A CLINICAL STUDY ON OCULAR MANIFESTATIONS OF INTRA CRANIAL TUMOURS

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CERTIFICATE

This is to certify that the dissertation entitled "**A CLINICAL STUDY ON OCULAR MANIFESTATIONS OF INTRACRANIAL TUMOURS"** is the bonafide original work of **Dr. GANESHKUMAR JAYAKRISHNAN** in partial fulfillment of the requirements for **M.S. (Ophthalmology) BRANCH – III** Examination of The Tamilnadu Dr. M.G.R. Medical University to be held in March 2007.

Dr. D.R. GUNASEKARAN, M.S.
DEAN
Govt. Stanley Medical College & Hospital,
Chennai-600 001.

Prof. Dr. A. PRIYA, M.S., D.O.
Professor & Head
Department of Ophthalmology,
Govt. Stanley Medical College
Chennai-600 001.

DECLARATION

I, **Dr. GANESHKUMAR JAYAKRISHNAN,** solemnly declare that this dissertation titled, entitled "**A CLINICAL STUDY ON OCULAR MANIFESTATIONS OF INTRACRANIAL TUMOURS"** is a bonafide work done by me at Govt. Stanley Medical College & Hospital during 2004-2007 under the guidance and supervision of **Prof. Dr. A. Priya, M.S., D.O.,** Professor and Head, Department of Ophthalmology, Stanley Medical College, Chennai-600 001.

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Date :

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CONTENTS

OPHTHALMIC MANIFESTATIONS OF INTRA CRANIAL TUMOURS

CASE PROFORMA

NAME :	**S. NO:**
AGE :	**IP NO:**
SEX :	**UNIT:**
ADDRESS :	**DOA:**
OCCUPATION:	**DOD:**
PRESENTING COMPLAINTS:	**DURATION:**

HISTORY OF PRESENTING COMPLAINTS

Complaints:

Headache

Vomiting

Motor Symptoms	Weakness
	Spasticity
	Seizures-Focal / generalized
	Aura / Hallucinations
Sensory disturbances	Tingling / Numbness/Paresthesias
Mental Changes	Withdrawn behaviour/ Apathy / Irritability/
	Depression / Memory Loss. / Poor Abstract
	Thinking/Lack of Judgment / Disorientation

Gait Disorder.

Nasal Symptoms — Obstruction/Discharge/Bleeding

Failure to thrive.

Endocrine Abnormalities

- Enlargement of hands & feet
- Prominence of jaw
- Decreased libido / Impotence
- Polydypsia / Polyuria
- Delayed body growth
- Intolerance to cold / heat
- Increased skin pigmentation

Increased pulse rate
Alteration of speech (Thickening of tongue)
Amenorrhea
Galactorrhoea
Increased sweating
Weight gain (Moon face) / Weight loss
Hot Moist palms

Cranial nerve abnormalities-

Anosmia
Dimness of vision
Diplopia
Abnormal EOM
Loss of sensation of one ½ 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: RE LE

Visual Disturbances :
Dimness of vision
Blunting of colour perception
Transient loss of Vision
Field Loss
Diplopia

Protrusion of eyeball :

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 & Rx taken

Any operations undergone

FAMILY HISTORY :

MENSTRUAL HISTORY :

PERSONAL HISTORY :

General Physical Examination:

Consciousness Pulse BP

Built / Nourishment

Pallor / Icterus / Cyanosis /Clubbing / Oedema / Lymphadenopathy

CNS Examination :

HMF : Consciousness (EMV)

Orientation Recent / Short term / Remote

Memory

Co-operation

Behaviour

Judgment

Speech Normal / Slurred / Dysarthria / Dysphasia

Handedness (Cerebral Dominance)

Mood

Cranial Nerves

1. Olfactory
2. Optic-
3. Oculomotor
4. Trochlear
5. Trigeminal
6. Abducens
7. Facial
8. Acoustic
9. Glossopharyngeal
10. Vagus
11. Accessory
12. Hypoglossal

Motor - Dealt in detail in next section
Sensory - Touch / Pain / Corneal Reflex
Motor - Upper part of face /Lower part of face
Sensory - Taste in Ant. 2/3 of tongue
Weber's test
Rinnie's test
Gag reflex / Palatal movements Voice / Swallowing
Shrugging of shoulders
Deviation of tongue
Wasting / Fibrillation of tongue

Motor : UL LL

Tone
Power
DTR
Plantar reflex
Co-ordination (cerebellar)
Involuntary movements

Sensory :

Touch
Temperature
Pain
Vibration

Skull & Spine :

Cerebellar Signs :

Meningeal Signs :

CVS Examination :

Heart Rate
Heart Sounds
Rhythm
Carotids – Pulsation / Bruit

RS Examination :

RR
Nature
Trachea
Breath Sounds

Abdominal Examination:

Hepatomegaly / Splenomegaly / Mass
Bowel Sounds

Summary :

Provisional Diagnosis

Investigations :

Preliminary

Blood Group

Hb	Blood Urea	ECG
TC	Serum Creatinine	Echo
DC	Serum Na^+	

Urine - Alb.
- Sug.

Special

X-ray Skull – AP / Lat
X-ray Orbit – AP / Lat
X-ray Chest – AP / Lat
X-ray Optic foramen (Rhese View)
CT Scan Brain – Plain / Contrast
MRI
CSF Analysis
Hormonal Assay – T3 / T4 / TSH
- Serum Estradiol
- Angiography (Cerebral)
- FFA
- EEG

MANAGEMENT

Medical

Surgical - Total Excision

- Subtotal excision and biopsy

Radiotherapy

Histopathology Report :

Post – Op / Post – Rx Visual Parameters: Improved / Status Quo / Deteriorated

Follow-up:

ABBREVIATIONS

A	-	Acting
Abn	-	Abnormal
B/L	-	Bilateral
B&B	-	Bowel & Bladder
BSE	-	Blind Spot Enlargement
Bitemp.Hp	-	Bitemporal Hemianopia
Ca	-	Carcinoma
CFCF	-	Counting Fingers Close to Face
ChT	-	Chemotherapy
CL	-	Contact Lens
CN	-	Cranial Nerves
CPA	-	Cerebello-Pontine Angle
EOM	-	Extra Ocular movements
F-T-P	-	Fronto-Tempero-Parietal
H	-	Headache
HM	-	Hand Movements
Hom	-	Homonymous
Hp	-	Hemianopia
HPE	-	HistoPathological examination
Inf.	-	Inferior
L	-	Left
LMN	-	Lower Motor Neuron
LND	-	Light Near Dissociation
LOC	-	Loss of Consciousness
ME	-	Macular Edema
N	-	Normal
NA	-	Not Acting

Nas.	-	Nasal
NP	-	Not Possible
Nys	-	Nystagmus
P	-	Papilledema
PL	-	Perception of Light
POA	-	Primary Optic Atrophy
Qp	-	Quadrantanopia
R	-	Right
RAPD	-	Relative Afferent Pupillary Defect
RT	-	Radiotherapy
SA	-	Sluggishly Acting
SOL	-	Space Occupying Lesion
Sup.	-	Superior
T-P	-	Tempero-Parietal
Temp	-	Temporal
UMN	-	Upper Motor Neuron
V/A	-	Visual Acuity
V-P	-	Ventriculo-Peritoneal
Vn	-	Vision
+	-	Present
-	-	Absent
↑	–	Increased
↓	–	Decreased

INTRODUCTION

Neuro-ophthalmology, an offshoot of the neurosciences, is a sub-speciality which has not received its due share of recognition, probably because it does not entirely belong to one faculty, comprising of a team of Ophthalmologists, Neurologists, Neurosurgeons, Radiologists and even includes Primary care Physicians.

This field is rapidly expanding with vastly improved and refined diagnostic and therapeutic modalities for various afflictions of the visual apparatus of neurologic origin.

Any form of congenital anomalies, trauma, inflammations, tumors and vascular disorders may involve the visual pathway manifesting as **Defective Visual Acuity, Ocular Motility Disorders, Pupillary Abnormalities, Visual Field Defects** and **Fundus changes**.

This is based on the unique structural anatomy wherein the entire visual system is confined within the brain but the globes are retained in the exterior which exhibit features due to lesions higher up in the cranium.

Intracranial tumours are relatively uncommon in adults when compared to other neoplasms accounting for about 2 % of all malignancies but are more common in childhood comprising around 20 % of neoplasms second only to leukaemias.

However, the incidence of intracranial tumours is on the rise due to multifactorial causes, the main causes being improved neurodiagnostic methods and increased life expectancy.

Ocular symptoms and signs appear with significant frequency in patients with brain tumours – upto 50% of patients with ocular symptoms and signs have neurological lesions above and about **60% present with ocular symptoms and signs as initial complaints**.

Hence it is essential for the Ophthalmologist to recognize a neurosurgical condition early when there is optimal chance for maximal treatment.

This is possible by **early referral** and **inter-disciplinary participation** which would save not only vision but also life, in certain instances. Early detection with adequate intervention would reduce the morbidity but not the mortality.

The knowledge of this sub-speciality, Neuro-Ophthalmology serves as the key to **early diagnosis** and **prompt management** of these patients.

Hence this study was done to analyse the Ocular manifestations of intra cranial tumours.

ANATOMY

VISUAL PATHWAY

Relations of visual pathway are of importance to localize the lesions with visual field defects because of the close anatomical relationship between visual apparatus and the brain allowing accurate localization of tumor lesions in around 25% of patients.

RETINA

This is the inner coat of the eyeball extending from optic disc to Ora serrata. Visual receptors (Rods and Cones) synapse with bipolar cells which relay to ganglion cells.

OPTIC NERVE

Axons of ganglion cells form optic nerve. It can be divided into four parts:

1. Intra-ocular part (1 mm) - includes optic disc and the portion that lies within the sclera.
2. Intra-orbital part (25 mm) - Optic nerve pierces the lamina cribrosa and acquires myelin sheath.
3. Intra-canalicular part (5 mm) - lies within the lesser wing of sphenoid bone.
4. Intra-cranial portion (10 mm) - passes backward, upward and medially, within the subarachnoid space, to reach optic chiasma.

Related above to olfactory tract, gyrus rectus and anterior cerebral artery; laterally to internal carotid artery.

OPTIC CHIASMA

Situated at the junction of anterior wall and floor of III Ventricle.

Contains decussating nerve fibres from nasal half of each retina(including nasal half of macula).

Inferiorly related to diaphragma sellae and pituitary gland. Blood supply is by branches from the Circle of Willis.

OPTIC TRACT

Contains ipsilateral temporal nerve fibres and contralateral nasal fibres. Passes posterolaterally, winding round lateral margin of cerebral peduncle and terminates in Lateral Geniculate Body(LGB).

LATERAL GENICULATE BODY

Receives caudal termination of lateral root of optic tract.

Layers 1, 4 & 6 receive crossed fibres, whereas uncrossed fibres terminate in layers 2, 3 & 5.

OPTIC RADIATION (Geniculocalcarine tracts)

Fibres from LGB fan out laterally and inferiorly, around inferior horn of lateral ventricle forming Loop of Meyer, then swing posteriorly to end in occipital cortex.

VISUAL CORTICAL AREAS

Primary (Area 17)

Situated in wall of calcarine sulcus on medial surface of cerebral hemisphere.

Secondary (Areas 18 & 19)

Surround primary visual area on medial and lateral surfaces of cerebral hemisphere.

CRANIAL NERVES

Oculomotor nerve / III CN

Nucleus:

Situated in the midbrain at the level of superior colliculus.

Course:

Passes forward between posterior cerebral and superior cerebellar arteries and laterally parallel to posterior communicating artery. On lateral side of posterior clinoid process, it 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 the tendinous ring.

Supply:

Superior rectus, Inferior rectus, Medial rectus, Inferior oblique and Levator Palpebrae superioris.

Trochlear Nerve / IV CN

Nucleus:

Situated in the midbrain at the level of inferior colliculus.

Course:

Emerges on posterior surface of brain stem, decussates and passes forward around cerebral peduncle. Enters lateral wall of cavernous sinus and

lies below III CN and above first division of V CN.

Passes through upper part of Superior Orbital fissure.

Supply: Superior oblique.

Trigeminal Nerve / V CN

Nucleus:

Extends from Pons, throughout medulla oblongata till second cervical segment in spinal cord.

Supply:

Ophthalmic division is entirely sensory and innervates the ocular structures through three branches - Lacrimal, frontal & nasociliary.

Abducens Nerve / VI CN

Nucleus:

Lies in upper part of fourth ventricle.

Course:

Emerges between pons & medulla oblongata, runs upward, forward and laterally, makes an acute bend across sharp upper border of petrous part of temporal bone & runs within cavernous sinus inferolateral to internal carotid artery. Enters orbit through superior orbital fissure between the two divisions of III CN.

Supply:

Lateral Rectus

Facial nerve / VII CN

Nucleus:

Lies within Pons.

Course:

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

Supply:

Motor supply to muscles of facial expression and orbicularis oculi. Sensory innervation carries taste sensation from anterior 2/3 of tongue.

PHYSIOLOGY

PUPIL

The iris contains two groups of smooth muscles with reciprocal action, sphincter and dilator pupillae and the pupillary size depends on the balance between the sympathetic and parasympathetic tone.

LIGHT REFLEX

Consists of four neurones accounting for both Direct & Consensual responses.

Light → Retinal Photoreceptors → Optic nerve → Optic chiasma → Optic tract → Pretectal nucleus → both Edinger Westphal nuclei →Ciliary ganglions→ Short ciliary nerves to innervate Sphincter pupillae.

NEAR REFLEX - Consists of (a) increasing accommodation

(b) convergence of visual axes

(c) pupillary constriction

Midbrain centre for near reflex is probably located in a more ventral location than light 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.

SYMPATHETIC SUPPLY

Posterior hypothalamus → Ciliospinal centre of Budge (between C8 & T2) → Superior cervical ganglion → Ascend along ICA → Ophthalmic division of V CN → Nasociliary branch → Dilator Pupillae.

ABNORMAL PUPILLARY REACTIONS

Total Afferent Pupillary Defect (Amaurotic Pupil)

- Caused by complete optic nerve lesion.
- Characterized by:

1. Involved eye is completely blind (No PL)
2. Both pupils are of equal size
3. Direct reflex is absent in involved eye, but consensual reflex is present
4. Near reflex is normal in both eyes

Relative Afferent Pupillary Defect (Marcus - Gunn Pupil)

- Caused by an incomplete optic nerve lesion.
- Elicited by "Swinging flash light" test.
- Consensual reflex is more pronounced than direct reflex in involved-eye.

Horner's Syndrome (Oculosympathetic Palsy)

- Features are mild ptosis, miosis and anhidrosis on affected side.
- Papillary reactions are normal to light and near, but involved pupil characteristically shows a dilatation lag.

OCULAR MOVEMENTS

The ocular system is basically aimed to

1. Fixate the vision at the object of interest.
2. Stabilise the retinal image on the fovea either when the target or the head is moving.
3. Change fixation to new areas of interest.
4. Maintain binocular vision.

These are done by three hierarchical circuits

1. Supranuclear mechanism of sensory guidance.
2. Internuclear mechanism of motor organization.
3. Nuclear and infranuclear mechanism.

Supranuclear mechanism

Contains various sub systems operating at supra nuclear level independent of each other and require visual guidance.

1. Pursuit
2. Saccades
3. Vergence
4. Fixation
5. Optokinetics &
6. Vestibulo-ocular systems.

Internuclear mechanism

Integrates the activity of all the motor nuclei of the brainstem involved in eye movements and are interconnected by medial longitudinal fasciculus (MLF) and are responsible for

1. Horizontal Gaze
2. Vertical Gaze
3. Vergence movements
4. Saccadic premotor signals

Nuclear and Infranuclear mechanism

They constitute the III, IV and the VI cranial nerve nuclei and the ensuing cranial nerves which innervate the extra ocular muscles. The connections are ipsilateral for all muscles excepting the superior oblique and the superior rectus which are innervated by the contra lateral IIIrd and IVth nuclei respectively.

Disorders of the supranuclear system

1. Saccadic pursuit
2. Impaired pursuit
3. Saccadic paralysis
4. Slow saccades
5. Hypometric saccades

Disorders of the internuclear system

1. Horizontal Gaze

VI nucleus - lesion causes ipsilateral conjugate palsy of saccades and not isolated VI or lateral rectus palsy.

MLF - lesion causes impaired ipsilateral adduction and contralateral abducting jerk nystagmus.

Total Inter Nuclear Ophthalmoplegia (INO) - adduction paralysed to saccadic, pursuit and vestibular stimulation. Vertical pursuit and VOR movements are slowed but the vertical saccades are normal.

One and a half Syndrome - combination of unilateral gaze palsy and INO with paralysis of horizontal movements ipsilaterally and paralysis of adduction in the other eye.

2. Vertical Gaze

Pretectal / dorsal midbrain syndrome – paralysis of upward saccades with or without involvement of upward pursuit and VOR is associated with binocular adduction and retraction and light near dissociation of pupils.

Bells phenomenon – upward deviation of eye during forced eye closure. Preservation of this indicates supranuclear cause for ophthalmoplegia.

Ventral pretectal lesion – selective down gaze palsy due to lesion in midbrain tegmentum rostral to IIIrd nerve nucleus

PATHOLOGY

Various classifications have been in use though for didactic purposes such as histological differences and surgical accessibility.

WHO CLASSIFICATION OF TUMOURS

a. Tumours of neuroectodermal tissue

b. Tumours of meninges and related tissues

c. Tumours of maldevelopmental origin

d. Tumours of pituitary gland

e. Tumours and hemartomas of blood vessels

f. Tumours of cranial and peripheral nerves

g. Tumours of hematopoietic cells and tissue

h. Secondary tumours affecting CNS.

Classification by surgical approach is based on the tentorium cerebelli which divides the cranial cavity into two spaces.

Supratentorial tumours	**Infratentorial tumours**
- Cerebral hemispheres	- Cerebello-Pontine Angle
Frontal Lobe	- Cerebellar hemispheres
Parietal Lobe	- Cerebellar Vermis
Temporal Lobe	- Pons
Occipital Lobe	- Medulla oblongata

- Pituitary gland and adnexa
- Anterior and Middle Fossa
- Diencephalon
- III Ventricle
- Thalamus
- Basal ganglia
- Mid Brain

Tumours of Mesencephalon can present in both forms

All primary brain tumours are more common in males, except meningiomas. In adults, about 1/3rd of brain tumours are seen below tentorium, whereas in children, 2/3rd are found in this location.

Average annual age adjusted incidence rate of

-Primary brain tumours is **10 /100,000 / year**.

-Brain metastasis is **8.3 / 100,000 / year**.

The frequency distribution among brain tumours is:

Adults	**Children**
Gliomas (40 - 67%)	Medulloblastomas (20 - 25%)
Meningiomas (9 - 27%)	Cerebellar Astrocytomas (12 -18%)
Pituitary Adenomas (15%)	Brain stem Gliomas (6 - 15%)
Supratentorial Astrocytomas (14 - 32%)	

CLINICAL FEATURES

TOPICAL DIAGNOSIS OF TUMOURS

Clinical features of Intra cranial tumours can be classified as

1. Non-localising signs and symptoms
2. False localising signs and symptoms
3. Truly localising signs and symptoms

NON-LOCALISING SIGNS AND SYMPTOMS

These are independent of site of tumours and primarily indicate an increase in intracranial pressure.

HEADACHE

- More than 50% (48% - 75%) of patients with intracranial tumours complain of headache. (Rushton&Becke, 1962; Mahaley et al, 1989; Snydner et al, 1993, Sunenwala et al, 1994)
- Classic brain tumours headache (extreme severity, worse in morning, associated with nausea and vomiting) is quite uncommon.
- Usually, headache presents as tension-type headache (77%) or migraine (9%) - mild to moderate severity, bifrontal, worse on side of lesion, worse with bending down, associated with nausea and vomiting.
- Headache is more common in tumours below tentorium. (The Childhood Brain Tumour Consortium, 1991; Sunenwala et al, 1994)
- Almost all patients with posterior fossa tumours have headache as first symptom.

- Causes of headache include Increased Intra Cranial Pressure (ICP), Increasing tumour size, Midline shift, Acute haemorrhage into tumour, History of prior headache, Venous sinus thrombosis, Acute obstructive hydrocephalus, meningeal invasion, contiguous infection and toxic effect of therapeutics.
- Use of neuroimaging in all patients with headache found very low prevalence of tumors among patients in general (1%) and those with migraine (0.3%).(Frishberg,1994)
- Nearly all children with brain tumors and headache have associated neurologic symptoms and signs. (Rossi & Varell, 1989; The Childhood Brain Tumour Consortium, 1991)

PAPILLEDEMA

- Most important and classical sign of elevated intracranial pressure. Paton(1935) & Uthoff (1915) found a prevalence in around 80% of patients while later studies by Petrohelos & Henderson(1950),Tannis(1959) and Huber(1976) showed this figure to be around 60%
- Modern imaging techniques has made earlier diagnosis possible and thus reducing prevalence at the time of presentation.
- 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 tumours (from cerebellum and fourth ventricle) tend to increase intracranial pressure by obstructing flow of CSF through aqueduct of sylvius and are more likely to produce papilledema(75.2%) than supratentorial tumours(53.4%)Petrohelos & Henderson(1950)
- Supratentorial and third ventricular tumours cause papilledema frequently due to the mass effect of the lesion or surrounding edema.
- Papilledema is rarely seen in pituitary gland tumours.
- There is greater tendency for papilledema to develop with slowly growing tumours than rapidly expanding lesions.
- Papilledema occurs not only based on tumor location but also on the type of tumour and rate of growth .e.g. Gliomas (76%), Meningiomas (40%), Glioblastomas (50%).(Hartmann & Guillarnat, 1938; Huber, 1976)

Mechanism of Increased Intracranial Pressure with Intracranial Tumours

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

Symptoms

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

Signs

1. Increase in disc diameter.

2. Blurring and indistinct margins in the nasal, temporal, upper and lower poles.
3. Elevation of the disc and mushrooming of the nerve head into the vitreous (doughnut or champagne cork shape).
4. Reddish discolouration of the disc (capillary stasis) with visible network of dilated capillaries all over the nerve head (hyperemic disc).
5. Venous congestion with tortousity of the veins with relatively normal arteries (increased ratio of calibre of veins and arteries).
6. Loss of spontaneous venous pulsation.
7. Deflection of the vessels at the disc margins.
8. Peripapillary flame shaped hemorrhages and also seen within the disc.
9. White exudates over the surface and at the margin of the disc.
10. Fundus fluorescein angiography initially shows dilated disc capillaries followed by increased hyperfllluorescence which extends beyond disc margins.

11. Fields show enlargement of the blind spot.

12. No primary disturbances of the sensory fundus.

SEIZURES

- Occurrence of a seizure in a previously healthy individual should be regarded to be due to intracranial tumours, until proved otherwise, as about 1/3rd adults who develop new onset of seizures harbour an intracranial tumours.
- There is no precise correlation between seizure pattern and tumour location (Adams & Victor,1993) - but usually a seizure associated with forced turning of head and eyes (versive movement) indicates a tumour contralateral to thedirection of head and eye movement.(Wyllie et al ,1980)
- Seizures occur more commonly in supratentorial and cortical lesions especially astrocytomas and oligodendrogliomas.(Giles et al, 1992; Fried et al, 1995) .Others noted high frequency in limbic regions especially temporal lobe.
- They may be the presenting sign of tumours in upto 50% of the cases (Giles et al, 1992; Snynder et al, 1993)

ABDUCENS NERVE PARESIS

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

- Unilateral or bilateral nerve paresis occurs in the presence of increased intra cranial pressure or mass lesion usually representing as a non localising sign. (Lundberg, 1960; Varsallus, 1967, Kleave, 1976)
- **Mechanism:**
    (1) VI nerve gets compressed between pons and basilar artery.
    (2) The nerve gets stretched along the sharp edge of petrous bone.

FALSE-LOCALISING 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 not a clinical sign that is misinterpreted)**.
- Usage of CT and MRI scanning in patients has made out false localising signs to be of less practical importance.
- They are seen in upto 12.5% cases of raised intra cranial tumours. (Collier, 1904)
- **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
- Others include Dementia, Visual field defects, Visual hallucinations,

Cranial neuropathies, Cerebellar signs, Pyramidal and Extra pyramidal signs, Nystagmus, Restriction of extra-ocular movements and Proptosis. All these can manifest as a false localising sign at one time or other.

- Most signs occur late in the the course of intra cranial tumors due to raised intra cranial pressure, brain movement and hydrocephalus.

TRULY LOCALISING SIGNS AND SYMPTOMS

TUMOURS INVOLVING ORBIT

These tumours 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 hemangiomas of orbit or optic nerve sheath meningiomas due to compromised blood supply to optic nerve and retina in that gaze.

Proptosis - Amount of protrusion of eyeball depends on

1) Speed of growth of tumour - Rapidly growing tumours produce greater mass effect.
2) Volume of tumour - Optic nerve sheath meningiomas, cause minimal proptosis and yet profound visual loss.
3) Consistency of tumour - Vascular tumours (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 disc swelling or optic atrophy form a triad which indicates chronic compression of intraorbital optic nerve, e.g. Spheno-orbital meningioma.

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

TUMOURS INVOLVING SUPRASELLAR REGION

- Intracranial part of optic nerve, optic chiasm, optic tract and hypothalamus are the regions commonly involved causing specific syndromes. (Adler et al, 1948; Walsh, 1956; Trobe et al, 1984; Sharpe 1985)
- The type of tumours 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 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 Chiasm

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 tumours cause compression from below, so the field defect starts as a superior temporal quadrantanopia and then progresses inferiorly

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 tumours.

Sudden decline in vision with extra ocular muscle palsy and altered consciousness indicates a **Pituitary Apoplexy** resulting from hemorrhage or infarction into a pituitary adenoma.

Syndrome of Optic tract

Complete homonymous hemianopia is the typical feature.

Afferent pupillary defect can occur without loss of visual acuity or colour vision in eye ipsilateral to hemianopia (i.e. contralateral to side of lesion).

Hypothalamic Syndrome

Disturbance in hormones secreted by hypothalamus can give rise to various problems like diabetes insipidus, disturbances of temperature

regulation and appetite, adiposogenital dystrophy (Frolich's syndrome), Gastric hemorrhages (Cushing's Ulcers) etc.

Syndrome of Pituitary Dysfunction

Features of hyper / hypo pituitarism can be seen depending on the nature of the lesion.

PARASELLAR TUMOURS

The manifestations can be divided into those arising from

1. Outer 1/3rd of sphenoid ridge.
2. Middle 1/3rd of sphenoid ridge.
3. Inner 1/3rd 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 and VI cranial nerves and occasional ipsilateral cornea hyposthesia.

TUMOURS OF FRONTAL LOBE

These account for 24% of all tumours and 51% of supratentorial tumors as they occupy 30% volume of cerebral hemispheres.

Gliomas (Astrocytoma, oligodendroglioma, glioblastoma multiforme) are the most common tumours involving frontal lobe, followed by Meningiomas.

A change in psyche seen as withdrawal behaviour, apathy, irritability, senseless joking etc may be seen leading on to the full picture of dementia.

Motor signs include forced grasping, groping and spasticity in the contralateral limbs. Aphasia develops if speech area is involved.

Slowly progressive monocular visual loss with central scotoma occurs due to pressure on optic nerve which may be reversible.

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

Ocular motor abnormalities may be seen along with inability to voluntarily close eyelids. Proptosis may also occur rarely.

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

Foster Kennedy Syndrome:

Ipsilateral optic atrophy with contralateral papilledema is a sign of great localising value, but it is not pathognomic of frontal lobe tumours and is also seen in meningiomas of the olfactory groove and sphenoid ridge.

OLFACTORY GROOVE TUMOURS

Meningiomas and neuroblastomas are the more commom tumours seen here and they exhibit features similar to the frontal lobe tumours.

They may cause papilledema and optic atrophy is seen in 30-70% of cases (Sclero et al, 1983; Baker, 1984)

TEMPORAL LOBE TUMOURS

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

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

Olfactory or gustatory hallucination, deja vu (feeling of familiarity) and Jamais vu (feeling of strangeness) are a frequent occurrence.

Visual hallucinations of formed images, micropsia, macropsia and teleopsia may be experienced.

Homonymous hemianopia or Superior quadrantantic defects which are largely incongruous with or without macular sparing are the typical presentation.

Papilledema and optic atrophy are seen commonly while cranial neuropathies and proptosis are rare

.**Cogan's Rule**:

Asymmetric opto-kinetic nystagmus response in a patient with temporal lesion indicates additional parietal lobe involvement – symmetric nystagmus indicates that an occipital lobe lesion is the cause.

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.

TUMOURS OF PARIETAL LOBE

Relatively uncommon, mostly meningiomas (30%), glioblastomas (20%), astrocytomas (10%) and then metastasis are seen.

Ocular manifestations include visual inattention, visual hallucinations, contralateral homonymous hemianopic defects, ocular motor abnormalities, corneal hyposthesia and rarely optic atrophy.

TUMOURS OF THE OCCIPITAL LOBE

Seen more commonly in adults than children, forming 3-5% of brain tumours, mainly as glioblastomas and meningiomas.

Clinical features include visual field defects as contralateral homonymous hemianopias, visual hallucinations, visual agnosia, saccadic disturbances, conjugate nerve palsies, symmetric optokinetic nystagmus and dilated pupils as a non localising sign.

TUMOURS OF THE PINEAL GLAND

Form only 0.4 – 1% of intra cranial neoplasms but more common in the Japanese race (4 -10%)

Mainly germ cell tumours, parenchymal tumours and to a lesser extent glial tumours and metastasis.

Papilledema, secondary optic atrophy, VI CN paresis and upward conjugate movements paresis with supranuclear gaze limitation can occur.

Parinaud's / Dorsal midbrain / Pretectal / Slyvian acqueduct Syndrome:

Associated with vertical gaze paresis ,convergence retraction nystagmus, Collier's bilateral lid retraction, convergence and accommodative spasm, light near dissociation and skew deviation.

TUMOURS OF THE CEREBELLOPONTINE ANGLE

The greatest numbers of tumours in this location are Acoustic neuromas. Characteristic features are paresis of V, VI, VII and VIIIth cranial nerves.

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

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

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

TUMOURS OF THE MEDULLA

Rare entities which cause ocular motor abnormalities, impaired voluntary saccadic and pursuit movements with skew deviation and horizontal, vertical, torsional or mixed nystagmus.

TUMOURS OF CEREBELLUM

Occur in all ages but more common in children, causing 65% of

childhood tumours. More common in males and types include astrocytomas and medullblastomas (upto 75% in children),haemangioblastomas and metastasis occur more in adults.

Manifestations include raised intra cranial pressure, dorsal midbrain syndrome, impaired vertical gaze, diplopia, strabismus, nystagmus and abnormalities of smooth pursuit.

METASTASIS

- Multiple lesions in brain are characteristics of secondaries.
- The signs depend on the site of involvement and histological type.
- Breast and lung malignancies are the most common primary tumours.

INVESTIGATIONS

PRELIMINARY INVESTIGATIONS

These are done to rule out any infection / diabetes / hypertension which may contradict surgery in the patient. The blood count and peripheral blood smear will also be useful in cases of suspected primary malignancy elsewhere giving rise to a secondary metastasis in the brain.

SPECIAL INVESTIGATIONS

Neuro-Imaging techniques - complement the patient history and physical examination.

COMPUTERISED AXIAL TOMOGRAPHY

Patient is moved through a circular structure with an X-ray emitter located across from a detector. The emitter is rotated 180° and the computer reconstructs an image by calculating the density of the target from the collected set of attenuation values.

CT - SCAN

Advantages	Disadvantages
Images can be obtained in less time	Relatively poor soft tissue contrast
Superior in detecting calcification, imaging bone and acute intracranial	Artifacts from bone and metallic objects
Wider availability	Lack of direct sagittal scanning capability
Costs less	Difficulty in positioning patients to perform a coronal scan

Does not miss significant lesions in orbit or CNS, although it may not image them as well as MRI	Potential side effects of intravenous contrast administration
	Accumulated radiation dose for many studies

MAGNETIC RESONANCE IMAGING

This is the neuro-imaging study of choice for demonstration of intracranial soft tissue anatomy and pathology. The target tissue is subjected to a strong magnetic pulse, and the energy emitted from the recovering tissue is converted to an image.

Views can be obtained directly in any plane (axial / coronal / sagittal) allowing excellent localisation and three dimensional reconstruction of lesions.

MRI - SCAN

Advantages	Disadvantages
Multi planar capability	Requires a scanning time of several minutes per sequence
Increased soft tissue contrast	Claustrophobic patients may have difficulty being surrounded by MRI machinery
Preferred for demonstration of vascular malformations and	Cardiac pacemakers and intracranial aneurysmal clips are a contraindication

TREATMENT

Treatment of brain tumours depends on the location of the tumour, its histopathology and the clinical setting

Management of intracranial tumours are primarily by neurosurgeons and **Surgery** is almost always the initial part of the treatment as it helps establish histopathologic diagnosis, decreases mass effect and also reduces tumour burden.

The surgery can be either - (1) **Total excision** or

(2) **Subtotal excision** and **biopsy**

Technical advances include microsurgical operating techniques, stereotactic surgery using CT or MRI guidance, intraoperative monitoring with evoked responses and applied laser technology for innovative resection instruments.(Black,1991; Nazzaro & Neuwalt,1996)

Another technique for focal treatment of brain tumours is **stereotactic radio frequency (thermal) ablation**.(Anzai et al,1995; Jolesz,1995)

Biopsied material is sent for histopathological examination based on which the patient is subjected to further surgery or radiotherapy.

Radiotherapy is the modality of choice in patients for whom surgery is not curative. **Stereotactic radiosurgery** and **interstitial brachytherapy** provide high doses of radiation to the tumour while minimizing irradiation of the adjacent normal brain.

However, it is customary to avoid irradiation in children younger than

2-3 years of age (Pollack, 1994), due to developmental delay, hormonal abnormalities and secondary malignancies.

Chemotherapy as a primary modality has yet to be fully established though it is the treatment of choice in young children with unresectable tumours, postoperative malignant tumours allowing for deferral of irradiation for several years.

The commonly used agents include Vincristine, Cisplatin, Procarbazine, Etoposide and Methotrexate.

Adjuvant therapy in the form of Haemopoietic growth factors, autologous bone marrow transplantation and reconstitution of peripheral stem cells are still under investigation. (Pollack, 1994)

The role of the ophthalmologist is to assist the neurosurgeon in decisions regarding the time of surgical intervention so as to prevent further progression of the lesion.

AIM OF THE STUDY

- To study the incidence of ocular signs and symptoms in intracranial tumours.

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

- To study the value of ocular symptoms and signs in predicting the presence of an intra cranial tumour

MATERIALS AND METHODS

The study was conducted at Govt. Stanley Medical College and Hospital, Chennai involving the Departments of Ophthalmology, Neurosurgery, Neuromedicine and Radio Diagnosis.

The period of the study was from October 2004 to October 2006. A total of 79 patients who presented with either ocular, neurological or both complaints, suggestive of a brain tumour, were thoroughly evaluated and followed up. Out of these 12 patients were discarded in the final analysis based on the guidelines followed and therefore a total of 67 patients were included in this study.

Inclusion Criteria

The patients who had typical CT scan abnormalities suggestive of a brain tumour were included, evaluated and followed up.

Even those with CT scans showing mass lesion, which later was confirmed to be a tumor by histopathological examination (after surgical excision and biopsy) were also included in this study.

Exclusion Criteria

Patients who had typical symptoms and signs suggestive of a space occupying lesion, but were found to have normal CT scans were excluded from the study. Out of the intracranial space occupying lesions, lesions other than tumours (e.g. abscesses, tuberculomas and haematomas) were not included in this clinical study.

Neurological Evaluation

A detailed history was taken about the complaints with which the patient first consulted a physician.

Out of the presenting complaints, special emphasis was laid on

- Headache - Site, type, association with vomiting
- Motor symptoms - Weakness/paralysis or any seizures
- Mental changes-Any irrational behaviour or sudden onset of depression
- Gait disorder
- Endocrine abnormalities - Any enlargement of hands & feet, alteration of speech, decreased libido, amenorrhea, galactorrhoea, delayed secondary sexual characters, polydipsia, polyuria, increased sweating etc.
- Cranial nerve abnormalities - History relating to abnormal cranial nerve function was elicited by direct questioning as many patients did not attribute much importance to the same and hence did not volunteer the information on their own.

A systematic central nervous system examination was carried out in all patients under the following groups:

- Higher mental functions
- Cranial nerve examination
- Motor & sensory system
- Examination for cerebellar / meningeal signs

Ophthalmological Examination

The history regarding any visual disturbances, occurrence of diplopia protrusion of eyeball (proptosis), drooping of upper eyelid (ptosis) and limitation of ocular movements was noted.

Ocular examination included a detailed evaluation of both eyes.

Facial asymmetry, globe position, adnexae and ocular movements were noted.

Pupils were examined for their size, shape, reaction to light-both direct and consensual and reaction to accommodation.

Corneal sensation was looked for with the aid of a wisp of dry cotton.

Visual acuity for distance was recorded with Snellen's charts with & without glass correction (if the patient was already wearing glasses) and with pinhole. Near vision was documented using reduced Snellen’s test type chart.

Colour Vision was also checked for assessing the optic nerve function.

Fundus-Detailed examination of the fundus was done after dilating the pupil using short acting mydriatic. Details of the optic disc (size, shape, colour, margins, Cup-Disc ratio) vessels and foveal reflex were noted.

Intra-ocular pressure was recorded using Schiotz (Indentation) tonometer.

Charting of the fields was done using Bjerrum's screen (both 1 m and 2 m charts) and Confrontation method. Few cases, with no field defect on Bjerrum's, but had a great degree of clinical suspicion were subjected to Automated Perimetry - (Octopus 1-2-3). Suprathreshold testing was done as a screening procedure for few patients.

Proptosis, when present was documented, with Hertel's exophthalmometer and all the related parameters (bruit/reducibility/ compressibility etc) were looked for.

Cranial nerve abnormalities-Diplopia Charting was done using red-green goggles. The type of diplopia was charted out to help in reviewing the patient as to progression / regression of the complaint.

Other Systemic Examination

Any relevant past history or family history related to diabetes or hypertension was recorded. General status of the patients including cardiac and respiratory functions was assessed. Evaluation of possible endocrine disturbances was done..

Investigations

Neurological Investigations

After screening the patients, the cases selected for the study were subjected to X-ray, CT-brain, MRI-brain to confirm and correlate to presence of an intracranial tumour.

Additional investigations like CSF analysis, hormonal assay, EEG etc were done as and when required.

Ophthalmic Investigations

X-rays of orbit and of both optic foramina were taken to delineate involvement of superior orbital fissure and optic canal.

Fundus fluorescein angiography was done in selected cases after injecting 3 ml of 20% fluorescein intravenously, to confirm the diagnosis of papilledema, when in doubt.

MANAGEMENT

Conservative management included antibiotics, steroids, and NSAIDs to reduce the inflammatory oedema and pain.

Hormone replacement was started for patients with endocrine dysfunction.

Surgical intervention was at the discretion of the neuro surgeon, being a total excision or sub total excision and biopsy taken.

Biopsy material was subjected to histopathological examination and further treatment was planned according to the tumour type.

Radiotherapy was given to patients whose tumours were inaccessible surgically or those who had recurrences after a primary excision.

Patients were reviewed periodically and any improvement/deterioration of their visual parameters was noted.

All the above data was recorded on a proforma.

ANALYSIS AND DISCUSSION

Sex Distribution

Table No.1

Total	Male	Female
67	32 (47.76%)	35 (52.24%)

Out of the 67 patients studied, 32 (47.76%) were males and 35 (52.2%) were females. Hence in this study a more or less equal sex predilection was noted for brain tumours on the whole.

Male preponderance is noted to be more in primary brain tumours (Radhakrishnan K. Mokri B, Parsi JE et al,1995)

The almost equal occurrence of males & females in the study can be due to the fact that this study was conducted at a hospital pertaining to a certain area with a limited time period and hence does not actually correlate with a true incidence among the general population.

Another possible reason can be attributed to emerging genetic susceptibility to brain tumors (Riggs 1995).

Age Distribution

Table - No. 2

Age (Years)	No. of Patients	Percentage
<10	3	4.48%
11-20	8	11.94%
21-30	18	26.86%
31-40	16	23.88%
41-50	11	16.41%
>50	11	16.41%
	67	

In this study population, the maximum number of patients (18) was seen in the 21-30 year age group (26.86%). The next most affected age group was in the 31-40 year period with 16 patients (23.88%).

So in this study, the 21-40 years age group was seen to be maximum involved. Quite a substantial no. of patients were of the > 40 year category (22 patients – 32.83%) This differs from the incidence rates quoted by National Survey 1973-74.

This increased incidence in the elderly population could be either due to availability of increased longevity and better diagnostic methods and management protocols followed.

Age and Sex Distribution

Table – No. 3

Age (Years)	Male (%)	Female (%)	Total (%)
<10	1(3.12%)	2(5.71%)	3(4.48%)
11-20	3(9.38%)	5(14.28%)	8(11.94%)
21-30	9(28.12%)	9(25.71%)	18(26.86%)
31-40	7(21.87%)	9(25.71%)	16(23.88%)
41-50	7(21.87%)	4(11.42%)	11(16.4%)
51-60	5(15.62%)	6(17.14%)	11(16.4%)
	32	35	67

Males & Females were equally affected in both 21-30 years & 31-40 Years age group (11 & 6 patients respectively).

There were more male patients (21.87%) in the older (41-50 Years) age group than female patients (11.42%) This could be because this study included people mainly of lower socio economic status wherein elderly women do not seek or get assistance for their problems.

However, the bulk of childhood tumours were seen in more females when compared with males (7 and 4 respectively).

Type of Tumour (Based on HPE Report)

Table - No. 4

Type of Tumour	No. of Patients	Percentage
Meningioma	13	19.40%
Acoustic Neuroma	7	10.44%
Pituitary Adenoma	10	14.92%
Craniopharyngioma	7	10.44%
Astrocytoma	3	4.47%
Glioma	6	8.95%
Pinealoma	1	1.49%
Medulloblastoma	1	1.49%
Ependymoma	1	1.49%
Not Known	18	26.86%
	67	

Meningiomas formed the majority of intracranial tumours in the study with 13 patients (19.40%) belonging to this category.

Pituitary adenomas were next in frequency with 10 patients (14.92%) in the group. Acoustic neuromas and Craniopharyngiomas were diagnosed in 7 patients (10.44%) in both groups.

Astrocytomas were few in number while Ependymomas and Pinealomas were seen only in one patient.

18 patients (26.86%) were not subjected to tumour biopsy & HPE or lost to follow up and hence, they could not be classified among any group.

Sex Distribution among Different Types of Tumours

Table No.5

Type of Tumour	Male (%)	Female (%)	Total (%)
Meningioma	6(18.75%)	7(20%)	13(19.4%)
Acoustic Neuroma	3(9.37%)	4(11.42%)	7(10.44%)
Pituitary Adenoma	4(12.5%)	6(17.14 %)	10(14.92%)
Craniopharyngioma	4(12.5%)	3(8.57%)	7(10.44%)
Astrocytoma	1(3.12%)	2(5.71%)	3(4.47%)
Glioma	3(9.37%)	3(8.57%)	6(8.95%)
Pinealoma	-	1(2.85%)	1(1.49%)
Medulloblastoma	1(3.12%)	-	1(1.49%)
Ependymoma	1(3.12%)	-	1(1.49%)
Not Known	9(28.12%)	9(25.71%)	18(26.86%)
	32	35	67

Comparing the incidence of types of tumours (Based on HPE reports) in both sexes, it was found that Menigiomas were seen more or less equally in females (7 Patients —20%) and males (6 patients – 18.75%).

Meningicmas are reported to be more common in females (Walsh). The fact that the number of female patients in the elderly group where meningiomas are more common was lesser may have been the cause for this data.

Pituitary adenomas, Craniopharyngiomas and Acoustic neuromas were also seen with almost equal frequency in both sexes.

Presenting Complaints

Table No. 6

Presenting Complaints	No. of Patients	Percentage
Ocular only	7	10.44 %
Non-Ocular only	27	40.29 %
Both	33	49.25 %
	67	

Most of the patients presented with both ocular and general symptoms - above 50% of the study group.

There were 7 patients (10.44%) who presented exclusively with ocular symptoms only and hence were seen by an ophthalmologist first.

Even of the remaining patients, 33of them (49.25 %) had both ocular and neurologic complaints.

Hence this underlines the role Opthalmologists play in detecting brain tumours at an earlier stage.

Ocular Symptoms

Table No. 7

Ocular Symptoms	No. of Patients	Percentage
Defective Vision	35	52.23%
Diplopia	11	16.41%
Ptosis	6	8.95%
Field defects	16	23.88%
Amaurosis fugax	2	2.98%
Proptosis	2	2.98%

Of the many symptoms, defective vision was the most common, affecting 35 patients (52.23%) and associated field defects coming second (16 patients, 23.88 %) correlating to the close proximity of the visual system with the intracranial structures

Diplopia was the next most common complaint, due to the ocular motor nerve abnormalities, correlating with sixth cranial nerve involvement occurring as a "false - localising" sign, in patients with increased intracranial pressure.

Typical complaint of amaurosis fugax (suggestive of papilledema) was volunteered by 3 patients (4.47%) only, out of the 23 patients (34.32 %) who were found to have papilledema on fundus examination. This could be because the study involved illiterate people from a poor socio-economic status who may not attach significance to the transient visual obscurations.

Correlation between Ocular Symptoms & Site of Brain Tumour

TableNo.8

Site of brain tumour	Headache	Defective vision (%)	Diplopia (%)
Frontal	8(16.3%)	2(5.88%)	1(9.09%)
Fronto-Parietal	3(6.12%)	3(8.82%)	-
Parietal	6(12.25%)	3(8.82%)	-
Temporal	7(14.28%)	3(8.82%)	1(9.09%)
Tempero-Parietal	11(22.45%)	2(5.88%)	1(9.09%)
Sellar/Parasellar/ Suprasellar region	4(8.16%)	14(41.17%)	2(18.18%)
CPA	5(10.2%)	3(8.82%)	4(36.36%)
Cerebellar	2(4.08%)	1(2.94%)	-
Sphenoid Bone	3(6.12%)	4(11.76%)	1(9.09%)
Pineal Gland	-	-	1(9.09%)
	49(73.1%)	34(50.74%)	11(16.41%)

Headache (with or without vomiting) was the single most consistent symptom occurring in 49 patients (73.1%). This correlates with the studies done elsewhere - Headache was seen to occur in 75% or more of patients (Rushton & Rooke, 1962; The Childhood Brain Tumour Consortium, 1991). Even when other complaints were present, headache was often told to be the first symptom by the patients in this study.

Headache was seen to occur in almost all the patients irrespective of the site of tumour & hence is regarded as a non-localising symptom. CT usage in all cases of generalized headache showed only 1% prevalence of tumours.

Correlation between Neurological Symptoms and Site of Tumour

Table No. 9

	Weakness	**Seizures**	**Other CN Abnormalit**	**Endocrine Dysfunctio**
Frontal	4(16.66%)	3(25%)	-	-
Fronto-Parietal	2(8.33%)	-	-	-
Parietal	3(12.5%)	4(33.33%)	1(10%)	-
Temporal	2(8.33%)	-	2(20%)	-
Tempero-Parietal	4(16.66%)	2(16.67%)	-	-
Sellar/Parasellar/ Suprasellar	1(4.16%)	2(16.67%)	-	6(100%)
CPA	3(12.5%)	1(8.33%)	7(70%)	-
Cerebellar	2(8.33%)	-	-	-
Sphenoid Bone	2(8.33%)	-	-	-
Pineal Gland	1(4.16%)	-	-	-
	24(35.82%)	12(17.91%)	10(14.9%)	6(8.95%)

Motor abnormalities including weakness / paresis of limbs or gait disorders were frequently complained (35.82%). Seizures whether focal or diffuse type accounted for another significant reason for seeking medical attention. (12 patients – 17.91%)

Among the 24 patients who complained of weakness or paralysis, 11 patients (45.8%) had a mass in the vicinity of parietal & temporal lobes. This is in concordance with the involvement of pyramidal tract in these areas & is mostly associated with a UMN type of seventh cranial nerve palsy (Huber, 1976).

Convulsions were seen to occur in 6 patients (50%) with lesions in and around parietal lobe (Parietal & Tempero - Parietal mass) - The occurrence of seizures due to a mass in this region is usually accompanied by visual hallucinations & aura (Forsyth & Posner, 1993).

Involvement of eighth cranial nerve causing deafness or tinnitus or vertigo occurred in 10 patients out of which 7 patients (70%) had a CPA mass. Hence any complaint of defective hearing in a patient suspecting to harbour a brain tumour should be immediately investigated for a CPA tumour.

6 patients had complaints of endocrine function (Amenorrhoea, Galactorrhoea, Enlargement of Hands & Feet, Delayed Growth etc.,) and all the 8 patients had a sellar or suprasellar mass - later found to be pituitary adenoma by HPE. Hence, occurrence of any hormonal imbalance should arouse a suspicion of a pituitary tumour.

Ophthalmic Signs

Table No. 10

Ophthalmic Signs	No. of Patients	Percentage
Papilledema	23	34.32%
Cranial nerves		
II	18	26.86%
III	6	8.95%
IV	1	1.49%
V	8	11.94%
VI	12	17.91%
VII	8	11.94%
Field defects	16	23.88%
Nystagmus	6	8.95%
Gaze palsy	3	4.47%
Proptosis	2	2.98%

On examining the patients, papilledema was found to be the major clinical sign occurring in 23 patients (34.32%). Though this formed the most common ophthalmic sign, the occurrence is less than studies done elsewhere

Papilledema occurred in 80% of the study group according to Uhtoff (1915) & Paton (1935). In this study FFA was done only in few cases with a clinical suspicion of papilledema - Probably routine FFA for all patients would yield a higher incidence of papilledema. Another reason may be due to the fact that lesions are being detected earlier due to advanced neuroimaging techniques.

The next frequent manifestation was of cranial nerve palsies out of which Optic nerve was most commonly affected (18 patients - 26.86%).

Out of the cranial nerves governing ocular motility sixth cranial nerve showed maximum involvement (12 patients – 17.91%) whereas third and fourth nerves were affected less frequently (8.95% and 1.49% respectively). This again shows the involvement of sixth cranial nerve as a false localising sign.

Field defects were seen in 16 patients (23.86%) which had a great localising value and aided the topical diagnosis of tumours profoundly.

Correlation of Ophthalmic Signs with the Site of Tumour

Table No. 11

Site of Tumour	Papilledema	II CN defect	Eye Movt. Defect	Field defects
Frontal	2(8.7%)	1(5.6%)	1(5.26%)	-
Fronto-Parietal	1(4.3%)	1(5.6%)	-	-
Parietal	4(17.3%)	-	2(10.52%)	3(18.75%)
Temporal	2(8.7%)	-	-	1(6.25%)
Tempero-Parietal	2(8.7%)	-	1(5.26%)	1(6.25%)
Sellar/Parasellar/ Suprasellar	1(4.3%)	14(77.8%)	2(10.52%)	10(62.5%)
CPA	8(34.8%)	2(11.1%)	7(36.84%)	1(6.25%)
Cerebellar	2(8.7%)	-	3(15.78%)	-
Sphenoid Bone	-	-	2(10.52%)	-
Pineal Gland	1(4.3%)	-	1(5.26%)	-
	23	18	19	16

On studying the occurrence of papilledema with tumours in different locations, it was found that 8 patients (34.8%) had a CPA mass. These patients had also complained of diplopia at the time of their presentation, which can thus be explained by sixth cranial nerve palsy occurring as a false localising sign.

SUMMARY

- This clinical study was conducted at Government Stanley Medical College & Hospital, Chennai, during the period October 2004 to October 2006.
- Out of the initial 79 patients, 67 were finally selected based on the criteria specified for the study.
- There were 32 male patients (47.76%) and 35 female patients (52.2%).
- The maximum incidence of brain tumours was seen in the 21-30 years age group (18 patients – 26.8%).
- Among these, males and females were equally affected (9 patients each). In age group > 40years, there were more number of male patients (21.87%), than female patients (11.42%).
- Headache was the major presenting complaint occurring in 49 patients (73.1%).
- Gradual loss of vision was the most prominent ocular complaint (52.23%) suggesting a compressive effect on the visual pathway.
- Severe and rapid visual loss indicated an intracranial tumour which was highly neoplastic (rapidly expanding), seen in 26 patients (38.8%).
- Ocular muscle palsies were dominated with sixth cranial nerve involvement which served as a warning sign for brain tumours and occurred as false localising signs.

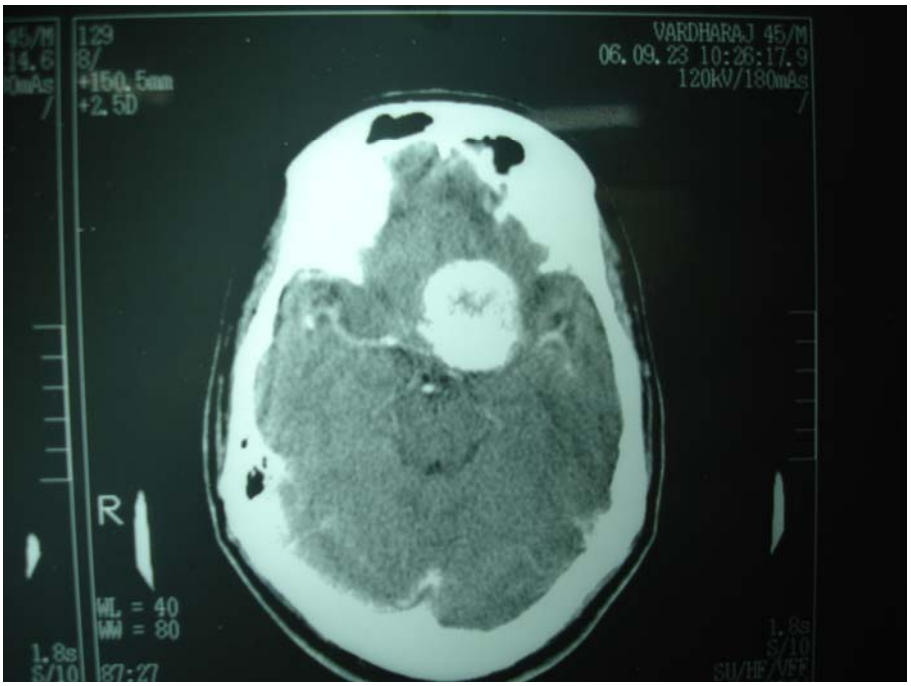
- Involvement of II cranial nerve either causing a fall in Vision or a field defect was found to be common in sellar lesions causing compression of optic chiasm.
- Papilledema was the salient ocular manifestation and it was always associated with increased intracranial pressure caused due to the mass effect of brain tumours.
- CT Scan was the main diagnostic tool used to confirm the presence or absence of an intracranial mass.
- Histopathological Examination of tumour specimens was done in 49 cases (73.13%).
- Meningiomas were the single most common type of tumour (13 patients – 19.4%) reported by HPE.
- Pituitary adenomas and Craniopharyngiomas together constituted of 17 patients (25.37%) which were significant due to their proximity with optic chiasma and hence the field defects that they produced.

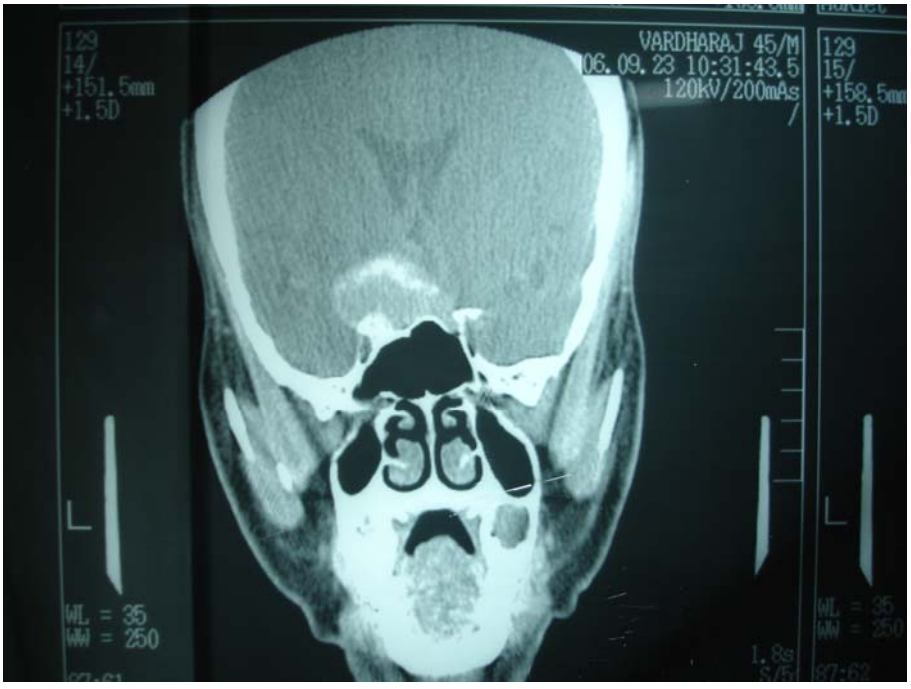
CONCLUSION

- Among the 67 patients studied, 21-30 year age group was the most commonly affected.
- Overall, there was no sex predilection on the whole in this study, though females were more commonly affected in the younger age group and the males more affected in the elderly group while the middle aged group were equally distributed.
- Headache, occurred as a non-localizing symptom, being present in almost all the patients irrespective of the location of tumours.
- About 10% of the patients in the study had presented with ocular symptoms only - hence ophthalmic manifestations are a predominant feature of intracranial tumours.
- Ocular signs aided in localising the site of tumour especially in association with neurological signs.
- Loss of vision or a field defect were seen to occur in lesions causing chiasmal compression (Sellar, Parasellar or Suprasellar mass).
- Sixth cranial nerve palsy occurred as a false localising sign, again most frequently in CPA tumours, as a result of the raised intracranial pressure.
- Papilledema was the most striking ophthalmic sign and it was seen to occur in CPA angle tumours most frequently

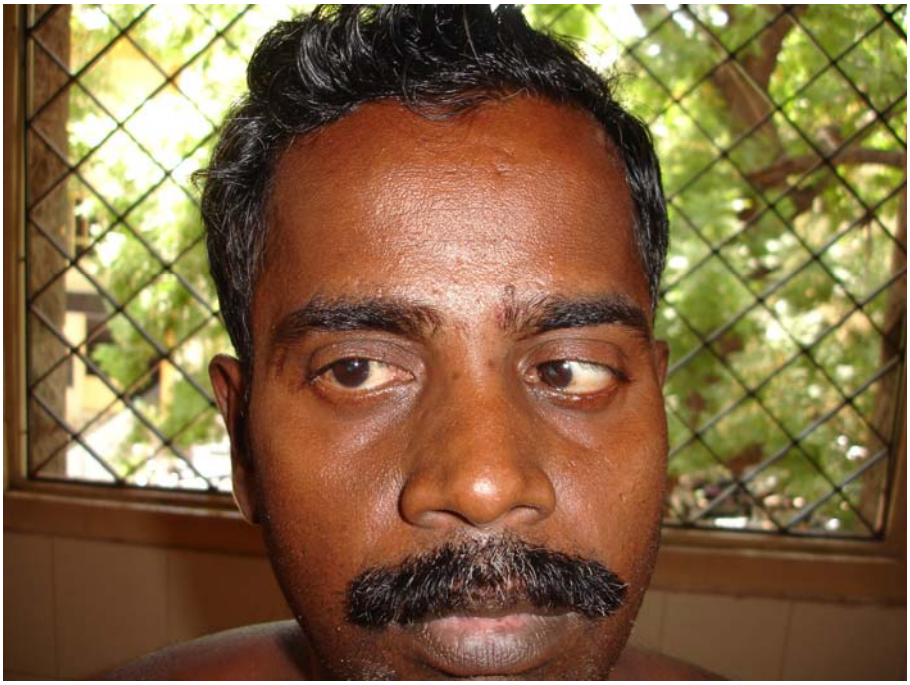
- Meningiomas were found to be the most common type of tumour as reported by HPE.
- Sellar tumours (pituitary adenomas and craniopharyngioma) formed a major bulk of the tumours, based on the site of tumours
- All the patients who complained of endocrine abnormality had a sellar mass, (pituitary adenoma).
- Ophthalmologists play a pivotal role not only in earliest detection of intra cranial tumours, but also are vital in assisting neurosurgeons with decisions regarding surgical intervention and post-operative management.
- Though newer and advanced radiology and neuro imaging techniques has improved the quality and localisation of intra cranial tumours, ocular signs prove to be a reliable and cost-effective clinical tool in the diagnosis of intracranial tumours.
- This study re-emphasizes the importance of Ophthalmologists, Neurosurgeons, Radiologists and Neurologists working as a unit towards early and accurate diagnosis with prompt management and patient care as far as intra cranial tumours are concerned to help reduce the morbidity.

CT BRAIN SHOWING SPHENOIDAL WING MENINGIOMA

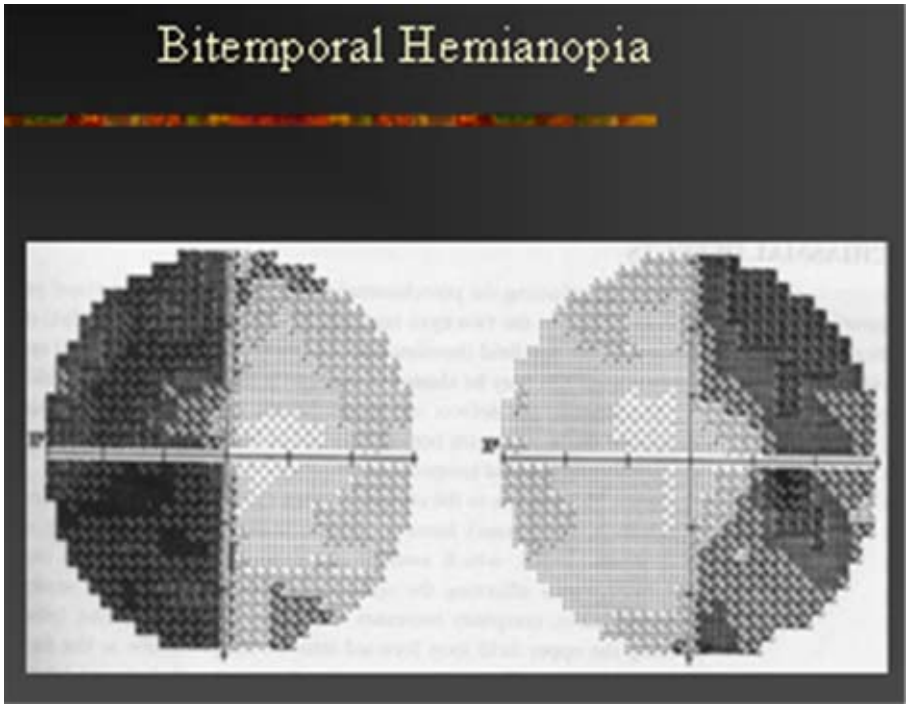




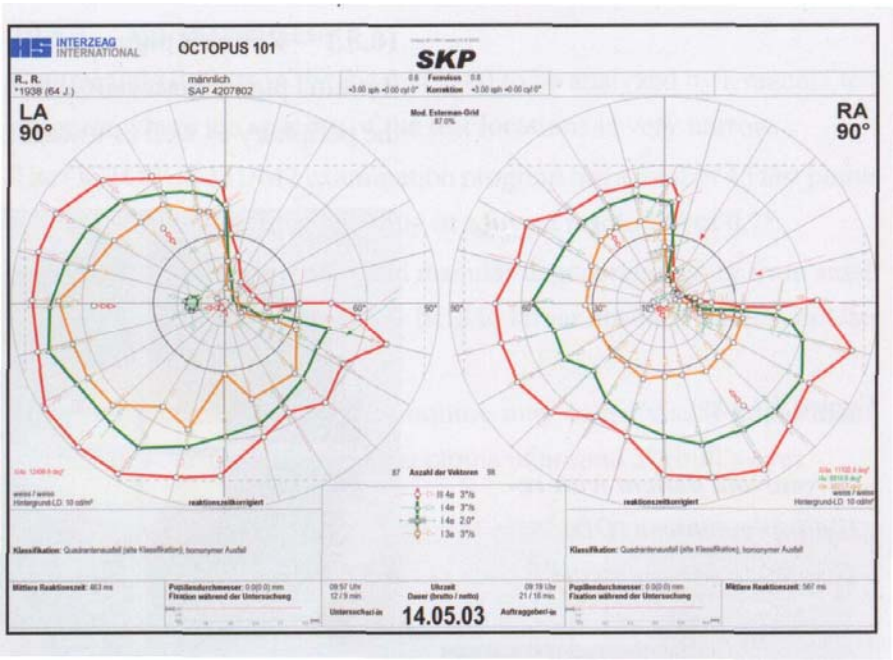
BILATERAL ABDUSCENS NERVE PALSY –
FALSE LOCALISING SIGN



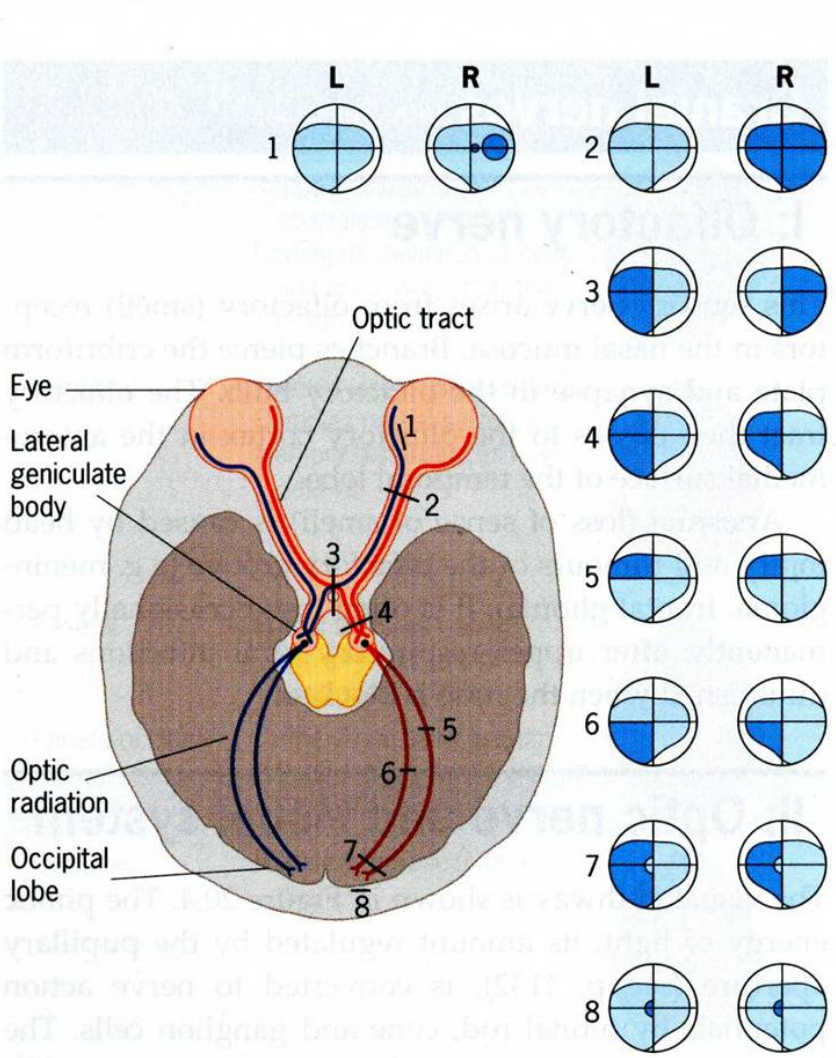
FIELD CHART OF PATIENT HAVING PITUITARY ADENOMA



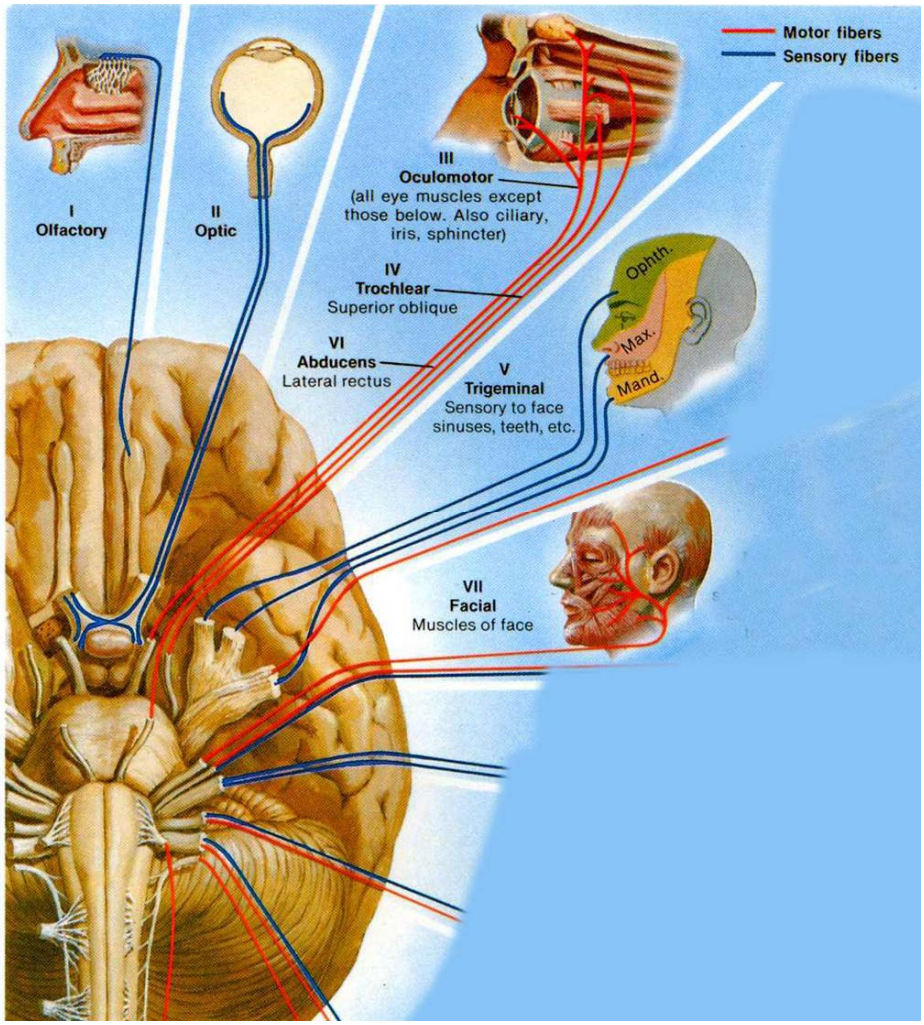
AUTOMATED PERIMETRY OF PATIENT SHOWING HOMONYMOUS SUPERIOR QUADRANTANOPIA



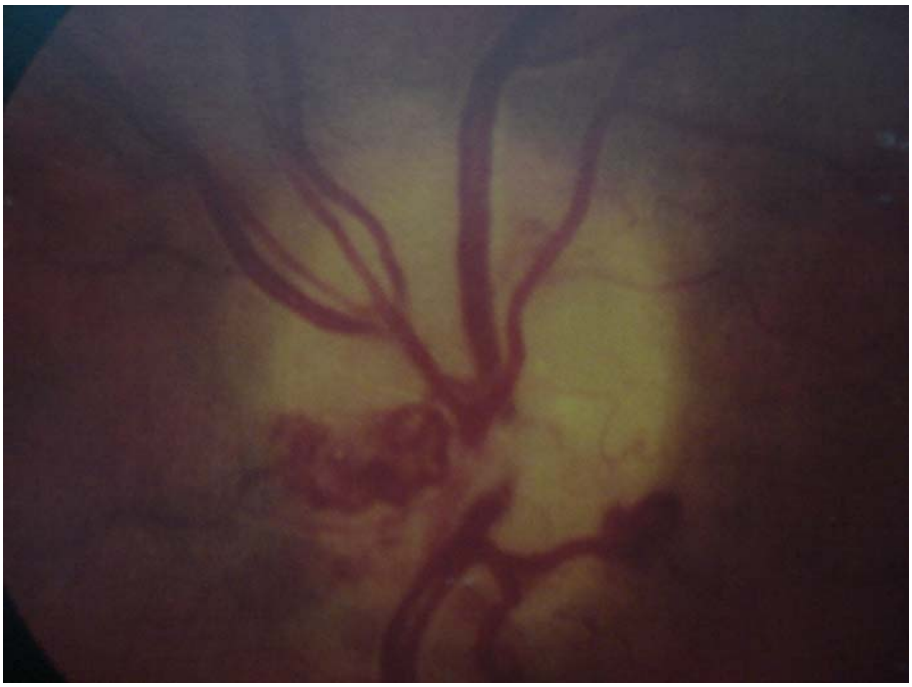
VISUAL PATHWAY DEFECTS CAUSED BY LESIONS AT VARIOUS LEVELS



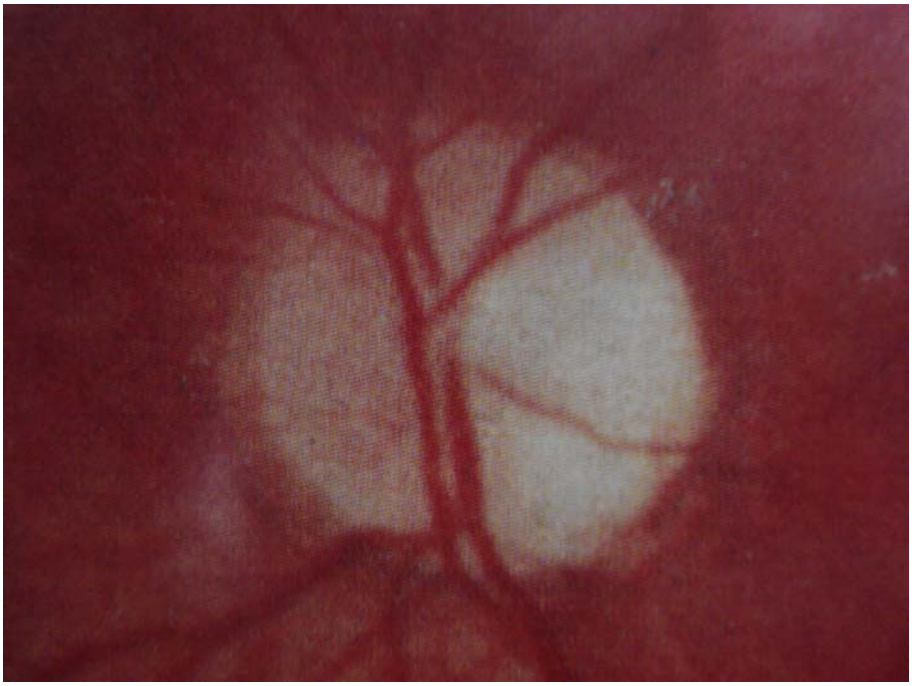
NEURO-OPHTHALMIC CRANIAL NERVES



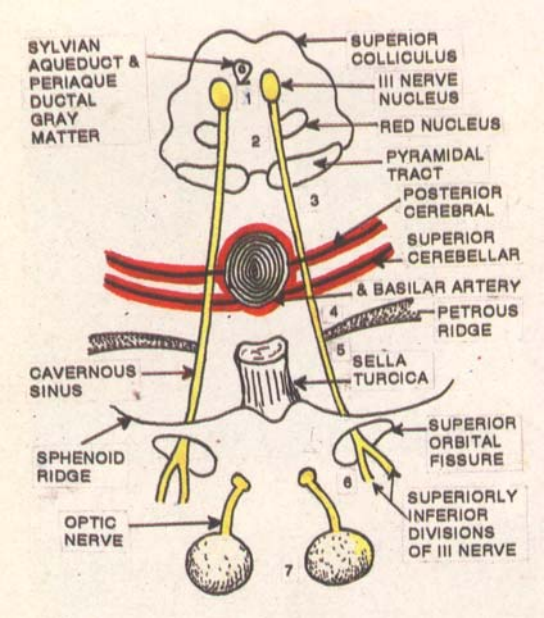
OPTO-CILIARY SHUNT



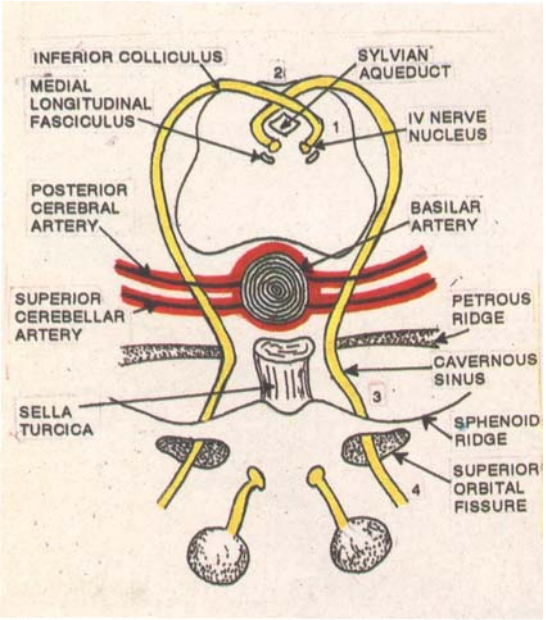
OPTIC ATROPHY



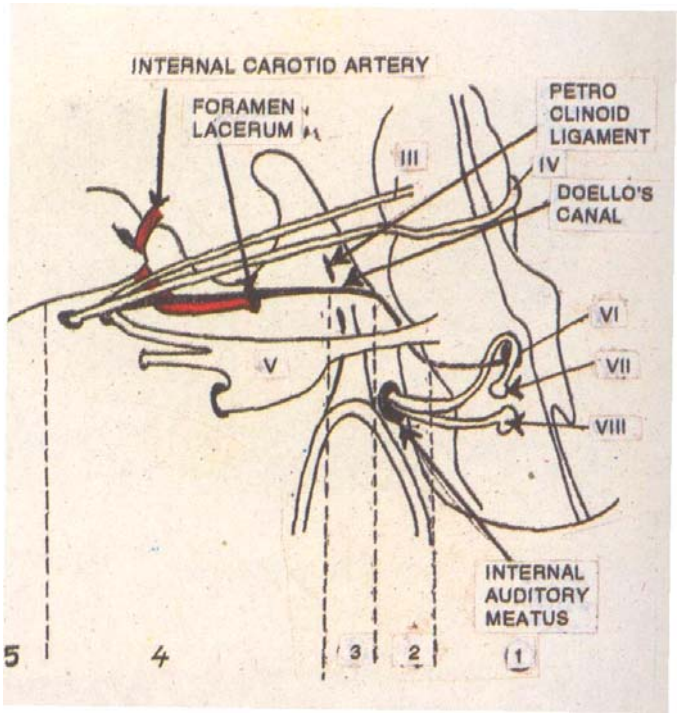
OCCULOMOTOR NERVE – COURSE AND POSSIBLE SITES OF LESIONS



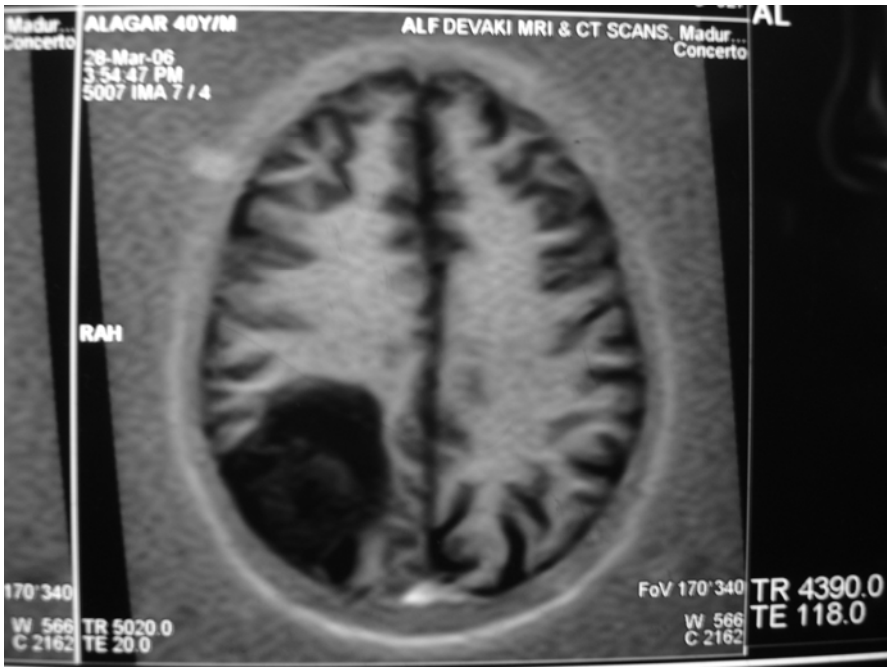
TROCHLEAR NERVE – COURSE AND POSSIBLE SITES OF LESIONS

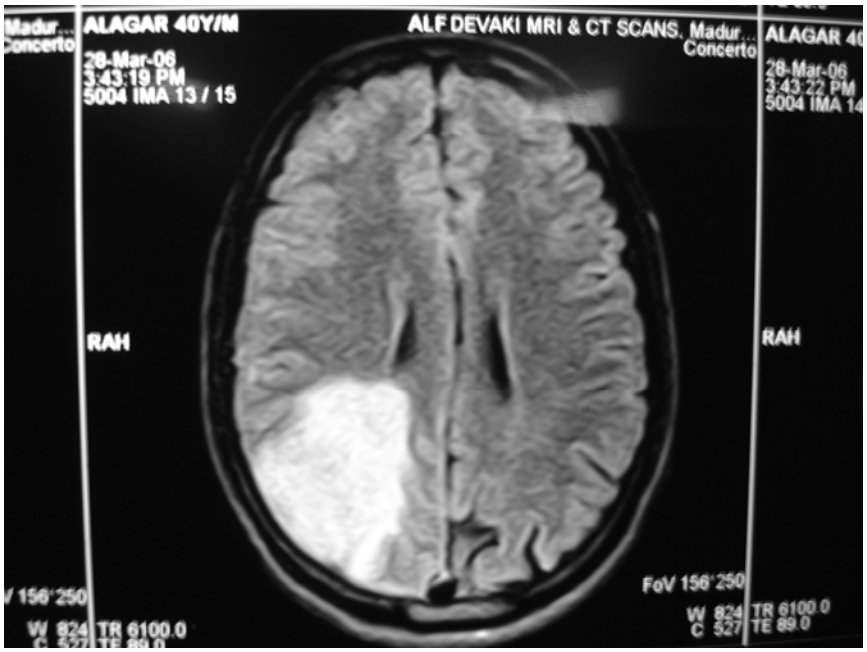


ABDUSCENS NERVE – COURSE AND POSSIBLE SITES OF LESIONS

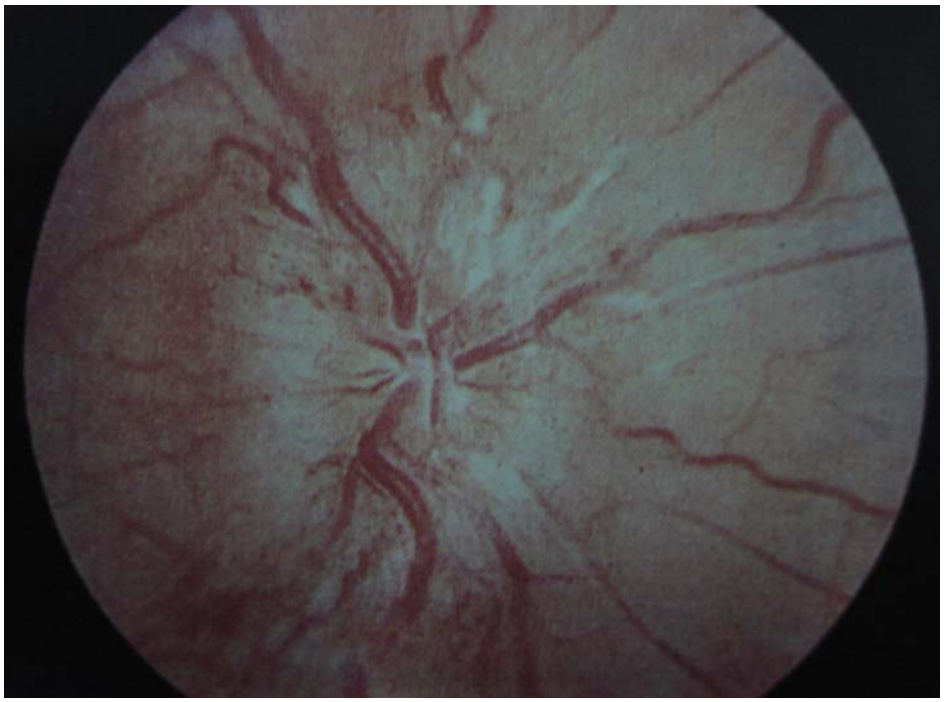


CT BRAIN SHOWING OCCIPITAL LOBE GLIOMA





PAPILLEDEMA



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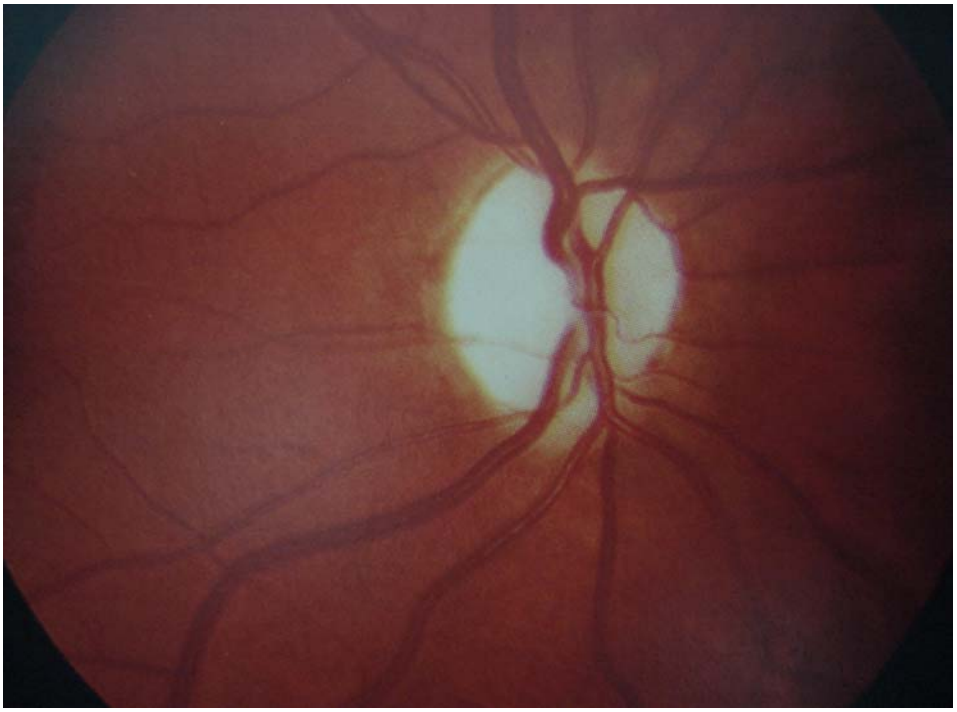
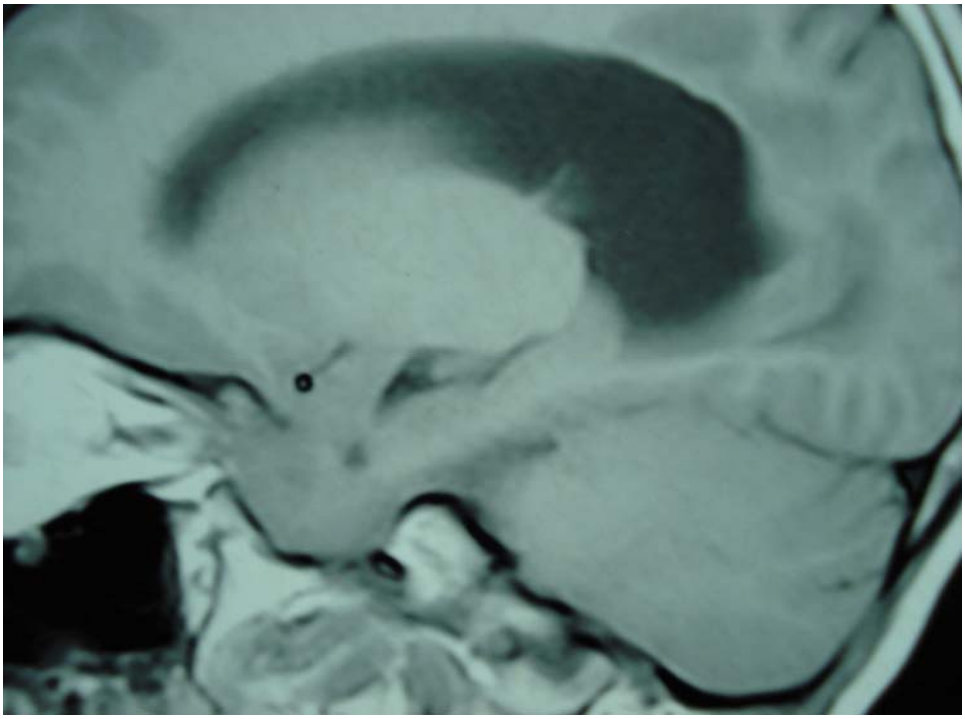
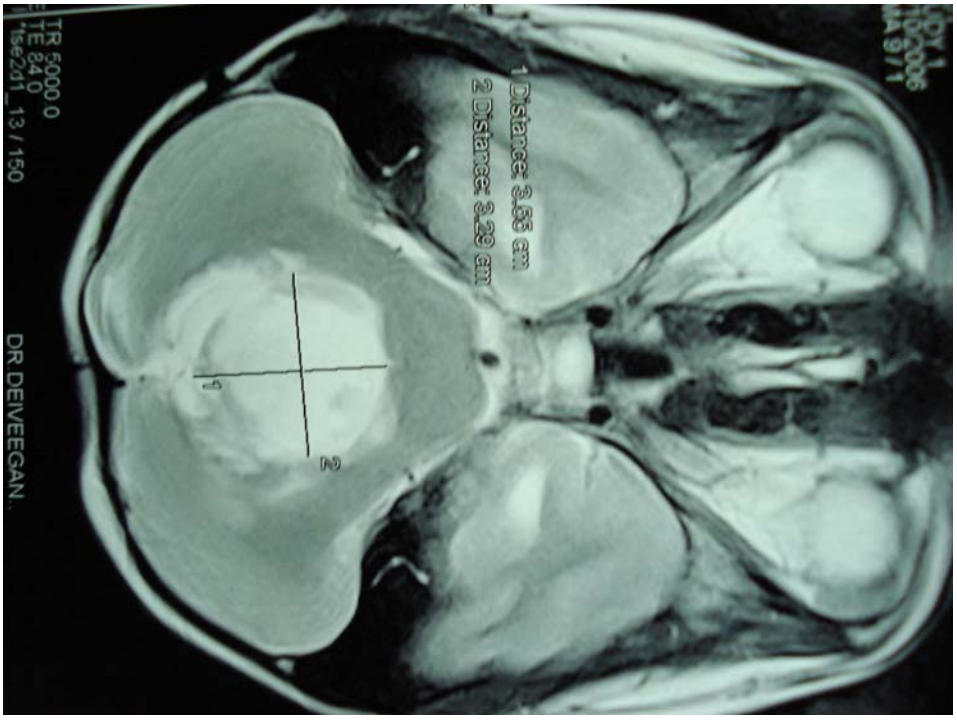
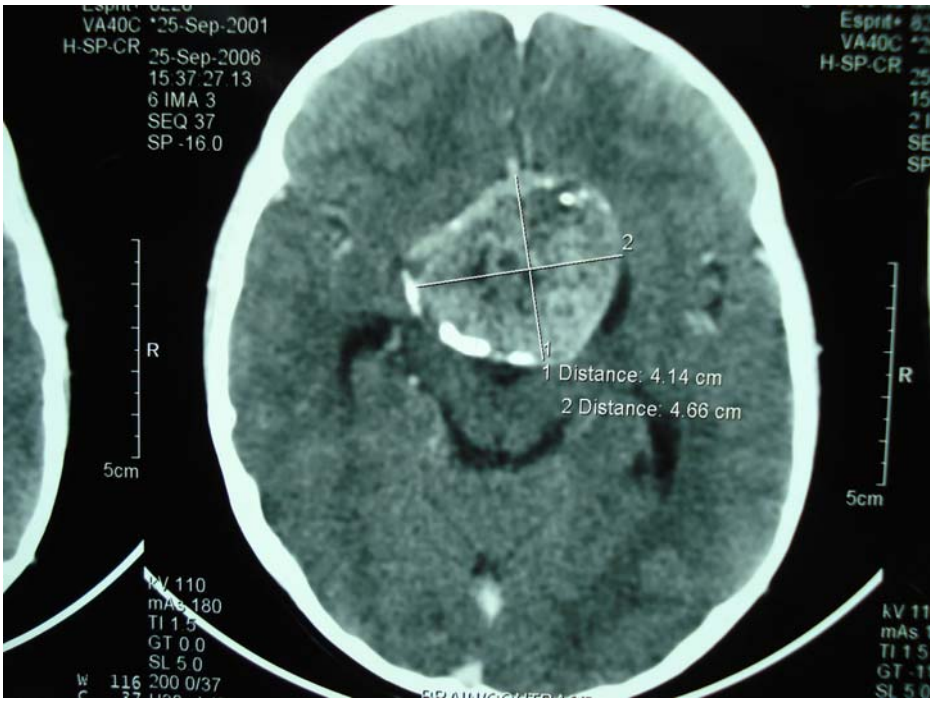


MRI BRAIN SHOWING A RECURRENT MEDULLOBLASTOMA WITH OBSTRUCTIVE HYDROCEPHALOUS

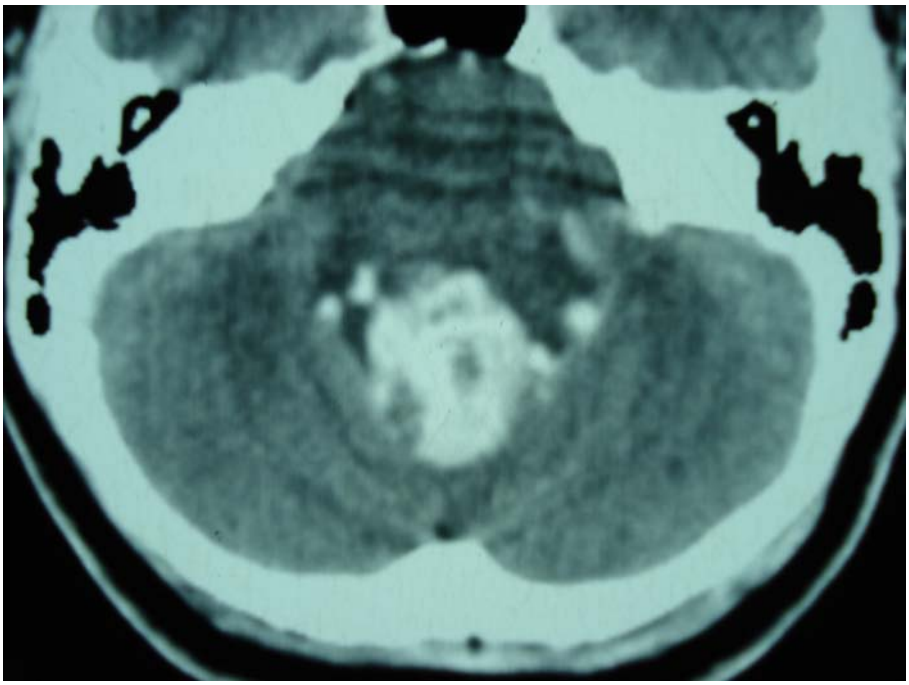




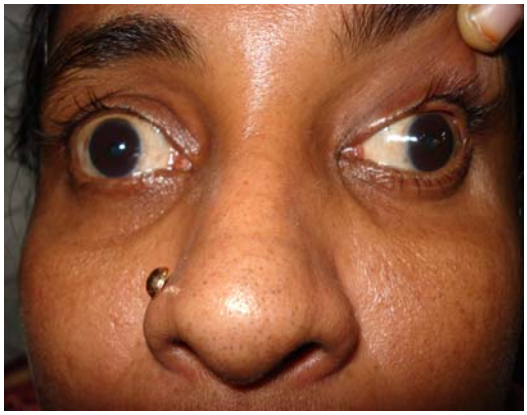
CT BRAIN SHOWING CRANIOPHARYNGIOMA

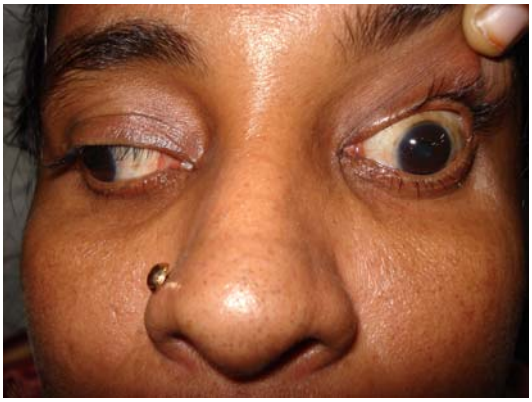


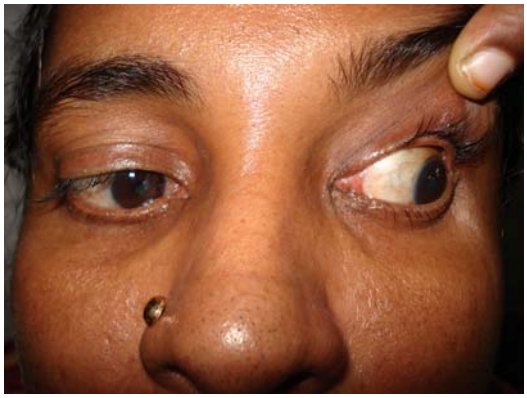
CT BRAIN SHOWING EPENDYMOMA WITH OBSTRUCTIVE HYDROCEPHALUS

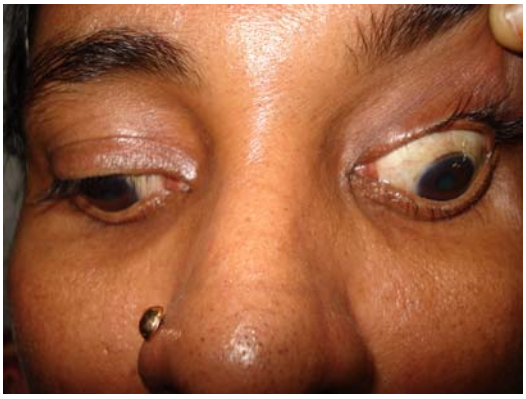


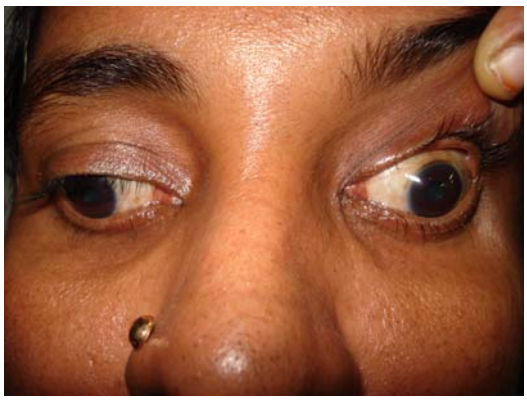
OCCULOMOTOR / III CN PARESIS



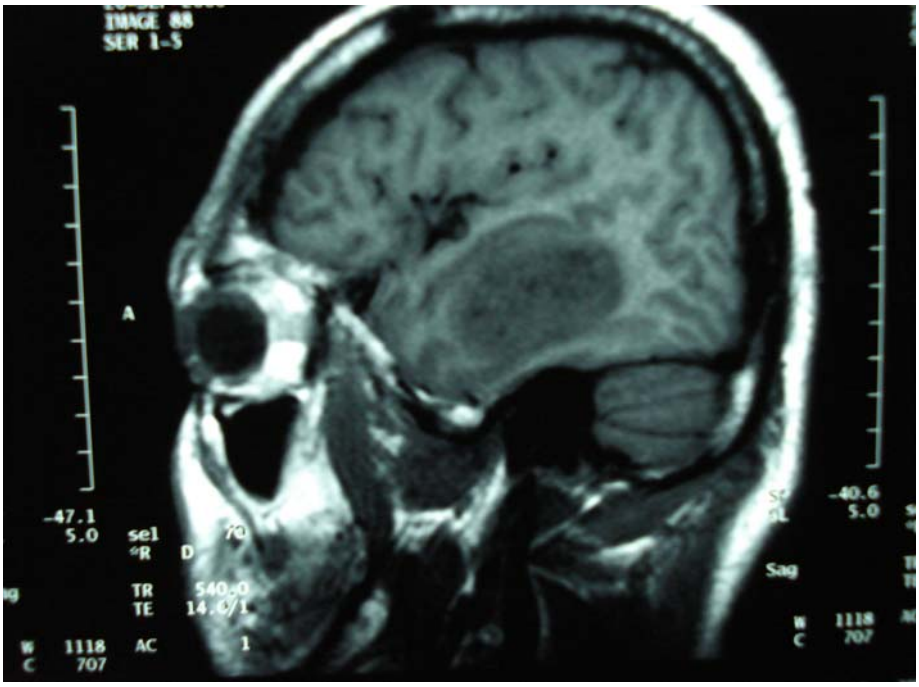


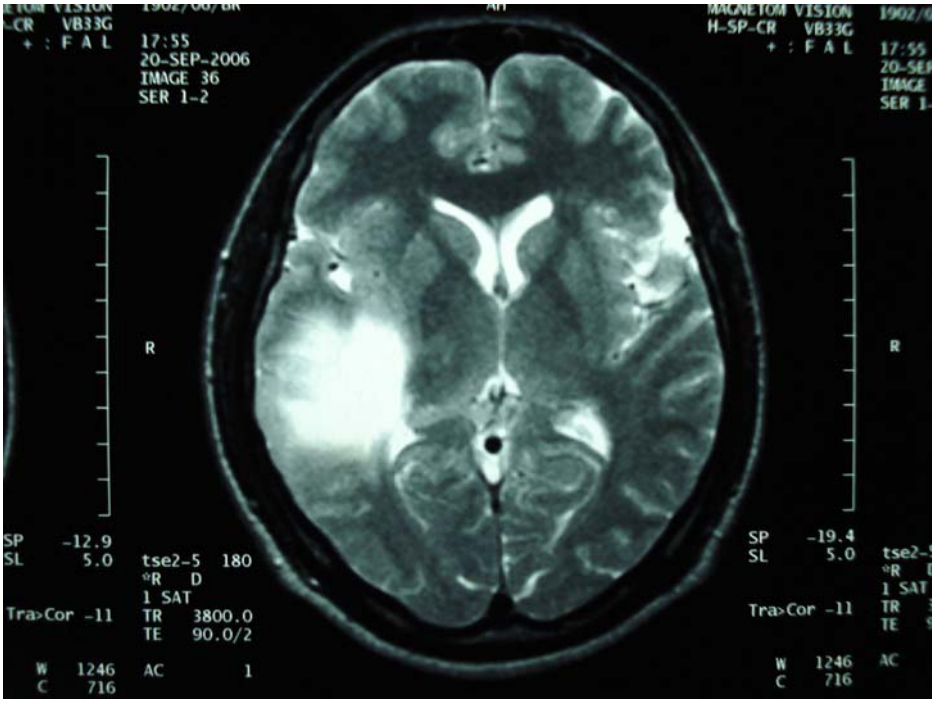




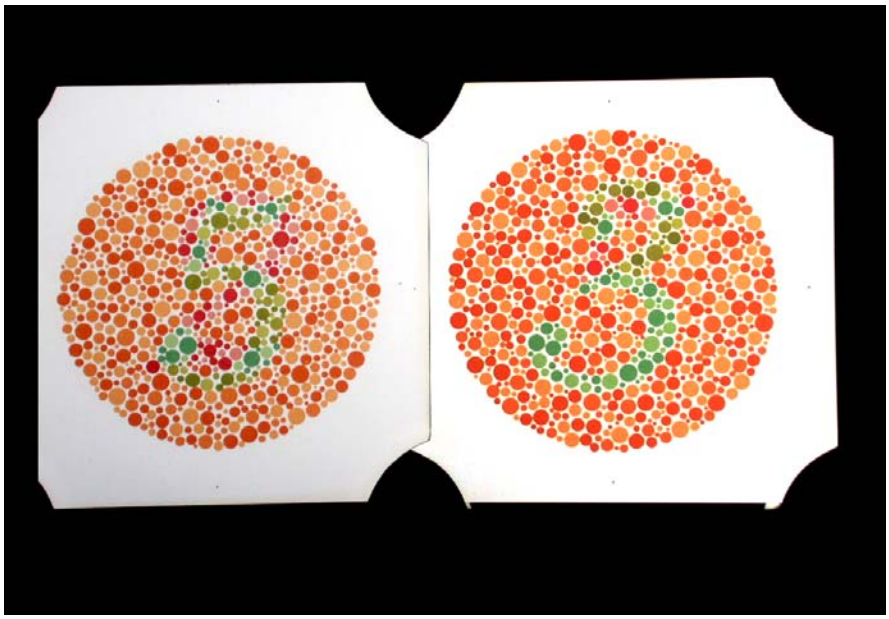


CT BRAIN SHOWING TEMPORAL LOBE GLIOMA

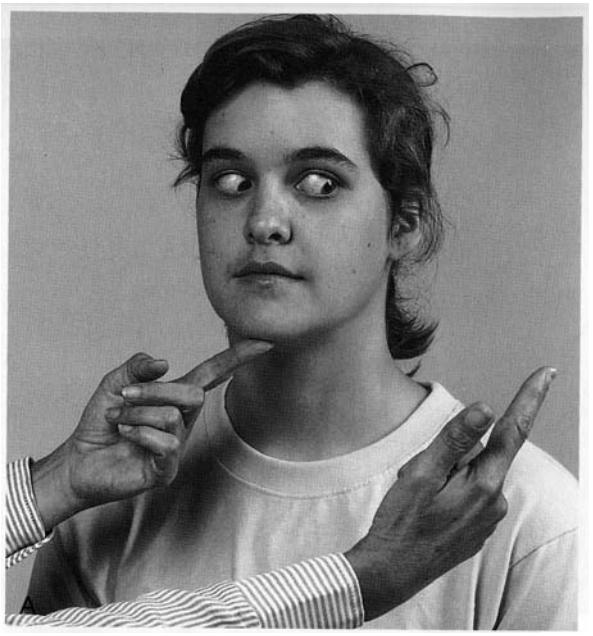




ISHIHARO'S ISOCHROMATIC
COLOUR VISION CARDS

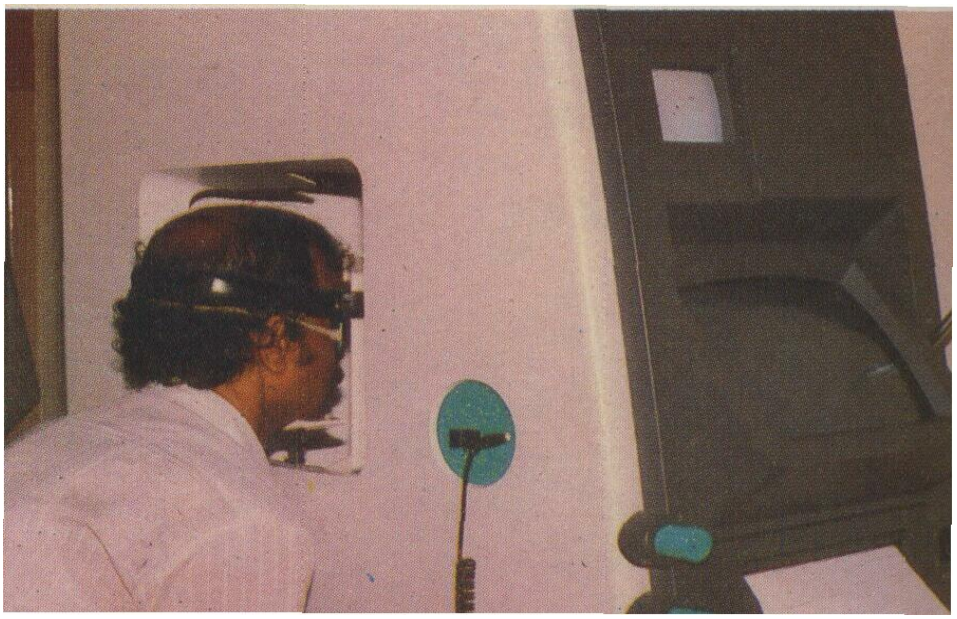


TESTING OF EXTRA OCULAR MOVEMENTS

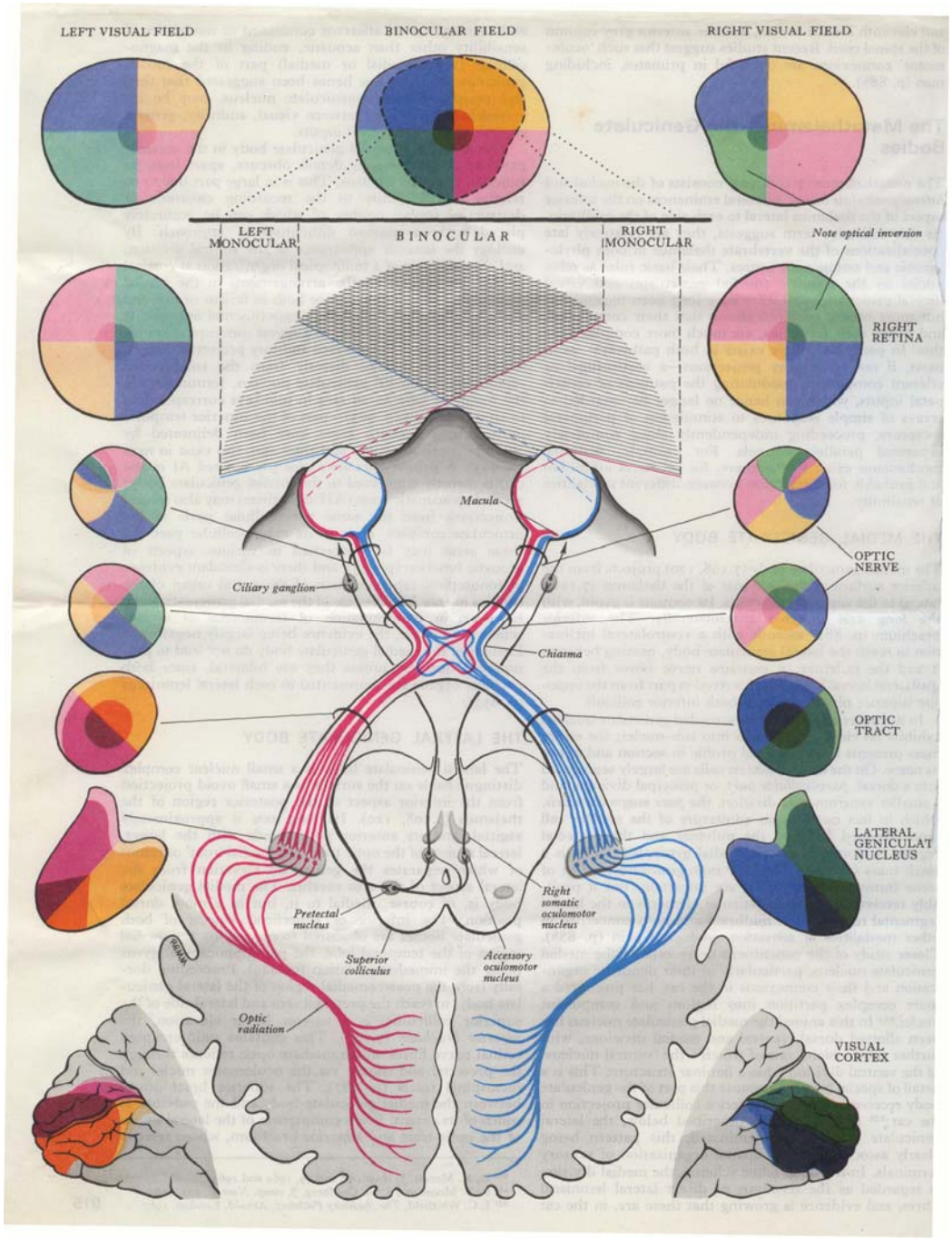


CONFRONTATION TESTING

AUTOMATED PERIMETRY



VISUAL PATHWAY – ANATOMY & ARRANGEMENT OF FIBRES



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