# STUDY ON NEURO OPHTHALMIC MANIFESTATIONS OF BRAIN TUMOR

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# CERTIFICATE

This is to certify that this dissertation entitled "**STUDY ON NEURO OPHTHALMIC MANIFESTATIONS OF BRAIN TUMOR**" has been done by ***DR. A. SYED MOHIDEEN ABDUL KHADAR*** under my guidance in the Department of Ophthalmology, Madurai Medical College, Madurai.

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

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# DECLARATION

I, ***Dr. A. SYED MOHIDEEN ABDUL KHADAR*** solemnly declare that the dissertation titled **"STUDY ON NEURO OPHTHALMIC MANIFESTATIONS OF BRAIN TUMOR"** has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement for the award of M.S.,degree (Ophthalmology) Examination to be held in MARCH 2008.

**Place : Madurai**

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# CONTENTS

# INTRODUCTION

Tumors of CNS represent a unique heterogenous population of both benign and malignant neoplasm.

Primary brain tumors constitute 2% of all cancers in Adults and 20% of all malignant tumors in children.

Brain tumors are second to leukemias in children to comprise a large group of solid tumors

The incidence of primary tumors is 10-17 per 1,00,000 per year.

Since the visual pathway extends from the frontal lobe anteriorly to the occipital lobe posteriorly any insult to the brain in the form of trauma, tumor, congenital defects can involve visual pathway which is reflected externally and detected by the ophthalmologist.

In developing countries like ours where expensive investigations like CT / MRI are yet to be of wide spread use a careful neuro ophthalmic evaluation can diagnose neurological disorder with a fair amount of accuracy.

Morethan 50% of the intracranial tumors first produce ocular signs and symptoms and hence are first detected by an ophthalmologist.

So Ophthalmologists play an important role in diagnosis and management of these patients.

# ANATOMY OF THE VISUAL PATHWAY

Light rays which enter the eyeball stimulate the receptors in the retina and the impulse from here are carried along the optic nerve, optic chiasma, optic tract, LGB & optic radiations to reach the visual area in the occipital lobe. These constitute the visual pathway or retino calcarine pathyway.

## RETINA :

Inner coat of the eye ball

The end receptors are rods and cones

Rods for scotopic and cones for photopic vision

Rods and cones synapse with bipolar cells which relay to ganglion cells

## OPTIC NERVE :

Axons of ganglion cells form optic nerve which extends from eyeball to optic chiasma

It can be divided into four parts :

1. **Intraocular part** (0.7 – 1 mm) is also called optic nerve head and is not covered by meninges. The non myelinated

fibres gain myelination as they pass through this part of optic nerve

2. **Intraorbital part** (25-30mm) : extends from eyeball to optic canal covered by meninges. The central retinal artery with the vein pierces the meninges and enters the substance of the optic nerve 1.5 cm behind the lamina cribrosa.
3. **Intracanalicular part** (6-10mm) : This is within the optic canal. Endosteal layer of dura blends with the periosteum. Related to the sphenoidal and posterior ethmoidal air sinus.
4. **Intracranial part (**10mm) : lies in the middle cranial fossa. The two optic nerves are directed backward and medially and converge at 60 degrees. They join the anterolateral angles of optic chiasma. Related to anterior perforated substance, anterior cerebral artery, olfactory tract, internal carotid artery, 80% of nerve fibres mediate visual perception. 20% formed by pupillary fibres.

**OPTIC CHIASMA :**

Flattened band measuring 12 mm transversely, 8mm anteroposteriorly, 35 mm vertically, connecting the two optic nerves

and two optic tracts. Situated at the junction of anterior wall and floor of third ventricle. Placed obliquely above diaphragma sella. Inferiorly related to pituitary gland.

**Optic Chiasma : Fibre Arrangements :**

a) The nasal retinal fibres of each eye cross in the chiasm to the contra lateral optic tract, temporal fibres are uncrossed. Thus a lesion here causes a bitemporal hemianopia

b) Lower retinal fibres project through the optic nerve and the chiasm to lie laterally in the tract : upper retinal fibres will lie medially.

c) Inferonasal retinal fibres cross in the chiasm and travel anteriorly in the contralateral optic nerve (Wilbrand's knee) before joining with the uncrossed infero temporal fibres in the optic tract.

d) Macular fibres are distributed throughout the chiasm, and if affected cause a central bitemporal hemianopic scotoma.

**OPTIC TRACT :**

Extends from the optic chiasm to the Lateral Geniculate Body. Contains ipsilateral temporal nerve fibres and contra lateral nasal

fibres. It terminates by dividing into two roots. Small medial and large lateral. Related to posterior perforated substance, III ventricle, cerebral peduncle, Internal capsule, Uncus, tuber cinereum. Medial root carries fibres to the superior colliculus, pretectal nucleus. Lateral root ends in the lateral geniculate body.

**LATERAL GENICULATE BODY :**

A saddle shaped nuclear mass having six layers. In the inferolateral aspect of pulvinar of thalamus. 1,4,6 receive crossed fibres, whereas uncrossed fibres terminate in layers 2,3 and 5. Lesion of retina result in degeneration of the 3 layers where retina ends. Lesion of visual cortex results in degeneration of all six layers.

**OPTIC RADIATION :**

Extends from LGB to the Calcarine cortex. First forms the optic peduncle. It runs in the retrolentiform part of the internal capsule in the temporal lobe, then the fibres fan out of the internal capsule, first horizontally and then vertically.

The upper fibres turn back and medially, lower fibres run forwards and loop backwards, lowest fibres run forward upto

anterior pole of the temporal lobe and then loop back, this is the loop of Meyer.

**VISUAL CORTEX :**

Located in the occipital lobe of the cerebral hemisphere. Primary Area "17" is situated in the wall of the calcarine sulcus on the medial surface of cerebral hemisphere. Secondary Areas 18,19 surround primary visual area on the medial and lateral surface of the hemisphere. Visual cortex of one side is concerned with the perception of the objects in contralateral visual field. Macular fibres are extensively represented and posteriorly located. The macular area is supplied by the branches of posterior and Middle cerebral arteries, which explains the Macular sparing in field defects due to lesions in the visual cortex.

**CRANIAL NERVES :**

**Oculomotor Nerve :**

**Nucleus :** Situated in the midbrain at the level of superior colliculus. Ventral to sylvian aqueduct

**Course :** Rootlets leave the midbrain on medial aspect of

cerebral peduncle. Passes forwards between posterior cerebral and superior cerebellar arteries and laterally parallel to posterior communicating artery. On lateral side of posterior clinoid process, pierces dura and enters lateral wall of cavernous sinus, above IV CN. Divides into superior and inferior divisions which enter the orbit through Superior Orbital fissure within annulus of zinn.

**Supply :** Superior rectus, Inferior rectus, Medial rectus, Inferior oblique and Levator Palpebrae superioris.

**Trochlear Nerve :**

Longest and slender intracranial nerve. Dorsal emergence from the tectum.

**Nucleus :** Situated in the midbrain at the level of inferior colliculus. Ventral to the sylvian aqueduct

**Course :** Emerges on posterior surface of brain stem, decussates and passes forward around cerebral peduncle between posterior cerebral and superior cerebellar artery. Enters lateral wall of cavernous sinus and lies below III Cranial Nerve and above first division of Fifth Cranial nerve.

Passes through the upper part of superior orbital fissure outside the tendinous ring.

**Supply :** Superior oblique muscle

**Trigeminal Nerve :**

**Nucleus :** Extends from Pons, throughout medulla oblongata upto second cervical segment in spinal cord

**Supply :** Ophthalmic division is entirely sensory and innervates the ocular structures through three branches – Lacrimal, Frontal and Nasociliary nerve.

**Abducens Nerve :**

**Nucleus :** Lies in Mid level of pons ventral to floor of fourth Ventricle

**Course :** Emerges between pons and medulla oblongata, runs upward, forward and laterally, makes an acute bend across sharp border of petrous part of temporal bone and runs within cavernous sinus infero lateral to Internal carotid artery. Enters orbit through superior orbital fissure between the two divisions of III cranial nerves.

**Supply :** Lateral rectus muscle.

## Facial Nerve :

**Nucleus :** Lies within pons

**Course :** Emerges on anterior surface of brain stem at the lower border of pons and passes laterally in the posterior cranial fossa. Travels through the internal acoustic meatus along with eighth cranial nerve, descends through posterior wall of the middle ear and emerges from skull via stylomastoid foramen.

**Supply :** Motor supply to muscles of facial expression and orbicularis oculi, sensory innervation to the anterior 2/3rds of the tongue i.e. taste.

## Sympathetic supply :

**Nucleus :** Postero lateral part of hypothalamus

**1st order neuron :** Passes from here through pons medulla and synapse at cilio spinal centre of Budge (C8 to T2)

**IInd order neuron :** Leaves the spinal cord and travels along the ventral roots of C8 to T2 and ascend with the sympathetic chain and synapse at the superior cervical ganglion at the base of the skull.

**III rd order neuron :** From here sends post ganglionic fibres, which ascends along internal carotid artery and then leaves the cavernous sinus and joins ophthalmic division of Vth nerve and reaches the dilator pupillae muscle without synapsing in the ciliary ganglion.

## PHYSIOLOGY

**PUPIL** : Normal size is 3-5 mm

**LIGHT REFLEX :** Direct and consensual reflex

**Pathway :** Light → Retinal photoreceptor → optic nerve → optic chiasm → optic tract → Pretectal nucleus → both EW nuclei → ciliary ganglion → short ciliary nerves to sphincter pupillae

**NEAR REFLEX :** Synkinesis involving

1) Accommodation
2) Convergence of visual axis
3) Pupillary constriction

**Centre :**

In the pons, more ventrally located than the pretectal limb of light reflex. Peristriate cortex (area 19) at the upper end of the calcarine fissure may be the origin of near reflex.

**Light – near Dissociation :**

condition in which light reflex is absent / diminished although near response is intact.

Seen in compressive lesions which preferentially involve dorsal pupillomotor fibres e.g. pinealomas

Pupillary involvement is more common in surgical lesions (trauma / tumors) which cause compressive damage to the more superficially located pupillomotor fibres.

**ABNORMAL PUPILLARY REACTIONS :**

1. Amaurotic pupil : due to complete optic nerve lesion. No PL in the affected eye, both pupil equal size, direct reflex absent, consensual present, near reflex normal.
2. RAPD (Marcus Gunn Pupil) : Due to incomplete optic nerve lesion. Elicited by “Swinging flash light” test. Consensual reflex is more pronounced than direct reflex in involved eye.

3. Argyll Robertson Pupil : Bilateral Miotic pupil. Light reflex absent, accommodation reflex present. Cause : Syphilis and Diabetes, chronic alcoholism, Multiple sclerosis sarcoidosis
4. Adie's Pupil : Large unilateral dilated pupil, poor light reflex, near response sluggish or tonic contraction of the iris due to ciliary ganglionitis.
5. Horner's syndrome : Ptosis, miosis, anhydrosis, pupillary reactions are normal to both light and near. Lesions involving sympathetic pathway.

## EPIDEMIOLOGY OF BRAIN TUMORS

The overall annual incidence of primary brain CNS tumors is 12.7 / 100000 person / year.

The incidence rate of primary malignant brain tumors is 6 / 100000 / yr with higher incidence in males than females.

The neuro epithelial tumors occur more frequently in males whereas benign meningiomas are almost twice as common in females.

Primary brain tumors are the second most common cancer in childhood with incidence of 3.8 per 100000 person year.

Frequency distribution of brain tumor

Adults : Glioma - 40 – 60%

Meningioma - 9 – 27%

Pitutary adenomas - 15%

Children

Medullo blastoma - 20 – 25%

Cerebellar astrocytoma- 12-18 %

Brain stem glioma - 6 – 15%

Supra tentorial astrocytoma-14-32%

The average age of onset of all primary brain tumors is 54 years. This figure varies with histologic type.

GBM & Meningioma - 62 years

Medullo blastomas - 16 years

Highest incidence for all brain tumors occurs in 75 to 84 year – old age group. In children the peak incidence occur in patients younger than 4 years of age

An increase in the incidence and mortality of brain tumors in adult has been noted over the past several decades reaches as high as 300%. For children the incidence has increased approximately 35%.

Some of these increased are certainly a result of better diagnostic imaging and improved access to medical care.

WHO classification of CNS tumors

**a) Primary**

1. Tumor of neuro ectodermal tissue
2. Tumor of meninges and related tissue
3. Tumors of mal developmental origin
4. Tumors of pituitary gland
5. Tumors and hamartomas of blood vessels
6. Tumors of cranial and peripheral nerves
7. Tumors of hematopoietic cells and tissue

**b) Secondary tumor**

Primary CNS tumors, comprise approximatly 50-70 % of intra cranial tumors with majority being neuro epithelial tissue tumors.

Neuro epithelial tumors are further sub classified into following types.

I - Astrocytic tumors
II - Oligo dendroglial tumors
III - Mixed tumors
IV - Choroid plexus tumors
V - Neuronal and mixed neuronal glial tumors
VI - Pineal tumor
VII - Embryonal tumor

VIII - Neuro blastic tumor

IX - Glial tumors of uncertain etiology

## CLINICAL FEATURES

**Diagnosis of Tumors** :

Clinical features of intracranial tumors can be categorized as

1. Non – localizing signs and symptoms
2. False – localizing signs and symptoms
3. Truly localizing signs and symptoms

**Non – Localizing signs and symptoms :**

These are independent of site of tumor and primarily due to increase in intracranial pressure. Include Headache, Nausea, vomiting, generalized seizures and change in level of consciousness, papilledema, ocular palsies particularly of the sixth nerve.

**A. HEAD ACHE :**

- More than 50% of patients with intracranial tumors complain of headache (48%-75%)
- Classic brain tumors presenting with headache of extreme severity, which is worse in morning, associated with nausea and vomiting are quite uncommon.

- Usually, headache presents, with a tension type of headache (77%) or migraine (9%) – mild to moderate severity, bifrontal worse on side of lesion, worse on bending down and associated with nausea and vomiting.
- Headache associated with an increase in intracranial pressure is often generalized, non focal and non lateralizing to the tumor site. Conversely headache not associated with increased intracranial pressure may localize the tumor. Tumors in anterior / middle cranial fossa have frontal, often supra orbital headache.
- Tumors in posterior fossa often present with sub occipital pain

**Mechanism of headache :**

1. Sudden changes of intracranial pressure (whether increase or decrease)
2. Traction upon / displacement of pain sensitive intracranial structures.
3. Acute hemorrhage into tumor
4. Venous sinus thrombosis
5. Acute obstructive hydrocephalus

6. Contiguous involvement of bone / meninges / sinus
7. Toxic effects of various therapies.

**Detailed Neurologic and Ophthalmic Examination is indicated in:**

1. Intense / prolonged / incapacitating headache
2. Changes in frequency / pattern / quality of headache
3. Recent onset of increased vomiting with headache
4. Associated with seizures or other focal neurological abnormalities
5. Headache that awakens individual from sleep

## B) VOMITING

- Usually associated with headache
- Projectile nature, especially if occurring without relationship to intake of food, should arouse suspicion of increased intracranial pressure.
- Related to rise of ICP or rarely tumor invasion of area postrema or vagal nucleus in the posterior fossa

## C) SEIZURES :

Occurrence of a seizure in a previously healthy individual should be regarded to be due to intracranial tumors, until proved otherwise, as

about 1/3 adults who develop new onset of seizures harbour an intracranial tumor.

There is no precise correlation between seizure pattern and tumor location – but usually, a seizure associated with forced turning of head and eyes (versive movement) indicates a tumor located opposite to the direction of head and eye movement.

**D) ABDUCENS NERVE PARESIS :**

Due to its long subarachnoid course, VI CN is susceptile to damage due to increased intracranial pressure.

Mechanism :

1) VI nerve gets compressed between pons and basilar artery
2) The nerve gets stretched along sharp edge of petrous bone

E) **Signs of increased ICP** include papilledema Diplopia, cranial nerve palsies, motor and sensory abnormalities and change in level of consciousness.

Mechanism of increased Intra cranial pressure with Intra cranial tumors

1. Increase in total amount of intracranial tissue

2. Increase in intracranial volume due to cerebral edema (focal / diffuse)
3. Blockage of CSF causing hydrocephalus (obstructive / communicating)
4. Decreased absorption CSF due to compromised cerebral venous outflow
5. Increased CSF production

**PAPILLEDEMA :**

Most important and classical sign of elevated intracranial pressure

Optic disc swelling associated with increased intracranial pressure

Papilledema is relatively unknown in infants due to non-fusion of cranial structures which allows room for enlargement

Infratentorial tumors (from cerebellum and fourth ventricle) tend to increase intracranial pressure by obstructing flow of CSF through aqueduct of sylvius.

Supratentorial and third ventricular tumors cause papilledema frequently due to the mass effect of the lesion or surrounding edema.

Papilledema is rarely seen in pituitary gland tumors

There is greater tendency for papilledema to develop with slowly growing tumors than rapidly expanding lesions.

SYMPTOMS :

1. Initially, visual symptoms are absent
2. Transient obscurations of vision, lasting for few seconds, may occur.
3. In long standing cases, if optic atrophy sets in visual acuity falls with progressive constriction of visual field.

SIGNS :

1. Hyperemic disc with indistinct margins, elevated from surrounding retina
2. Loss of spontaneous venous pulsation
3. Venous engorgement and peripapillary flame shaped hemorrhages
4. Fundus fluorescein angiography initially shows dilated disc capillaries followed by increased hyper fluorescence which extends beyond the disc margin.
5. Visual fields show enlargement of blind spot.

# FALSE – LOCALIZING SIGNS AND SYMPTOMS

Defined as a sign which potentially causes confusion in diagnosis by suggesting an abnormality at a distance away from the actual site of lesion. (It is a clinical sign that is often misinterpreted)

**Mechanism**

General compression of a nerve having a long course

Meningitis

Edema and gliosis

Metastatic infiltration

Hydrocephalus

Gross brain displacement with shift of sagittal plane

Dementia, visual field defects, visual hallucinations, cranial neuropathies, cerebellar signs, pyramidal and extra pyramidal signs, nystagmus, restriction of extra ocular movements, proptosis etc. All these can manifest as a false localizing sign at one time or other.

## TRULY LOCALIZING SIGNS AND SYMPTOMS

### TUMORS INVOLVING ORBIT :

These tumors tend to produce ocular manifestations early due to their mass effect.

### Gaze-evoked amaurosis :

Transient obscurations of vision in one particular gaze, occurs in cavernous hamangiomas of orbit or optic nerve sheath meningiomas due to compromised blood supply of optic nerve and retina in that gaze.

Proptosis : Amount of protrusion of eyeball depends on

1. speed of growth of tumor – Rapidly growing tumors produce greater mass effect.
2. Volume of tumor – Optic nerve sheath meningiomas, cause minimal proptosis and yet profound visual loss.
3. Consistency of tumor – Vascular tumors (soft in consistency) usually do not produce proptosis early, whereas gliomas manifest early with proptosis.

**Optociliary shunt Vessels :**

Along with ipsilateral visual loss and optic swelling or optic atrophy form a triad which indicates chronic compression of intraorbital optic nerve eg. Spheno-orbital meningioma.

Choroidal folds and limitation of ocular movements with a positive forced duction test are the other indicators of a tumor invading the orbit.

## TUMORS INVOLVING SUPERIOR ORBITAL FISSURE AND CAVERNOUS SINUS

Tumors in this region can usually manifest as :

**Sphenocavernous syndrome -** Consists of paralysis / paresis of more than one ocular motor nerve, with first and second division of trigeminal nerve, Horner's syndrome and visual loss, Peri orbital or hemicranial pain.

**The orbital apex syndrome** should be reserved for multiple ocular motor cranial nerve palsies plus optic nerve dysfunction.

III CN paresis associated with small pupil can occur due to involvement of both III CN and the sympathetic pathway in the cavernous sinus, where they are closely related.

VI CN paresis with ipsilateral Horner's syndrome can result because the sympathetic fibres join briefly with VI CN before they merge with ophthalmic division of V CN.

Primary oculomotor nerve synkinesis (Primary aberrant regeneration) – It is a condition in which misdirected innervation of extraocular muscles normally innervated by III CN occurs without any previous history of acute III CN paresis. Commonly seen with slowly growing lesions of cavernous sinus. e.g. meningiomas or aneurysms.

**TUMORS INVOLVING SUPRASELLAR REGION :**

Intracranial part of optic nerve, optic chiasm, optic tract and hypothalamus are the regions commonly involved.

The type of tumors at this site are pituitary adenomas, meningiomas, craniopharyngiomas and gliomas.

**Syndrome of distal optic nerve** (Anterior chiasmal syndrome) :

Compression of intracranial part of optic nerve leads to central or arcuate scotomas, peripheral constriction or hemianopic field loss.

Decussating fibres from lower nasal half of retina loop anteriorly into the contralateral optic nerve before they go on to the

optic tract damage to this area (Wilbrand's knee) produces a characteristic field defect called Junctional scotoma of Traquair.

**Syndrome of Optic Chiasma :**

Characteristic feature is a bitemporal quadrantanopia or hemianopia that respects the vertical midline.

Pattern of progression of visual field can give an indication to the site of lesion – pituitary tumors cause compression from below : so the field defect starts as superior temporal quadrantanopia and then progresses to inferior quadrant.

Craniopharyngiomas, on the contrary, compress the optic chiasm from above – hence in this case, field defect first occurs in the inferior quadrant and proceeds upwards.

Deterioration of vision is the first complaint of individuals who suffer from intrasellar tumors.

Sudden decline in vision with extraocular muscle palsy and altered consciousness indicates a PITUITARY APOPLEXY resulting from hemorrhage or infarction into a pituitary adenoma.

## Syndrome of optic tract :

- Contra lateral homonymous hemianopia is the typical feature.
- Afferent pupillary defect can occur without loss of visual acuity or colour vision in the eye ipsilateral to hemianopia (i.e. contralateral to side of lesion)
- Incongrous field defects
- Bilateral retinal nerve fibre layer atrophy
- Wernicke's pupil
- Behr's pupil (inequality of pupil with large pupil on the side of hemianopia)
- Negative OKN

## Hypothalmic syndrome :

Disturbance in hormones secreted by hypothalamus can give rise to various problems like diabetes, adiposogenital dystrophy (Frolich's syndrome), Gastric hemorrhages (Cushing's Ulcers) etc.

## Syndrome of pituitary dysfunction :

Features of hypo or hyper pituitarism can be present depending on the nature of the lesion.

## Parasellar Tumors :

The manifestations can be divided into

1. Those arising from outer 1/3 of sphenoid ridge
2. Those arising form middle 1/3 of sphenoid ridge
3. Those arising from inner 1/3 of sphenoid ridge

They produce ocular symptoms if they expand in a medial direction and involve the optic nerve and superior orbital fissure.

Characteristic feature is a unilateral exophthalmos with variable paresis of III, IV & VI cranial nerves and occasional hypoesthesia of the ipsilateral cornea.

## TUMORS OF FRONTAL LOBE :

Gliomas (astrocytoma, oligodendroglioma, glioblastoma multiforme) are the most common tumors of frontal lobe followed by meningiomas.

A change in psyche manifesting as withdrawn behaviour, apathy, irritability, senseless joking loss of social inhibition etc may lead on to the full picture of dementia.

Lesion that affect premotor area produce apraxia and rigidity, whereas those that affect motor cortex produce contralateal weakness. Frontal release sign, include abnormal grasp, sucking, snout and palmomental reflexes.

Aphasia develops if dominant hemisphere is involved. Slowly progressive monocular visual loss with central scotoma occurs due to pressure on optic nerve.

Damage to frontal gaze centre manifests as defective saccades to opposite side.

**Foster Kennedy syndrome :**

Ipsilateral optic atrophy with contra lateral papilledema is a sign of great localizing value.

Aversive seizures (conjugate horizontal deviation of eyes with head turning away from side of tumor) can occur.

**TEMPORAL LOBE TUMORS :**

About 10% of all intracranial and 20% of all supratentorial tumors involve this lobe.

Seizures – psychomotor / partial complex type constitute outstanding feature of these tumors.

Cause Abnormalities of speech, hearing, memory and vision.

Dominant temporal lobe syndrome includes auditory hallucinations, dysnomia, sensory aphasia and impairment of recent memory and visual field defect. Visual hallucination of formed images, micropsia, Macropsia and Teichopsia may be experienced.

Visual field defects are the most conspicuous. Most common type of defect is incongruous homonymous hemianopia with sloping margins. The field defect can begin as a contralateral upper homonymous quadrantanopia called ‘pie-in-sky’ defect.

Non dominant temporal lobe lesion produce spatial disorientation and problem with perception of taste, hearing, vision or movement as well as difficult in memory retention.

Temporal lobe tumors extending posteriorly may cause alexia and anomia.

## TUMORS OF PARIETAL LOBE :

Involvement of sensory cortex (Post central gyrus) produces cortical sensory loss an opposite side which manifests as loss of touch, pain and position sense.

### Gerstmann syndrome :

Finger agnosia, right-left disorientation, acalculia and agraphia constitute a lesion in the dominant parietal lobe.

Visual hallucinations are of unformed variety

Field defects start as inferior homonymous quadrantanopia or ‘pie-on-floor’ defect.

Neglect syndrome may occur with tumors of the non dominant parietal lobe including lack of awareness of objects in contralateral visual field or of movement or position of the body on the opposite side.

Asymmetric opto-kinetic nystagmus is an important indicator of lesions at this site.

General signs of increased intra-cranial pressure are uncommon. Visual field defects which are largely congruous are the

main feature complete hemianopia with macular sparing is the typical presentation.

**Cogan's Rule :** Asymmetric opto-kinetic nystagmus response in a patient with occipital lesion indicates additional parietal lobe involvement. This suggests that the occipital lobe lesion is neoplastic in nature.

Visual agnosia (failure to recognize objects in absence of visual impairment), colour agnosia, prosopagnosia (inability to recognize familiar faces) and pallinopsia (persistence of visual image after the stimulus is removed from the visual field) are the other manifestations.

**TUMORS OF THE CEREBELLOPONTINE ANGLE :**

The commonest tumors in this location are Acoustic neuromas.

Characteristic features are paresis / paralysis of fifth, sixth, seventh and eighth cranial nerves.

Earliest symptom is tinnitus (high-pitched) and deafness due to involvement of eighth cranial nerve.

The first sign to occur is decreased corneal sensation manifesting as absent corneal reflex.

Other than the lower cranial nerve palsies, there can be cerebellar signs and papilledema.

**TUMORS OF OCCIPITAL CORTEX :**

A neurologically silent area except for ocular symptoms

Headache

Rarely fits

Field defects

(Contra lateral to the side of lesion)

1. Macular sparing homonymus type
2. Tubular fields
3. Congruous field defects
4. Riddoch phenomenon - perception of form and movement separately
5. Visual hallucination are of unformed images as flash of light of various shapes
6. Alexia without agraphia : Extension of the occipital lobe tumor into splenium of corpus callosum.

Anton syndrome - bilateral lesions – patient cortically blind but not aware of his blindness.

TUMORS OF MIDBRAIN :

Localising signs depends on involvement of pyramidal tracts and the oculomotor nerves.

All of them may be associated with homonymous herninopia owing to pressure on the optic tracts.

**In upper part of midbrain**

- Ptosis, together with loss of conjugate movement upwards
- Light near dissociation
- Vertical Nystagmus and adduction movements on attempted vertical gaze

**At intermediate level :**

Webers syndrome- Ipsilateral ptosis & complete third nerve paralysis associated with contralateral hemiplegia due to involvement of cerebral peduncles

Benedict syndrome – if red nucleus is involved, tremors & Jerky movements occur in contralateral side combined with ipsilateral third nerve palsy.

**At the level of Pons :**

In the upper part, third nerve paralysis with contralateral hemiplegia and UMN type facial palsy.

In the lower part of Pons, the lateral rectus may be paralysd with a contralateral hemiplegia and ipsilateral facial palsy. (Millard – Gubler syndrome)

In Foville syndrome, there is loss of conjugate movement to the same side instead of sixth nerve palsy.

**Metastasis :**

Multiple lesions in brain are characteristics of secondaries

The signs depend on the site of involvement

Breast and lung carcinoma are the leading primary tumors.

## INVESTIGATIONS

1. **General :**

    Blood count

    Peripheral blood smear

    ESR

    Serum Calcium

    Chest X ray

**2. Ophthalmic Invest :**

1. Visual acquity By Snellen chart
2. Refraction & BCVA
3. Colour perception by Ishihara
4. Fields – Confrontation

   Tangent screen

   Automated perimetry
5. Contrast sensitivity
6. Extraocular movement
7. Diplopia charting
8. Hess screening
9. Slit lamp examinations
10. Fundus examination
11. Hertels exophthalmometry
12. Fundus flourescein Angiography

**3. Radiological**

Skull X ray → Look for calcification– Oligodendroglioma

Meningioma

Craniopharyngioma

Osteolytic lesion → 1 or 2 tumor

Dermoid / epidermoid

Chordoma

Sign of raised ICP → Beaten Brass appearance

Sutural separation in children

**CT Scanning**

Look for Site

Mass effect – Midline shift

Ventricular compression

Hydrocephalus

Effect on adjacent bone – eg Meningioma – hyperostotic

Single or multiple lesions

Effect of contrast enhancement

MRI – Site / Mass effect / Lesion multiplicity

Angiography

# TREATMENT

Treatment of any intracranial tumor is basically by surgery. First – the increased ICP is controlled by drugs like osmotic diuretics.

The tumor is either totally excised or subtotally excised. Tumor is sent for HPE. Radiotherapy is another modality of treatment .

Role of ophthalmologist is to identify those cases of headache with field effects and aid in diagnosing a brain tumor and refer the patients in time to Neuro surgeon.

# AIM OF THE STUDY

To study the value of ocular signs in predicting the presence of an intracranial tumor.

To correlate the ocular symptoms and signs with the location of the brain tumor.

# MATERIALS AND METHODS

The study was conducted in the Department of ophthalmololgy, Government Rajaji Hospital, Madurai. The period of study was between January 2006 to August 2007.

The total of sixty patients with signs and symptoms suggestive of brain tumor were examined and then referred to neuro surgery department, GRH and followed up. Also the patients referred from neuro surgery department to our department were also included in the study.

**INCLUSION CRITERIA :**

Patients who had typical CT scan findings of a brain tumor were included.

Patients with signs and symptoms suggestive of brain tumor but whose CT was normal were excluded.

Patients with SOL which were diagnosed as an abcess / tuberculoma / cysticercosis were also excluded (confirmed by neuro surgery department).

**Neurological Evaluation :**

At first, detailed history was elicited

**Emphasis laid on the following complaints**

1. Headache – site, duration, associated with vomiting
2. Motor symptoms – paresis / paralysis

3. Convulsions – focal / generalised
4. Mental changes – depression / altered behaviour
5. GAIT abnormality
6. Cranial nerve lesion – deviation of mouth / altered smell / hearing loss
7. Endocrine systems – amenorrhea – galactorrhoea / enlarged hands / impotence / polyuria / polydipsia

**Ophthalmic evaluation :**

1. Visual acuity : Decreased vision / Decreased field of vision
2. Diplopia
3. Colour vision : with ishihara chart
4. Visual hallucinations (Flashing of lights / forced / unformed images)
5. Oscillopsia
6. Visual Acuity tested with snellens distant vision chart
7. IOP measured with schiotz tonometer
8. Fundus : After dilating the pupil, details of disc noted
9. Field charting :Lister's perimeter
   Confrontation method
   Bjerrum's 2m screen
   Automated perimetry with Medmont
10. Proptosis : measured with Hertel's exophthalmometry
11. Diplopia charting with Red – Green goggle

**Systemic Evaluation :**

History of diabetes / Hypertension / Endocrine abnormalities cardiac / respiratory functions were assessed.

**Investigations :**

**Ophthalmic :**

X ray of orbit, optic foramen to rule out any lesion

Fundus Fluorescein angiography in selected cases to confirm papilledema when in doubt

Neurological : Some patients had CT / MRI (referred from NS)
Others were subject to X ray skull

**Management :**

First, the raised intracranial tension was controlled with medical management (osmotic diuretics / steroids)

Then neuro surgical intervention was advocated

Total excision and Biopsy / sub total excision as the case warranted. Then the biopsy material were sent for histopathological examination and further treatment was instituted if necessary

Radiotherapy given to inaccessible tumor or surgical recurrences.

Hormonal treatment given for patient's with Endocrine abnormality.

# RESULTS

**Sex Distribution :**

Out of 60 patients, 34 were male and 27 were female indicating a greater pre disposition in males.

| Sex | No.of patients | Percentage |
|---|---|---|
| Male | 34 | 56 |
| Female | 26 | 44 |
| Total | 60 | 100 |

Age Distribution :

| Age in years | No.of patients | Percentage |
|---|---|---|
| < 10 | 2 | 3.33 |
| 11-20 | 10 | 16.66 |
| 21-30 | 10 | 16.66 |
| 31-40 | 13 | 21.66 |
| 41-50 | 21 | 35.00 |
| 51-60 | 4 | 6.66 |
| Total | 60 | 100 |

Maximum number of patients were seen in the 41-50 age groups. Next was in the 31-40 age groups.

**Age and Sex Distribution :**

| Age | Male | Female | Total |
|---|---|---|---|
| < 10 | - | 2 (7.69%) | 2 |
| 11-20 | 4(11.76%) | 6 (23.07%) | 10 |
| 21-30 | 6(17.64%) | 4(15.38%) | 10 |
| 31-40 | 8 (23.52%0 | 5 (19.23%0 | 13 |
| 41-50 | 13 (38.23%) | 8 (30.76%) | 21 |
| 51-60 | 3 (8.82%) | 1 (3.84%) | 4 |
| Total | 34 | 26 | 60 |

Males and females were equally affected in 21-30 age group. There were more no. of male patients 38.23% in the older (41-50 years) age group than female patients.

Type of tumor (based on HPE report)

| Age in years | No.of patients | Percentage |
|---|---|---|
| Meningioma | 11 | 18.33 |
| Acoustic Neuroma | 6 | 10 |
| Pituitary Adenoma | 8 | 13.33 |
| Craniopharyngioma | 5 | 8.33 |
| Astrocytoma | 7 | 11.66 |
| Glioma | 7 | 11.66 |
| Pinealoma | 2 | 3.33 |
| Medulloblastoma | 2 | 3.33 |
| Not known | 12 | 20.00 |

Glial tumors (10) both astrocytoma and glioma form the major chunk of intracranial tumors i.e. 14 cases followed by Meningioma 11 cases. (18.33%).

Pituitary adenomas were next in frequency with 8 patients in this group and they cause compression of optic chiasm from below and cause visual symptoms and field defects.

**Sex Distribution among tumors :**

| Type of Tumor | Male | Female | Total |
|---|---|---|---|
| Meningioma | 4 (11.76%) | 7(26.92%) | 11 |
| Acoustic Neuroma | 2(5.88%) | 4 (15.38%) | 6 |
| Pituitary Adenoma | 4 (11.76%) | 4 (15.38%) | 8 |
| Craniopharyngioma | 3 (8.82%) | 2 (7.69%) | 5 |
| Astrocytoma | 5 (14.70%) | 2 (7.69%) | 7 |
| Glioma | 6(17.64%) | 1(3.84%) | 7 |
| Pinealoma | 1 (2.94%) | 1 (3.84%) | 2 |
| Medulloblastoma | 2(5.88%) | - | 2 |
| Not known | 7 (20.58%) | 5 (19.23) | 12 |

Comparing the incidence of different types of tumor (based on HPE report) in both sexes, it was found that Meningiomas were seen more frequently in females (26.92%) then males 4(11.76%).

Pituitary Adenomas were found with equal frequency among both male and female patients 4 cases each as were pinealoma.

**Presenting complaints :**

Both ocular and non-ocular complaints were recorded. The no.of patients first seen by an ophthalmologist were 38 in number. Few patients were seen in neuro surgery department and referred to the ophthalmology department. Hence ophthalmologist play a major role in recognizing brain tumors at an early stage.

**Ocular Symptoms :**

| Ocular Symptoms | No.of patients | Percentage |
|---|---|---|
| Diminished vision | 26 | 43.33 |
| Field defects | 11 | 18.33 |
| Diplopia | 10 | 16.66 |
| Ptosis | 5 | 8.33 |
| Amaurosis fugax | 2 | 3.33 |
| Proptosis | 2 | 3.33 |

The most common ocular symptom recorded was diminished vision. Diplopia was due to ocular cranial nerve palsies (mostly sixth nerve palsy) which is a “false localizing” sign in patients with increased intracranial pressure.

**Ocular symptoms and site of Brain tumor :**

| Site | No.of patients with decreased vision | No.of patients with Diplopia |
|---|---|---|
| Frontal | 1 (3.85%) | 1 (10%) |
| Frontoparietal | 1 (3.85%) | - |
| Parietal | 4 (15.38%) | - |
| Temporal | 3 (11.535) | 1 (10%) |
| Tempero parietal | 1 (3.85%) | 1 (10%0 |
| Sellar / Parasellar suprasellar | 10 (38.5%) | 2 (20%) |
| CPA | 3 (11.53%) | 4 (40%) |
| Cerebellar | 1 (3.85%) | - |
| Sphenoid bone | 2 (7.69%) | 1 (10%) |
| Total | 26 | 10 |

Out of the 26 patients who complained of diminished visual acuity, 10 patients (38.46%) had a space occupying mass in the sellar, parasellar or suprasellar region.

Among the 10 patients complaining of diplopia majority were found to have a CPA mass on CT (40%).

**Non Ocular symptoms :**

| Non ocular symptoms | No.of patients | Percentage |
|---|---|---|
| Head ache | 50 | 83.33 |
| Weakness | 20 | 33.33 |
| Convulsion | 18 | 30.00 |
| Deafness, vertigo, Tinnitus | 10 | 16.67 |
| Endocrine abnormality | 6 | 10 |
| Numbness | 2 | 3.34 |
| Deviation of angle of mouth | 4 | 6.67 |
| Abnormal behaviour | 3 | 5 |

Headache (with or without vomiting) was the commonest symptom reported by patients. This was found to be consistent with the standard teachings from text books. This is a non-localizing symptoms as mentioned previously.

Ophthalmic Signs :

| Ophthalmic signs | No.of patients | Percentage |
|---|---|---|
| Papilledema | 28 | 46.67 |
| Cranial nerves | | |
| II | 14 | 23.34 |
| III | 4 | 6.67 |
| IV | 1 | 1.59 |
| V | 6 | 10 |
| VI | 13 | 21.66 |
| VII | 10 | 16.66 |
| Field defects | 15 | 25 |
| Nystagmus | 5 | 8.34 |
| Gaze palsy | 4 | 6.67 |
| Proptosis | 2 | 3.33 |

Papilledema was the commonest sign. This was a major finding in CPA tumors. Patient's presented early and late with optic atrophy also. Few were confirmed by FFA.

**Correlation of ophthalmic signs with the site of tumor**

| Site of tumor | Papilledema | II CN defect | Eye movement defect | Field defect |
|---|---|---|---|---|
| Frontal | 3 (10.71%) | 1(7.14%) | 1(6.25%) | - |
| Fronto parietal | 1 (3.57%) | 1(7.14%) | - | - |
| Parietal | 5(17.85) | - | 1(6.25%) | 4(26.67) |
| Temporal | 2(7.14) | - | - | 1(6.67%) |
| Temporo parietal | 3(10.71) | - | 1(6.25%) | 1(6.67%) |
| Sellar / parasellar suprasellar | 3(10.71) | 11(78.5%) | 2 (12.5) | 8(53.34) |
| CPA | 7 (25%) | 1(7.14%) | 7(43.75%) | 1 (6.67) |
| Cerebellar | 3(10.71) | - | 2(12.5%) | - |
| Sphenoid bone | - | - | 1(6.25%) | - |
| Pineal gland | 1(3.57%) | - | 1(6.25%) | - |
| Total | 28 | 14 | 16 | 15 |

Occurance of papilledema was common with CP angle tumors. These patients also had diplopia due to VI N palsy – false localizing signs. Sellar mass patients had II N involvement and had visual symptoms.

# DISCUSSION

Out of 60 patients, 34 were male and 27 were female indicating a greater pre disposition in males.

Maximum number of patients were seen in the 41-50 age groups. Next was in the 31-40 age groups.

Males and females were equally affected in 21-30 age group. There were more no. of male patients 38.23% in the older (41-50 years) age group than female patients.

Glial tumors (10) both astrocytoma and glioma form the major chunk of intracranial tumors i.e. 14 cases followed by Meningioma 11 cases. (18.33%).

Pituitary adenomas were next in frequency with 8 patients in this group and they cause compression of optic chiasm from below and cause visual symptoms and field defects.

Craniopharyngiomas were diagnosed in 5 patients (8.33%). These tumors have tendency to cause compression of optic chiasm from above and hence lead to inferior field defects.

Acoustic neuromas were seen in 6 patients (12.4%). They formed a major chunk of CP angle tumors and infra tentorial in a majority of children and 1/3 of adult brain tumors. They are part of type I neuro fibro matosis.

12 patients (20%) were not subjected to excision biopsy of tumor & HPE, hence not classified. The reason being either they didn't follow our advice or didn't come for follow up despite our advice.

Comparing the incidence of different types of tumor (based on HPE report) in both sexes, it was found that Meningiomas were seen more frequently in females (26.92%) then males 4(11.76%).

Pituitary Adenomas were found with equal frequency among both male and female patients 4 cases each as were pinealoma.

The most common ocular symptom recorded was diminished vision. Diplopia was due to ocular cranial nerve palsies (mostly sixth nerve palsy) which is a "false localizing" sign in patients with increased intracranial pressure.

Out of the 26 patients who complained of diminished visual acuity, 10 patients (38.46%) had a space occupying mass in the sellar, parasellar or suprasellar region. These space occupying lesions were later confirmed to be pituitary adenomas and craniopharyngiomas by HPE. This finding is in concordance with the anatomy showing that any sellar mass is bound to cause a compressive damage to the optic chiasm and hence causes a lowering of visual acuity.

Among the 10 patients complaining of diplopia majority were found to have a CPA mass on CT (40%). The incidence of papilledema

was also high in this group only. Hence the diplopia can be accounted for by sixth cranial nerve palsy due to increased intracranial pressure (false localizing sign)

11 patients complained of defective field of vision out of which 6 patients were found to have suprasellar mass. But majority of the patients were unaware of the loss of field of vision. This could be due to occupation or depressed mentation.

Headache (with or without vomiting) was the commonest symptom reported by patients. This was found to be consistent with the standard teachings from text books. This is a non-localizing symptoms as mentioned previously.

Sudden onset of deviation of angle of mouth with drooling of saliva and inability to close the eyelids on one side was attributed to seventh nerve palsy as in CPA tumors.

Seizures were seen in 18 patients. They had a lesion in the parietal lobe (Temporo-parietal / parietal) due to mass effects.

Patients with weakness of limbs had tumor in the parietal / temporal lobes. This is due to the involvement of pyramidal tract.

Deafness / Tinnitus / Vertigo occurred in patients with CPA mass. they also had facial nerve palsy.

The patient's with endocrine complaints like amenorrhoea, Galactorrhoea, enlargement of hands and feet, delayed growth etc., had a sellar / suprasellar mass – Later found to be pituitary adenoma by HPE.

Papilledema was the commonest sign. This was a major finding in CPA tumors. Patient's presented early and late with optic atrophy also. Few were confirmed by FFA.

Next sign was cranial nerve palsies and due to compressive tumors or increased intracranial tension.

Optic nerve was commonly affected. The next common cranial nerve to be involved was the sixth nerve but it is a false localizing sign. The involvement of the IIIrd and IVth nerve was less common when compared to the sixth nerve. Here again CPA tumors are known to be associated with increased intracranial tension.

Field defects were of great importance in localizing the tumor anatomically.

Sellar / suprasellar mass lead to a bitemporal hemianopia / quadrantanopia. Not all patients were aware of the field defects.

Occurance of papilledema was common with CP angle tumors. These patients also had diplopia due to VI N palsy – false localizing signs. Sellar mass patients had II N involvement and had visual symptoms.

# CONCLUSION

Of the 60 patients studied.

- 30-50 age group was commonly affected.
- Male predilection noted.
- Pathologically the commonest tumor was Glioma.
- Headache was the commonest non localizing symptom. Papilledema was the commonest ophthalmic sign particularly in CP angle tumors.
- Patients with Sellar / parasellar tumors (Pituitary adenoma and craniopharyngioma) presented with endocrine complaints with a characteristic visual field defect.
- CP angle tumor manifested with sixth nerve palsy along with seventh and eighth nerve lesion with papilledema.

The ocular signs and symptoms helped in localizing the site of the tumor.

Ophthalmologists by and large play a major role in early diagnosis of the brain tumors, so with ocular findings as the best clinical tool and aided by radio imaging techniques, the brain tumors can be virtually fished out and the patient given early treatment thereby preventing further morbidity and mortality.

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# PROFORMA

Name : Age : Sex :

**Neurological complaint :**

- Headache
  - Duration
  - Time of the day
  - Aggravating / relieving factors
- Weakness
  - Generalised
  - Right / Left
  - Peresis / Paralysis
- Convulsions
  - Focal
  - Generalised
- Mentation
  - Depression
  - Altered Behaviour
- Gait Disturbance

**Endocrine Symptoms :**

- Amenorrhoea
- Galactorrhea
- Weight Gain
- Enlarged Hands / feet
- Impotence
- Polyuria
- Polydipsia
- Increased sweating
- Hot / Moist palms
- Increased skin pigmentation

**Cranial nerve abnormalities**

- Anosmia
- Dimness of vision
- Diplopia
- Abnormal EOM
- Loss of sensation of one half of face
- Difficulty in chewing
- Deviation of angle of mouth
- Drooling of saliva on one side
- Accumulation of food on one side
- Inability to close eye properly
- Hearing loss
- Tinnitus
- Vertigo

- Nasal twang of voice
- Nasal regurgitation of food

Deviation of Tongue

**Ocular complaints**

Visual disturbances

Dimness of vision

Blunting of color perception

Transient loss of vision

Field loss

Diplopia

Protrusion of eye ball

Drooping of upper eyelid

Difficulty in closure of eyelids

(Watering)

Eye movement abnormality

**Past History :**

DM / HT / Pulm. TB / BA / IHD / CVA

Any significant illness and Rx taken

Any operations undergone

Family History :

Menstrual History :

Personal History :

**General Physical Examination :**

Consciousness Pulse BP

Build / Nourishment

Pallor / Icterus / Cyanosis / Clubbing / Oedema / Lymphadenopathy

**CNS Examination :**

HMF : Consciousness (EMV)

Orientation : Time / Place / Person

Memory : Recent / short term / Remote

Co-operation :

Behaviour :

Judgement :

Speech : Normal / Slurred / Dysarthria / Dysphasia

Handedness (Cerebral Dominance)

Mood

**Cranial Nerves**

1. Olfactory
2. Optic
3. Oculomotor
4. Trochlear
5. Abducent
6. Trigeminal - Motor
   Sensory – Touch / pain / corneal reflects
7. Facial - Motor – upper part of face / lower part of face
   - Sensory – taste – Ant. 2/3 of Tongue

| Examination | Right eye | Left eye |
|---|---|---|

Proptosis / Exophthalmos

Orbital margins

Lids / periocular area

Conjuctiva / cornea

AC / iris

Pupil size shape

Light — Consensual

Direct

RAPD

Near reflex

Lens

Sensation — Corneal

Lids & face

OPTC N & Visual pathway

- Vision - bedside
- Ref. error & BCVA
- Colour perception – simple red target,
- Ishihara
- Brightness sense
- Fields
    1. Confrontation
    2. Tanget screen,
- Extra ocular movements
- Nystagmus
- Diplopia charting
- Conjugate movement

Oculocephalic / Oculo vertibular reflex

Optokinetic reflex

Fundus examination

Slit lamp examination

Hertels exophthalmometry

Tension

Investigation

- Skul X ray
- CT Scan
- MRI

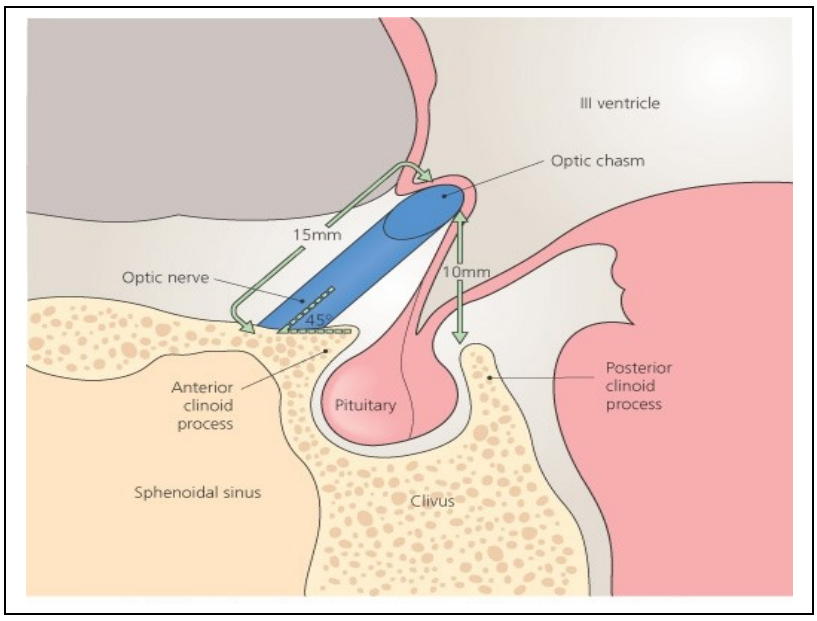
Provisional diagnosis

HPE Examination

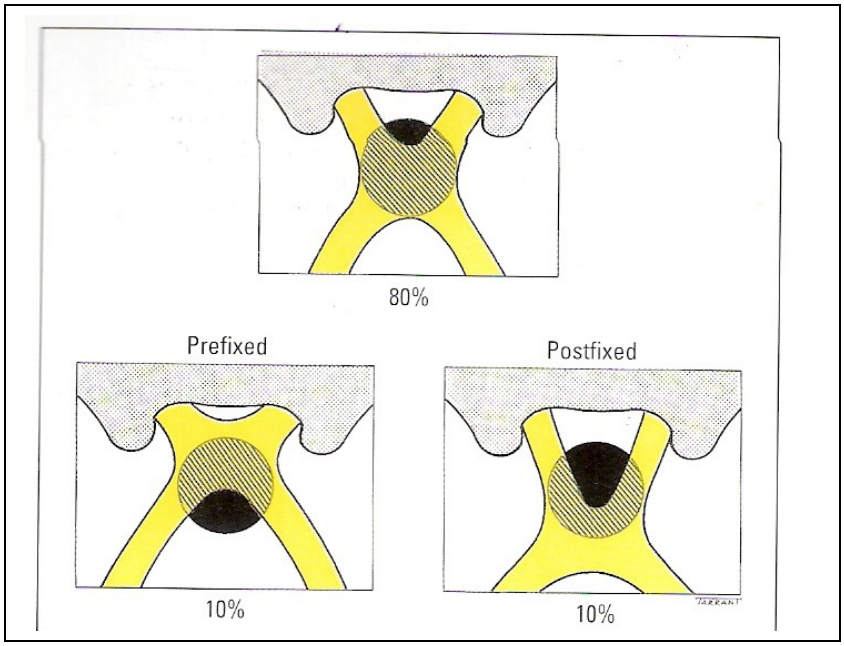
Progress and Result

Follow up

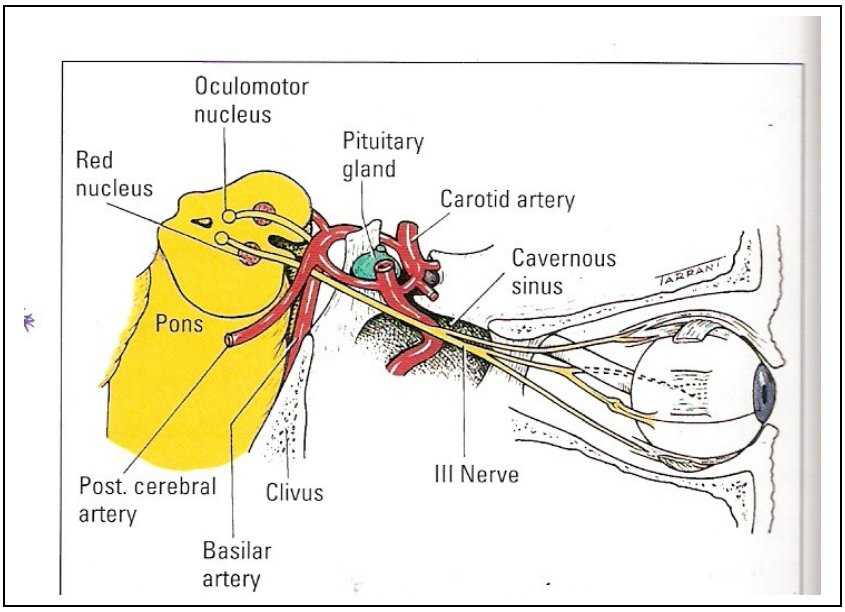
# OPTIC CHIASMA IN RELATION TO PITUITARY GLAND



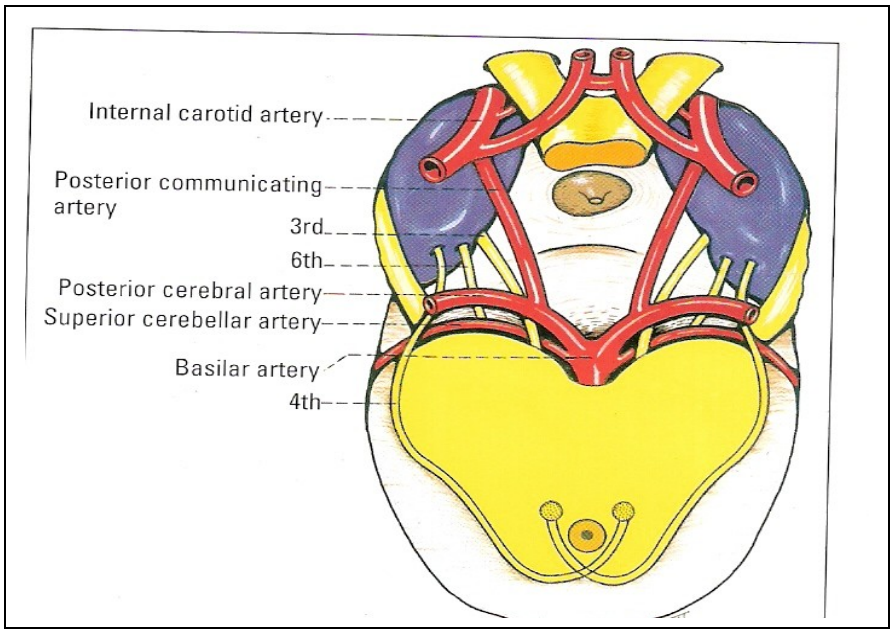

# ANATOMICAL VARIATIONS IN POSITION OF OPTIC CHIASMA



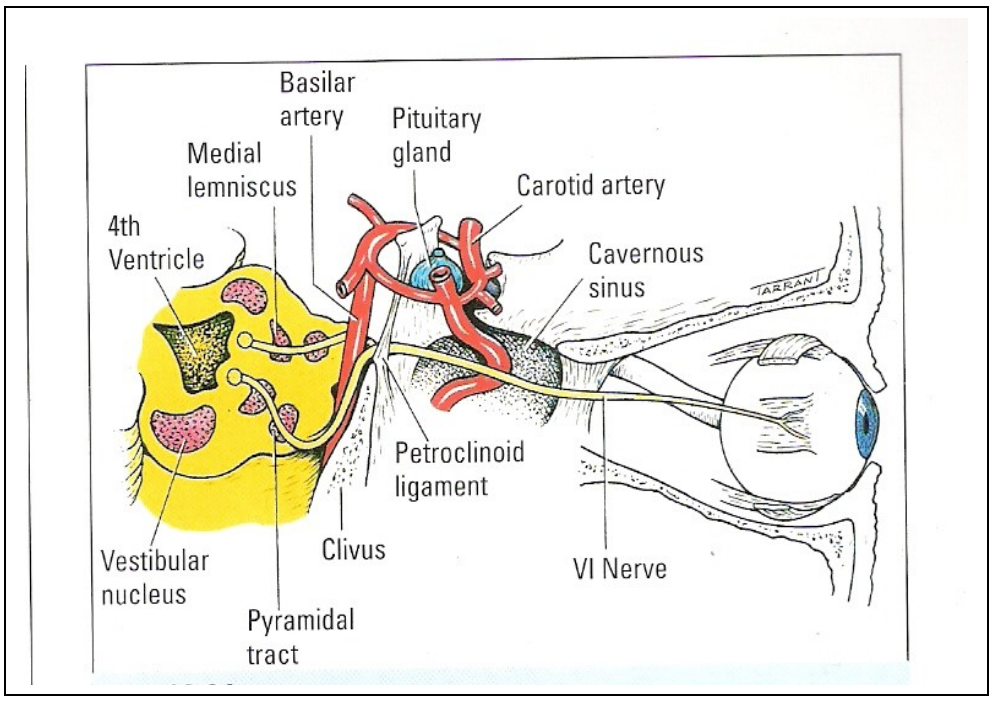
# LATERAL VIEW OF COURSE OF III NERVE



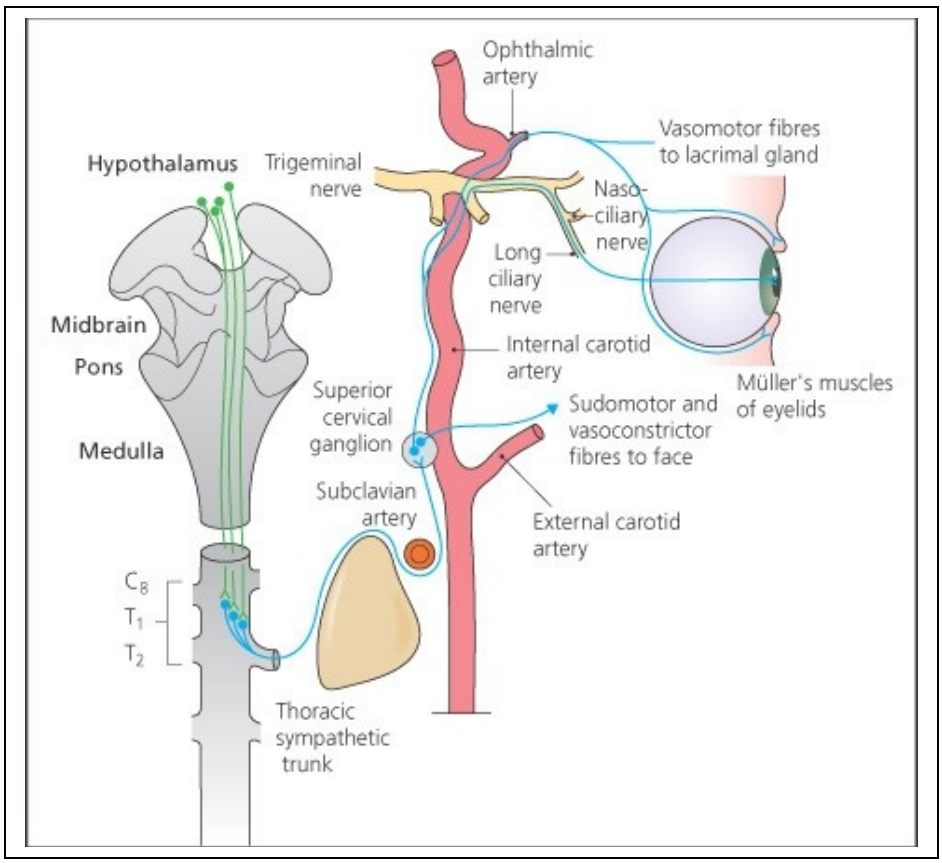

# DORSAL VIEW OF COURSE OF THE IV NERVE



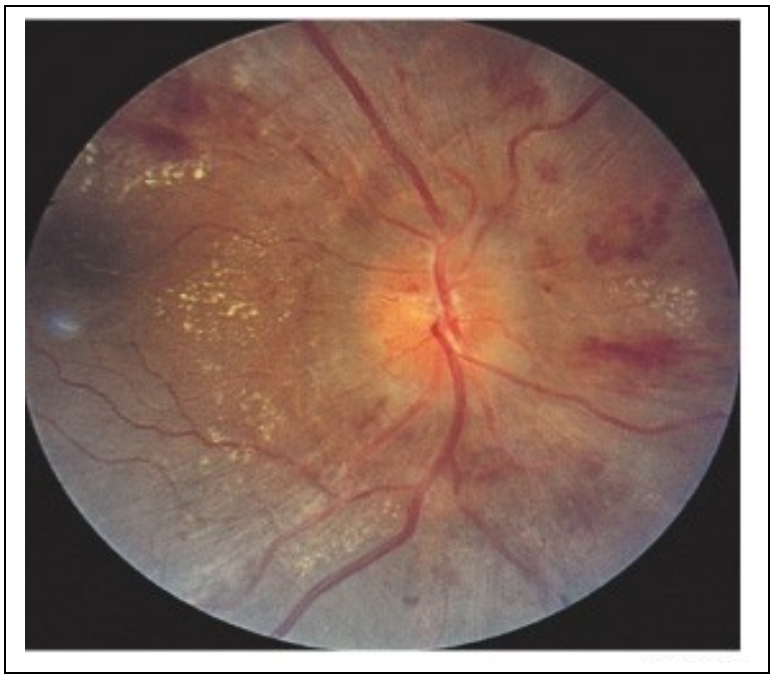
# LATERAL VIEW OF VI NERVE



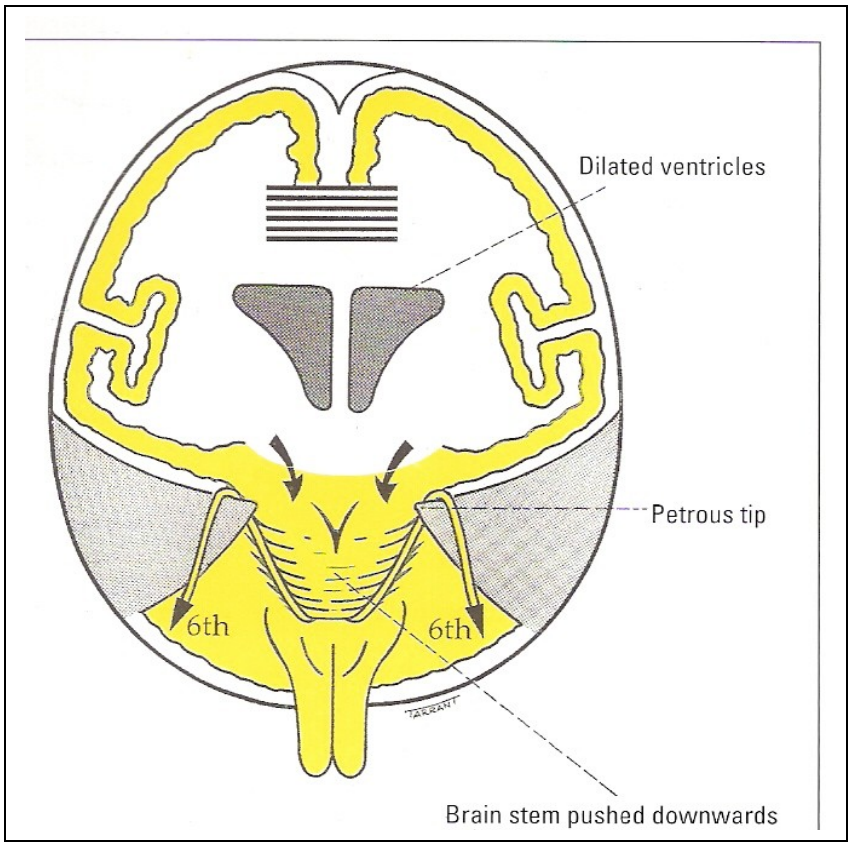
# SYMPATHETIC NERVE SUPPLY



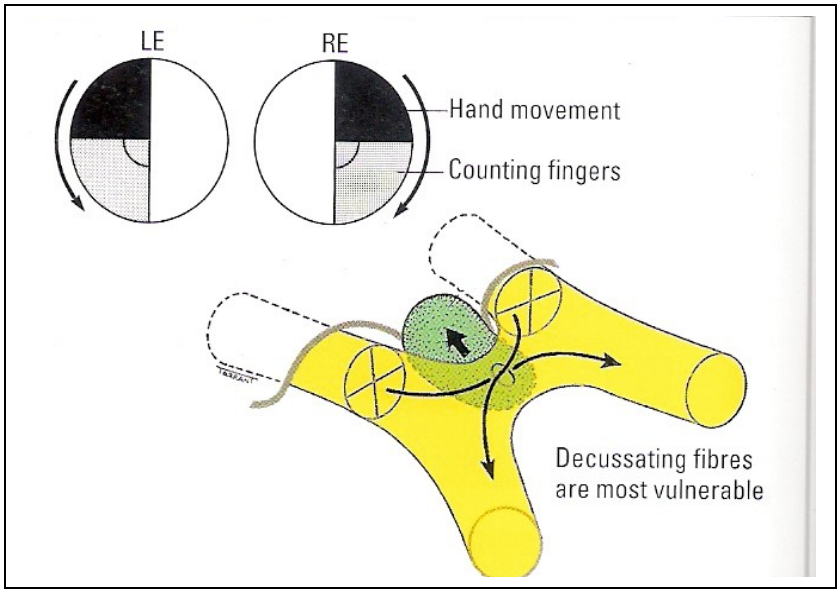
# PAPILLEDEMA



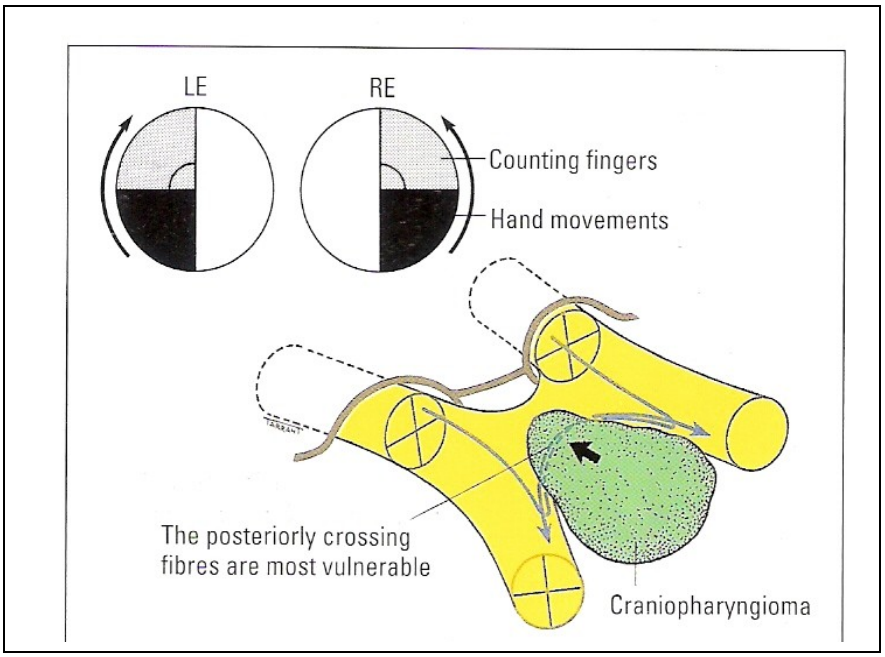
# MECHANISM OF BILATERAL VI NERVE PALSY FROM RAISED ICP



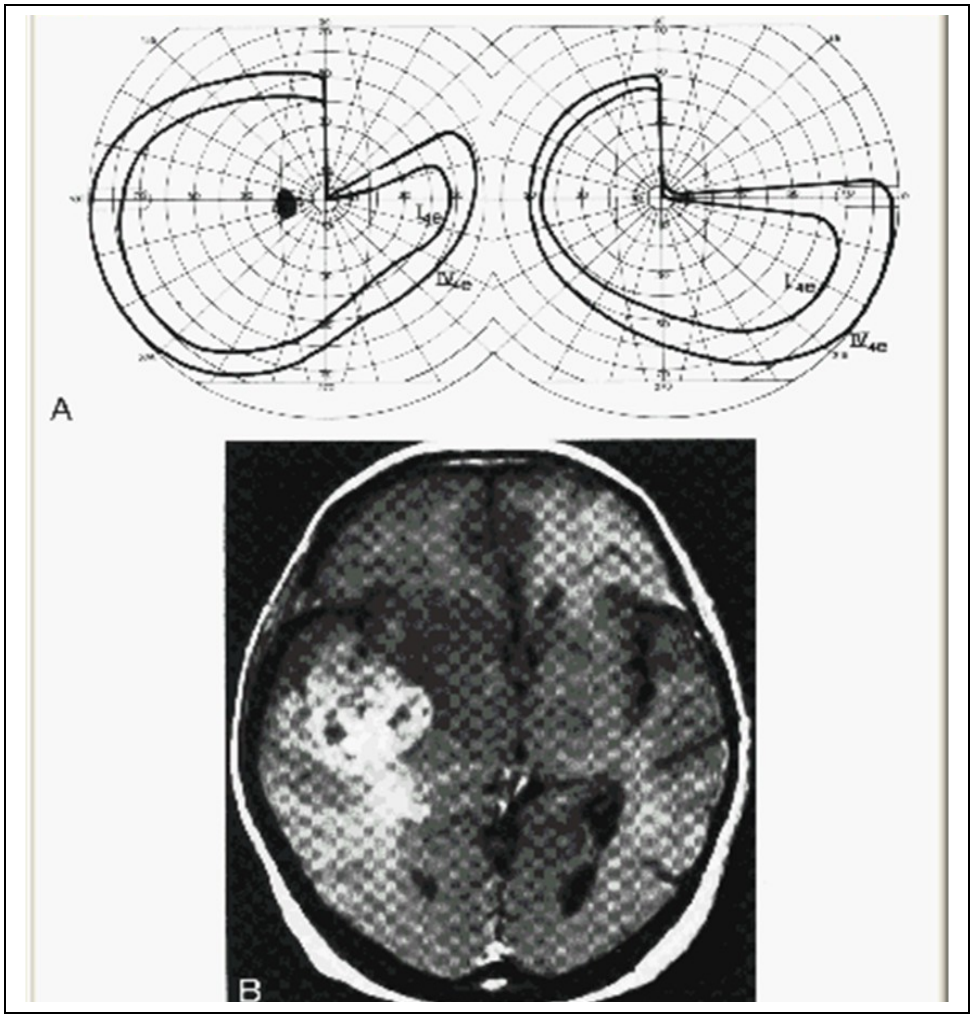
# PROGRESSION OF VISUAL FIELD DEFECT
# PITUITARY TUMOR



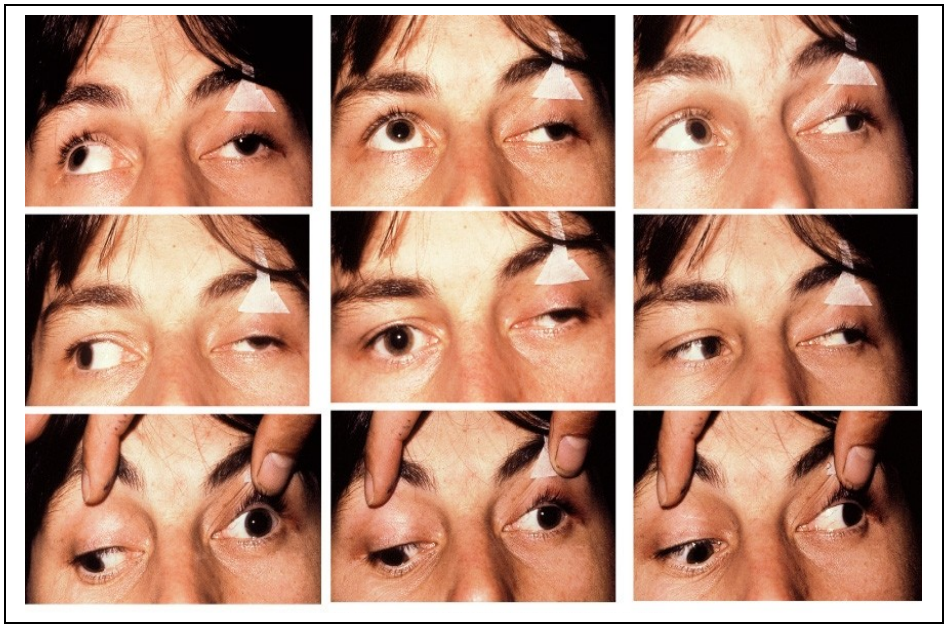

# CRANIO PHARYNGIOMA



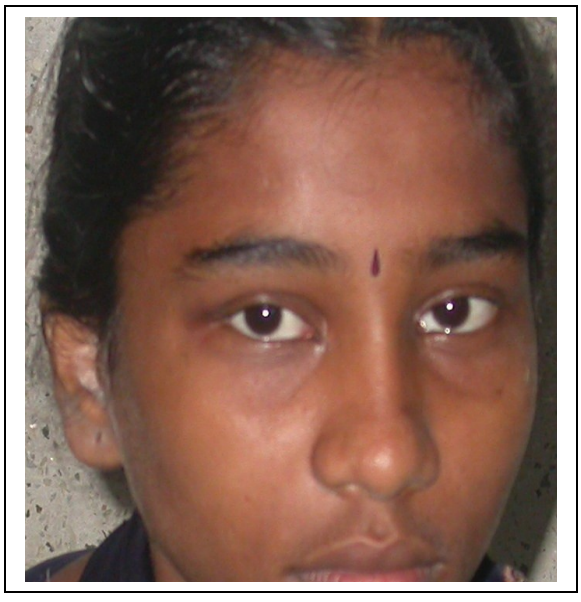
# PIE IN SKY DEFECT (TEMPORAL LOBE LESION)

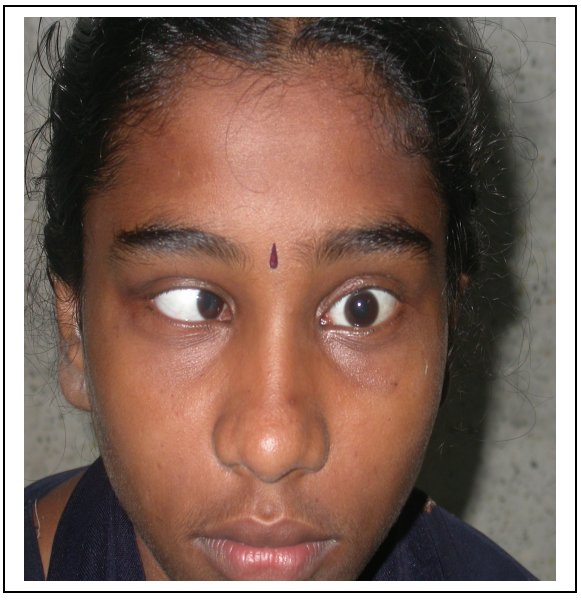


## LEFT TEMPORAL LOBE MENINGIOMA
## WITH III NERVE PALSY

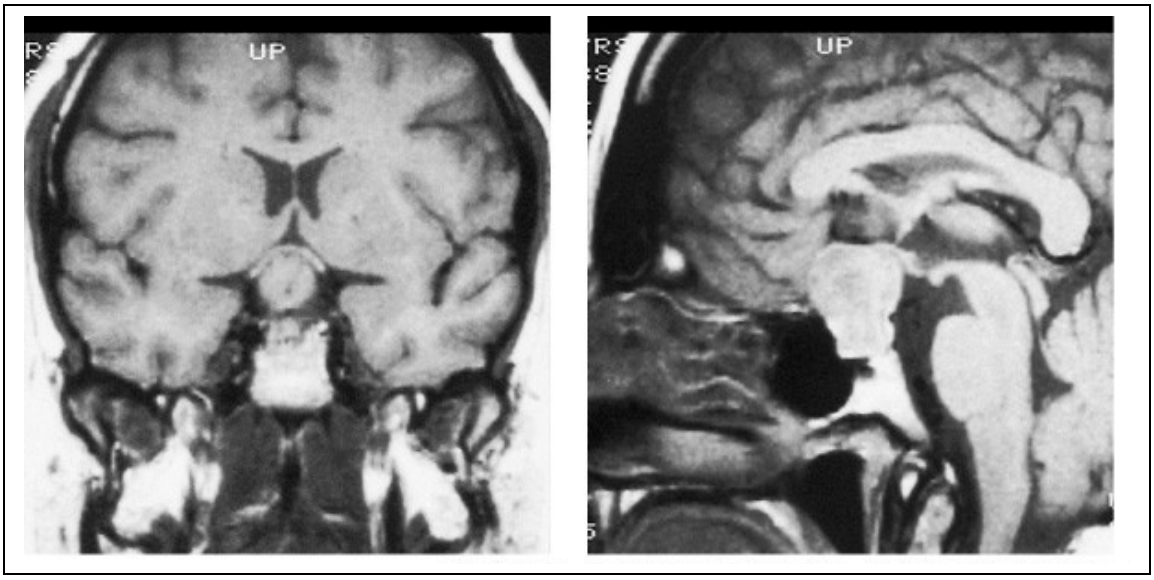


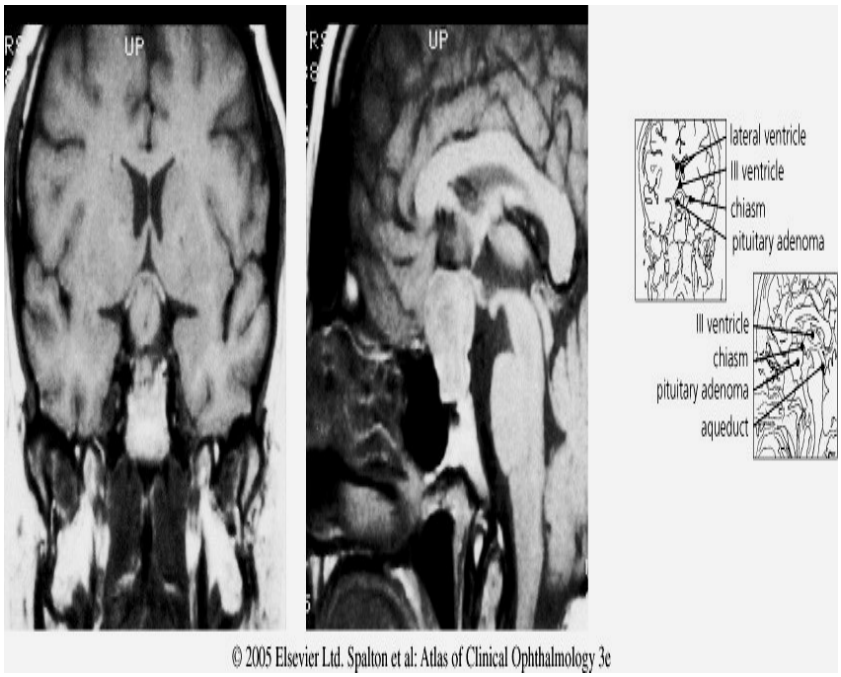
## CP ANGLE TUMOR WITH VI NERVE PALSY



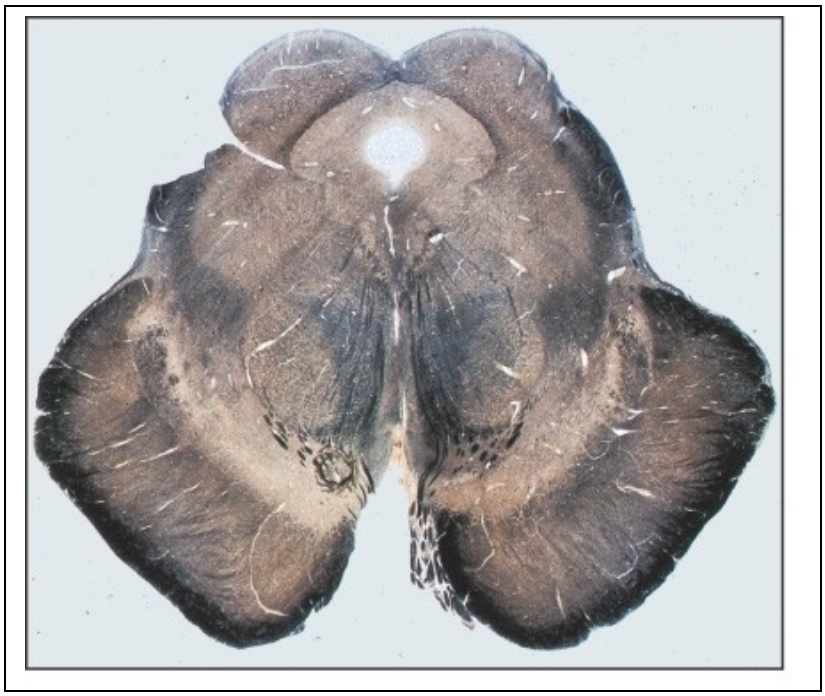


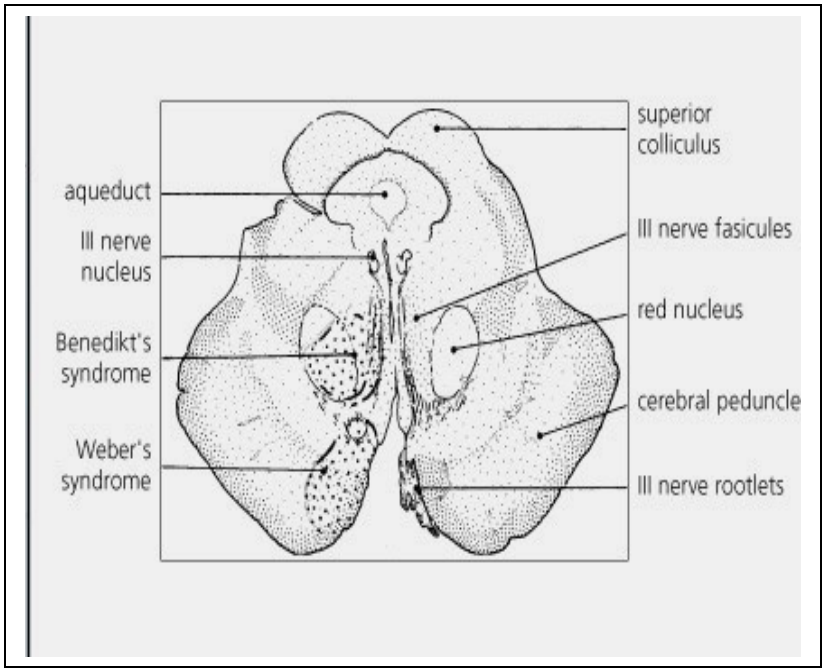
# CT PITUITARY ADENOMA



# MID BRAIN SYNDROMES





# MASTER CHART

| S.No. | Name | Age / Sex | DOV | Seizures | Diplopia | Headache | Papilledema | CN defect | OKN | FD | Site of tumor |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. | Ganesan | 45/M | + | + | - | + | + | + | + | - | F |
| 2. | Ramu | 18/M | + | + | - | + | - | - | - | - | F |
| 3. | Sivakami | 22/F | + | + | - | + | + | - | - | + | FP |
| 4. | Saradha | 35/F | - | - | - | + | + | - | + | + | P |
| 5. | Sundari | 16/F | - | - | + | + | - | + | - | - | F |
| 6. | Shanmugam | 26/M | + | + | - | - | - | - | - | + | CP |
| 7. | Sivanandi | 35/M | - | - | - | + | + | - | - | + | S |
| 8. | Suganthi | 6/F | - | - | - | + | - | + | - | - | FP |
| 9. | Subbiah | 36/M | + | + | - | - | + | + | - | - | CP |
| 10. | Saraswathy | 36/F | - | - | + | + | + | + | + | - | F |
| 11. | Sunder | 28/M | - | - | - | + | + | - | - | - | CP |
| 12. | Salaika | 24/F | - | + | - | - | - | - | - | - | F |
| 13. | Mahamed | 30/M | + | + | - | + | + | - | - | - | FP |
| 14. | Selvi | 8/F | + |  | - | + | + | - | - | - | F |
| 15. | Ramesh | 38/M | - | + | - | + | + | + | - | + | S |
| 16. | Ramanan | 50/M | - | + | - | - | - | - | - | + | T |
| 17. | Saroja | 16/F | + | + | - | + | - | - | - | + | T |
| 18. | Saravanan | 48/M | - | - | - | + | + | + | - | + | CP |
| 19. | Sivaranjan | 18/M | - | - | - | + | + | - | + | - | FP |
| 20. | Sudali | 38/F | + | + | - | + | + | - | + | + | FP |

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 21. | Mariappan | 28/M | - | - | - | + | - | + | - | - | CP |
| 22. | Ponnu | 15/F | - | - | - | - | - | - | + | + | P |
| 23. | Malayandi | 36/M | + | + | + | + | + | + | - | + | S |
| 24. | Parvathi | 52/F | - | + | - | + | + | + | - | - | CP |
| 25. | Muthulaxmi | 38/F | + | - | - | - | - | - | + | + | P |
| 26. | Muthiah | 39/M | + | + | - | + | - | - | - | - | CP |
| 27. | Vellayutham | 38/M | + | - | - | + | - | - | - | - | F |
| 28. | Kamatchi | 13/F | - | - | - | - | - | + | - | - | F |
| 29. | Vinayagam | 14/M | + | - | + | + | + | - | + | - | CP |
| 30. | Kaveri | 31/F | - | + | - | - | - | - | - | - | S |
| 31. | Vignesh | 11/M | + | + | - | - | - | + | - | + | T |
| 32. | Ramalaxmi | 50/F | + | - | - | + | + | - | + | - | CP |
| 33. | Virumandi | 36/M | - | - | - | + | - | - | - | - | T |
| 34. | Veerayan | 38/M | + | + | - | - | - | + | - | + | FP |
| 35. | Pethinammal | 38/F | - | - | + | + | + | - | - | - | S |
| 36. | Kandasamy | 50/M | - | - | - | - | - | + | + | + | P |
| 37. | Kalyani | 17/F | - | - | - | + | + | - | - | + | S |
| 38. | Palpandi | 50/M | + | + | - | + | - | + | - | - | T |
| 39. | Balaraman | 28/M | + | + | - | + | - | - | + | - | FP |
| 40. | Kaliammal | 26/F | + | - | - | + | + | + | - | - | S |
| 41. | Kannapan | 48/M | - | - | + | + | + | + | - | + | S |
| 42. | Aseem | 35/M | - | - | - | + | - | - | - | - | F |
| 43. | Arulvelan | 50/M | + | + | - | - | - | - | - | - | F |
| 44. | Subhammal | 65/F | + | + | - | - | + | - | - | - | CP |
| 45. | Jeyakumar | 49/M | - | - | - | - | - | - | - | - | F |
| 46. | Kumaravelu | 50/M | + | - | + | + | + | - | - | - | FP |
| 47. | Sivanammal | 46/F | + | + | - | + | + | + | - | + | S |

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 48. | Karthick | 50/M | + | + | - | + | + | + | - | - | C |
| 49. | Seethalaxmi | 28/F | - | - | - | - | + | - | - | + | S |
| 50. | Karuppiah | 60/M | + | - | - | - | - | + | - | - | C |
| 51. | Velammal | 55/F | - | - | - | - | - | - | - | - | CP |
| 52. | Sudalai | 51/M | - | + | + | + | + | + | + | - | F |
| 53. | Visalaxi | 45/F | - | + | + | + | - | + | + | - | FP |
| 54. | Selvam | 46/M | - | + | - | - | - | - | - | + | F |
| 55. | Veeralaxmi | 60/F | + | - | - | + | + | - | - | - | T |
| 56. | Rahmathulla | 50/M | + | + | - | + | + | + | + | + | P |
| 57. | Chelliah | 48/M | + | - | - | - | - | + | - | - | F |
| 58. | Kalairani | 18/F | - | - | - | + | + | - | - | + | T |
| 59. | Kanniah | 45/M | + | + | - | + | + | - | - | - | F |
| 60. | Viswanathan | 50/M | + | + | + | + | + | + | + | - | CP |