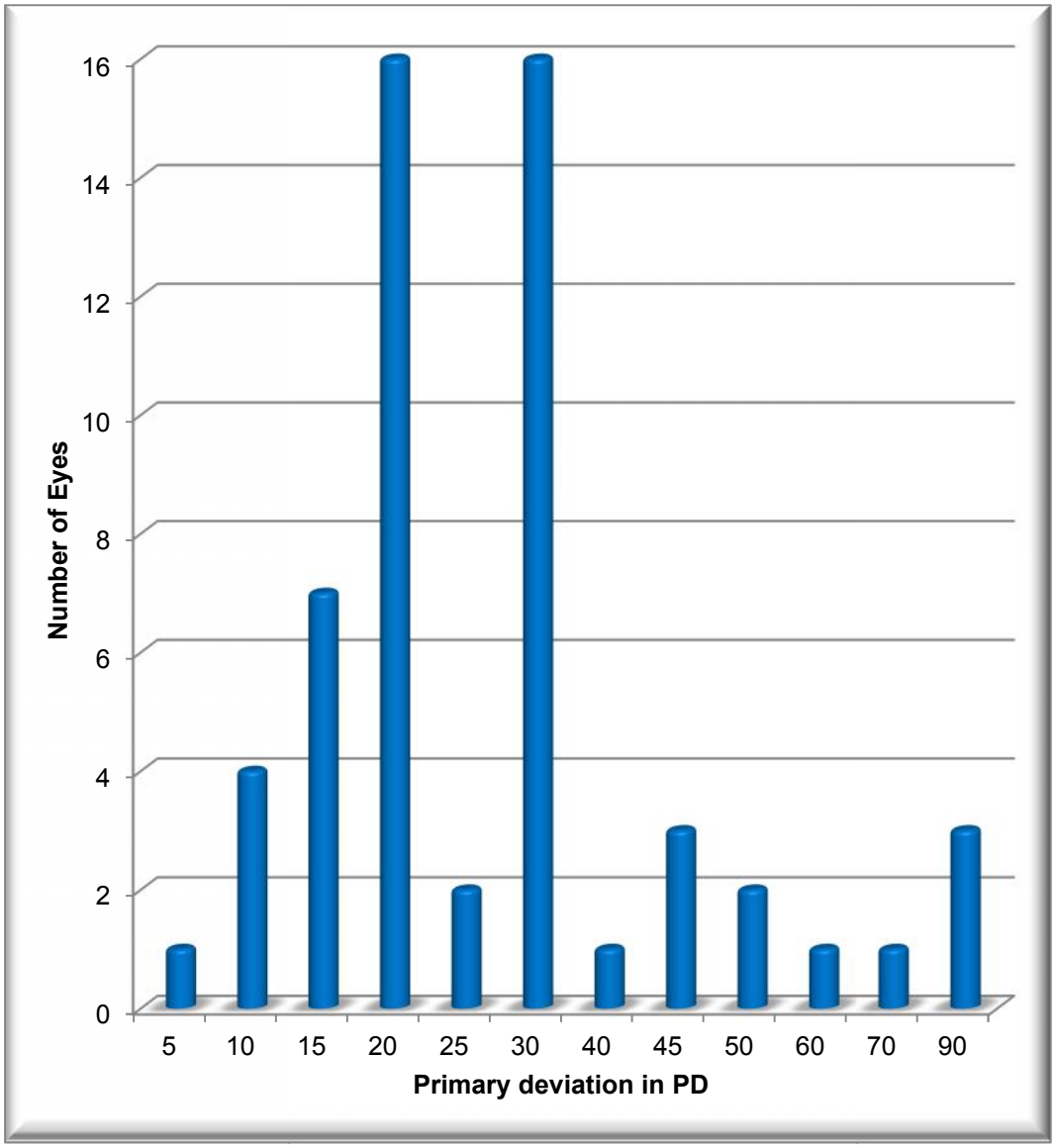
5. PRIMARY DEVIATION IN AFFECTED EYES

TABLE VI

Primary deviation in PD	Frequency	%
5	1	1.8
10	4	7.0
15	7	12.3
20	**16**	**28.1**
25	2	3.5
30	**16**	**28.1**
40	1	1.8
45	3	5.3
50	2	3.5
60	1	1.8
70	1	1.8
90	3	5.3
Total	**57**	**100.0**

The primary deviation was around 20 PD and 30 PD each in 16 eyes (28.1 %) with lateral rectus palsy. 15 PD in 7 eyes (12.3 %), 10 PD in 4 eyes (7 %), 45 PD and 90 PD in 3 eyes (5.3 %) each, 25 PD and 50 PD in 2 eyes (3.5 %) each. Rest of the eyes had a primary deviation of 5PD, 40 PD, 60 PD and 70 PD respectively.

GRAPH – 5: PRIMARY DEVIATION IN PD



6. AGE DISTRIBUTION ACCORDING TO ETIOLOGY

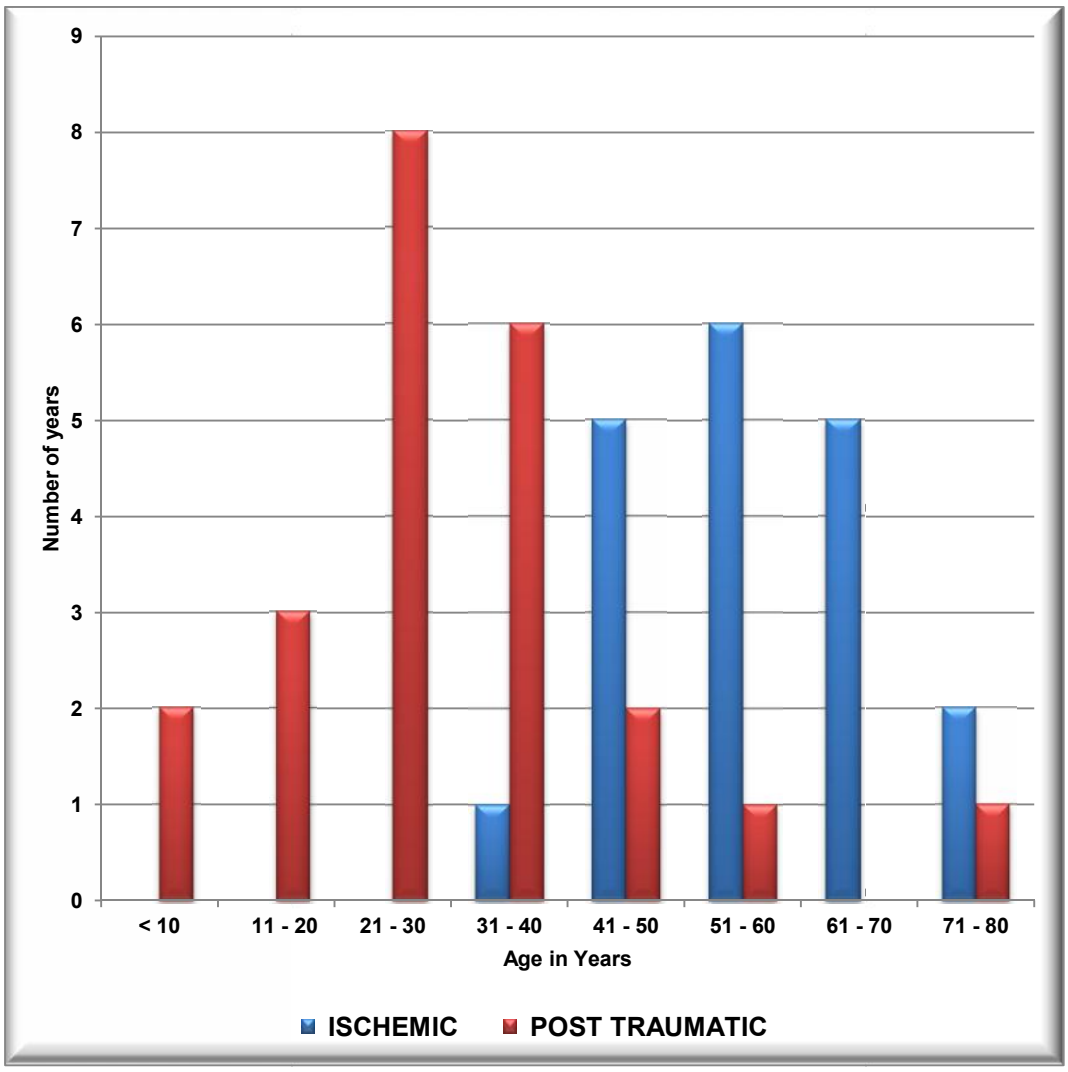
TABLE - VII

Age in years	Etiology		Total	p < 0.001
	Ischemic	Traumatic		
Upto 10	0	2	2	
11-20	0	3	3	
21-30	0	8	8	
31-40	1	6	7	
41-50	5	2	7	
51-60	6	1	7	
61-70	5	0	5	
71-80	2	1	3	
Total	**19**	**23**	**42**	

Most of the eyes (6) with ischemic sixth nerve palsy belonged to the age group of 51 to 60 years contributing to 31.6 % of all similar cases. Whereas most of the eyes (8) with traumatic sixth nerve palsy fell in the age group of 21 to 30 years contributing to 34.8 % of all traumatic cases. No patient less than 30 years suffered from ischemic lateral rectus palsy. Incidence of ischemic sixth nerve palsy significantly increases with age until 60 years. 5 eyes were from patients in the age group of 41 to 50 years and 61

to 70 years each, 2 eyes in age group from 71 to 80 and one eye in age group 31 to 40. The incidence of traumatic sixth nerve palsy significantly decreases with age. 6 eyes were of patients in the age group of 31 to 40 years, 3 in the age group of 41 to 50, 2 in 11 to 20, and 1 each in age groups of 51 to 60 and 71 to 80.

GRAPH – 6: AGE DISTRIBUTION IN SIXTH NERVE PALSY DUE TO VARIOUS ETIOLOGIES



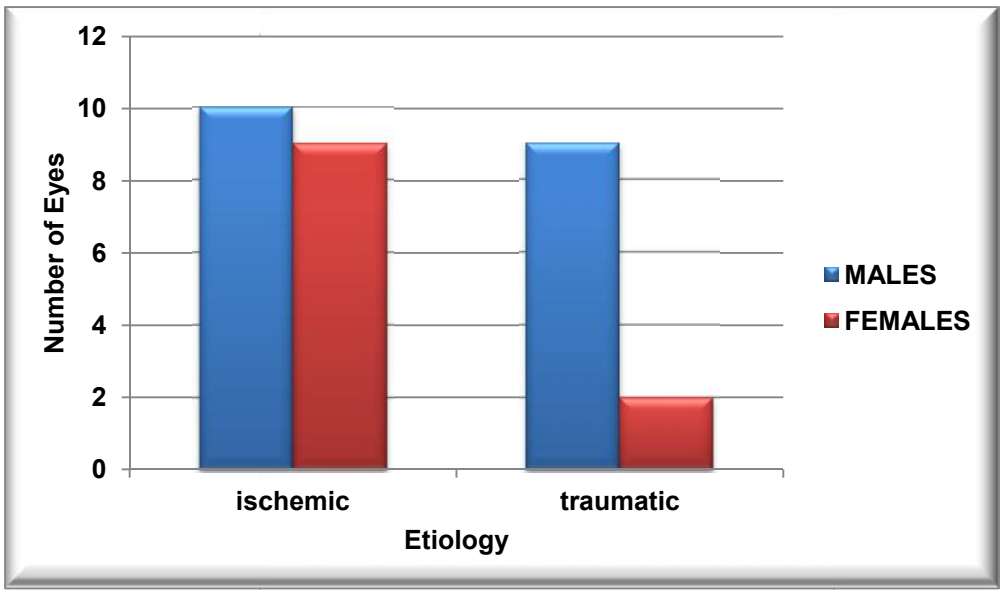
7. SEX WISE DISTRIBUTION OF SIXTH NERVE PALSY DUE TO VARIOUS ETIOLOGIES

TABLE - VIII

Sex	Etiology		Total	p = 0.005
	Ischemic	Traumatic		
Male	10	21	31	
Female	9	2	11	
Total	**19**	**23**	**42**	

21 out of 23 eyes (91.3 %) were of males with history of trauma; only 2 eyes (8.7 %) were of females. 10 out of 19 eyes (52 .6 %) were of males with ischemic pathologies, whereas 9 eyes (47.4 %) were of females.

GRAPH – 7: SEX DISTRIBUTION IN SIXTH NERVE PALSY DUE TO VARIOUS ETIOLOGIES



8. RECOVERY PATTERN AFTER 6 MONTHS DURATION

TABLE – IX

	Frequency	Percent
Recovered	37	64.9
Not Recovered	18	31.6
Lost to follow up	2	3.5
Total	**57**	**100.0**

37 eyes (64.9 %) with isolated sixth cranial palsy due to all causes recovered spontaneously within a period of six months from the onset of their symptoms. 18 eyes (31.6 %) had residual muscle paresis at the end of six months. 2 eyes (3.5 %) were lost to follow up.

GRAPH – 7: RECOVERY AT END OF 6 MONTHS

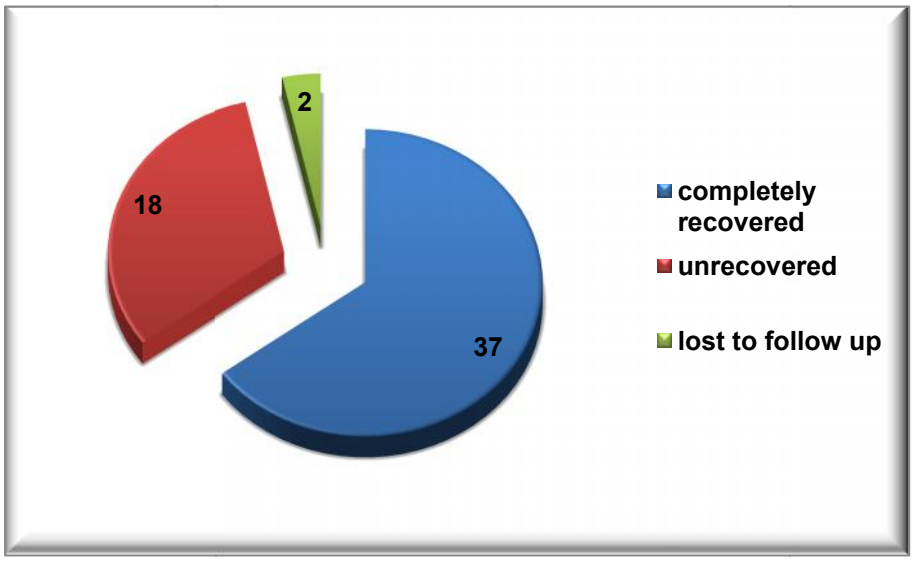


TABLE - X

After 6 months	Etiology					Total
	Ischemic	Traumatic	idiopathic	Post - viral	Post-surgical	
Recovered	15	13	6	3	0	37
Non - Recovered	3	9	4	0	2	18
Lost to follow up	1	1	0	0	0	2
Total	**19**	**23**	**10**	**3**	**2**	**57**

Among the ischemic group, 78.9 % of the eyes recovered, whereas in 15.8 % of all eyes some residual deviation persisted even after a period of 6 months and 5.3 % were lost to follow up.

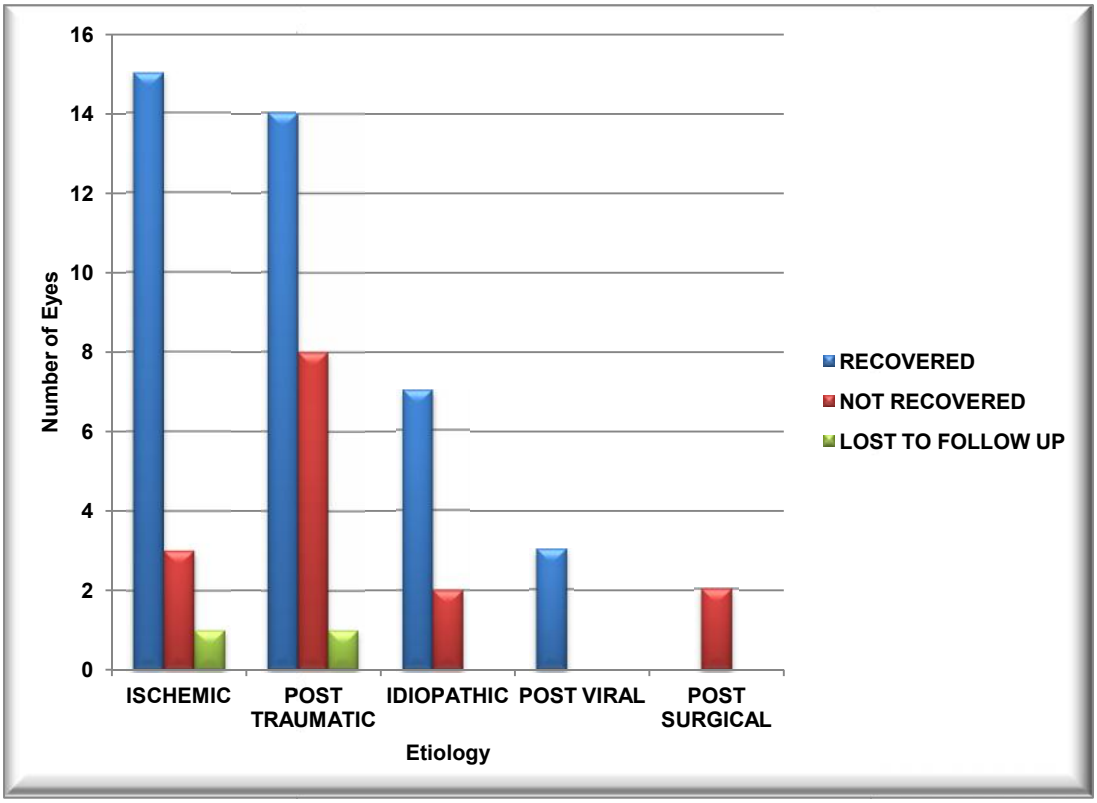
Among the post traumatic group, 56.5 % of all eyes recovered the action of lateral rectus, however 39.1 % of all eyes had some degree of residual deviation at the end of 6 months, and 4.3 % eyes were lost to follow up.

Among the recovered group, 15 eyes (40.54 %) were of patients with ischemic etiology, 13 eyes (35.14 %) were of patients with traumatic etiology, 6 eyes (16.2%) were patients without any known cause of lateral rectus palsy and 3 eyes (8.1%) were of patients with viral pathology.

Among the non – recovered group, only 3 eyes (16.67%) were of patients with ischemic etiology, whereas 9 eyes (50%) were of patients with traumatic etiology and 6 eyes (33.33%) were of patients with idiopathic etiology.

Both patients who acquired sixth nerve palsy following mastoidectomy did not recover at the end of 6 months. Whereas all the patients with post viral fever muscle weakness recovered completely within 6 months. Among the idiopathic causes 75 % patients showed complete recovery within six months, while 25 % patients suffered from residual muscle weakness.

GRAPH – 8: CLINICAL COURSE OF LATERAL RECTUS PALSY OVER 6 MONTHS ACCORDING TO ETIOLOGY



9. CORRELATION BETWEEN THE PRIMARY DEVIATION IN THE EYE AT PRESENTATION AND RECOVERY AT THE END OF 6 MONTHS

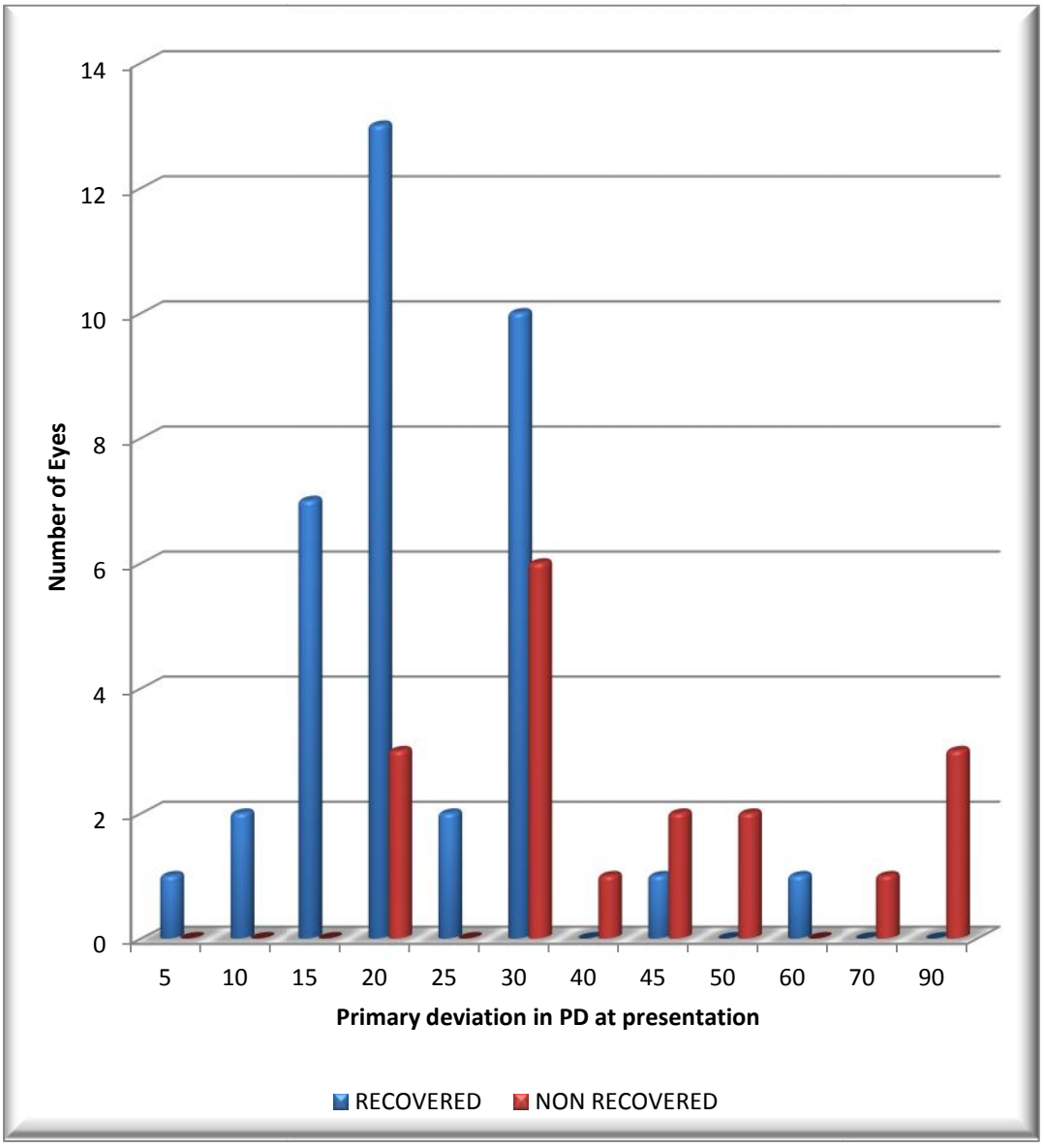
TABLE XI

Primary deviation in PD	After 6 months		p < 0.001
	Recovered	Non Recovered	
5	1	0	
10	2	0	
15	7	0	
20	**13**	**3**	
25	2	0	
30	**10**	**6**	
40	0	1	
45	1	2	
50	0	2	
60	1	0	
70	0	1	
90	0	3	
	37	**18**	

35 out of 37 eyes (94.59 %) that recovered had a primary deviation of < 45 PD and only 2 eyes (5.4%) had primary deviation =/> 45 PD. Whereas in the non - recovered group, 10 out of 18 eyes (only 55.5 %) had primary

deviation < 45 PD, and remaining 8 eyes (44.44 %) had primary deviation =/ > 45 PD.

GRAPH - 9: CORRELATION BETWEEN THE PRIMARY DEVIATION IN THE EYE AT PRESENTATION AND RECOVERY AT THE END OF 6 MONTHS



10. CORRELATION BETWEEN AGE AND RECOVERY AFTER PERIOD OF 6 MONTHS.

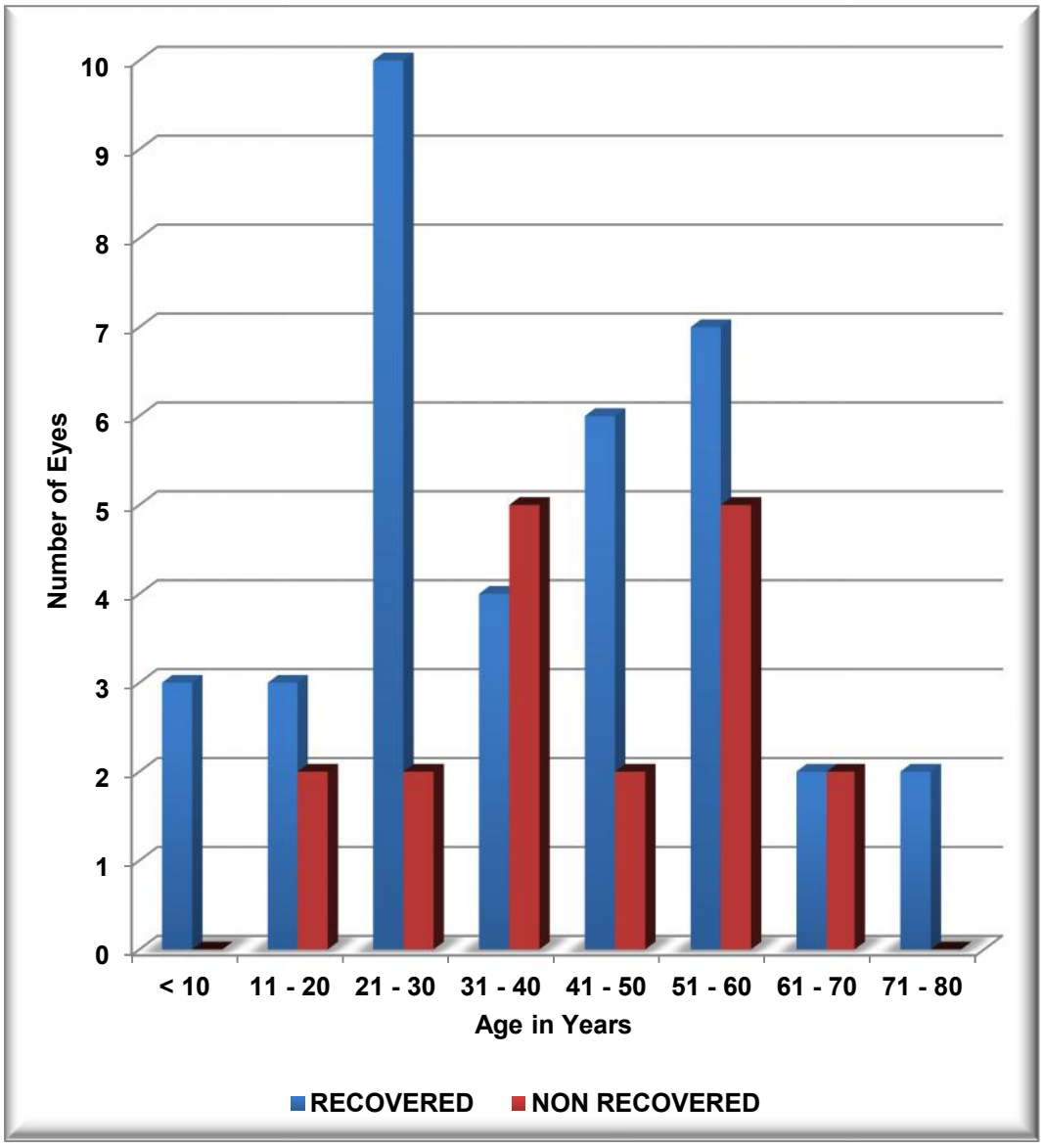
TABLE XII

Age in years	After 6 months		Total	p = 0.4
	Recovered	Non Recovered		
Upto 10	3	0	3	
11-20	3	2	5	
21-30	**10**	**2**	**12**	
31-40	4	5	9	
41-50	6	2	8	
51-60	7	5	12	
61-70	2	2	4	
71-80	2	0	2	
Total	**37**	**18**	**55**	

In the recovered group, 10 eyes (27%) were of patients of age group 21 to 30, 7 eyes (18.9%) of age group 51 to 60, 6 eyes (16.2%) of age group 41 to 50, and 4 eyes (10.8%) of age group 31 to 40. Patients within the age group of < 10 years and between 11 to 20 years contributed to 3 eyes (8.1%) each, and those in the age group of 61 to 70 years and 71 to 80 contributed to 2 eyes (5.4%) each.

In the non - recovered group, 5 eyes (27.8%) were of patients of age group 31 to 40 and 51 to 60 each. Patients within the age group of 11 to 20, 21 to 30, 41 to 50 and 61 to 70 years contributed to 2 eyes (11.1%) each, and those in the age group of < 10 years and 71 to 80 had no non recovered eyes.

GRAPH – 10: CORRELATION BETWEEN AGE AND RECOVERY AFTER PERIOD OF 6 MONTHS.



11. INCIDENCE OF RECOVERY AFTER 6 MONTHS IN MALES AND FEMALES

TABLE XIII

Sex	**After 6 months**		**Total**	**p = 0.638**
	Recovered	**Non Recovered**		
Male	25	11	36	
Female	12	7	19	
Total	**37**	**18**	**55**	

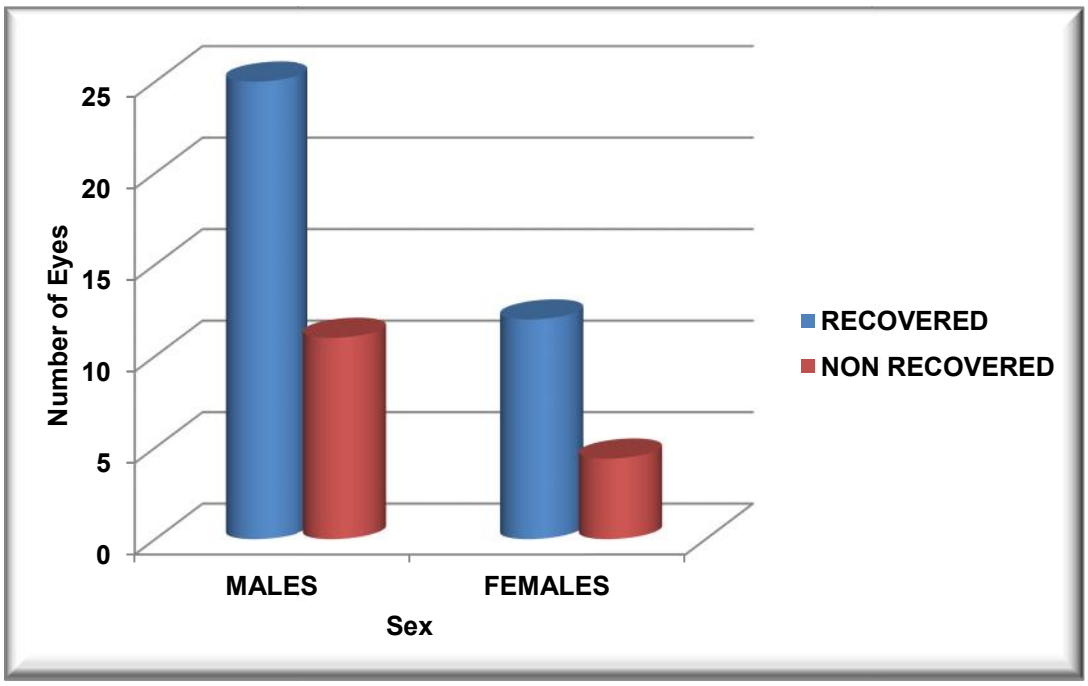
Among men, 25 out of 36 eyes (6**9**.4%) recovered whereas 11 of them (30.6%) did not recover even at the end of six months period.

Among women 12 out of 19 eyes (63.2%) recovered whereas 7 of them (36.8%) did not recover at the end of six months period.

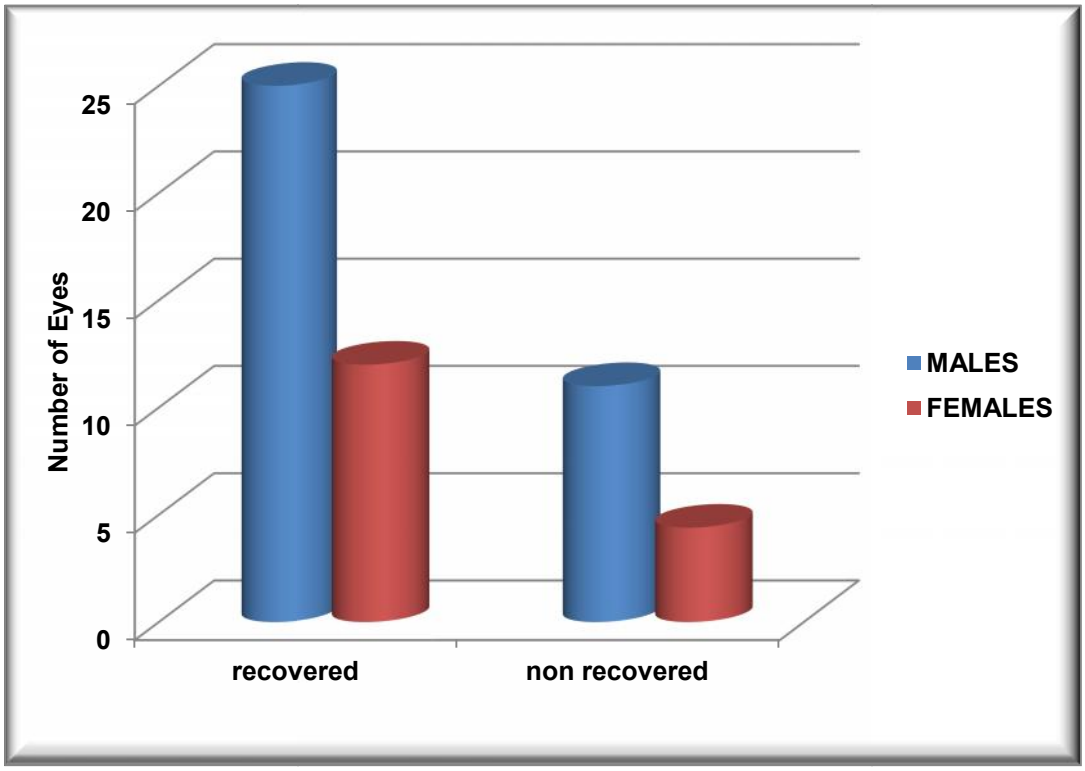
Among the recovered group, 25 out of 37 eyes (67.6%) were of men, whereas, 12 of them (32.4%) were of women.

Among the non recovered group, 11 out of 18 eyes (61.1%) were of men, whereas, 7 of them (38.9%) were of women.

GRAPH – 11: RECOVERY ACCORDING TO SEX



GRAPH – 12 : SEX DISTRIBUTION ACCORDING TO RECOVERY



12. IN UNRECOVERED CASES, at the end of 6 months

TABLE XIV

Diplopia	Patients
Present	6
Absent	9
Total	**15**

Diplopia was persistent in 6 (40 %) patients that did not recover from sixth nerve palsy at the end of 6 months. The remaining 9 (60 %) non recovered patients had no diplopia probably due to suppression.

TABLE XV

Forced generation test	Eyes
Positive	8
Negative	10
Total	**18**

In 8 eyes (44.44 %) had Forced Generation Test positive at the end of six months. 10 eyes (55.56 %) had a negative Forced Generation Test.

AT THE END OF 6 MONTHS

TABLE XVI

Surgery	Patients	Eyes
Yes	10	11
No	5	7
Total	**15**	**18**

10 out of 15 non recovered patients (66.67%) underwent squint surgery at the end of 6 months; remaining 5 patients (33.33%) were experiencing diplopia or cosmetic problems and hence did not undergo squint surgery. One patient required surgery in both eyes. Thus, 11 out of 18 eyes (61.11%) underwent surgery.

TABLE XVII

Indication for surgery	Patients
Diplopia	6
Cosmetic	4
Total	**10**

In 6 out of 10 patients (60%), diplopia was the primary indication of surgery while in the remaining 4 patients (40%), the primary indication was unacceptable cosmetic appearance.

SURGERY PERFORMED

TABLE XVIII

Type of surgery performed	Patients
Ipsilateral LR resection + MR recession	7
Bilateral LR resection and MR recession	1
Ipsilateral transposition of superior and inferior recti to LR with MR recession	2
Total	**10**

In 3 patients with diplopia with a positive FGT and in 4 patients in whom surgery was indicated for cosmetic purposes accounting to 7 out of 10 (70%), two muscle surgery was performed i.e ipsilateral LR resection and MR recession. In one patient both eyes were affected, hence bilateral LR resection and MR recession was done. In two patients with diplopia and negative FGT, ipsilateral transposition of superior and inferior recti to the LR with MR recession was performed.

SURGICAL OUTCOME

After the appropriate surgeries were performed, the patients were followed up at 1 week, 2 weeks and 4 weeks post operatively. At the end of 4 weeks, all the patients achieved a deviation of less than 20 PD in primary gaze, which was cosmetically acceptable by them. All the six patients with diplopia were relieved of diplopia in the primary gaze as desired. There were no untoward complications noted in any of the strabismus surgeries and the patient satisfaction for the procedure was high.

CHEMODENERVATION

In our study, we injected 3 – 5 I.U. of Botulinum toxin, in ipsilateral MR of five patients with acute sixth nerve palsy. Three of them were traumatic in origin and two were of ischemic origin. All five patients were relieved of diplopia in primary gaze within a period of one week of administration of the injection. At the end of 6 months four patients completely recovered, whereas one patient failed to recover completely but was asymptomatic. There were no untoward complications noted following botulinum toxin injection. The results were very satisfying.

INTERVENTIONS UNDERGONE BY PATIENTS IN OUR STUDY

TABLE XIX

	Frequency	Percent
NIL	39	64.9
Surgery	11	22.8
Botulinum inj.	5	8.8
Lost to follow up	2	3.5
Total	**57**	**100.0**

39 eyes i.e 68.42 % of all eyes did not undergo any intervention in our study group. 11 eyes (19.3 %) were taken up for surgical correction of squint. 8.8 % were given injection Botulinum as primary treatment. 3.5 % eyes were lost to follow up.

DISCUSSION

DISCUSSION

1. AGE

In this study 57 eyes of 50 patients with isolated sixth nerve palsy were studied. The maximum number of patients belonged to the age group of 21 – 30 years and 51 to 60 (21.1% each). At extremes of age < 10years and > 70 years the incidence of isolated sixth cranial nerve palsy is least (5.3% each).

The etiology defers according to the different age groups. In our study, 94.74% of the eyes affected due to trauma belonged to patients < 40 years of age. In contrast, 82.6 % of eyes of patients with ischemic etiology fell in the age group of > 40 years. This correlation is highly significant as tested by Pearson Chi Square test has a p value of < 0.001.

The recovery pattern of sixth nerve palsy does not depend on the age of the patient (p = 0.4).Younger patients however are able to overcome diplopia due to suppression better than patients of older age group. Cosmetic appearance is of greater concern in patients of younger age group and was one of the main indications for surgical intervention in these individuals.

2. SEX

Incidence of isolated sixth cranial nerve palsy in males was higher than females in our study. In our study there was a significant gender difference, with males out numbering females – 63.2 % males against 36.8 % females. Ratio of Males: Females was 1.71: 1. However this difference was reflected most in patients with history of trauma where 91.3 % of the eyes were of males. In patients with sixth nerve palsy due to ischemic causes, the incidence among males and females was almost equal, 52.6 % and 47.4 % respectively.

The greater incidence among males of younger age group is attributed to the fact that they are more prone to trauma and road traffic accidents than females and older individuals.

In our study 69.4 % eyes from males and 63.2% eyes from females completely recovered within a period of 6 month. The recovery pattern in males and females did not show any difference ($p = 0.6$) and is not statistically significant.

3. ETIOLOGY

In our study, for most of the eyes (40.4 %) were of patients with history of trauma. 35.5 % eyes were of patients who suffered ischemic insult to the sixth nerve either due to diabetes or hypertension or both. 3 patients had history of fever in the week preceding the symptoms of diplopia and hence

their paresis was attributed to viral infection. All these patients were less than 20 years of age and they completely recovered in a period of less than 3 months of the insult. 2 patients had undergone mastoidectomy, following which they developed acute complete sixth nerve palsy with a negative forced generation test. None of the patients showed any recovery even after 6 months duration.

In 17.5 % of the eyes, the cause of isolated sixth nerve palsy could not be determined and was presumed to be due to several causes like; ear infections, migraine, post ictal and idiopathic.

There was no significant influence of the duration or severity of diabetes or hypertension on the clinical course of the disease.

78.9% of the eyes with ischemic etiology recovered at the end of 6 months follow up period whereas only 56.5 % of the post traumatic eyes showed complete recovery in that period. However the etiology of sixth nerve palsy in the traumatic and ischemic group, have no correlation with the pattern of recovery according to the analysis done by Pearsons Chi Square test ($p = 0.248$), and that the data is not statistically significant.

Lopez and colleagues found hypertension as a major cause of isolated sixth nerve palsy, however in our study only around 16% patients were diagnosed with hypertension.[1]

In a study by Michael P Ehrenhaus, MD; Chief Editor: Hampton Roy Sr, MD, sixth nerve palsies fell into the following categories: 8-30% idiopathic, 10-30% miscellaneous, 3-30% trauma, 0-6% aneurysm, and 0-36% ischemic.[7]

The sixth cranial nerve is the most commonly affected of the ocular motor nerves. In children, it is the second most common after the fourth nerve, with an incidence of 2.5 cases per 100,000 in the population.[24]

Intracranial tumours and raised intracranial tension due to various congenital malformations, etc are the most common causes of isolated sixth nerve palsy especially in children and young adults.[24] As clinicians it is our responsibility to rule out all malignant and life threatening causes of nerve palsy before coming to any final conclusion regarding the etiology.

In a long-term follow-up study of children with benign abducens nerve palsy by V Sturm and C Schöffler, Department of Ophthalmology, University Hospital of Hamburg, Hamburg, Germany; migraine, infections and inflammation were the most common causes.[9]

4. LATERALITY

In our study, both eyes were almost equally affected with a slight predominance of right eye over left eye by 2 %. In 14 % of individuals both eyes were affected.

5. DEVIATION AT PRESENTATION

In our study, most of eyes had a primary deviation of around 20 to 30 PD, 28 % each. 94.9 % eyes with a primary deviation of < 45 PD recovered at the end of 6months follow up. Thus the degree of insult at presentation in the form of primary deviation, is a very good predictor of the pattern of recovery from sixth nerve palsy ($p < 0.001$)

6. RECOVERY

In this study, 64.9 % of all eyes with isolated sixth cranial palsy due to all causes recovered spontaneously within a period of six months from the onset of their symptoms. 31.6 % of the eyes had residual muscle paresis at the end of six months. 2 patients were lost to follow up.

60 – 80 % of all patients with history of trauma and ischemia recovered at end of six months. Patients with injury to the sixth nerve during surgical procedure did not show any signs of recovery, probably due to anatomic disruption of the nerve fibres and lack of regeneration.

The age at presentation, sex and etiology have no influence on the speed of recovery in patients with isolated sixth nerve palsy; however the primary deviation of < 45 PD is a strong predictor of the recovery pattern.

6. SURGRICAL OUTCOME

Only 19.29 % of all e eyes in our study were subjected to strabismus surgery. Primary indication for surgery for 60% of these patients was intractable diplopia in either primary or lateral gaze. Cosmetic appearance was the primary indication for surgery in 40 % of unrecovered patients. Either two muscle i.e ipsilateral LR resection and MR recession, four muscle, i.e bilateral LR resection and MR recession and ipsilateral transposition of superior and inferior recti to the LR with MR recession was performed on all patients depending on the residual deviation, action of the muscle and forced generation test for lateral rectus at the end of six months. At the end of 4 weeks, all the patients achieved a deviation of less than 20 PD in primary gaze, which was cosmetically acceptable by them. All the six patients with diplopia were relieved of diplopia in the primary gaze as desired. There were no untoward complications noted in any of the strabismus surgeries and the patient satisfaction for the procedure was high.

The results of our surgical procedures are similar to the “Outcomes of Surgical and Non-Surgical Treatment for Sixth Nerve Palsy” – a study by Abbas Bagheri, MD; Babak Babsharif, MD; Mohammad Abrishami, MD; Hossein Salour, MD; Maryam Aletaha, MD Labbafinejad Medical Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran. Their conclusions in a study of 37 eyes of 33 patients were that various procedures

are effective for treatment of sixth nerve dysfunction; all improve ocular deviation, head turn and abduction deficit. The rate of reoperation is not high when treatment is appropriately selected according to clinical condition.[19]

7. ROLE OF BOTULINUM TOXIN

In our study, we injected Botulinum toxin, in ipsilateral MR of five patients with acute sixth nerve palsy. All five patients were relieved of diplopia in primary gaze within a period of one week of administration of the injection. The results were very satisfying. According to a study - Approach to Traumatic Sixth Nerve Palsy with Botulinum Toxin A by H. E. Killer, B. K. Blumer and A. Bähler Department of Ophthalmology, Kantonsspital Aarau, Switzerland; intramuscular injections of botulinum toxin A into the direct antagonist of the paretic muscle shortly after the onset of the palsy may help to prevent the development of spastic contracture of the ipsilateral antagonist and prevent restriction to abduction.[10] It also assists to reestablish earlier the field of single binocular vision in primary position. This concept is supported by many other studies.[12-18]

CONCLUSION

CONCLUSION

1. The incidence of isolated sixth cranial nerve palsy had a bimodal peak, first peak between ages 21 to 30 years and second between ages 51 to 60.

2. Incidence of isolated sixth cranial nerve palsy in males was higher than females in our study. Ratio of Males : Females was 1.7 : 1

3. Right and left eyes were almost equally affected in our study.

4. Primary deviation was around 20 to 30 PD in most of the patients and serves as a good predictor of complete recovery from lateral rectus palsy for that eye.

5. Trauma and ischemia are amongst the most common causes of isolated sixth cranial palsy.

6. Incidence of ischemic insult to sixth cranial nerve was almost equal in both males and females

7. Incidence of trauma was 10.5 times more in male patients compared to females.

8. In patients with ischemic sixth nerve palsy, the incidence in patients older than 40 years was 18 times more than patients younger than 40 years.

9. In patients with post traumatic sixth nerve palsy, the incidence in patients younger than 40 years was 4.75 times more than patients older than 40 years.

10. There was no correlation between the glycemic control and development of ischemic paralysis of sixth nerve in the diabetic patients.

11. There was also no association between diabetic patients who developed retinopathy and sixth nerve palsy.

12. Most of the patients with post traumatic sixth nerve palsy were not associated with fracture of the bony orbit.

13. There is no correlation between the duration of diabetes mellitus or hypertension on the incidence of sixth nerve palsy.

14. The modality of treatment with insulin or oral hypoglycemic drugs does not alter the course or the severity of sixth nerve palsy.

15. Invariably diabetes was detected by an ophthalmologist, following the presentation of sixth nerve palsy.

16. 64.9 % of all eyes recovered completely within six months of the onset of paralysis.

17. The age at presentation, sex and etiology had no correlation with the pattern of recovery in eyes with lateral rectus palsy

18. All the patients of post viral etiology showed an early complete recovery.

19. All the patients who underwent mastoidectomy showed no recovery even after six months, indicating an anatomic insult to the abducens nerve during surgery.

20. At the end of six months, most of the patients were relieved of diplopia probably due to facultative suppression.

21. The most common indication for surgery however remained diplopia in the primary gaze for most patients and for cosmesis.

22. Appropriate selection of patients ensures good surgical outcome in sixth nerve palsy.

23. Injection of botulinum toxin in acute sixth nerve palsy relieves patient of diplopia in primary gaze. However the effect is short lived and needs repeated injections at 3 – 6 months interval.

PART III

BIBLIOGRAPHY

1. Wolff's anatomy of the eye and orbit, Eighth edition, Pg No.189 -191

2. Clinical Anatomy of the Eye – Richard Snell, M.D, PHD. Second edition, Pg. No. 307 – 311.

3. Walsh and Hoyt's Clinical Neuro – Ophthalmology, The essentials, Second edition, Pg- 398 – 404

4. Binocular Vision and Ocular Motility, Theory and Management of Strabismus, by Gunter K.Von Noorden, 6th Edition, Pg. No 439

5. Profile of isolated sixth cranial nerve palsy: A hospital based study by S Shakya

6. Manual of Strabismus Surgery by Caroline Mac Even and Richard Gregson. First edition, Pg. No. 121 – 130, 163 – 168

7. Abducens Nerve Palsy, Author: Michael P Ehrenhaus, MD; Chief Editor: Hampton Roy Sr, MD,Medscape Updated: May 21, 2009

8. Isolated sixth nerve palsy, Contributors:Andrew G Lee MD, author. Dr. Lee of the Methodist Hospital and Weill Cornell College of Medicine in Houston, Texas; Paul W Brazis MD, author. Dr. Brazis of the Mayo Clinic in Jacksonville, Florida; Jonathan Trobe MD,Editor. Dr. Trobe of the University of Michigan.

9. Long-term follow-up of children with benign abducens nerve palsy,V Sturm and C Schöffler, Department of Ophthalmology, University Hospital of Hamburg, Hamburg, Germany; Eye (2010) 24, 74–78; doi:10.1038/eye.2009.22; published online 13 February 2009

10. Approach to Traumatic Sixth Nerve palsy with Botulinum Toxin A, by H. E Killer, B. K. Blumer, A. Bahler. Department of Ophthalmology, Kantonssipital Aran, Switzerland.

11. Clinical strabismus management: principles and surgical techniques by Arthur Rosenbaum, Alvina Pauline Santiago, Pg 259; Am J Ophthalmol. 2001 Mar; 131(3):359-63.

12. Botulinum Toxin A in the Early Treatment of Sixth Nerve Palsy-Induced Diplopia in Type 2 Diabetes, Anna Broniarczyk-Loba, MD, PHD, Leszek Czupryniak, MD, PHD, Olimpia Nowakowska, MD, PHD and Jerzy Loba, MD, PHD

13. Paralytic strabismus: the role of botulinum toxin. Elston JS, Lee JP: Br J Ophthalmol 69:891–896, 1985

14. Initial treatment outcomes in chronic sixth nerve palsy. Holmes JM, Leske DA, Christiansen SP. J AAPOS 5:370–376, 2001

15. Results of a prospective randomized trial of botulinum toxin therapy in acute unilateral sixth nerve palsy. Lee J, Harris S, Cohen J, Cooper K, MacEwan C, Jones S: J Pediatr Ophthalmol Strabismus 31:283–286, 1994

16. Early botulinum toxin treatment of acute sixth nerve palsy. Murray AD: Eye 5:45–47, 1991

17. A review of 5 years' experience in the use of botulinum toxin A in the treatment of sixth cranial nerve palsy at the Singapore National Eye Centre. Quah BL, Ling YL, Cheong PY, Balakrishnan V: Singapore Med J 40:405–409, 1999.

18. Botulinum toxin injection into extraocular muscles as an alternative to strabismus surgery. Scott AB. Ophthalmology. 1980 Oct; 87(10): 1044-9.

19. Outcomes of Surgical and Non-Surgical Treatment for Sixth Nerve Palsy Abbas Bagheri, MD; Babak Babsharif, MD; Mohammad Abrishami, MD; Hossein Salour, MD; Maryam Aletaha, MD. Labbafinejad Medical Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran.

20. Modified transposition procedure of the vertical recti in sixth nerve palsy. Neugebauer A, Fricke J, Kirsch A, Rüssmann W. University Eye Clinic, D-50924 Cologne, Germany.

21. Unaugmented vertical muscle transposition surgery for chronic sixth nerve paralysis. Bansal S, Khan J, Marsh IB. Department of Ophthalmology, Royal Liverpool University Hospital, Liverpool, UK. 2006 Dec; 14(4):177-81

22. Vertical rectus muscle transposition and botulinum toxin for complete sixth nerve palsy. Flanders M, Qahtani F, Gans M, Beneish R. 20 .Can J Ophthalmol. 2001 Feb;36(1):18-25 Department of Ophthalmology, McGill University Health Centre, Montreal General Hospital and Montreal Children's Hospital, Montreal, Que.

23. Partial rectus muscle-augmented transpositions in abduction deficiency. Britt MT, Velez FG, Thacker N, Alcorn D, Foster RS, Rosenbaum AL. J AAPOS. 2003 Oct;7(5):325-32. Jules Stein Eye Institute, University of California-Los Angeles, 100 Stein Plaza, Los Angeles, CA 90095, USA.

24. Sixth cranial nerve (abducens nerve) palsy in children , AuthorsAndrew G Lee, MDPaul W Brazis, MD, Evelyn A Paysse, MDDouglas R Nordli, Jr, MD, Mary M Torchia, MD.

PROFORMA

SQUINT CLINIC NO:

OP/IP NO:

DATE:

UNIT:

NAME:

AGE: SEX: M/F

OCCUPATION:

EYE INVOLVED: RE/ LE

MODE OF ONSET:

VISUAL COMPLAINTS

HEADACHE

WEAKNESS

DIPLOPIA

PTOSIS

PAST HISTORY OF: TRAUMA, CONVULSIONS , LOC , NAUSEA, VOMITTING , Rx FOR STD , FEVER , EAR COMPLAINTS ,NECK SWELLING , EPISTAXIS, NASAL OBSTRUCTION, VOICE CHANGE.

SYSTEMIC DISEASES - DM, HYPERTENSION, CONNECTIVE TISSUE DISORDER

PAST HISTORY OF SIMILAR EPISODE

TREATMENT HISTORY

GENERAL EXAMINATION:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

ABDOMINAL SYSTEM:

CENTRAL NERVOUS SYSTEM: HIGHER FUNCTIONS

CRANIAL NERVES

MOTOR SYSTEM

SENSORY SYSTEM

CEREBELLUM

OCULAR EXAMINATION:

1. HEAD POSITION: HEAD TILT, FACE TURN, CHIN ELEVATION/DEPRESSION
2. SKULL ABNORMALITIES
3. EYELIDS: PTOSIS, / RETRACTION / LID LAG / MARCUS GUNN JAW WINKING PHENOMENON / DUANE'S RETRACTION SYNDROME
4. PROPTOSIS / ENOPHTHALMOS
5. BELLS PHENOMENON / SACCADES / PURSUIT / NYSTAGMUS
6. EXTRA OCULAR MOVEMENTS

   UNIOCULAR

   BINOCULAR

   VERGENCE

FDT

FGT

EMG

7. ANTERIOR SEGMENT:

PUPIL: SIZE

SHAPE

REACTION

FUNDUS

VISION with PINHOLE NEAR VISION

REFRACTION

COLOUR VISION

FIELDS

IOP

DIPLOPIA CHARTING

ORTHOPTIC EVALUATION

SACCADIC VELOCITY

INVESTIGATIONS:

BP

COMPLETE HAEMOGRAM

FBS/ PP

URINE ROUTINE

MANTOUX

VDRL

RADIOLOGICAL STUDY:

X-RAY ORBIT

X-RAY SKULL – AP/ LATERAL

PNS

ORBITAL FORAMINA

CT SCAN

MRI

NEUROLOGIST OPINION

ENT OPINION

OBRIT CLINIC OPINION

DIAGNOSIS

ADVICE:

PLAN OF SURGERY:

IMMEDIATE POST OP ASSESSMENT

APPEARANCE

DIPLOPIA

ORTHOPTIC EVALUATION

LATE FOLLOW UP 2 WEEKS/6 WEEKS

COSMETIC APPEARANCE: BINOCULAR SINGLE VISION:

FINAL OUTCOME

EXCELLENT / VERY GOOD / GOOD / FAIR / POOR

KEY TO MASTER CHART

M - MALE

F - FEMALE

RE - RIGHT EYE

LE - LEFT EYE

NAD - NO ABNORMALITY DETECTED

DR - DIABETIC RETINOPATHY

DM - DIABETIC MELLITUS

HTN - HYPERTENSION

R - RECOVERED

NR - NOT RECOVERED

FR - FIXING WITH RIGHT EYE

FL - FIXING WITH LEFT EYE