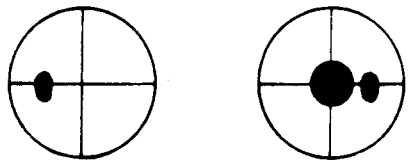
CHAPTER 11 LESIONS OF THE SENSORY AND MOTOR PATHWAYS

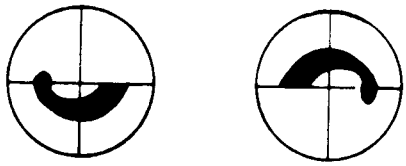
A. Field defects

1. CENTRAL SCOTOMA



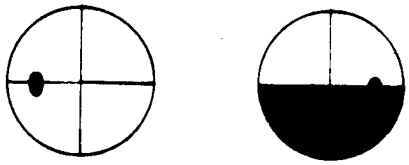
AETIOLOGY:
a) Optic neuritis(multiple sclerosis
b) Macular degeneration
c) Foveal oedema or haemorrhage(diabetes)

2. ARCUATE SCOTOMA



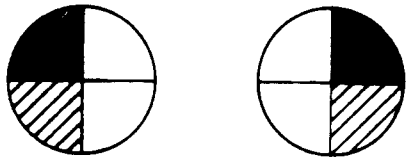
AETIOLOGY:
a) Glaucoma
b) Lesions at the margin of disc e.g.drusen or melanoma

3. ALTITUDINAL DEFECT



AETIOLOGY:
Lesion must be in the retina or optic nerve
a) Branch occlusion of first order retinal artery
b) Retinal detachment
c) Ischaemia of optic nerve head eg.Temporal arteritis

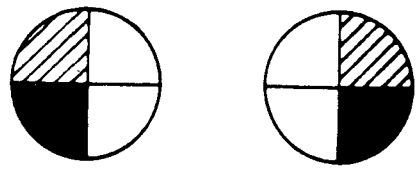
4. BITEMPORAL QUADRANTANOPIA OR HEMIANOPIA



AETIOLOGY:
Lesion pressing on the optic chiasm from below - usually a chromophobe adenoma

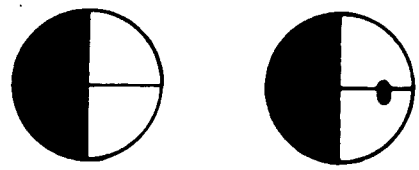
note: Altitudinal visual field defects generally are of vascular origin (retina and optic nerve). Vertical visual field defects generally are of neurological aetiology.

5. BITEMPORAL QUADRANTANOPIA OR HEMIANOPIA (INFERIOR)



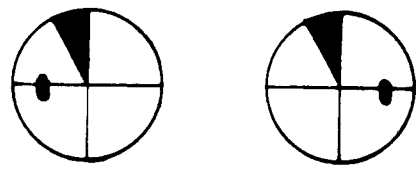
AETIOLOGY:
Lesions pressing on the chiasm from above:
a) Craniopharyngioma
b) Meningioma
- olfactory groove
- lesser wing of sphenoid
- tuberculum sellae
c) Glioma of frontal lobe
d) Aneurysm of int.carotids
e) Dermoid
f) Chordoma

6. HOMONYMOUS HEMIANOPIA - COMPLETE



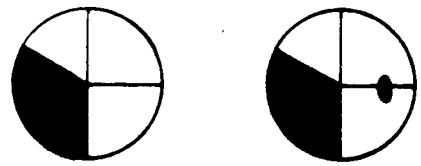
AETIOLOGY:
Lesions of the optic tract, Lateral geniculate nucleus:
a) Tumour
b) vascular lesions
+/- macular sparing

7. HOMONYMOUS HEMIANOPIA - SECTOR OR QUADRANTIC (SUPERIOR)



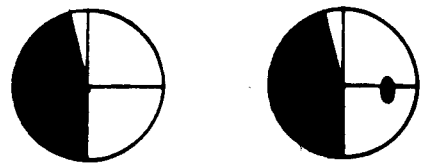
AETIOLOGY:
Temporal lobe (geniculo-calcarine pathway)
Symptoms: Loss of memory hallucinations, déjà-vu phenomena

8. HOMONYMOUS HEMIANOPIA QUADRANTIC (INFERIOR)



AETIOLOGY:
Lesion in parietal lobe (geniculo-calcarine pathway)
Signs: Positive optokinetic sign,dominant or non-dominant parietal lobe signs

9. HOMONYMOUS HEMIANOPIA



AETIOLOGY:
Lesion in occipital lobe
Symptoms:
a) very congrous
b) Patient is alert
c) 20% have macular sparing

B. EXTRAOCULAR MUSCLE PROBLEMS CAUSING DIPLOPIA AND STRABISMUS.

DIPLOPIA

Questions to ask the patient:

- When was the onset?
- Is it worse looking to the right or left, up or down?

The patient's first complaint often may be simply blurred vision and the diplopia is not observed. An acute observer will cover one eye and then note that the vision is improved.

Diagnostic possibilities:

If diplopia is vertical - usually IVth nerve palsy.

If diplopia is horizontal:

1. VIth nerve palsy - uncrossed diplopia (eyes convergent)
2. IIIrd nerve palsy - crossed diplopia (eyes divergent).

If patient walks in with one eye turned out and NO diplopia, one may assume:

- It is probably not a neurological problem.
- If it has a neurological basis, it is long-standing (check photographs of patient).
- It may be due to an old strabismus.

Special points

1. Sudden onset of diplopia or ptosis with severe headache should be considered diagnostic of an intracerebral aneurysm until proven otherwise.
2. Sudden onset of ptosis or diplopia always deserves a Tensilon (edrophonium) test for myasthenia gravis.

THIRD NERVE PALSY supplies all except lateral rectus + superior oblique

A. Clinical findings of a total IIIrd nerve palsy:

1. Ptosis, due to weakness of the levator muscle.
2. Abduction, due to the unopposed action of the lateral rectus.

3. Intorsion of the globe on attempted downgaze, due to the action of the superior oblique.

4. Limitation of depression, due to weakness of the inferior rectus.

5. Limitation of adduction, due to weakness of the medial rectus.

6. Limitation of elevation, due to weakness of the superior rectus.

7. A fixed and dilated pupil, due to interruption of the parasympathetic supply to the sphincter.

8. Weakness of accommodation, due to interruption of the parasympathetic supply to the ciliary muscle.

B. Aetiology:

1. Diabetes - This usually spares the pupil and resolves in 4+ weeks.

2. Aneurysm - This usually involves the pupil, is painful and is usually permanent

3. Demyelination

4. Trauma

5. Syphilis

6. Tumour

7. Miscellaneous eg. sinusitis, TB

C. Anatomical Diagnosis

1. Nuclear

2. Dorsal fascicular (Benedikt's Syndrome) due to damage to the dorsal part of the fasciculus as it passes through the red nucleus. This gives rise to an ipsilateral IIIrd nerve palsy and a contralateral ataxia and flapping tremor.

3. Ventral fascicular (Weber's Syndrome) is to damage involving the ventral part of the fasciculus as it passes through the cerebral peduncle. This is characterised by ipsilateral IIIrd nerve palsy with a contralateral hemiplegia due to involvement of the corticospinal tract.

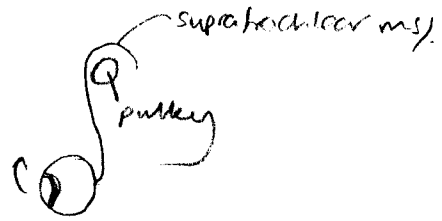
4. Basilar. The IIIrd nerve passes between the posterior cerebral artery and the superior

cerebellar artery. It then passes lateral to and parallel with the posterior communicating artery. Isolated IIIrd palsies are frequently basilar in origin and most often are due to aneurysms, a tentorial pressure cone, or Diabetes.

5. Cavernous - usually associated with other cranial nerve palsies.

6. Orbital - usually associated with other cranial nerve palsies including damage to the optic nerve.Additional symptoms include decrease in visual acuity and proptosis

FOURTH NERVE PALSY



A. Clinical findings

1. Vertical diplopia

2. Weakness in downward gaze with the eye adducted

3. Head tilt to the opposite shoulder

(Look for fourth nerve paralysis in children with torticollis).

B. Aetiology

1. Occurs with many of the conditions that cause paralysis of third nerve (diabetes, aneurysms, lesions of cavernous sinus, etc.)

2. Lesions peculiar to the fourth nerve:

a) Trauma about the superior orbital rim involving the trochlea.

b) Fourth nerve paresis is the commonest cranial nerve sequela of a closed head injury.

SIXTH NERVE PALSY lateral rectus.

A. Clinical Findings

1. Weakness in abduction of the eye.

2. Turning of the head towards the paralysed muscle.

3. Diplopia worse for distance than for near.

4. In recent palsies, the strabismus is greater with the paralysed eye fixing than with the non-paralytic eye fixing. (Secondary deviation is greater than primary deviation).

B. Aetiology

1. Occurs with many of the conditions that cause third nerve and fourth nerve palsies. Diabetes, Aneurysms.

2. Basal meningitis, sarcoidosis.

3. Raised intracranial pressure - the sixth nerve is stretched over the sharp petrous temporal bone.

4. Gradenigo's syndrome (osteitis of the petrous tip).
Symptoms: Diplopia (sixth nerve)
pain (fifth)
deafness (eighth)

5. Metastatic carcinoma

6. Wernicke's encephalopathy (thiamine deficiency).

7. Cerebellar pontine angle tumours (acoustic neuroma). Fifth, seventh and eighth nerves also may be involved. Beware of patient with unilateral deafness and severe intractable corneal ulceration on the same side.

8. Millard-Gubler syndrome - lesion within the pons causing ipsilateral sixth nerve palsy and pyramidal tract signs on the contralateral side.

SUPRANUCLEAR PALSY Palsy due to ~~abor~~ (brain) cortical defects.

A. Clinical findings:

These defects of ocular movement are always conjugate, therefore:

1. Defects of motility are symmetrical

2. There is no diplopia

B. Anatomical Diagnosis:

1. Internuclear ophthalmoplegia - the lesion is in the medial longitudinal fasciculus and causes weakness of the medial rectus on conjugate lateral gaze, but preservation of medial rectus function on convergence.There is thus weakness of adduction often combined with nystagmus of the abducting eye.

   Aetiology:

   - If bilateral, it is usually due to Multiple sclerosis
   - If unilateral, it may be due to occlusion of a small branch of the basilar artery

2. Parinaud's syndrome - due to a lesion in the area of the superior colliculus, usually a pinealoma.

   The findings are:

   a) Paralysis of upward gaze

   b) Pupils which do not react to light but react to accommodation i.e. light-near dissocation

   c) Convergence - retraction nystagmus

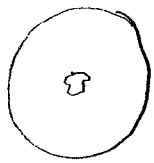
ABNORMALITIES OF THE PUPILS

A. ARGYLL ROBERTSON PUPIL

Aetiology:

1. Syphilis (heart murmur + A R pupil)
2. Alcoholism
3. Encephalitis
4. Diabetes

Signs:



prostitute
: will accomodate
: won't relax.

1. Good vision
2. Meiosis
3. Irregular pupil margin
4. No reaction to light } Light - near
5. Reaction to accommodation } ~~dissociation~~

B. ADIE'S PUPIL (Holmes-Adie, tonic pupil)

1. Slow constriction of the pupil with accommodation or light stimulus.
2. Once pupil is constricted it dilates slowly.
3. Accommodation is often affected - patient complains of blurred vision for near.
4. Constricts to a dilute solution of a miotic which has no effect on a normal pupil.

3. AMAUROTIC PUPIL (blind eye)

1. Does not react to direct light.
2. Reacts to consensual light stimulus.
3. Peservation of near reflex (consensual accommodation).

4. HEMIANOPIC PUPIL (Wernicke)

1. Occurs in homonymous hemianopia.
2. Light from side of the hemianopia gives a lesser response than light from opposite side.

5. HORNER'S SYNDROME (interruption of sympathetic supply to the pupil)

Aetiology:

a) Congenital
b) Idiopathic
c) Tumour in neck region, and apex of lung (pancoast syndrome)
d) Any lesions from the hypothalamus to the superior cervical ganglion to the cavernous sinus.

Signs:

a) Ptosis
b) Meiosis
c) Anhydrosis (lack of sweating)
d) Apparent enophthalmos

If the iris of the involved eye is a lighter colour, it almost certainly is congenital Horner's syndrome.

Response to Mydriatics:

	At rest	Cocaine 4%	Adrenaline 0.1%
Normal pupil	normal	dilates	normal
Horner's pupil	miotic	miotic	dilates

NYSTAGMUS

Definition: A disturbance of ocular posture characterised by involuntary, rhythmic, and fully co-ordinated oscillations of the eyes.

A. Pendular (ocular) nystagmus

1. Due to impairment of central visual acuity

   - If onset of impairment is before the age of two years, nystagmus is almost always present.
   - If onset is between age two and six years unsteady nystagmus, which is not marked, is seen.
   - If onset is after age six, no nystagmus occurs.

2. Miner's nystagmus - due to decreased illumination for long periods.

B. Jerky nystagmus

1. Optokinetic nystagmus

- Normally occurs when one watches telephone poles from a moving car or train window. It can also be produced by moving a tape or drum with stripes on it in front of the patient.

- Can be used to check visual acuity in malingerers or children (by decreasing the width of the stripes).

2. Vestibular nystagmus (horizontal or rotary)

Aetiology

a) Labyrinthitis
b) Multiple sclerosis
c) Syringobulbia
d) Encephalitis
e) Alcoholism (Wernicke's encephalopathy)
f) Cerebellar-pontine angle tumour
g) Barbiturates or Epanutin

Patients who are on drugs will often exhibit a horizontal nystagmus in right and left gaze and also a vertical nystagmus in upward gaze.

3. Neuromuscular deficiency (end-point nystagmus)

In many normal individuals when the eyes move into an extreme position of gaze a few beats of nystagmus may occur.

4. Latent nystagmus

This occurs when one eye is occluded, and is often associated with congenital esotropia.