A STUDY OF ISOLATED AND MULTIPLE CRANIAL NERVE PALSIES

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CERTIFICATE

It is hereby certified that this dissertation 'the study of isolated and multiple cranial nerve palsy' is the bonafide work done by Dr. S.Venkatesh, postgraduate student in ophthalmology, studying at Madras Medical College ,between august 2003 and September 2005, at Regional Institute of Ophthalmology and Government Ophthalmic Hospital ,Chennai.

Head Of The Department	Director & Superintendent R .I .O.&G.O.H	Dean, Madras Medical College.

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CONTENTS

A STUDY OF MULTIPLE CRANIAL NERVE PALSIES AFFECTING THE EYE

Introduction

There are several cranial nerves which are supplying the eye. They are namely the second, third, fourth, fifth, sixth, and seventh nerves. They are concerned with the eye movements, pupillary reactions corneal sensation and lid movements. Restricted eye movements can be due to paralysis of those cranial nerves which are responsible for ocular movement. The ocular motor nerves are third nerve [oculomotor nerve],fourth nerve[trochlear nerve], and the sixth nerve[abducent nerve].So a paralytic squint can occur from paralysis of these nerves[neurogenic] or from neuromuscular origin[myasthenia gravis] or from myopathic origin or from restrictive origin. Clinically differentiating these etiologies is important because their treatment also differs.

In our study we are concerned with paralytic squint [where angle of deviation varies with different gaze positions] of neurogenic origin .Such nerve palsies can involve the ocular motor system at various levels from nucleus to the effector organ, that is, the extra ocular muscles.There are six extra ocular muscles controlling the eye movements, namely medial, lateral,superior,and inferior recti and superior and inferior oblique muscles.

Lesions in the cerebral cortex causes supranuclear conjugate gaze palsies where both eyes are affected equally and no diplopia occurs.But lesions affecting the lower portions of the neurone like the nuclei, infranuclear portion of the nerve in the brain stem,and after leaving the the brainstem in the subarachnoid space,and in the cavernous sinus and in the orbit etc., can lead to paralytic squint where the relative co-ordinaton of the eye is affected and the patient becomes symptomatic with diplopia and other symptoms.

The paralytic squint can be due to single or multiple cranial nerve involvement. When multiple nerves are involved the localization of the lesion becomes

very important .When multiple cranial nerves are involved , presence of associated signs of neurological importance ,gives diagnostic clues about the site of lesion.[e.g. brainstem]

Common locations of where multiple cranial nerves are involved without other neurological manifestations include the subarachnoid space , the cavernous sinus, the superior orbital fissure, and the apex of the orbit .

Common etiologies causing such multiple cranial nerve palsies include trauma , vascular pathology , tumour ,and inflammation of infectious and noninfectious origin.

ANATOMY and PHYSIOLOGY

Our understanding of ocular motor control has advanced rapidly during the last 30 yr, in part, because the purpose of eye movements can be defined so clearly: To maintain high acuity, images must be held steadily on the retina. Image motion of only a few degrees per second leads to significant blurring. The disturbances potentially causing image motion across the retina are movement of the observer and motion of the visual object of interest.Five distinct types of eye movements have evolved to counteract these disturbances or to bring new objects of interest onto the fovea .

Vestibuloocular and optokinetic eye movements stabilize images on the retina during head rotation by counterrolling the eyes through an angle equal to that of the head, but opposite in direction.

Thus, the VOR and OKN keep images stationary on the retina and maintain constant gaze (eye position relative to the earth) by generating slow eye movements equal to and opposite to the head motion.

In contrast saccadic, smooth pursuit, and vergence eye movements shift gaze to bring new objects of interest onto the fovea. Saccades are rapid conjugate eye movements that redirect the fovea toward an object in the retinal periphery.

. Smooth pursuit movements utilize visual feedback to track small objects and thus keep their image on the fovea. The smooth pursuit system may also have a role in the suppression of the VOR, while tracking an object with head movements . Vergence movements are slow disconjugate changes in horizontal eye position that redirect each fovea independently toward an object in response to disparity between the images of one object on both retinas or as an accompaniment of accommodation in response to blurring.

The Nerves Governing Extraocular Motility

The final common pathway for oculomotor control consists of the three pairs of ocular motor nerves and the muscles that they innervate. The nerves originate in paired nuclei within the midbrain and pons, and their axons course as fascicles through the brain stem parenchyma, run freely for variable distances within the subarachnoid space, pass through the cavernous sinus, and enter the orbit to supply the extraocular muscles.

The Third Nerve

The oculomotor nerve (third cranial nerve) supplies motor innervation for the superior rectus, medial rectus, inferior rectus, inferior oblique and levator palpebrae superioris muscles and also parasympathetic input to the pupillary constrictor and ciliary muscles. Cell bodies reside in the midbrain in a nuclear mass straddling the vertical midline . Each target muscle has a subnucleus devoted exclusively to its function. Most rostral and dorsal are the visceral nuclei (Edinger-Westphal nuclei and adjacent structures) that supply parasympathetic innervation to the pupillary sphincters and ciliary muscles via the ciliary ganglia. Caudal and dorsal, the “caudal central nucleus” is a single midline structure that innervates the levator palpebrae superioris muscles, subserving upper lid elevation. The cell bodies in the superior rectus subnuclei send their axons directly across the midline to join the contralateral oculomotor fascicles. The other subnuclei project ipsilaterally to their individual extraocular muscles.

The oculomotor fascicles refer to the portion of the nerve that travels through the brain stem parenchyma to exit ventrally. The fascicles pass through the substance of the red nuclei and the medial cerebral peduncles. Upon leaving the brain stem, the nerve enters the subarachnoid space and courses forward and laterally between the posterior cerebral artery above and the superior cerebellar artery below to run briefly alongside the posterior communicating artery. At this level, the pupillary fibers are located dorsally and peripherally. The nerve then pierces the dura and enters the cavernous sinus.

Within the anterior aspect of the cavernous sinus, the third nerve divides so that two divisions of the oculomotor nerve pass through the superior orbital fissure and enter the orbit. The superior division contains axons destined for the levator palpebrae superioris and superior rectus muscles. The inferior division carries the motor fibers to the medial rectus, inferior rectus, and inferior oblique muscles, as well as the preganglionic parasympathetic pupillomotor fibers to the ciliary ganglion.

The Fourth Nerve

The trochlear nerve (fourth cranial nerve) provides innervation to the superior oblique muscle only. Its nucleus is located in the midbrain, just beneath the cerebral aqueduct, caudal to the oculomotor nuclear complex . The axons exit dorsally and cross within the anterior medullary velum that lies just caudal to the inferior colliculi. Thus, innervation to the superior oblique muscles is strictly contralateral. After decussation, the nerve runs forward in the subarachnoid space around the mesencephalon and cerebral peduncles, along the free edge of the tentorium, between the posterior cerebral and superior cerebellar arteries. It enters the cavernous sinus just below the oculomotor nerve. Although the trochlear nerve passes through the superior orbital fissure , it enters the orbit outside of the annulus of Zinn (a position unique for the ocular motor nerves) and crosses above the superior rectus muscle en route to innervate the superior oblique muscle.

Sixth Nerve

The The abducens nerve (sixth cranial nerve) supplies the lateral rectus muscle. Its nucleus is located in the pons in the floor of the fourth ventricle, at the level of the facial colliculi Loops of the fascicular portion of the facial nerve (seventh cranial nerve) course ipsilaterally around the abducens nucleus, creating the facial genu.

The abducens nucleus contains both cell bodies destined to innervate the lateral rectus via the abducens nerve and interneurons whose axons run in the contralateral medial longitudinal fasciculus to the medial rectus subnucleus.

The abducens fascicles run ventrally through the substance of the pons, in close proximity to the facial nucleus and nerve, the trigeminal nuclei and tract, the superior olivary complex, the central tegmental tract, and the corticospinal fibers .

The nerve enters the subarachnoid space at the junction of the pons and medulla, just lateral to the pyramids. It courses upward along the surface of the clivus to pierce the dura and run beneath the petroclinoid ligament into the cavernous sinus . Entrance into the orbit is through the superior orbital fissure and the annulus of Zinn. Branches of the sixth nerve innervate the lateral rectus muscle .

Cavernous sinus and the nerve palsy

The cavernous sinus is a trabeculated venous sinus located between folds of dura on either side of the sella turcica . Within the substance of the sinus runs the intracavernous portion of the internal carotid artery (as it bends into its siphon), surrounded by the sympathetic plexus. Also “free floating” within the sinus, just lateral to the carotid, is the abducens nerve. In distinction, the third and fourth cranial nerves are located within the dural folds of the lateral wall of the cavernous sinus, along with the first division of the trigeminal nerve. The second division of the trigeminal nerve travels in the dura of the middle fossa just lateral to the cavernous sinus.

This anatomic relationship is maintained as the ocular motor nerves and the first division of the trigeminal nerve travel forward through the superior orbital fissure. Most superior and lateral within this bone canal are the lacrimal and frontal branches of the trigeminal nerve, with the fourth nerve just below. More ventral and medial are the superior division of the third nerve, the nasociliary branch of the trigeminal nerve, the inferior division of the third nerve, the sixth nerve, and the superior ophthalmic vein.

Third Nerve Palsy

Third-nerve palsies may be partial or complete, congenital or acquired, isolated, or accompanied by signs of more extensive neurologic involvement. They can result from muscle lesions anywhere along the anatomic pathway from the nucleus to the end motor organ .

Congenital palsy

Oculomotor nerve palsies present at birth are presumed secondary to maldevelopment, intrauterine injury, or birth trauma. Although relatively rare compared with acquired lesions, they constitute nearly half of the third-nerve palsies documented in children. Typically, they are unilateral and isolated, although bilaterality and accompanying neurologic signs are reported occasionally. Some degree of ptosis and ophthalmoplegia is the rule. The pupil is usually involved (either miotic because of presumed aberrant regeneration or dilated), but pupillary sparing has been noted in some cases. The location of the lesion probably varies among cases. The significant occurrence of aberrant regeneration suggests that some of the lesions are along the peripheral course of the nerve, but modern neuroimaging has demonstrated more central loci. Except in cases of obvious birth trauma in which some recovery is expected, most congenital oculomotor nervalsies are permanent.

Acquired Palsy

Nuclear lesion

Acquired third nerve palsy due to lesions involving the oculomotor nucleus should have a particular constellation of clinical signs reflecting the unique anatomy as described earlier. Although a unilateral, stereotactically placed experimental lesion could conceivably result in bilateral ptosis, contralateral superior rectus dysfunction, and abnormalities of the remaining muscles ipsilaterally, the clinical picture is more likely that of a complete ipsilateral oculomotor nerve palsy with additional contralateral ptosis and superior rectus dysfunction.

If the nuclear lesion is rostral, pupillary involvement is likely and lid function may be spared. Conversely, with caudal lesions, bilateral ptosis may be a prominent or even isolated finding.

The most common cause of lesions of the oculomotor nucleus is vascular compromise, usually a result of thrombotic occlusion of small perforating vessels off the basilar artery or embolic or thrombotic occlusive disease of larger vessels (“top of the basilar syndrome”). Other causes to consider include small intraparenchymal hemorrhage from presumed vascular malformations, metastatic neoplasms, and abscesses.

Fasicular Lesion

Classically, the differentiating feature of fascicular from peripheral nerve lesions has been the accompanying neurologic signs reflecting the fascicles' location within the parenchyma of the brain stem. Several syndromes have been recognized .

Third-nerve palsy and ipsilateral cerebellar ataxia may result from involvement of the fascicles and the brachium conjunctivum (Nothnagel's syndrome).

Oculomotor palsy and contralateral tremor may reflect a lesion in the region of the red nucleus (Benedikt's syndrome).

Third-nerve dysfunction plus contralateral hemiparesis implicates involvement of the ipsilateral cerebral peduncle (Weber's syndrome).

With the advent of more sensitive neuroimaging such as magnetic resonance imaging (MRI), isolated and even pupil-sparing oculomotor nerve dysfunction has been shown to occasionally result from fascicular lesions. MRI has also demonstrated that a lesion of the fascicles can cause isolated dysfunction of either the superior or inferior division of the third nerve. This suggests that functional organization of the oculomotor nerve into divisions may occur within the fascicles prior to the anatomic separation seen grossly within the anterior cavernous sinus. The causes of fascicular oculomotor nerve lesions are nearly identical to those of lesions of the nuclear complex, with vascular causes heading the list, followed by infiltrative and inflammatory causes. Because the fascicles are white matter tracts, the diagnosis of demyelinating disease must also be considered.

Subarachnoid space lesion

The subarachnoid space is the most likely site of injury in cases of isolated oculomotor palsies. Involvement may be partial or complete, although most commonly there is progression to total involvement over time.

Because of the dorsal and peripheral location of the pupillary fibers, a dilated pupil may be the first sign of a compressive lesion in the subarachnoid space.

A common cause of an isolated oculomotor nerve palsy with significant pupillary involvement in adults is an intracranial aneurysm,typically originating at the junction of the posterior communicating and the internal carotid arteries . Almost without exception, there is pain (although sometimes modest) and eventually other evidence of oculomotor

nerve involvement. Other locations of aneurysmal dilatation that have been shown to cause third-nerve palsies include the top of the basilar artery and the junction of the basilar and superior cerebellar arteries.

Diabetic “microvascular” oculomotor palsies are also commonly painful and can have some pupil involvement in 10 to 20 percent of cases.

Other causes of oculomotor nerve dysfunction in the subarachnoid space include compressive or infiltrating neoplasms or inflammatory lesions, ischemia, meningitis (infectious, inflammatory, or neoplastic), compression by large dolichoectatic vessels or cerebral structures shifted by expanding supratentorial lesions or edema, and trauma.

The trauma necessary for third-nerve damage is typically severe enough to have caused skull fractures and loss of consciousness.The oculomotor palsy apparent after minor trauma needs careful evaluation, because it may reflect an underlying mass lesion.

Third-nerve dysfunction may be a component of a generalized polyneuropathy or the Miller Fisher variant of the Guillain-Barré syndrome.

Cavernous sinus lesion

There are no specific distinguishing features of third-nerve involvement in the cavernous sinus. Although bifurcation of the nerve into its two divisions typically occurs in the anterior cavernous sinus, there is evidence that a functional bifurcation occurs more proximally along the course of the oculomotor nerve, probably within the brain stem, making localization of a divisional paresis problematic.

To clinically distinguish a cavernous sinus location of an oculomotor nerve palsy, we must note the accompanying involvement of fourth, fifth and sixth nerve and the oculosympathetics. Dysfunction of the venous drainage of the eye and orbit may be apparent. Pain may be a prominent feature. The pupil may be small or midsized and poorly reactive because of concurrent oculosympathetic involvement. Causes include neoplastic (pituitary tumors, craniopharyngioma, meningioma, nasopharyngeal carcinoma, schwannoma, metastatic lesions), inflammatory (Tolosa-Hunt, sarcoid), aneurysmal compression, ischemia, cavernous sinus thrombosis, and arteriovenous fistulas.

Orbital lesions may cause third-nerve palsies that respect the anatomic bifurcation of the nerve or that reflect individual muscle involvement. Commonly

associated clinical features include proptosis and visual loss. Causes include trauma, neoplasms, and inflammation.

Most studies reviewing the causes of oculomotor nerve palsies do not distinguish isolated from nonisolated palsies, nor do they list causes by location. A large number of cases are recorded as “undetermined” cause and a larger proportion still, those of vascular origin, remain of uncertain location.

Newer, more sensitive neuroimaging modalities, such as MRI, are localizing more of these lesions and even providing clues about pathogenesis, but most third-nerve palsies remain poorly characterized.

Vascular lesions causing oculomotor nerve palsies were believed to occur most frequently along the nerve's subarachnoid or intracavernous course, although fascicular involvement was later demonstrated in some cases. Conditions frequently associated with third-nerve dysfunction include diabetes mellitus, hypertension, giant cell arteritis, systemic lupus erythematosus, syphilis, and migraine. The site of third-nerve involvement in “viral” syndromes remains unknown.

Pupillary sparing in third nerve palsy

The phenomenon of “pupillary sparing” bears special mention. True pupillary sparing implies that each of the extraocular muscles innervated by the oculomotor nerve is involved to some extent, but the pupil remains of normal size and reactivity. Oculomotor nerve palsies without dysfunction of all of the muscles innervated by the third nerve that also do not involve the pupil are not “pupillary sparing.” The distinction becomes very important in management .

The cause of most isolated pupil-sparing third-nerve palsies is believed to be vascular, frequently associated with diabetes mellitus or systemic hypertension. The explanation for this may be anatomic in that the peripherally located pupillary fibers may receive more collateral blood supply than the main nerve trunk. Vascular third-nerve palsies may be quite painful but usually resolve after 2 to 4 mo. Rarely, isolated pupillary-sparing oculomotor nerve palsies may be secondary to compressive lesions, although the majority of these cases have incomplete palsies.

Special Syndromes

CYCLIC OCULOMOTOR PARESIS

This is an uncommon but dramatic condition typically seen in patients with known congenital third-nerve palsies. The classic scenario is that of alternating baseline paresis with episodes lasting seconds of pupillary miosis, increased accommodation, elevation of a previously ptotic upper lid, and adduction of the eye.

ABERRANT REGENERATION

Although this phenomenon has been demonstrated or suspected in virtually every peripheral nerve that has had partial damage, the oculomotor nerve is particularly interesting in this regard because of its many branches and target structures.

Signs include elevation of the upper lid with attempted downgaze (pseudo-Graefe's sign) , upgaze, or adduction, segmental constriction of the pupil with movement in the direction of action of muscles innervated by the third nerve, retraction of the globe with attempted vertical gaze (presumably secondary to co-contraction of the superior and inferior recti), or adduction of the eye with attempted up- or downgaze.

Oculomotor synkinesis is generally believed to reflect the misdirection of regenerating fibers after partial damage to the peripheral portion of the nerve. Oculomotor nerve synkinesis is most commonly seen 2 to 3 mo after injury to the nerve by trauma or compression from aneurysms or tumors. Ischemic lesions (i.e., secondary to diabetes) do not result in aberrant regeneration, and the presence of synkinesis requires a careful search for a nonischemic cause.

Slowly growing mass lesions are likely to be responsible for the phenomenon of “primary” oculomotor nerve synkinesis, in which a recognizable paretic phase does not precede development of the aberrant movements.

All patients under the age of 40 who present with an isolated third-nerve palsy of any extent should also have complete neurologic evaluation, including a cerebral angiogram. There is some controversy about the application of this rule to children under the age of 10, in whom aneurysms are extremely rare.

Patients over the age of 40 who present with an isolated, pupil-sparing, but otherwise complete third-nerve palsy, even in the presence of pain, can usually be assumed to have a vasculopathic etiology. Minimal work-up in the known diabetic

patient would consist of a measurement of systemic blood pressure, serum glucose, and sedimentation rate. If there is no history of diabetes, a glucose tolerance test or a serum hemoglobin Alc level should be obtained. These patients must be observed closely for the next week for evidence of pupillary involvement.

The patient over age 40 with an isolated complete oculomotor nerve palsy with pupillary involvement or a partial third-nerve palsy presents the most difficult management issue. All these patients should have at least the minimal blood work-up as outlined earlier and a neuroimage performed, preferably the more sensitive MRI. The majority of these patients ultimately require a cerebral arteriogram. The use of new noninvasive techniques of MRI angiography in this setting awaits further experience .

The majority of third-nerve palsies of ischemic etiology resolve within 3 months. Compressive or traumatic oculomotor nerve palsies may take longer to improve, and incomplete recovery with or without synkinesis is more likely. Despite rare reports of continued improvement in third-nerve palsies years after onset, once the deficit has stabilized (usually within 6 months after injury) it is unlikely that there will be further recovery.

FOURTH NERVE PALSY

Trochlear nerve palsies are the most common cause of vertical malalignment of the eyes. Given the mechanical arrangement of the superior oblique muscle, this vertical deviation is worse with attempted downgaze and gaze to the side opposite the paretic muscle.

This is the basis for the first two steps of the “Three-Step Test” in which first the higher eye is identified (reflecting the relative decrease in tonic downward input from the superior oblique), then the direction of horizontal gaze in which this vertical deviation worsens is noted. In the third step, the patient's hyperdeviation is compared on head tilt left and right.

Patients typically compensate for a superior oblique palsy by tilting the head to the opposite side. Another clue to trochlear nerve paresis in the observant patient is a

torsional component to the vertical diplopia: The two vertically separated images are not parallel, but rather approach each other as if to meet in the direction of the involved eye.

Detection of bilateral fourth-nerve palsies may be difficult, especially if the damage is asymmetric and the less involved nerve paresis is "masked." Clues to bilaterality include greater than 12 degrees of subjective excyclotorsion, a chin-down posture, lack of head tilt, a large "V" shift, observation of fundus torsion on ophthalmoscopy, and a reversal of hypertropia, however minimal, on cover testing in the oblique fields of gaze or on contralateral head tilt.

Other causes of vertical strabismus, such as paralysis of more than one vertical muscle, dissociated vertical divergence, previous muscle surgery, contracture of the vertical recti, myasthenia gravis, thyroid ophthalmopathy, and skew deviation, may have a positive three-step test and cause errors in diagnosis. History and associated findings on examination, as well as careful observation of versions into all the diagnostic fields of gaze, help to distinguish isolated trochlear palsy from these other conditions.

Fourth nerve palsy can be congenital or acquired.

Congenital

Although the cause of congenital trochlear nerve palsies is unknown, they occur commonly. They may remain compensated and unrecognized during childhood, only to become manifest later either spontaneously or after minor trauma. Clues to their congenital nature include a head tilt seen on old photographs and the demonstration of large vertical fusional amplitudes (nonstrabismic patients usually only fuse 3- to 6-prism diopters vertically).

Acquired

Involvement of the trochlear nerve at the nuclear or fascicular level may be recognized by associated brain stem findings. If the process occurs before the decussation of the fascicles, the superior oblique palsy is seen contralateral to the other parenchymal signs such as a "first-order neuron" Horner's syndrome. The causes of such lesions include small infarcts and hemorrhages, intrinsic mass lesions, and external compression or trauma.

Bilateral fascicular damage at the level of the superior medullary velum may be a primary site of trochlear nerve damage secondary to trauma or hydrocephalus. Trauma is the most common cause of bilateral fourth-nerve palsy.

The dorsal exit of the trochlear nerve and its long course around the brain stem and along the tentorial edge make it particularly susceptible to the effects of trauma and neurosurgical manipulation. It is here in the subarachnoid space that inflammatory and possibly ischemic processes may result in fourth-nerve dysfunction.

Lesions of the fourth nerve within the cavernous sinus are rarely if ever isolated. Associated oculomotor, abducens, or trigeminal nerve or oculosympathetic dysfunction helps to localize the problem. Neoplastic, inflammatory, and infiltrative causes are essentially the same as those discussed earlier in relation to oculomotor nerve abnormalities in the same location.

Neoplastic, inflammatory, ischemic, and traumatic processes within the orbit may affect the trochlear nerve, the superior oblique muscle, or the trochlear tendon or its sheath.

It is likely that the actual incidence of fourth-nerve palsy is higher than usually reported, reflecting the frequent difficulty in routine recognition and diagnosis. When an etiology is known, the most common cause is trauma, although the exact locus of involvement remains unclear.

The main differential diagnosis of isolated trochlear nerve paresis is skew deviation, ocular myopathies, and myasthenia gravis. A fourth-nerve palsy can usually be distinguished from skew deviation by the latter's frequent association with other brain stem signs or symptoms and the former's torsional diplopia with cyclodeviation.

Orbital myositis affecting only the superior oblique is typically very painful. Disorders of the neuromuscular junction can mimic any single ocular muscle paralysis, and diagnosis depends on the presence of diurnal variation and ultimately the development of other associated signs.

The nonisolated fourth-nerve palsy requires an evaluation determined by the presumed location of the lesion. Thus, if a mesencephalic or cavernous sinus locale is likely, MRI is indicated. If a meningeal process is suspected, cerebrospinal fluid analysis is necessary. Orbital lesions may be best evaluated with high-resolution CT scanning.

The majority of acquired trochlear nerve palsies of traumatic, vascular, or “undetermined” etiology improve over time, usually within 6 mo. In the interim, monocular occlusion or vertical prisms may help the patient symptomatically.

Sixth Nerve Palsy

Although the abducens nerve may appear to be a simple arrangement of one other cranial nerve supplying a single ipsilateral extraocular muscle, the unique organization of its nucleus and the surrounding brain stem structures provides for a rich assortment of congenital and acquired clinical syndromes. An examination of the patient with suspected sixth-nerve palsy must include a close inspection of the function of the oth s of pontine origin and of the motility of the contralateral eye.

Congenital

A unilateral, isolated, congenital abduction deficit is an unusual finding, which is usually transient and most likely related to birth trauma. Less rare, but also uncommon, are isolated congenital horizontal gaze palsies. The most common constellation of congenital findings involving sixth-nerve dysfunction fall under the headings of Möbius' syndrome and Duane's retraction syndrome.

In Möbius' syndrome, facial diplegia is associated with abnormalities of horizontal gaze, usually complete absence of horizontal motility. Head movements and convergence (if preserved) are substituted.

Duane's retraction syndrome is characterized by unilateral or bilateral abduction deficits, variable abnormalities of adduction, and globe retraction with consequent palpebral fissure narrowing on attempted adduction. Clinical findings are bilateral in about one fifth of patients. Most patients are female and the left eye is involved more often than the right. Visual abnormalities such as diplopia or amblyopia are surprisingly infrequent.

Acquired

Because of the diffuse intermixing within the sixth-nerve nucleus of neuronal cell bodies that project to the ipsilateral abducens nerve and those whose axons are destined

for the contralateral medial longitudinal fasciculus, lesions of the nucleus result in abnormalities of ipsilateral horizontal gaze. Furthermore, the close proximity of the facial nerve fascicle as it loops around the abducens nucleus frequently results in a peripheral facial palsy as part of the clinical picture. The causes of lesions of the abducens nucleus are infarction; infiltration; hemorrhage; trauma; and neoplastic, infectious, or inflammatory compression In addition, the presumed metabolic lesions in Wernicke-Korsakoff syndrome are believed to involve the sixth-nerve nucleus.

Abducens nerve dysfunction secondary to involvement of the fascicle should be recognizable by the associated brain stem findings. Several specific syndromes reflecting particular localization have been described.

Foville's syndrome combines an abduction or horizontal gaze deficit with ipsilateral facial weakness, ipsilateral loss of taste, ipsilateral facial analgesia, ipsilateral Horner's syndrome, and ipsilateral deafness. The structures affected are the abducens fascicle or nucleus, the facial nerve fascicle, the nucleus of the tractus solitarius, the spinal tract of the trigeminal nerve, the central tegmental tract, and the cochlear nuclei, respectively.

If the lesion is located more ventrally in the pons, Raymond's syndrome or Millard-Gubler syndrome may be manifest.

Raymond’s syndrome is a combination of sixth-nerve palsy and contralateral hemiplegia secondary to involvement of the abducens fascicle as it courses through the ipsilateral pyramidal tract.

Millard-Gubler syndrome combines an abduction deficit with contralateral hemiplegia and ipsilateral facial paralysis, implying involvement of the abducens fascicle, the pyramid, and the facial nerve fascicle.

Causes of fascicular damage are the same as those listed for nuclear lesions, with the addition of demyelinating disease.

Abducens nerve lesions in the subarachnoid space may be isolated or associated with other peripheral cranial nerve involvement, unilateral or bilateral. Meningeal processes, including infectious, inflammatory, and neoplastic etiologies, may present with abduction deficits.

Elevated intracranial pressure from any cause frequently results in unilateral or bilateral sixth-nerve palsies, presumably from nonspecific “stretching” of the nerves along their course between the pons and the petrous apex.

The same pathogenesis may be implicated in cases of abducens palsy following trauma, neurosurgical manipulation, cervical traction, and lumbar puncture.

Tumors may involve the subarachnoid portion of the sixth nerve, either in isolation (e.g., with clivus chordomas, nasopharyngeal carcinomas, intrinsic neurinomas, and meningiomas) or with evidence of other neurologic abnormalities, as typically seen with exophytic intrinsic posterior fossa tumors or acoustic neuromas .

Vascular compression of the nerve by dolichoectatic vessels is also possible. Although less common than third-nerve involvement, aneurysmal compression may result in a sixth-nerve palsy, almost always in association with a severe headache.

GRADINEGO′S SYNDROME

Where the sixth nerve enters the dura above the clivus and along the petrous ridge, it is susceptible to processes involving the adjacent mastoid air cells, such as mastoiditis. The resultant symptom complex, Gradenigo's syndrome, reflects inflammation in this region and consists of abducens palsy, severe facial and eye pain (from involvement of the adjacent gasserian ganglion), and sometimes facial paralysis.

PSEUDO GRADINEGO′S SYNDROME

Lateral sinus thrombosis with inferior petrosal sinus involvement or tumors along the petrous ridge may mimic this syndrome. This is presumably also the site of traumatic sixth-nerve palsies associated with basal skull fractures of the temporal bone.

The combination of abducens palsy and loss of tearing with or without involvement of the second division of the trigeminal nerve localizes the lesion to the sphenopalatine fossa. The cause is commonly metastatic tumor or nasopharyngeal carcinoma.

In the cavernous sinus, the sixth nerve is susceptible to involvement by all the same processes mentioned earlier with regard to third- and fourth-nerve lesions in this location. The particular location of the abducens nerve floating relatively freely within the substance of the sinus rather than within a flap of dura, however, makes it frequently

the first inhabitant of the cavernous sinus to be involved by any of these pathologic processes.

This is particularly true when the cause of the lesion is vascular, such as carotid cavernous fistulas, dural shunts, and intracavernous aneurysms.

Ischemia, inflammation, both infectious and noninfectious, and neoplasms may also involve the intracavernous sixth nerve, usually in association with other cranial nerve involvement.

The combination of oculosympathetic dysfunction and ipsilateral abduction deficit is usually localized to the cavernous sinus, where the sympathetics anatomically run with the abducens nerve over a short course.

Isolated orbital involvement of the abducens nerve is rare because of the relatively short course of the nerve prior to its innervation of the lateral rectus muscle. An orbital location of paralysis has been postulated for those sixth-nerve palsies occurring after dental anesthesia.

As with presumed ischemic lesions of the oculomotor and trochlear nerves, the exact location of isolated sixth-nerve palsies that occur in the setting of diabetes mellitus, hypertension, giant cell arteritis, or migraine remains unknown. It is probable that the majority of these lesions localize to the subarachnoid or intracavernous portion of the sixth nerve. As noted earlier, however, at least some of these ischemic lesions involve the fascicular, intraparenchymal nerve. The transient abducens palsy, which is seen especially in children, is presumed to be of viral or postinfectious origin and is also of indeterminant location.

Sixth-nerve palsies in conjunction with other cranial nerve or neurologic involvement require sophisticated neuroimaging, ideally MRI for a sensitive view of the brain stem and cavernous sinus. Processes that localize to the subarachnoid space require cerebrospinal fluid analysis, including measurement of the opening pressure, and possibly cerebral angiography. If facial pain or numbness is an associated feature, immediate neuroimaging of the petrous ridge is appropriate.

In children with or without an antecedent viral illness or vaccination, observation alone is sufficient after careful neurologic assessment. Initially, an examination every 2 wk is necessary to determine if progression or additional neurologic involvement occurs.

In the elderly population, an isolated sixth-nerve paresis is likely to be ischemic in etiology and therefore ultimately transient and not indicative of underlying neurologic disease. A minimum work-up would include a glucose tolerance test (or serum glucose measurement in the already diagnosed diabetic), and an erythrocyte sedimentation rate, looking for evidence of giant cell arteritis. Even those isolated ischemic sixth-nerve palsies localized by MRI to the fascicular portion of the nerve usually recover, suggesting little prognostic importance in determining the exact location of these lesions.

The initial evaluation of the young adult who presents with a truly isolated sixth-nerve palsy remains a controversial issue. Most clinicians obtain at least a neuroimage and possibly a lumbar puncture on these patients, although the majority of these work-ups prove to be negative. A thorough systemic and neurologic evaluation is mandatory. Bilateral sixth-nerve palsies suggest elevated intracranial pressure or a meningeal process and require neuroimaging. If the result is negative, lumbar puncture should be done.

Presumed ischemic abducens lesions generally resolve within 3 to 4 months of onset. Similarly, the isolated sixth-nerve palsies in children usually completely recover within 4 months. These patients must be followed carefully to ensure nonprogression and continued clinical isolation. If at any time additional neurologic abnormalities develop, more extensive investigation should be pursued. The chronic, isolated sixth-nerve palsy may reflect slow-growing basilar skull neoplasms, such as schwannomas or meningiomas.

Simplest management for a sixth-nerve palsy is monocular occlusion, sometimes only over the portion of the temporal field in which horizontal diplopia occurs. Prisms are rarely helpful. After a sixth-nerve palsy has stabilized and underlying causes have been either ruled out or deemed nonprogressive, various surgical procedures can be performed to better align the eyes in primary position. Most surgeons prefer 6 to 12 mo of stable measurements before intervention.

The use of botulinum toxin in the treatment of abducens nerve palsies has added a new therapeutic option for transient symptomatic relief. The toxin is injected into the ipsilateral unopposed medial rectus muscle, thus reducing the tonic opposition to the weak lateral rectus and allowing for fusion. It is possible that the use of this treatment in acute lesions may reduce the tendency for fibrosis and contracture to develop.

MULTIPLE CRANIAL NERVE PALSY

A special situation exists when more than one ocular motor nerve is involved either unilaterally or bilaterally. Of primary importance is the exclusion of processes that may mimic neurogenic palsies, such as ocular myopathies and disorders of neuromuscular transmission. Once the problem has been diagnosed as multiple ocular motor nerve paralysis, localization is paramount. Common locations for multiple infranuclear nerve involvement are the subarachnoid space and the cavernous sinus/superior orbital fissure.

Although the brain stem contains all the ocular motor nerves and their nuclei, it would be difficult to have a pathologic process that involved the cranial nerves without also affecting the adjacent brain stem parenchyma with resultant disorders of supranuclear motility and neurologic motor and sensory dysfunction. There are rare reports of patients with severe amyotrophic lateral sclerosis who have abnormal ocular motility, possibly on the basis of lower ocular motor neuron degeneration, although supranuclear mechanisms may be primary.

Meningeal disease, however, may affect multiple cranial nerves with minimal systemic or neurologic abnormality. Infectious and neoplastic seeding of the subarachnoid space can result in dysfunction of numerous cranial nerves, including the ocular motor nerves. Noninfectious inflammatory causes, such as neurosarcoidosis and Behçet's syndrome, have also been implicated. Head trauma with presumed shearing of multiple cranial nerves, base of the skull tumors, particularly along the clivus, and possibly dolichoectatic vasculature may also result in a picture of multiple cranial neuropathies.

The anatomy of the cavernous sinus makes it a likely location of lesions resulting in multiple ocular motor nerve dysfunction. Traversing this paired trabeculated structure that flanks the sphenoid sinus and pituitary fossa are the third, fourth, and sixth cranial nerves, the first and, posteriorly, the second divisions of the trigeminal nerve, and the carotid artery with its surrounding sympathetic plexus . In close proximity is the pituitary gland medially within the sella turcica and the optic nerves and chiasm superiorly. Disease processes within or adjacent to the cavernous sinus can result in clinical

syndromes reflecting dysfunction of various combinations of these structures, both unilateral and bilateral. To clinically distinguish cavernous sinus from superior orbital fissure syndromes is difficult and is of questionable value. The same structures continue forward into the fissure, and the disease processes usually do not respect any anatomic boundary.

Of the ocular motor nerves, the sixth nerve is probably affected most commonly by lesions intrinsic to the cavernous sinus because of its relatively free-floating position. Because of its more superior position adjacent to the pituitary fossa, the third nerve may be the first to be involved with lateral expansion of tumors within the pituitary fossa. The combination of oculosympathetic and oculomotor nerve dysfunction may result in a small or midsized, poorly reactive pupil. Blockage of venous outflow can cause proptosis, ocular chemosis, and infection.There may be concurrent visual loss and visual field defects localizing to the optic chiasm or posterior optic nerves. Pain is a common manifestation of cavernous sinus disease and likely reflects involvement of the trigeminal nerve.

The disease processes that can involve the cavernous sinus are many and varied. They fall into three major categories: neoplasms, vascular lesions, and inflammation. In allthe three, the lesions may result from disease intrinsic to the sinus or from compression or extension from adjacent structures. Lateral extension of pituitary adenomas may result from either tumor growth or sudden expansion from pituitary apoplexy. Meningiomas, craniopharyngiomas, nasopharyngeal carcinomas, and metastatic tumors comprise the other common neoplasms in this region. Schwannomas within the sinus usually originate from branches of the trigeminal nerve, although the ocular motor nerves are rarely implicated.

Vascular lesions within the cavernous sinus include aneurysms, arteriovenous fistulas, and venous thrombosis. The last two commonly result in bilateral clinical manifestations. Aneurysms within the sinus are frequently large upon presentation and cause much of their dysfunction by compression of adjacent structures.

Cavernous sinus thrombosis presents with a clinical picture similar to carotid-cavernous fistulas, but the patients commonly have prominent systemic manifestations of sepsis. Paranasal sinus and local skin infections are the most frequent cause. Fungal infections, such as mucormycosis and aspergillus, may mimic cavernous sinus

thrombosis, because their pathogenesis probably also involves some element of thrombophlebitis and obstruction of venous outflow.

Inflammation within the cavernous sinus may result from infectious causes or be idiopathic.

Ophthalmoplegia associated with herpes zoster infection may be localized to the cavernous sinus. The third, fourth, and sixth nerves may be involved, either isolated or in combination, and frequently to partial degree.

In the Tolosa-Hunt syndrome, nonspecific, noninfectious granulomatous inflammation results in hemicranial or periorbital pain associated with ipsilateral ophthalmoplegia of any or all of the ocular motor nerves. Sensory loss in the distribution of the ipsilateral ophthalmic or maxillary division of the trigeminal nerve and oculosympathetic paralysis may also be present. Typically, there is a dramatic response of symptoms and signs to corticosteroid therapy.

AIM OF THE STUDY

1 . To analyse the various etiologies and pathogenic factors leading to multiple ocular motor nerve paralysis.

2 . To analyse the common clinical patterns arising in the background of acquired multiple ocular motor nerve palsy .

3 . To analyse the recovery pattern in multiple ocular motor nerve palsies and to compare this with that of isolated nerve palsies.

MATERIALS AND METHODS

The study was conducted at the Regional institute of ophthalmology and Government Ophthalmic Hospital in Chennai. The period of study was from august 2003 to September 2005.

All patients with paralytic squint have been included in the study .

Congenital cranial nerve palsies have been excluded .

Patients with incomitant squint due to myogenic, myasthenic and restrictive causes have been excluded .

HISTORY TAKING

Patients with paralytic squint were identified and details regarding their name, age, sex, symptoms, and its duration and any change in the symptoms between their presentation and their onset have been recorded.

Detailed history regarding the incidences that preceded the onset of symptoms like trauma [trivial or severe] , headache fainting attacks ,numbness, etc., was taken .

Past history of any previous episodes of similar nature and the treatment given for the same has been noted .History of systemic illnesses like hypertension, diabetesmellitus, thyroid abnormalities, and seizure disorder and previous neurological involvement in any other disorders like tuberculosis ,syphilis, were noted.

History specific to ocular complaints such as double vision , blurring of vision ,field defects ,and vestibular complaints like vertigo ,tinnitus,ear discharge ,and bleeding per ear have been recorded .

Personal history regarding smoking, alcohol intake and diet pattern were asked and recorded .

GENERAL EXAMINATION.

Detailed general examination with regard to build,nutritional status, orientation and presence of., anaemia ,lymphadenopathy., and hepatosplenomegaly has been done for all patients. Blood pressure and pulse rate has been recorded in sitting position and in the supine position .

EXAMINATION OF THE EYE

A thorough clinical examination of eye was done including examination of the head for any position change like head tilt or face turn or chin position change .Abnormalities of the skull and asymmetry of the face were looked for. .Lid was examined for ptosis, lid retraction, synkinetic movements like Marcus Gunn jaw winking phenomenon.

Eye movements were examined and position of the eye and presence and amount of deviaton in all cardinal positions of gaze were noted and angle of deviation was recorded in prism dioptres with each eye fixing[primary and secondary angle of devition].The angle of deviation has been recorded with prism bar cover test. The range and restrictions of eye movements were recorded and the underaction and overaction were graded 1 to 4 as defined by Guibour. Bell's eye phenomenon was tested to rule out supranuclear palsy .Presence of nystagmus and past pointing were looked for and noted if any.

The anterior segment was carefully examined with slit lamp and pupil was examined for size, shape, reaction to direct and consensual light, and reaction to accommodation were noted

The unaided visual acuity and best corrected visual acuity were recorded. Colour vision was recorded using Ishihara colour plates . Both the central and peripheral fields were tested and recorded .Fundus was examined with direct and indirect ophthalmoscopy and abnormalities were recorded.

Diplopia charting wearing red-green goggles was done .Hess screening was done in all those patients who were reviewed for recovery .

NEUROLOGICAL EXAMINATION

A complete neurological examination was done regarding the higher functions, all other cranial nerves ,motor and sensory systems ,reflex systems and cerebellar functons .

INVESTIGATIONS

A complete haemogram ,urine analysis for albumin, sugar,and deposits ,blood sugar VDRL, Mantoux test , X-ray of skull(both anteroposterior and lateral)and paranasal sinuses and optic foramen has been done for all patients . CT-scan was done in almost all trauma cases and in other cases whenever possible .MRI-scan was done whenever it was possible. Certain special tests like neostigmine test has been done in certain cases to diagnose and to differentiate myasthenia gravis from nerve palsies.

Follow up of these cases has been done at the end of 4 weeks , 8weeks, 12weeks,and 6 months in case of patients living nearby and at an interval of 4weeks , 12weeks , and 6months in case of patients coming from far off places

RESULTS

Age Distribution

S.No	AGE	Third nerve	Fourth nerve	Sixth nerve	Total	3&6	3&4	3,4&6	Total
1	13to20	3	0	6	9	0	1	2	3
2	21to40	3	3	8	14	1	4	6	11
3	41to60	5	2	9	16	1	3	4	8

4	61to80	2	2	5	9	2	4	4	10
		13	7	28	48	4	12	16	32

Of the 80 cases of acquired ocular motor nerve palsy cases, isolated nerve palsy cases constitute 60 % (48 cases) and multiple nerve palsy cases constitute 40% (32 cases) .In this study , maximum number of isolated nerve palsy cases has occurred in the 5th and 6th decades while that of multiple nerve palsy cases has occurred in the 4th and 5th decades .Maximum number of third and sixth nerve palsies has occurred in the 5th and 6th decades while that of 4th nerve palsy has occurred in 3rd and 4th decades, and in the case of multiple nerve palsies 60% of the cases had occurred between 5th and 8th decades reflecting ischaemia as the most common etiology in this age group .Of all the isolated nerves involved , sixth nerve is the most common nerve involved . Amongst cases of multiple nerve palsies most common pattern of involvement is involvement of 3rd ,4th and 6th nerves i.e., involvement all the three nerves .

Gender distribution

		Isolated	Isolated	Multiple	Multiple
S.No.	Age	male	female	male	female
1	13 to 20	4	5	1	2
2	21 to 40	8	6	6	5
3	41 to 60	9	7	7	4
4	61 to 80	5	4	4	3
Total		26	22	18	14

Of the 80 cases taken for study , 44 cases were males and 36 cases were females and in percentage , there is a slight male preponderance with 55% of isolated cases and 56 % of multiple nerve palsy cases being males . There is uniform distribution among all age groups studied except for 1 3 to 20 age group where there is slight female preponderance .

Distribution Of Nerve Palsies.

S.No	AGE	Third nerve	Fourth nerve	Sixth nerve	Total	3&6	3&4	3,4&6	Total
1	13to20	3	0	6	9	0	1	2	3

2	21to40	3	3	8	14	1	4	6	11
3	41to60	5	2	9	16	1	3	4	8
4	61to80	2	2	5	9	2	4	4	10
		13	7	28	48	4	12	16	32

Of the 48 cases of isolated nerve palsies , maximum of cases belonged to 6th nerve palsies (28 cases-58.3%) . Of next common occurrence was 3rd nerve palsies .The least common cases of isolated nerve palsies belonged to isolated 4th nerve palsy (7Cases-14%) . Of the 32 cases of multiple ocular motor nerve palsies, most common pattern of involvement being that of 3rd 4th &6th nerve palsies.

Laterality

S .No.	Eye	Third	Fourth	Sixth	Multiple	Total
1	Right	8	3	12	12	35
2	Left	5	4	11	14	34
3	Both			5	6	11
	Total	13	7	28	32	80

In this study of the total 80 cases, in 35 cases right eye was involved and in 34 cases left eye was involved .In 11cases both eyes were involved .In this study there was bilateral involvement of isolated sixth nerve palsies, but there were no cases with bilateral isolated 3rd nerve palsy and bilateral isolated 4th nerve palsy .There were 6 cases of multiple nerve palsies where both eyes were involved .

Symptoms At Presentation

S.No.	Symptoms	No. of patients
1	Double Vision	71
2	Drooping of lids	45
3	Headache	61
4	Pain in the eye	22
5	Fits &H/of LOC	17
6	Ear Discharge	38
7	Vestibular symptoms	18
8	Defective vision	29
9	Fever Prior to onset	20

Most common symptom that has been noticed in the study population of 80 patients was diplopia or double vision followed next in order by ptosis & headache .One fourth of the patients had painful eyes and nearly half of the cases had ear discharge

and defective vision was complained by 29 cases i.e., around one third of the study population. Head injury and loss of consciousness were reported in 17 patients i.e.,21% of the study population.

Etiology of acquired nerve palsy

S.No.	Etiology	No. of cases	Percentage
1	Ischaemia	22	27.5%
2	Trauma	24	30%
3	Tumour	05	6.25%
4	Aneurysm	03	3.75%
5	Undetermined	26	32.5%

Among the etiologies given in the table, apart from undetermined causes the commonest etiology was trauma which was followed by ischaemia.Tumour and aneurysm were less common causes .

Among those patients with nerve palsy of undertermined origin, ability to do higher investigations like MRI and CT SCAN might be revealing the exact lesion responsible.

Etiology Of Isolated and Multiple Nerve Palsy

Etiology	Isolated Ner. Palsy	Multiple N.Palsy
Ischaemia	11	11
Trauma	17	07
Tumour	03	02
Aneurysm	03	
Etiology known cases –total	34	20
Undetermined	14	12
Total	48	32

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Of the 80 cases taken for the study etiology was determined with certainity in 54 cases (67.5%) and the cause was not detected in 24 cases (32.5%)either due to unknown etiologies or due to economical inability to perform higher investigations.

Of those patients with known etiology, trauma was the commonest cause among the isolated nerve palsy cases. And among the multiple nerve palsy cases ischaemia was the commonest cause.

Isolated nerve palsy-Distribution of etiologies

Etiology	Third N.	Fourth	Sixth	Total	Percentage
Ischaemia	5(38.6%)		6(21%)	11	23%
Trauma	3(23%)	4(57.5%)	10(36%)	17	35.5%
Tumour	1(7.7%)	1(14%)	1(3.5%)	3	6.25%
Aneurysm	1(7.7%)		2(7%)	3	6.25%
Undetermined	3(23%)	2(28.5%)	9(32.5%)	14	29%
Total	13	7	28	48	

Of the 48 cases of isolated nerve palsy, sixth nerve palsy was the commonest .Trauma was the most common cause (36%) followed by etiology of undetermined origin (32.5%). Ischaemia was third common cause (21%).

Of the 13 cases of third nerve palsy ischaemia was the most common (38.6%) cause followed by trauma (23%).Of the rest 38.4% of cases, 23% of third nerve palsy cases were of etiologies from unknown origin. Tumour and trauma constitute 7.7% of cases each.

Of the 7 cases of isolated fourth nerve palsy cases included in the study, the most common etiology was trauma (57.5%) which was followed by causes of unknown origin(28.5%) and tumour (14%-one case).

Multiple nerve palsy-Distribution of etiologies

Etiology	Multiple nerve palsy cases	Total(isolated and multiple)	Percentage of etiology due to multiple nerve palsy
Ischaemia	11	22	27.5%
Trauma	7	24	30%
Tumour	2	5	6.25%
Aneurysm		3	3.75%
Undetermined	12	26	32.5%
Total	32	80	

Among those cases of multiple nerve palsy,20 patients had known etiologies. Of these 20 cases with known etiologies, the most common being ischaemia and the next common being trauma, while two patients had tumours causing multiple nerve palsy. None of the patients of multiple nerve palsies with known etiologies (20 cases) had aneurysm.

RECOVERY PATTERN

Pattern	Third Nerve	Fourth Nerve	Sixth Nerve	Multiple Ns	Total	Percentage
Fully Recovered	7	1	11	10	29	36.25%
Partially Recovered	2	2	7	9	20	25%
Not Recovered(*)	4(2+2)	4(1+3)	10(4+6)	13(4+9)	31(11+20)	38.75%(13.75%+ 25.0%)
Total	13	7	28	32	80	100%

(*-not recovered cases+ cases who lost follow up = 11+20)

In about 50% of cases included in the study , there was recovery; though half of those cases recovered ,showed only partial recovery.

Those cases with complete recovery, the etiology was either microangiopathy due to diabetes or hypertension or nonspecific neuritis . These patients showed almost full or at least partial recovery at 6 months.

Those patients with traumatic etiologies there was a poor recovery pattern with partial recovery more common than complete recovery.

Of the 80 cases, 25% 0f cases lost follow up and hence of the 75% 0f cases(60 cases) followed ,14% of the cases did not recover and 66% recovered either partially or completely.

Of the 48 cases of isolated nerve palsy, 40% of the cases showed complete recovery, while 22.5 % of cases showed only partial recovery14.% of cases did not recover and 23.5% of cases lost follow up.

Of the 32 cases multiple nerve palsy cases, 32% of cases showed complete recovery ,while 28% of cases showed only partial recovery.12.5% of cases did not recover and 27.5% of cases lost follow up.

DISCUSSION

Ocular motor nerve palsy either isolated or multiple can lead to certain diagnostic and therapeutic dilemma ,i.e, whether to treat the primary cause or not, when to treat and when to interfere with regard to ocular complaints.

In the following discussion, our study has been compared with the following studies namely, Rush JA,Younge BR,(1981-Jan), Sheu YJ,Lin LL, Ko LS , (Taiwan-Jul-1986), Carlow TJ(Jan-1989), Berlit P(Jan-1991), Kubatko-Zielinska (May-1995), Sowka J(Jan-1996), and Tiffin PA, MacEven CJ(Jan-1996).

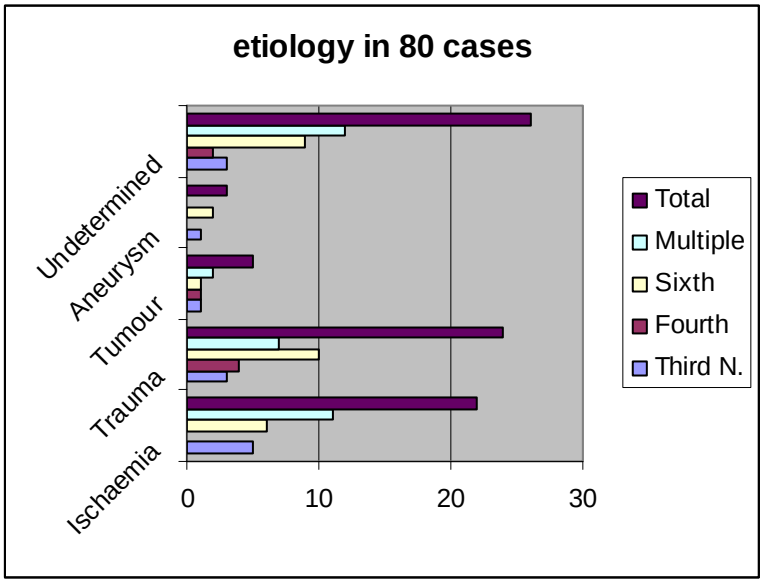
Rush JA and Younge BR analysed 1000 cases of acquired 3^{rd} , 4^{th} , and 6^{th} cranial nerve palsy ,retrospectively. The number of patients with no known cause for paralysis was surprisingly high (>25%) inspite of availability of CT-Scaning for all the patients.And 51% of the patients with unknown etiology had spontaneous remission. Cranial nerve palsy secondary to vascular cause was only temporary in 71% of cases (i.e, full recovery) irrespective of the cranial affected. Patients with palsies due to aneurysm trauma and tumour were less likely to recover.

In the study conducted by Kubatko-Zielinska et al,120 patients with acquired cranial nerve palsy were studied . There was a male preponderance .And trauma was the major cause of nerve palsy in the age group of 21 to 40 years. The most common symptom at presentation was diplopia.

In the study conduted by Berlit P, 412 patients of both isolated and combined nerve palsy were analysed in a retrospective study. Here third nerve and fourth nerve palsies were of almost equal incidence i.e,41% and 40% respectively. Of the patients with combined nerve palsy the common patterns of involvement were combinations of third and sixth cranial nerve palsy(42%) and pareses of all three cranial nerves(34%). In cases of oculomotor nerve palsy, there was pupillary sparing in about 70% of cases. In inflammatory diseases and brain tumour, 6^{th} nerve palsy was commonest to occur , while in palsy of aneurysmal origin third nerve was the commonest to be involed . In about 50% of cases , there was complete recovery and in about 15% of cases there was partial recovery. The most favourable prognosis was with inflammatory and vascular lesions.

In the study conducted by Tiffin PA,MacEvenCJ,CraigEA,and Clayton G, retrospective analysis of 165 cases had been done. Sixth nerve palsies were the commonest to occur. Next common was third nerve palsy. Most common etiology was due to undetermined causes (35%) followed by vascular etiology (32%) which by the way was the most common cause among the patients with known etiology. 80% of the patients showed recovery (at least partial) though only 57% showed complete recovery

In our present study, 80 cases have been included, the most common etiology has no known origin .It is in concordance with the results of the study conducted by Tiffin et al and also with the reports of the study conducted by Richard, Jones and Younge from Mayo clinic (4298 cases) which states that the largest group had palsy of unknown origin and the commonest nerve involved was sixth nerve.



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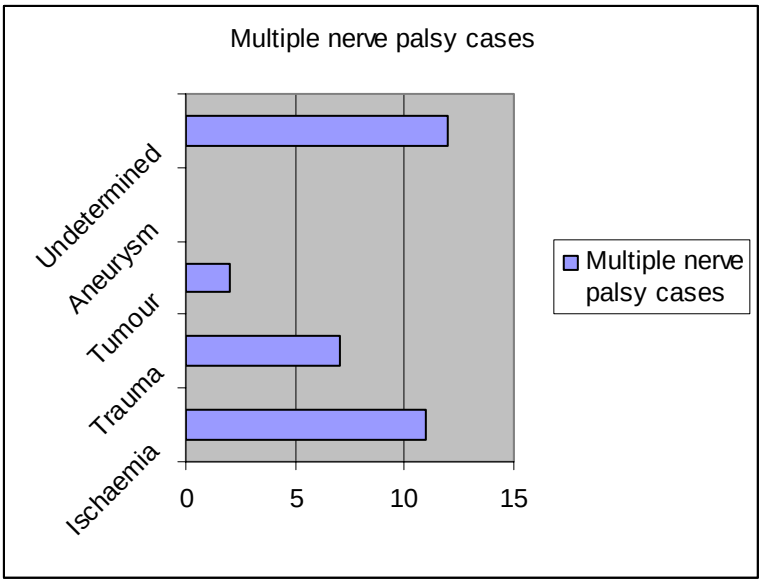
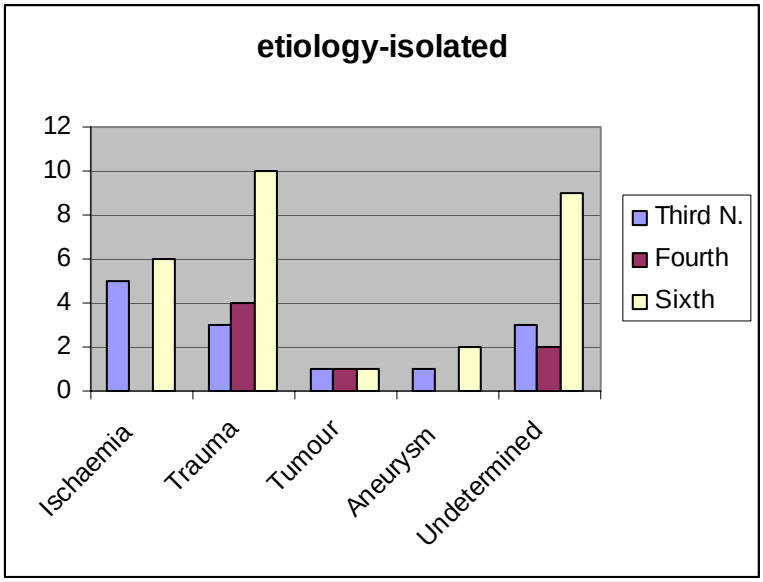
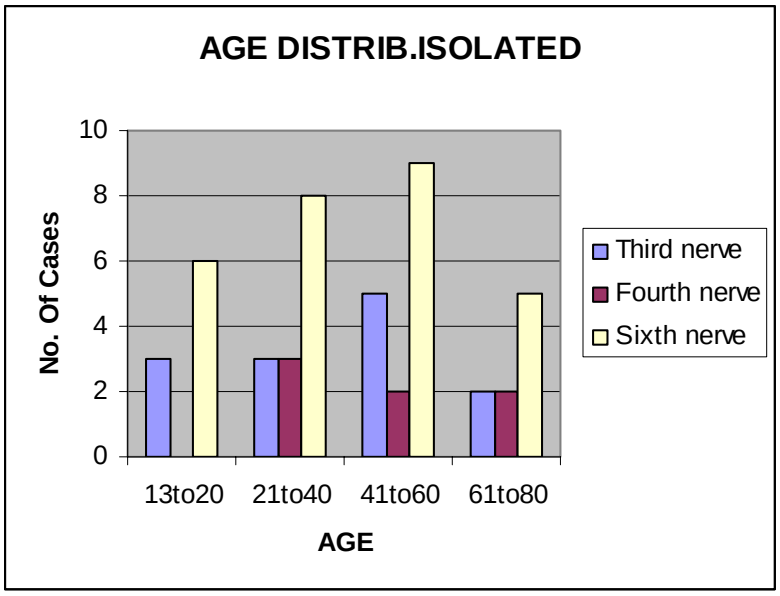
Most common etiology in multiple nerve palsy cases also was of unknown origin. Next common etiology in the multiple nerve palsy cases was ischaemia, which was in agreement with studies conducted by Tiffih et al.



In cases of isolated nerve palsy cases ,the most common etiology was trauma .This is in concordance with studies conducted by Kubatko –Zielinska et al, who studied 120 cases of ocular motor nerve palsy.

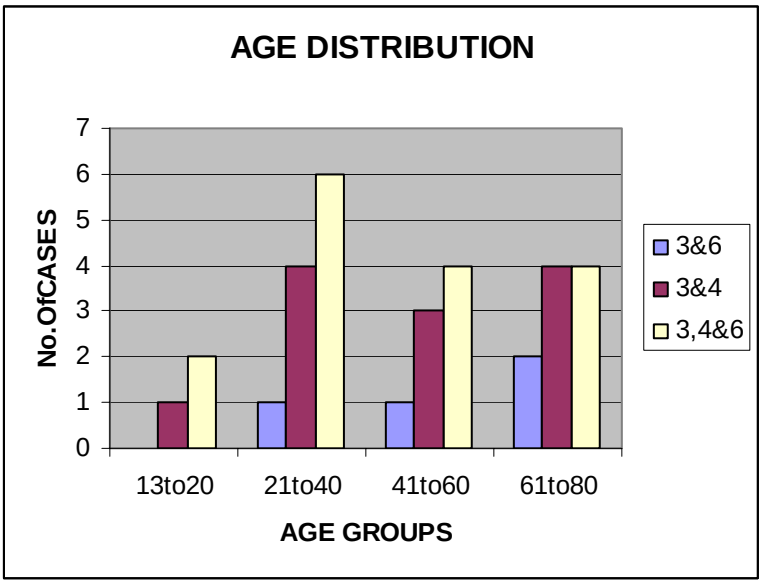


AGE DISTRIBUTION



Most common age group involved in our study was 41 to 60 years in cases of isolated nerve palsy .

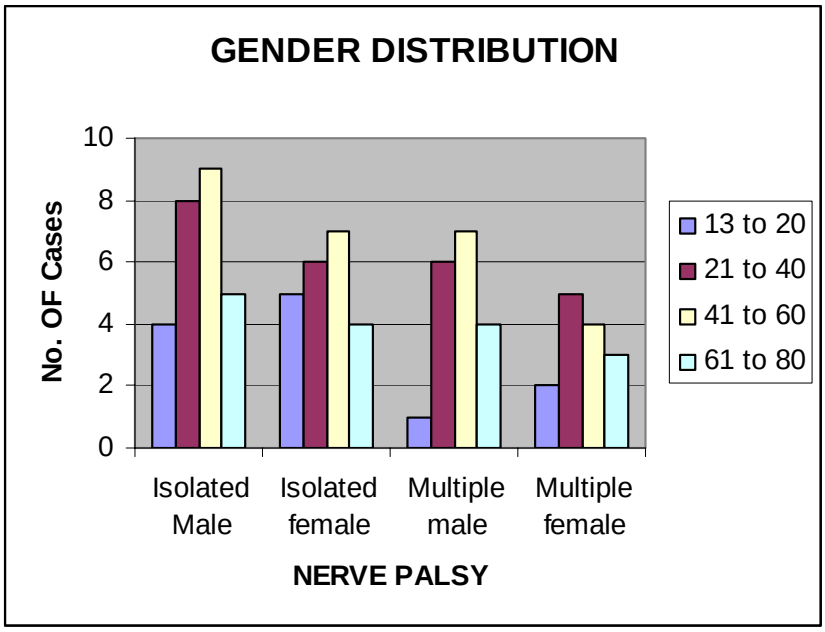
In cases of multiple nerve palsy the common age group involved was 61 to 80 years.



The above picture also shows that the most common pattern of involvement of multiple nerve palsy was 3,4,& 6nerves involvement.This is not in agreement with the pattern of involvement described by Berlit P in her series of 412 cases which states that 3rd and 6th nerve involvement was the common pattern noticed .

GENDER DISTRIBUTION

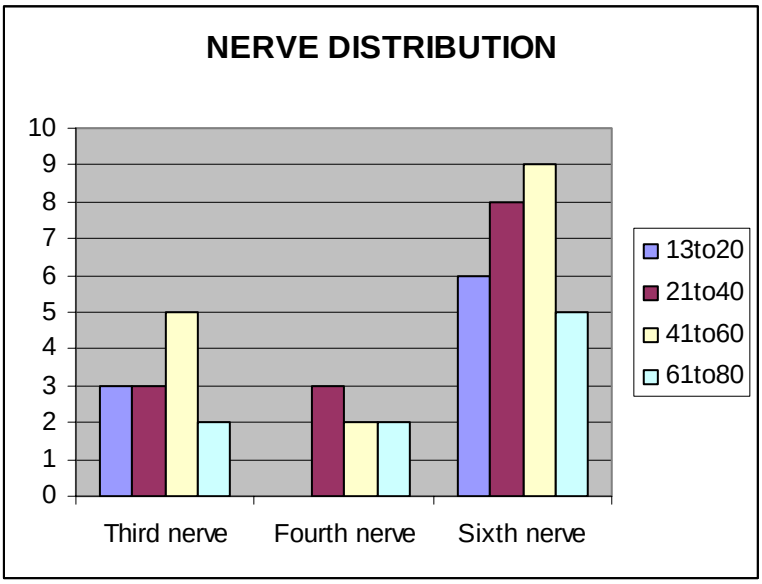
In our study there is a slight male preponderance. In the study conducted by Kubatko-Zielinska et al , there was a definite male preponderance especially in the 21 to 40 age group . In our study also the age group between 21 to 60 showed male preponderance



DISTRIBUTION OF NERVE INVOLVEMENT

Among the isolated nerve palsy patients ,the most common nerve involved was sixth nerve. This is in agreement with the results of many studies conducted in the past including one by Tiffin et al. In the study conducted by Berlit P ,the occurrence of

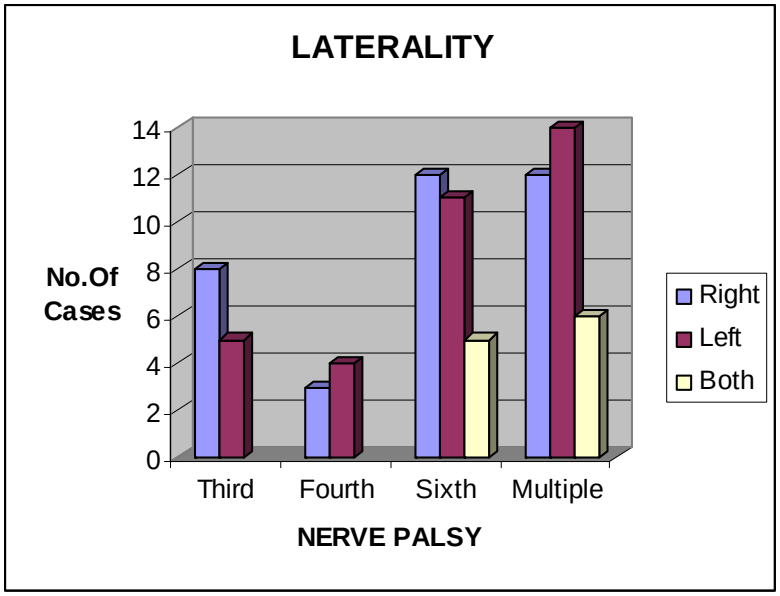
third nerve palsy was slightly more common than that ofsixth nerve palsy (41% and 40% respectively).



LATERALITY

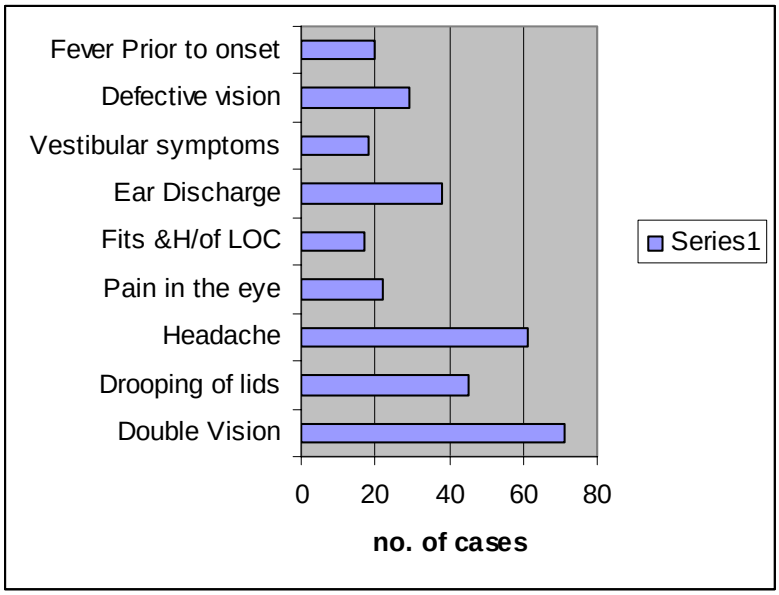
The right eye was more commonly involved in isolated sixth and isolated third nerve palsy while isolated fourth nerve involvement showed a slight left preponderance. Among patients with multiple nerve palsy there was a left eye

preponderance, while bilateral involvement was noted in isolated 6th nerve and multiple nerve palsies.

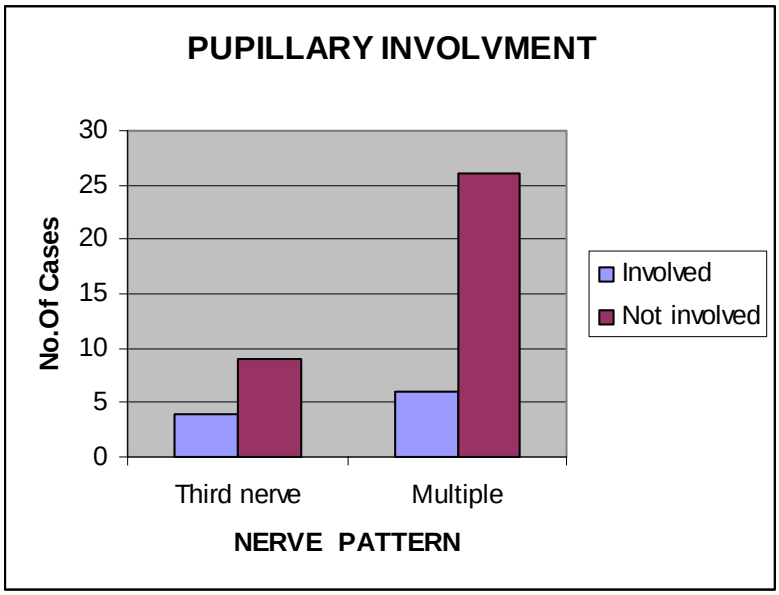


PRESENTING SYMPTOMS

Regarding the presenting symptoms, in our study ,more than 90% of patients had diplopia as their primary symptom. This was in accecptance with the results of Kobatko-Zielinska et al in whose series of 120 patients ,106 patients complained of diplopia.



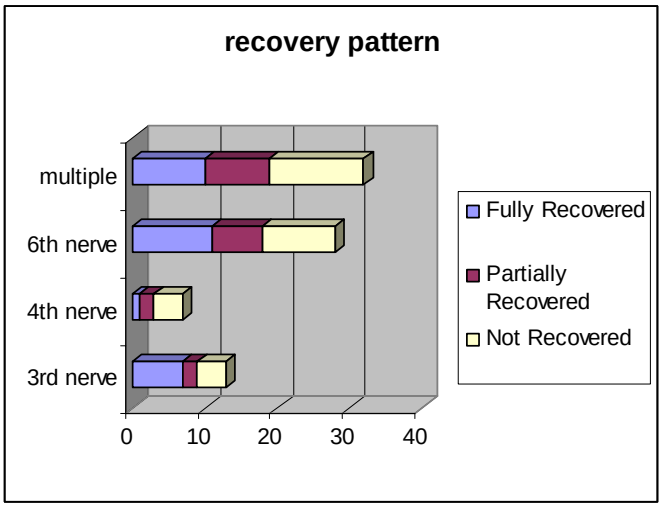
PUPILLARY INVOLVEMENT



In this series of 80 cases 21% of cases showed pupillary involvement while 79% of cases did not show any involvement .And another important thing to be considered in this regard is only 3rd nerve and multiple nerve palsy cases were included in the chart for analysis.

In the study conducted by Berlit P et al, (412cases) ,there was papillary sparing in 70% of cases .This result is in concordance with the results of our study.

RECOVERY PATTERN



In the study conducted by Tiffin et al ,80% of the patients showed recovery (atleast partial) though only 57% showed complete recovery.

According to the study conducted byBerlit P , in about 50% of cases, there was complete recovery and in about 15% of cases there was partial recovery. The most favourable prognosis was with inflammatory and vascular lesions.

In our study, there were 80 cases included in the study.

Of the 80 cases, 25% 0f cases lost follow up and hence of the 75% 0f cases(60 cases) followed ,14% of the cases did not recover and 66% recovered either partially or completely.

Of the 48 cases of isolated nerve palsy, 40% of the cases showed complete recovery, while 22.5% of cases showed only partial recovery.14% of cases did not recover and 23.5% of cases lost follow up.

Of the 32 cases multiple nerve palsy cases, 32% of cases showed complete recovery ,while 28% of cases showed only partial recovery.12.5% of cases did not recover and 27.5% of cases lost follow up.

SUMMARY

1. Of the 80 cases taken for the study, more than 80 percent of cases have occurred between 21 and 70 years. The maximum number of isolated nerve palsies occurred between 21and 60 years .In particular ,third nerve palsy cases occurred in maximum numbers between 21 and 60 years, while sixth nerve cases occurred with a uniform distribution in the age groups between second and eighth decades .The maximum number of multiple nerve palsy cases have occurred between 41 and 80 years.

2. The distribution of the cases are as follows:48 cases of isolated nerve palsy and 32 cases of multiple cranial palsy. Among the isolated nerve palsy cases, there were 13 third nerve palsy cases,7 fourth nerve cases, 28 sixth nerve palsy cases.

3. Number of male patients affected was 48 while that of female patients was32.

4. In 44% of cases right eye was involved, while 42% of cases left eye was involved. In about 14% of cases both eyes were involved.

5. Most of the patients reported to the hospital within one month from the onset of symptoms. After the discharge, most of the patients were followed for about 6 months time, at fortnightly intervals.

6. Most common general complaint was headache while the commonest ocular symptom being double vision in 6^{th} and 4^{th} nerve palsy cases and drooping of the eyelid was the commonest ocular complaint at presentation in cases of third nerve palsy.

7. The common associations were trauma, diabetes mellitus and systemic hypertension.

8. Of the 80 cases, the etiology was undetermined in 24 cases and in 67.5% of the cases the etiology was identified .

9. Of the 48 cases of isolated nerve palsies, most common was sixth nerve palsy (28cases) and the commonest cause for the sixth nerve palsy was trauma. Of the 13 cases of third nerve palsies, the commonest cause was microangiopathy due to diabetes or hypertension and among the 7 cases of fourth nerve palsy, trauma was the most common cause.

10. Of the 32 cases of multiple nerve palsy, in16 cases ,all the three nerves were involved and in12 cases third and fourth nerves were involved and in 4 cases third and sixth nerves were involved .

11. Of the 32 cases of multiple nerve palsy, the most common etiology was nonspecific neuritis of unknown origin. Of the cases with known etiology (20cases), 11 cases were due to microangiopathy and 7 cases were due to trauma and 2 cases were due to tumour.

12. Of the 80 cases of nerve palsy, 56 cases had known etiology and of these 80 cases 30% was due to trauma and 27.5% was due to microangiopathy and only 6.25% was due to tumours and the rest were due to unknown etiology.

13. Of the 80 cases, 25% of cases have lost follow up and 36.25% of cases recovered fully and and 25% of cases recovered partially and 13.75% of cases did not recover at six months.

14. Among the isolated nerve palsy cases (48 cases),40% cases recovered fully and 22.5% cases recovered partially and 14% cases did not recover up to the study follow up time of 6 months and 23.5% cases lost the follow up.

15. Among the multiple nerve palsy cases (32cases),32% cases recovered fully and 28% cases recovered partially, while 12.5% cases did not recover at 6 months and 27.5%lost the follow up.

16. The cases which recovered well were mostly due to non specific neuritis and due to microangiopathy while nerve palsy due to traumatic origin recovered poorly.

CONCLUSION

From this study of 80 cases of nerve palsies , it is concluded that :

1. The sixth cranial nerve palsy is the commonest nerve palsy and it is followed by third nerve palsy and fourth nerve palsy.
2. Among the multiple nerve palsy cases ,one or all the three nerves can be involved depending on the etiology causing it.
3. The commonest presenting symptom was double vision in sixth and third nerve cases .
4. Drooping of the eyelids was the common presenting symptom in cases of third nerve palsy, and pain and headache were the common symptoms complained often on the side of palsy.
5. Among the systemic associations, diabetes followed by hypertension was the most commonly occurring association.
6. Trauma was the common cause of isolated sixth nerve and isolated fourth nerve palsy, while microangiopathy was the commonest cause of third nerve palsy. Even trivial trauma was associated with fourth nerve palsy, severe closed head trauma was commonly associated with third and sixth nerve palsies.
7. Patients belonging to older age group (more than 65 years) had palsies commonly due to microangiopathic pathology while younger patients suffered these palsies commonly because of trauma.
8. The most common etiology was nonspecific neuritis and these cases improved well with good recovery at six months.
9. Nerve palsies arising secondary to microangiopathy (diabetes or hypertension) recovered well in six months time regardless of the nerves affected.
10. Regarding the recovery, the percentage of recovery of isolated nerve palsy patients was more than that of multiple nerve palsy.
11. Complete recovery of patients at six month was more common among isolated nerve palsy patients than multiple nerve palsy patients.
12. Levator palpebrae superioris was the first muscle to recover in cases of both isolated third nerve palsy and multiple nerve palsy involving third nerve also.

PROFORMA

A STUDY OF ISOLATED AND MULTIPLE CRANIAL NERVE PALSIES

Name: Age: Sex: Address:
Complaints: Laterality:
Duration:
GOHIP/OP Number: Date:

History

General:

Ocular/Ophthalmic:

Medical/ Surgical:
Past History:
Personal History:

Examination

General:

Systemic (CNS):
Higher Function/Cranial Nerves/Sensory/Motor/Reflex/Cerebellar Systems

Ocular:
Head Position/Facial Asymmetry/Lids/EOM/Anterior Segment/Pupil/V|A/Fundus

Investigations

General:
Pulse/BP/Temperature

Ocular:
V|A/Refraction/Tension/Hess charting/ Diplopia Charting/Fields

Orthoptic evaluation:
Cover Test/PBCT/WFDT/BSV/SMP

Lab Investigations:
Haemogram/RBS/Urine Routine/Mx/VDRL/X-Ray/CT Scan/MRI Scan

Specialist Opinion:
Neuro/ENT/Other Specialities

Diagnosis:
Follow Up:
V|A/EOM/Orthoptic Evaluation/Diplopia Chart/Hess Chart

MASTER CHART

S.No	AGE	SEX	EYE	Pri. Symp.	V/A	Ant. Segm.	Pupil	Fun Dus	Nerves Inv.	CNS	Sys. Dis.	X ray	CT	MRI	Follow up
1	67	F	L	D/P	<6/18	Imc	N	N	M	N	N	N	N	N	N
2	16	F	R	Pto.	6/6	N	N	N	3rd	EDH	N	N	EDH	N	N
3	23	M	L	Pain	6/6	N	N	N	4th	N	N	N	N	N	N
4	70	F	R	Dip.	<6/18	Imc	N	DR	4th	N	DM/HT	N	N	N	N
5	68	M	R	Pto.	<6/18	Imc	N	N	M	Hemiplegia	N	N	Inf.	N	N
6	20	F	L	Pain	6/6	N	N	N	6th	N	N	N	N	N	N
7	42	F	L	Dip.	6/6	N	N	N	6th	N	N	N	N	N	N
8	32	M	R	Pain	6/6	N	N	N	6th	N	N	N	N	N	N
9	66	F	R	Dip.	<6/18	Imc	N	N	M	Hemiparesis	N	N	Cal	N	N
10	18	M	L	Pto.	6/6	N	N	N	M	SDH	N	N	SDH	N	N
11	18	F	R	Dip.	6/6	N	N	N	6th	N	N	N	N	N	N
12	39	F	L	Pain	6/6	N	N	DR	6th	N	DM/HT	N	N	N	N
13	48	F	R	Pain	<6/18	Imc	N	N	M	N	N	N	N	N	N
14	62	M	R	Pto.	<6/18	Imc	N	N	M	N	N	N	N	N	N
15	40	F	L	Dip.	6/6	N	N	N	4th	N	N	N	N	N	N

16	69	M	L	Dip.	<6/18	Imc	N	DR	6th	N	DM	N	N	N	N
17	67	F	R	Pain	<6/18	Imc	N	DR	4th	Meningioma	DM/HT	N	Cal	Tr	N
18	71	M	R	D/P	<6/18	Imc	N	DR	3rd	N	DM	N	N	N	N
19	67	F	R	Dip.	<6/18	Imc	N	N	6th	N	N	N	N	N	N
20	17	M	R	Pto.	6/6	N	N	N	M	N	N	N	N	N	N
21	69	F	BE	D/P.	<6/18	Imc	N	N	M	N	N	N	N	N	N
22	26	M	R	Pto	6/6	N	N	N	M	CP angle tr	N	N	SOL	Tr	N
23	29	F	L	Pain	6/6	N	N	N	6th	N	N	N	N	N	N
24	41	F	R	Pain	6/6	N	N	N	6th	N	N	N	N	N	N
25	80	F	L	Dip.	<6/36	MC	N	N	6th	Parkinsonsim	DM/HT	N	N	N	N
26	63	M	R	Pain	<6/24	Lens Changes	N	Mac.Dru.	6th	N	N	N	N	N	N
27	76	M	L	Pain	<6/36	MC	N	DR	6th	Quadriparesis	DM	N	N	N	N
28	65	F	BE	D/P.	<6/18	Imc	N	N	M	N	N	N	N	N	N
29	16	F	R	Pto.	6/6	N	N	N	3rd	Cysticercosis	N	N	Ring.	N	N
30	40	M	L	Dip.	6/6	N	N	N	6th	N	DM	N	N	N	N
31	27	M	R	Pto	6/6	N	N	N	M	N	N	N	N	N	N
32	55	F	L	Dip	6/6	N	N	N	6th	N	DM	N	N	N	N

33	66	F	R	Pto	<6/18	Imc	N	N	M	N	N	N	N	N	N
34	67	M	L	D/P	6/12	Imc	In	N	M	Meningioma	N	N	Cal	Tr	N
35	71	F	L	Pto	6/24	Imc	In	N	M	N	N	N	N	N	N
36	23	M	L	D/P	6/6	N	N	N	M	N	N	N	N	N	N
37	51	F	R	Pain	6/60	Imc	N	N	6th	Aneurysm ICA	N	N	N	An	N
38	29	M	L	D/P	6/6	N	N	N	M	N	N	N	N	N	N
39	57	M	R	Dip	6/9	Cor Opa	N	DR	6th	N	DM	N	N	N	N
40	39	M	L	D/P	6/6	N	N	N	M	N	N	N	N	N	N
41	13	M	L	Pain	6/6	N	N	N	6th	N	N	N	N	N	N
42	47	F	Be	D/P	6/6	N	N	N	6th	N	N	N	N	N	N
43	49	F	Be	D/P	6/6	N	N	N	M	N	N	N	N	N	N
44	22	M	L	D/P	6/6	N	N	N	M	N	N	N	N	N	N
45	46	F	Be	D/P	6/6	N	N	N	M	N	N	N	N	N	N
46	52	F	Be	D/P	6/24	Imc	N	N	M	N	N	N	N	N	N
47	31	F	R	Dip/Pain	6/6	N	N	N	6th	Aneurysm	N	N	N	An	N
48	55	M	R	D/P	6/24	imc	N	N	M	N	N	N	N	N	N
49	17	M	R	D/P	6/6	N	N	N	M	N	N	# base of skull	#	N	N
50	53	F	R	D/P	6/60	MC	N	DR	M	N	DM	N	N	N	N
51	50	M	Be	D/P	6/6	N	N	N	M	N	N	N	N	N	N
52	28	F	R	D/P	6/6	N	N	N	3rd	N	N	N	N	N	N
53	49	F	R	D/Pain	6/6	N	N	DR	6th	N	DM	N	N	N	N

54	57	F	L	D/Pain	6/36	MC	N	DR	6th	N	DM/HT	N	N	N	N
55	18	M	Be	D/P	6/6	N	N	N	3rd	N	N	#zygoma	#	N	N
56	33	M	Be	D/P	6/6	N	N	N	3rd	N	N	#parietal bone	#	N	N
57	47	M	R	Dip	6/6	N	N	N	4th	N	N	N	N	N	N
58	34	M	Be	D/P	6/6	N	N	HR	3rd	N	HT	N	N	N	N
59	61	F	L	Pto	<6/18	Im Cat.	N	N	3rd Nerve	N	N	N	N	N	N
60	49	M	R	D/P	6/60	MC	N	N	3rd	Aneurysm	N	N	N	N	N
61	15	M	R	D/Pain	6/6	N	N	N	6th	N	N	N	N	N	N
62	37	M	R	D/Pain	6/6	N	N	HR	6th	N	HT	N	N	N	N
63	45	M	R	D/Pain	6/6	N	N	N	4th	N	N	N	N	N	N
64	48	M	R	D/P	6/60	NC	N	HR	3rd	N	HT	N	N	N	N
65	14	M	R	D/P	6/6	N	N	N	6th	N	N	N	N	N	N
66	32	F	R	Pto	6/6	N	N	N	M	SOL TB	N	N	N	N	N
67	57	M	L	D/P	5/60	NC	N	DR	M	N	DM	N	N	N	N
68	36	F	L	Pain		N	N	N	4th	N	N	N	N	N	N
69	59	M	L	D/P	6/36	PCC	N	DR/HR	3rd	N	DM/HT	N	N	N	N
70	14	M	L	Pain	6/6	N	N	N	6th	N	N	#frontal bone	#	N	N
71	54	M	Ll	Dip	6/6	N	N	N	6th	N	N	N	N	N	N
72	27	M		Dip	6/6	N	N	N	6th	N	N	N	N	N	N
73	52	M	R	Pto	6/60	NC	N	N	3rd	N	DM	N	N	N	N
74	16	M	L	Dip	6/6	N	N	N	6th	N	N	N	N	N	N

75	33	F	L	Pto	6/6	N	N	N	M	N	N	N	N	N	N
76	56	M	L	D/P	6/24	NC	N	DR	3rd	N	DM	N	N	N	N
77	28	M	L	Dip	6/6	N	N	N	6th	N	N	N	N	N	N
78	37	f	L	D/P	6/6	N	N	N	M	N	N	N	N	N	N
79	50	M	L	D/P	6/6	N	N	N	M	N	DM	N	N	N	N
80	40	f	L	D/P	6/6	N	N	N	M	N	N	N	N	N	N

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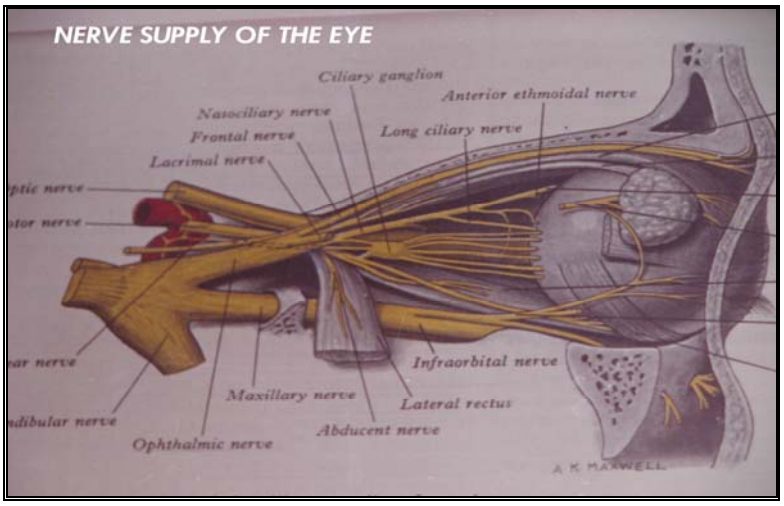
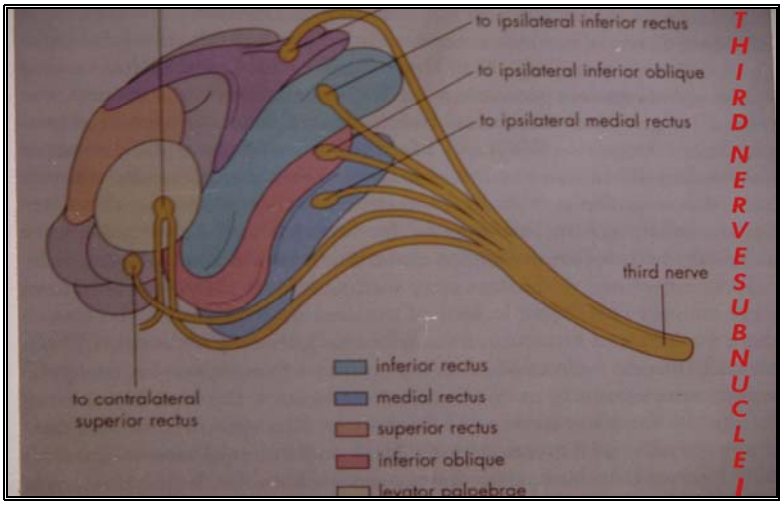
ABBREVIATIONS

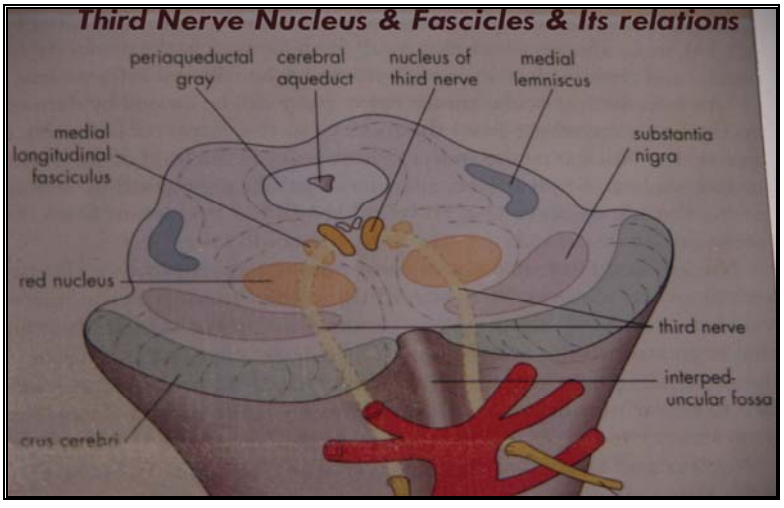
RE	-	Right Eye
LE	-	Left Eye
Dip	-	Diplopia
Pto	-	Ptosis
#	-	Fracture
DM	-	Diabetic Mellitus
HT	-	Hypertension
DR	-	Diabetic Retinopathy
HR	-	Hypertensive Retinopathy
N	-	Normal/ No Abnormality Detected
MC	-	Mature Cataract
IMC	-	Immature Cataract
In	-	Involved (Pupil)
An	-	Aneurysm
EDH	-	Extra Dural Haemorrhage
SDH	-	Sub Dural Haemorrhage
Mac. Dru.	-	Macular Drusen
Sys. Dis.	-	Systemic Disease
Tr.	-	Tumour

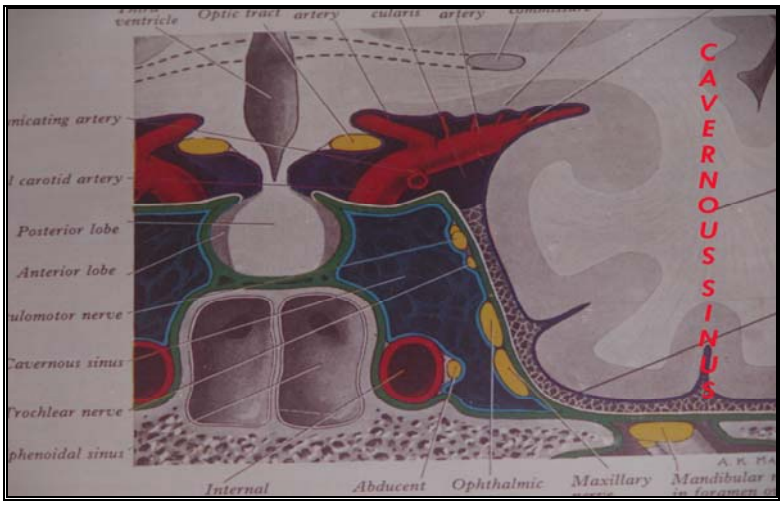
LIST OF SURGERIES

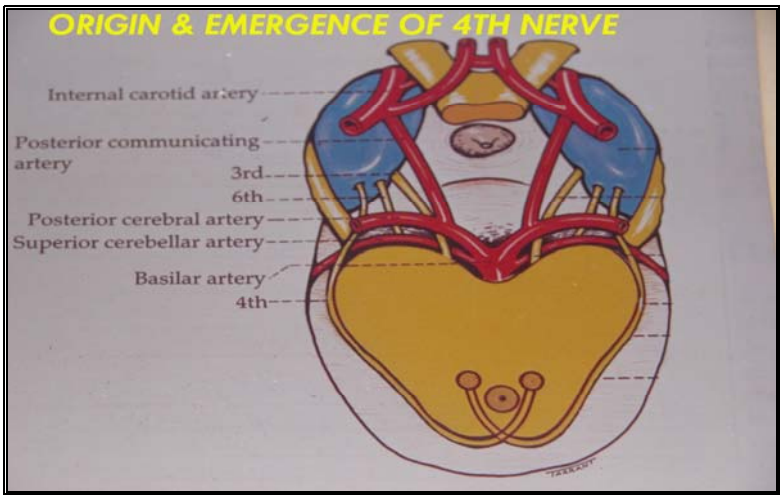
Sl.No.	Date	Name	Age	Sex	Surgery
1	16.9.2003	Chandrasekar	60	M	ECCE with PCIOL - RE
2	16.10.2003	Gandhimathi	70	F	ECCE with PI – RE
3	26.10.2003	Sarala	40	F	Evisceration – RE
4	1.3.2004	Kasiammal	60	F	ECCE with PCIOL – RE
5	10.3.2004	Shanmugam	70	M	DCT – LE
6	17.3.2004	Devaraj	20	M	CHALAZION – I & C – RE
7	26.4.2004	Krishnaveni	46	F	DCR – RE
8	20.5.2004	Vijaya	25	F	Pterygium Excision - LE
9	26.6.2004	Thilaga	32	F	Lower Lid Tear – Suturing – RE
10	28.8.2004	Govindaraj	60	M	Enucleation - LE
11	15.10.2004	Panneer	7	M	Probing – LE
12	15.11.2004	Kamatchi	26	F	TKP – LE
13	3.2.2005	Lakshiammal	46	F	Trab with Lens Removal – RE
14	17.6.2005	Jamuna	17	F	Corneal Tear Suture – RE
15	20.8.2005	Venkatesan	40	M	Tarsoraphy – LE
16	21.12.2005	Geetha	50	F	SICS with PCIOL – RE
17	22.2.2006	Kanaga	16	F	Symblepharon Release – RE
18	26.3.2006	Sekar	56	M	SICS with PCIOL – RE
19	28.3.2006	Vijaya	22	F	Squint Surgery - LE

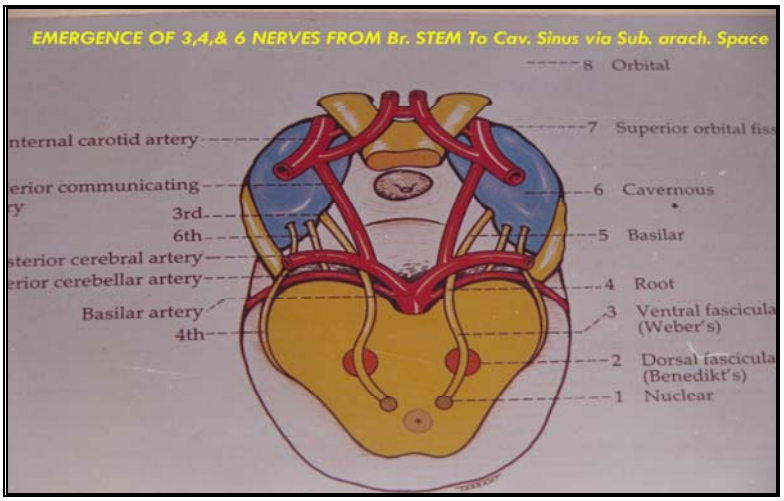

NERVE SUPPLY OF THE EYE
Ciliary ganglion
Anterior ethmoidal nerve
Nasociliary nerve
Long ciliary nerve
Frontal nerve
Lacrimal nerve
Infraorbital nerve
Maxillary nerve
Lateral rectus
Abducent nerve
Ophthalmic nerve
A. K. MAXWELL

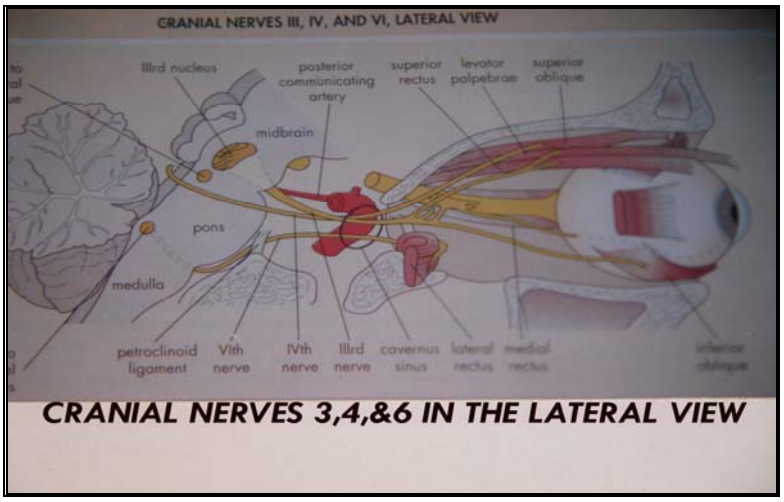

to ipsilateral inferior rectus
to ipsilateral inferior oblique
to ipsilateral medial rectus
third nerve
to contralateral superior rectus
inferior rectus
medial rectus
superior rectus
inferior oblique
THIRD NERVE SUBNUCLEI

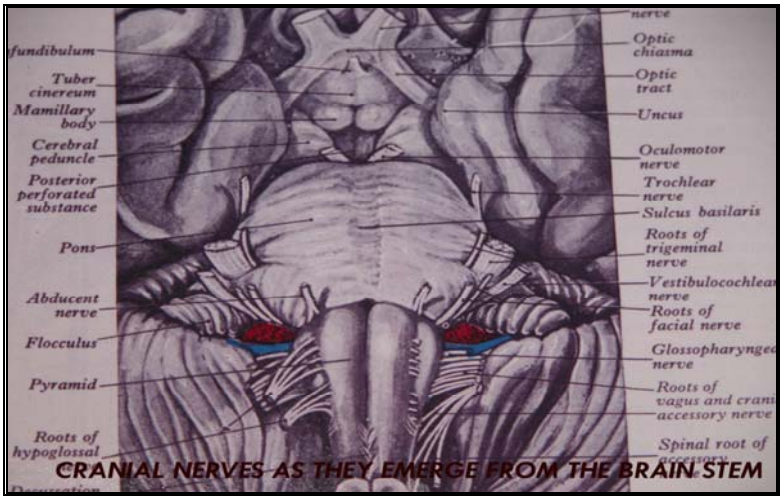

Third Nerve Nucleus & Fascicles & Its relations
periaqueductal gray
cerebral aqueduct
nucleus of third nerve
medial lemniscus
medial longitudinal fasciculus
substantia nigra
red nucleus
third nerve
interped-uncular fossa
crus cerebri

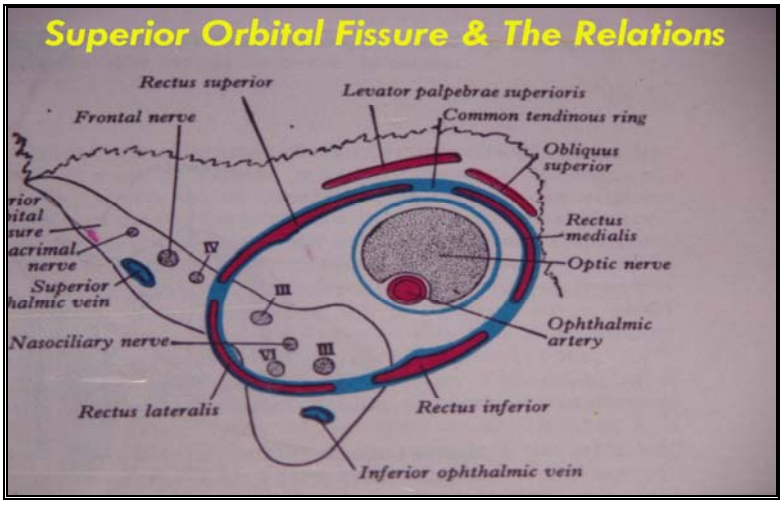

CAVERNOUS SINUS
Optic tract
Posterior lobe
Anterior lobe
Cavernous sinus
Internal
Abducent
Ophthalmic
Maxillary


ORIGIN & EMERGENCE OF 4TH NERVE
Internal carotid artery
Posterior communicating artery
3rd
6th
Posterior cerebral artery
Superior cerebellar artery
Basilar artery
4th

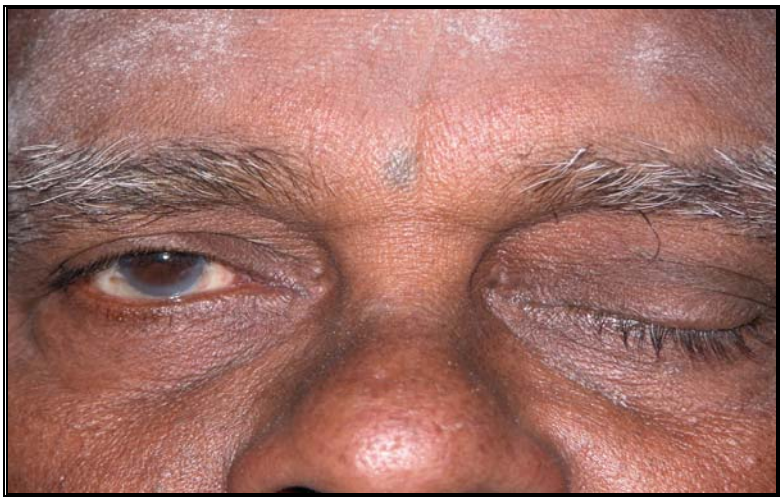

EMERGENCE OF 3,4,& 6 NERVES FROM Br. STEM To Cav. Sinus via Sub. arach. Space
8 Orbital
7 Superior orbital fiss
6 Cavernous
5 Basilar
4 Root
3 Ventral fascicula (Weber's)
2 Dorsal fascicula (Benedikt's)
1 Nuclear
3rd
6th
Basilar artery
4th

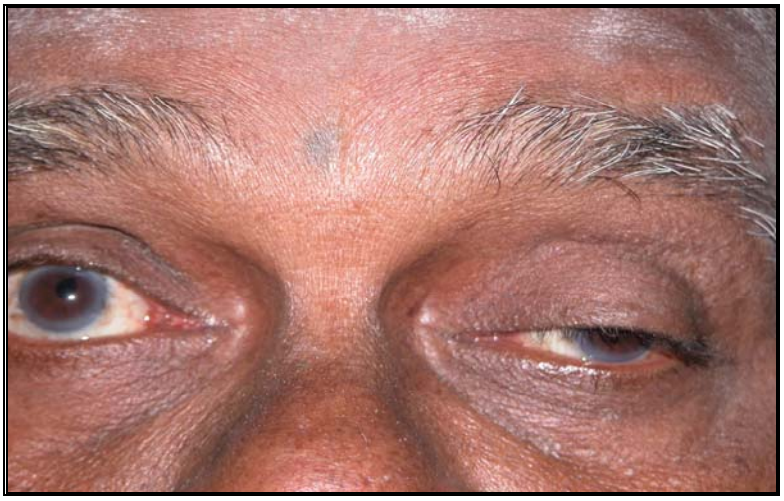

CRANIAL NERVES III, IV, AND VI, LATERAL VIEW
IIIrd nucleus
posterior communicating artery
superior rectus
levator palpebrae
superior oblique
midbrain
pons
medulla
petroclinoid ligament
Vth nerve
IVth nerve
IIIrd nerve
cavernus sinus
lateral rectus
medial rectus
CRANIAL NERVES 3,4,&6 IN THE LATERAL VIEW

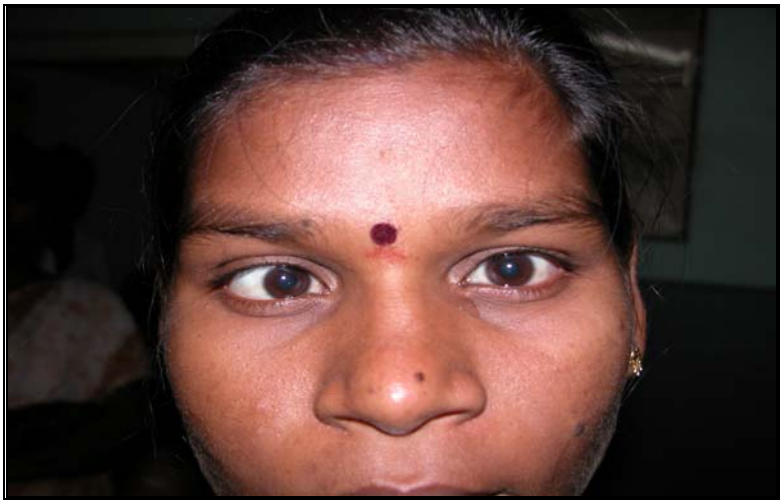

fundibulum
Tuber cinereum
Mamillary body
Cerebral peduncle
Posterior perforated substance
Pons
Abducent nerve
Flocculus
Pyramid
Roots of hypoglossal
Optic chiasma
Optic tract
Uncus
Oculomotor nerve
Trochlear nerve
Sulcus basilaris
Roots of trigeminal nerve
Vestibulocochlear nerve
Roots of facial nerve
Spinal root of
CRANIAL NERVES AS THEY EMERGE FROM THE BRAIN STEM


Superior Orbital Fissure & The Relations
Rectus superior
Levator palpebrae superioris
Frontal nerve
Common tendinous ring
Obliquus superior
Rectus medialis
Optic nerve
Ophthalmic artery
Nasociliary nerve
Rectus lateralis
Rectus inferior
Inferior ophthalmic vein

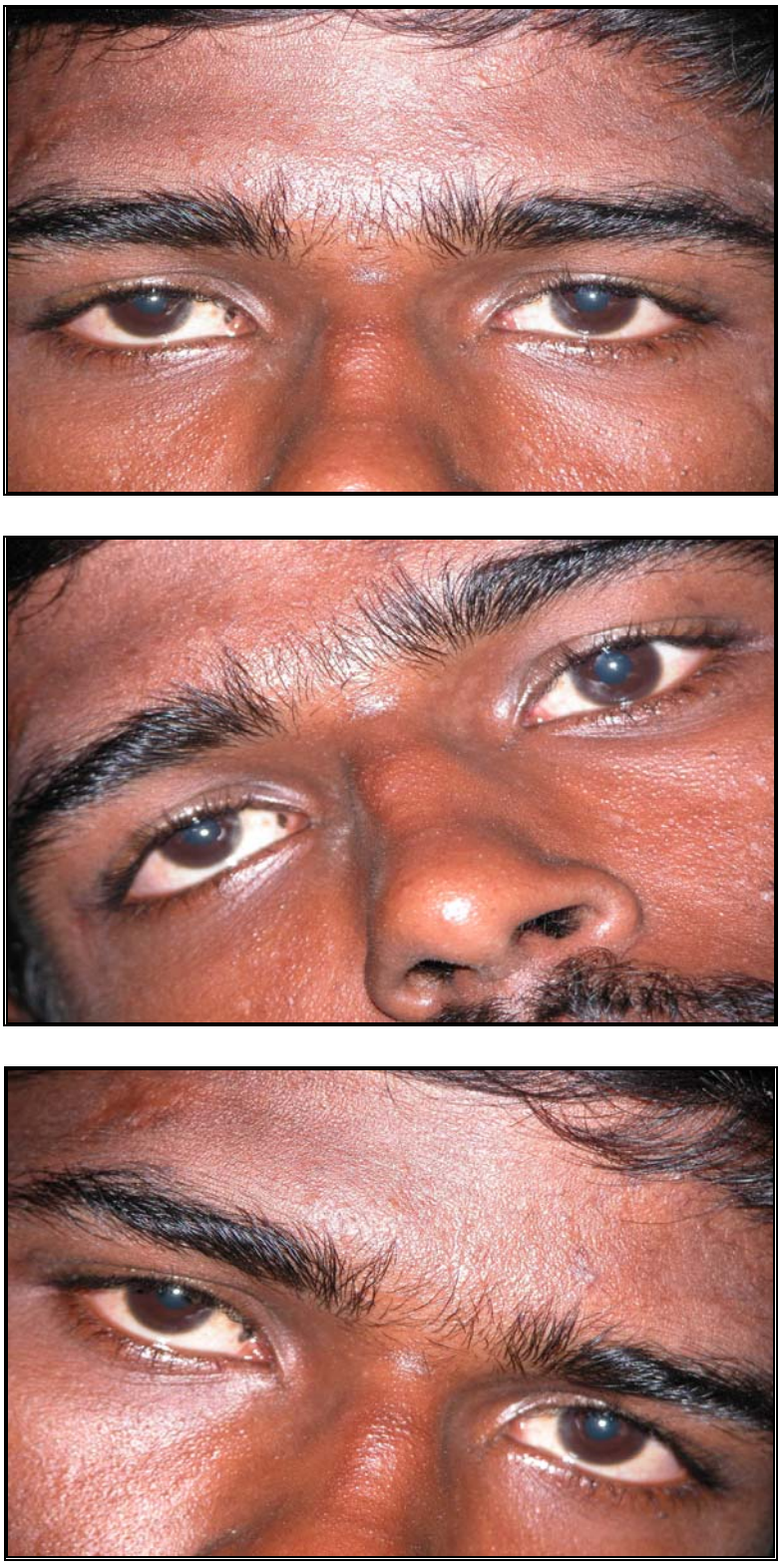
Third nerve palsy



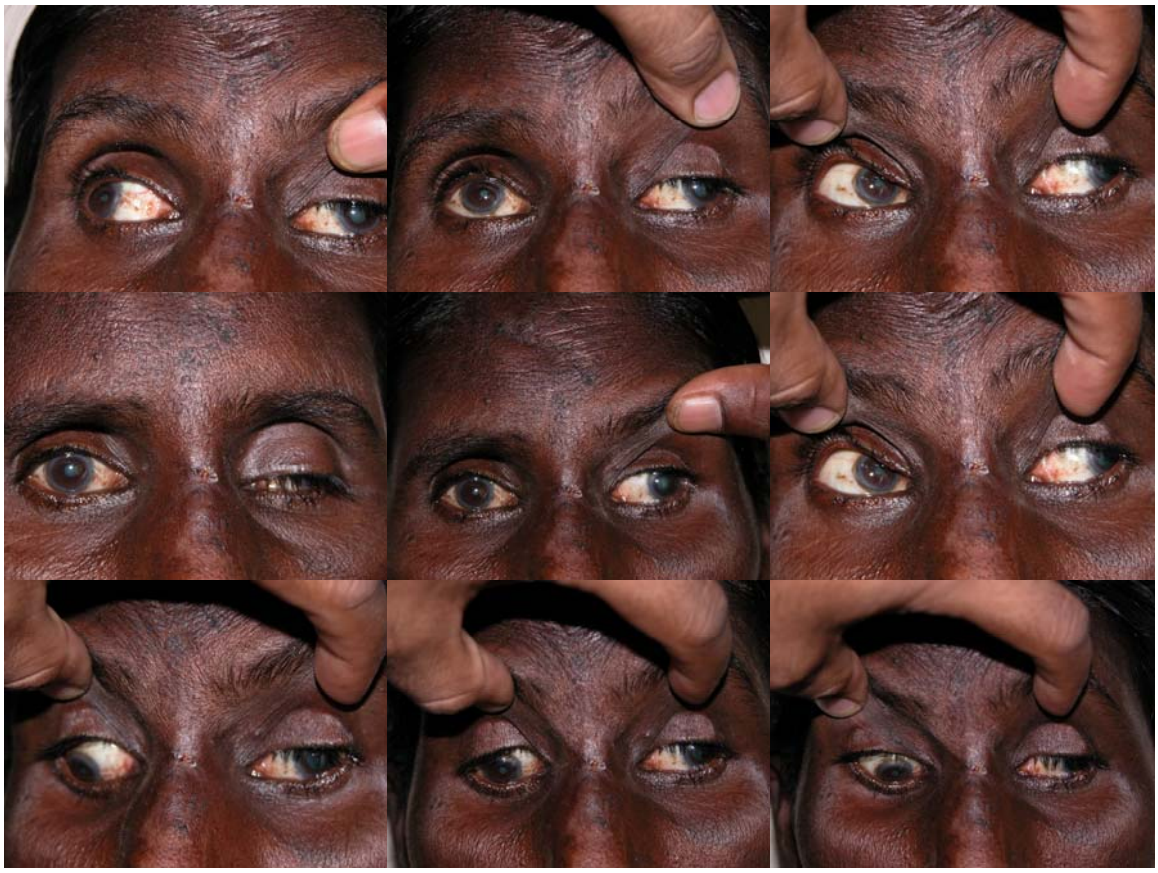




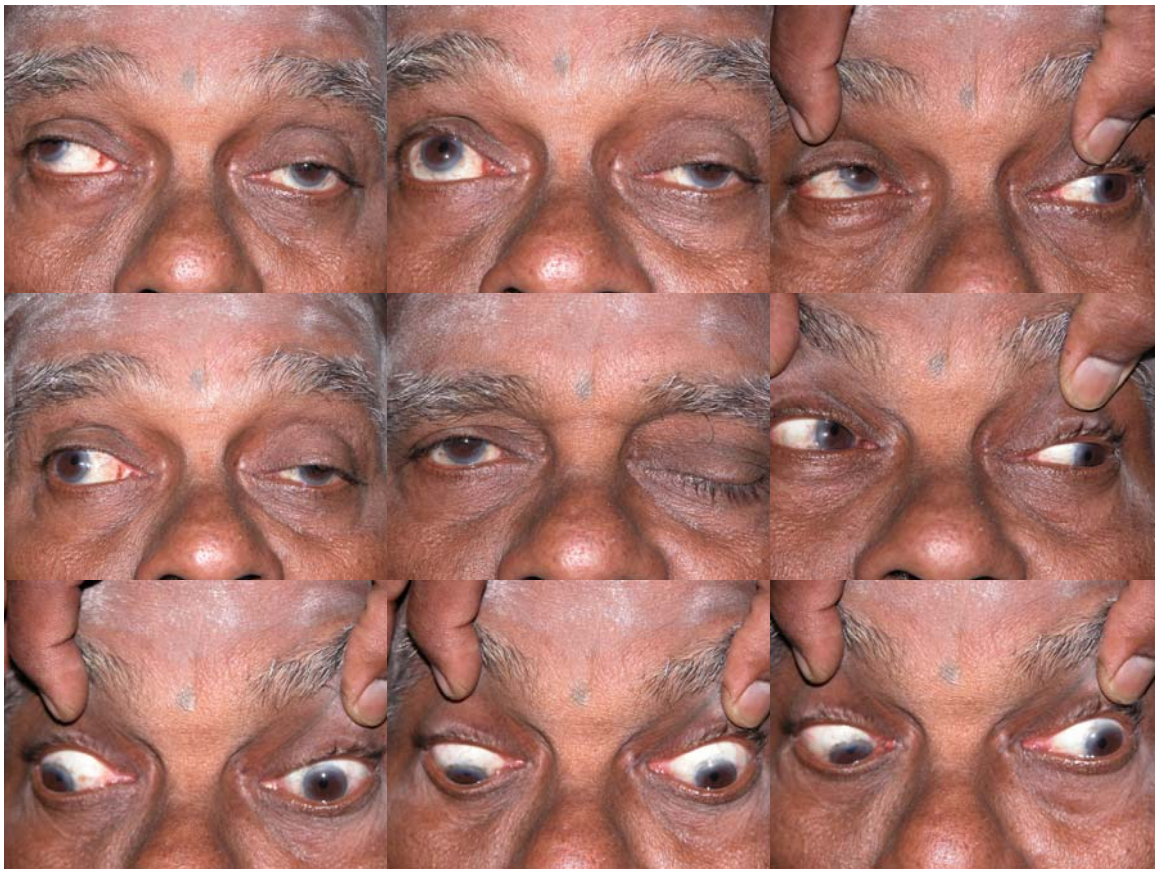
Sixth nerve Palsy



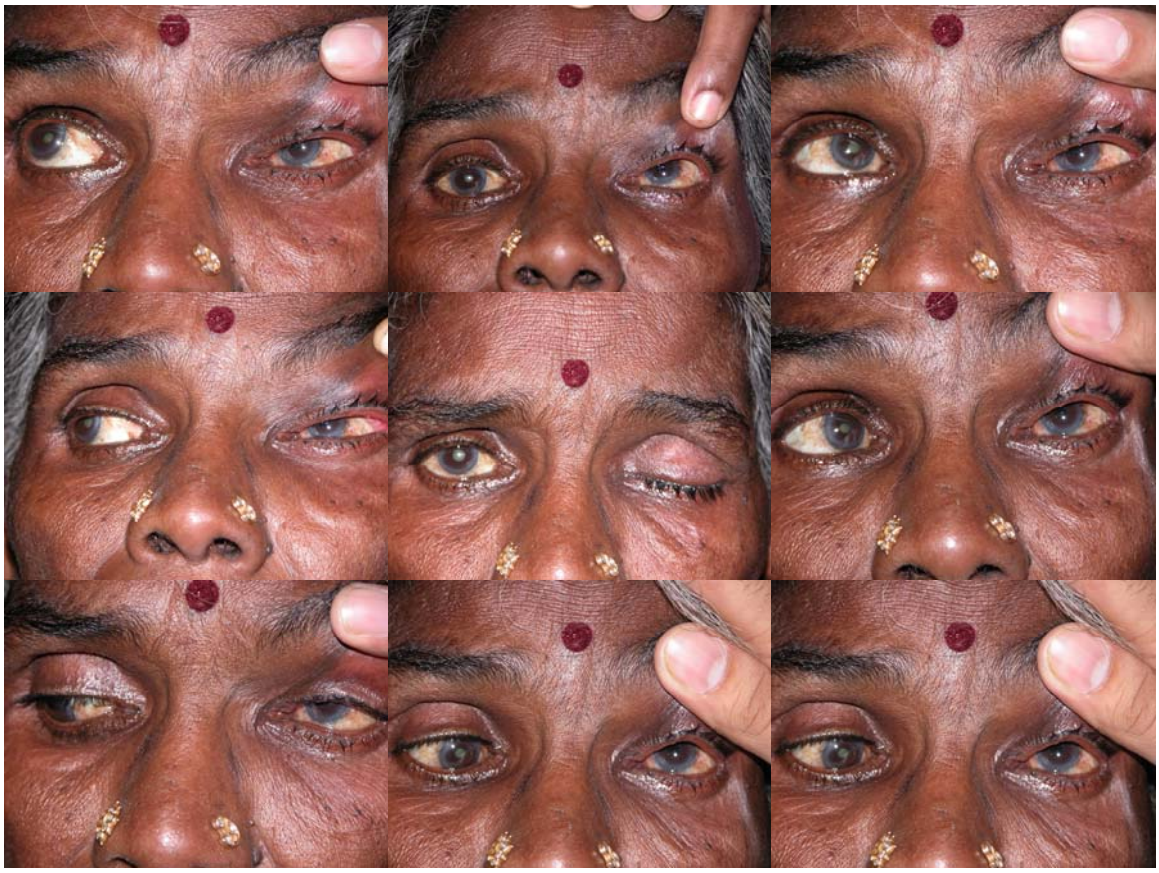
BIELCHOWISKY'S Three Step Test in Fourth nerve palsy



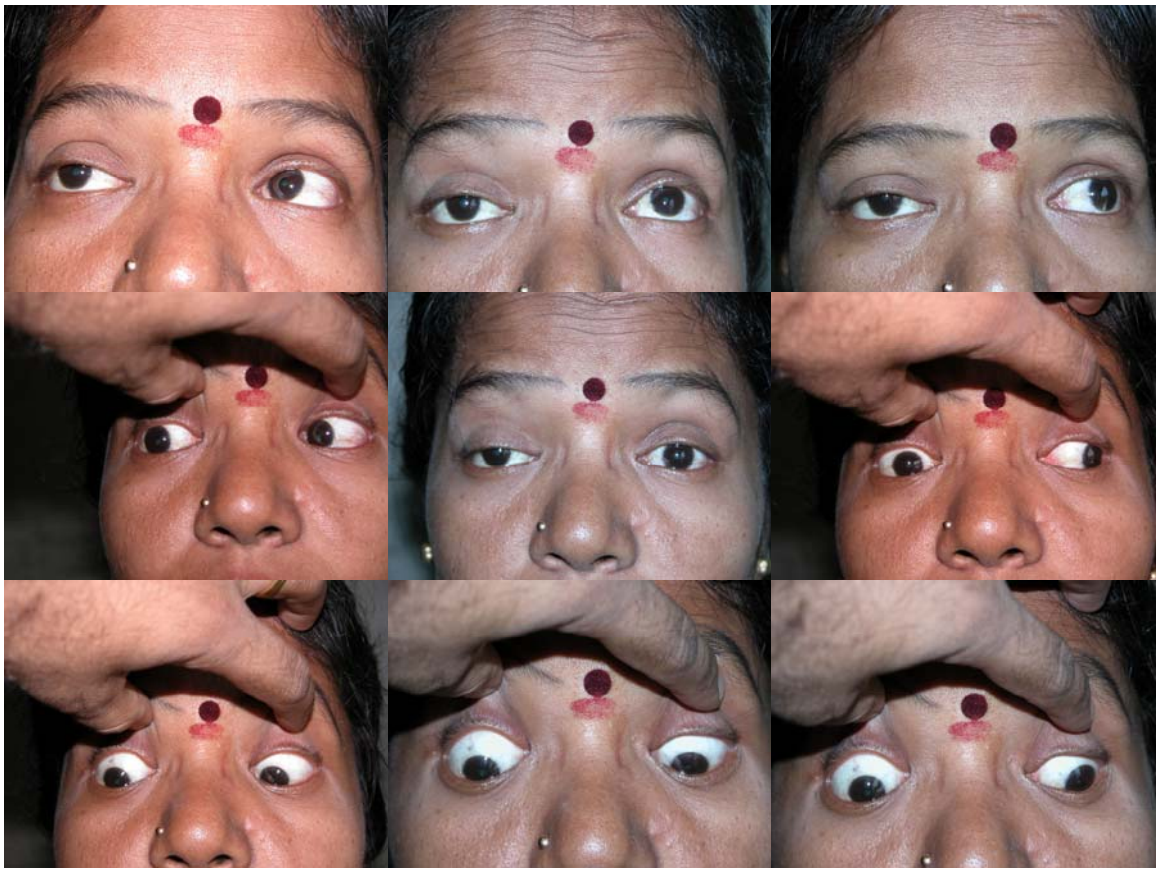
Isolated third nerve palsy



Isolated third nerve palsy



Left 3, 4 and 6 nerve palsy



Resolving 3 and 6 nerve palsy