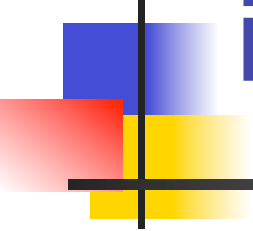


VI nerve palsy Clinical Approach and Treatment in adult population



Dr. Lieve Van Eeckhoutte

Anatomy of nervus VI

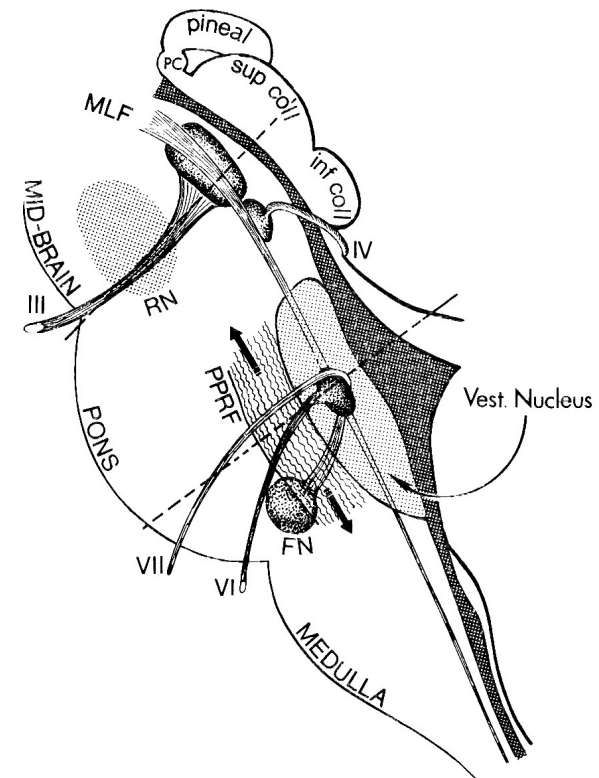
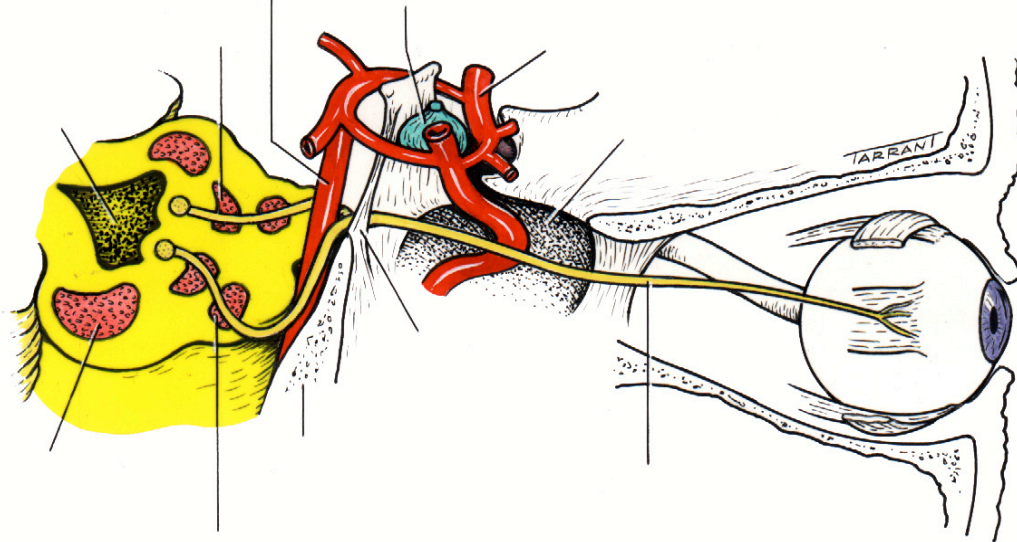
- **Nucleus**

- caudal portion of pontine tegmentum beneath floor 4th ventricle

- n VII fibers loop around VI

- MLF passes medial of VI

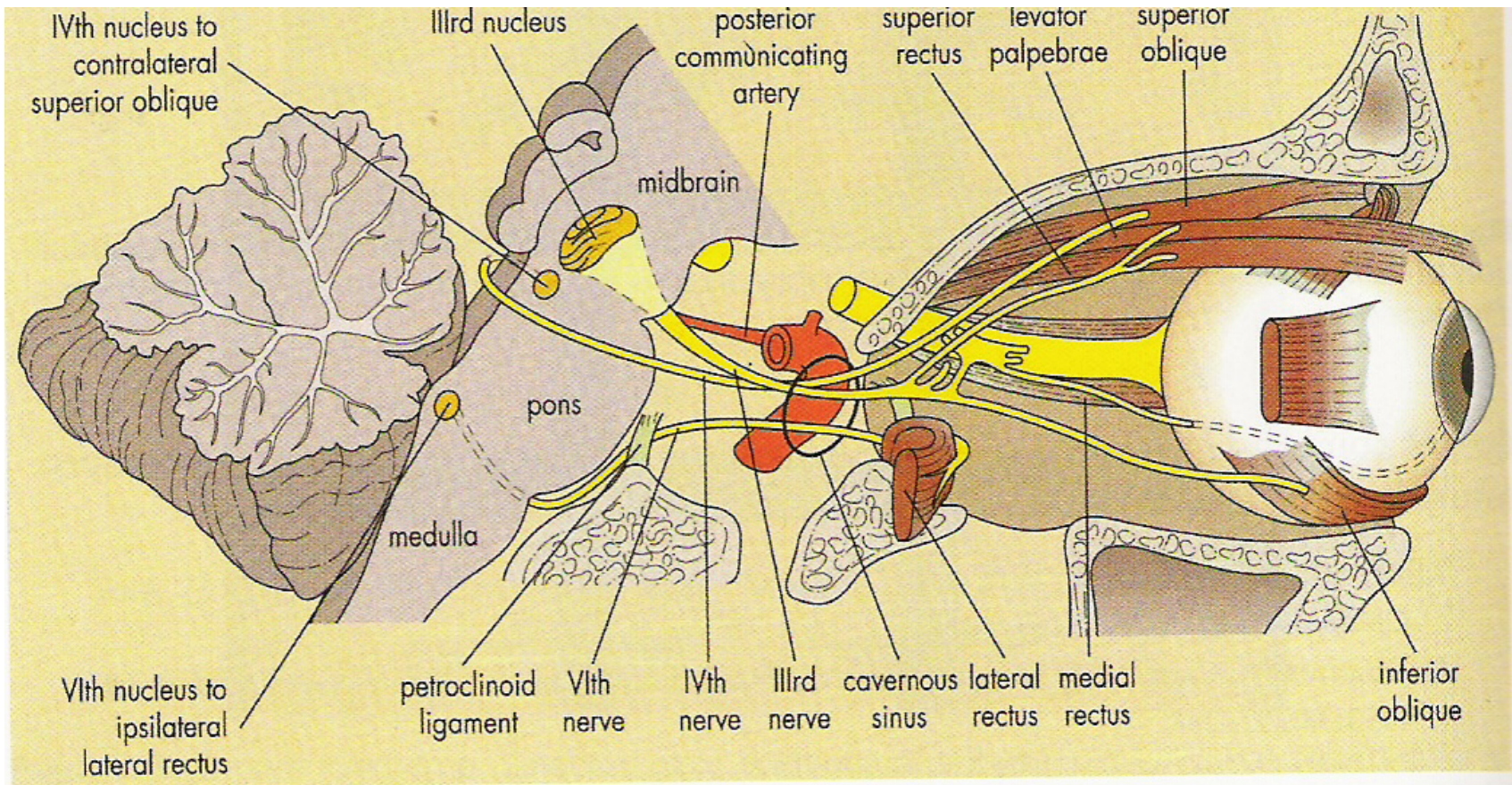
- **Medial longitudinal fasciculus (conjugate horizontal gaze)**



**Basilar: ventral face of the pons
pierces dura of the clivus, runs beneath the petroclinoid
ligament**

Sinus Cavernosus : VI lies freely within the body

Orbita : superior orbital fissure





Etiology VI palsy

All lesions on the path of the long tortuous course of the VI (intracerebral to intraorbital)

- Neoplasm, infection, trauma, neurologic disorders, ...
- Vasculopathies frequently > 50 y
- Isolated VI or multiple cranial nerve palsies

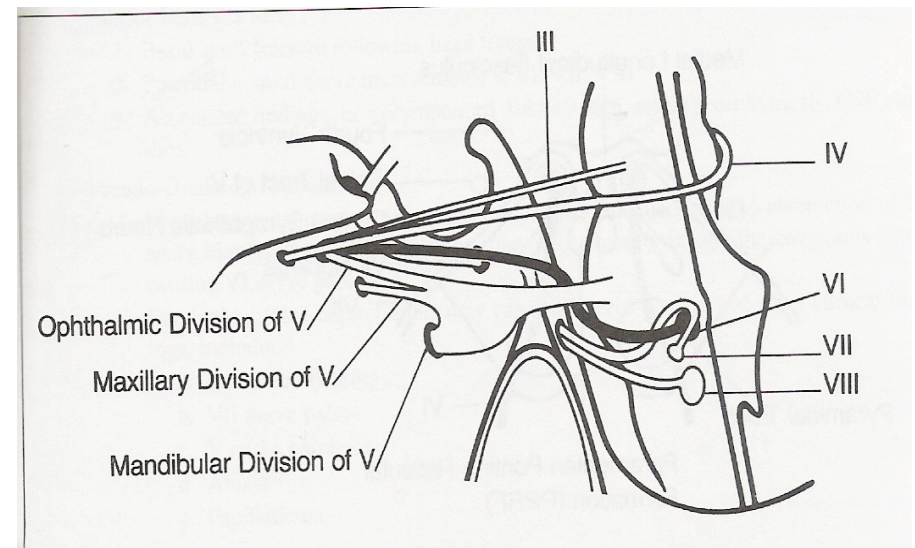


VI and intracranial pressure

- Downward movement of the brain stem
- As the VI ascends the clivus in the subarachnoid space it is vulnerable
- Uni or bilateral VI
- Causes:
neoplasms, insults, infection, trauma,
benign ICH
- Symptoms: headache, nausea, vomit,
papilledema, visual disturbance

VI and VII, VIII, V

- VII : facial palsy
- V : cornea hypoesthesia, facial paresthesias, eye or facial pain
- VIII : loss of hearing, deafness, vestibular symptoms





VI and apex petrosus syndrome (= Gradenigo syndrome)

- Involvement of VI in combination with:
 - VII (facial palsy)
 - V (facial or eye pain)
 - VIII (loss of hearing)
- Cause: inflammation of the petrous bone secondary to middle-ear infections



VI with V, VII and VIII

- Other causes
 - Acoustic neurinoma
 - Meningioma
 - Nasopharyngeal tumor : proliferation through basal foramina (nosebleeding, nose obstruction)



VI and aneurysma a. carotis interna intracavernous

- VI lies central in the sinus cavernosus ,
not in the wall
- Combination with ipsilateral Horner
- Slow progressive unilateral
ophthalmoplegia
- May become painful
- May rupture (fistel, rarely
subarachnoidal bleeding)

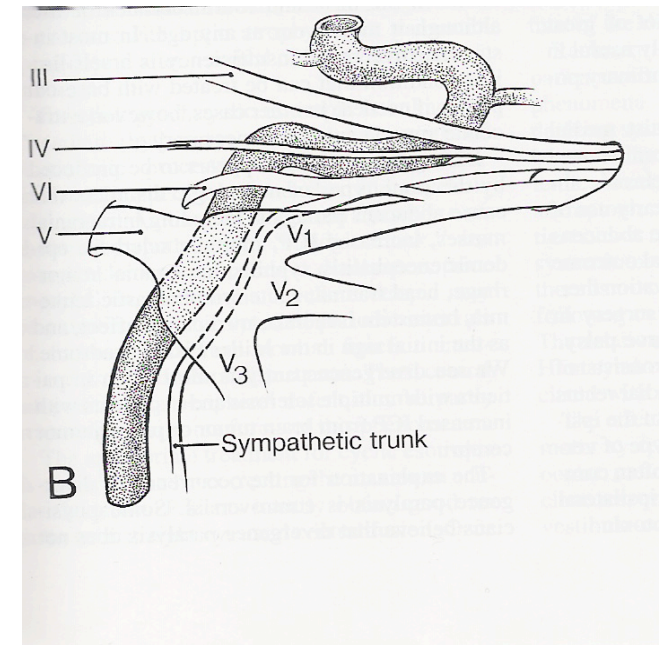
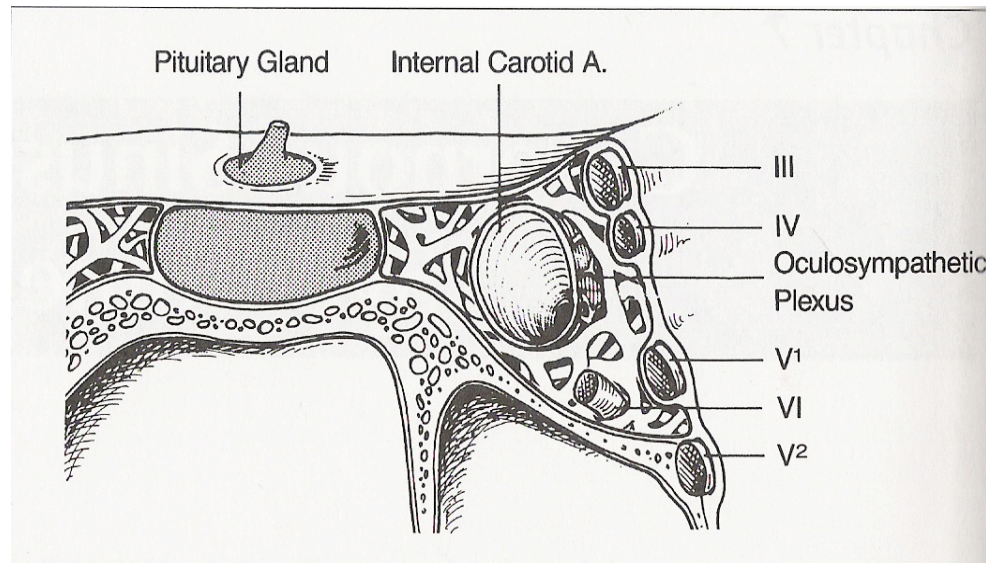


VI and carotid-cavernous fistulas

- Spontaneous dural shunts
- Frequently in elderly people
- VI paresis, sometimes painful, ocular tension, red eye, tortuous blood vessels, proptosis, postauricular noise
- Sometimes spontaneous recovery

Cavernous sinus thrombosis

- VI may be the first sign
- III (ptose) ,IV , V1ophthalmic trigeminus (pain)
- Horner





Cavernous sinus thrombosis

- Etiologie:
 - 70% neoplasm
 - vascular (aneurysma, fistulas)
 - inflammation (infectieus,
non-infectieus = Tolosa-Hunt)
 - trauma



Isolated VI nerve palsy

- Peripheral microvascular ischemic lesion (vasa nervorum)
- Vascular risk factors (diabetes, hypertension, cholesterol)
- Acute palsy (in 7-10 days)
- No other neurological signs 1 month before and 4 months after onset
- Sometimes pain
- Recovery within 3-6 months



Clinical characteristics

- Complaint of horizontal diplopia
far > near
- Esotropia (incomitant)
- Limitation of abduction
- Compensatory face turn if meaningful
field of binocular single vision

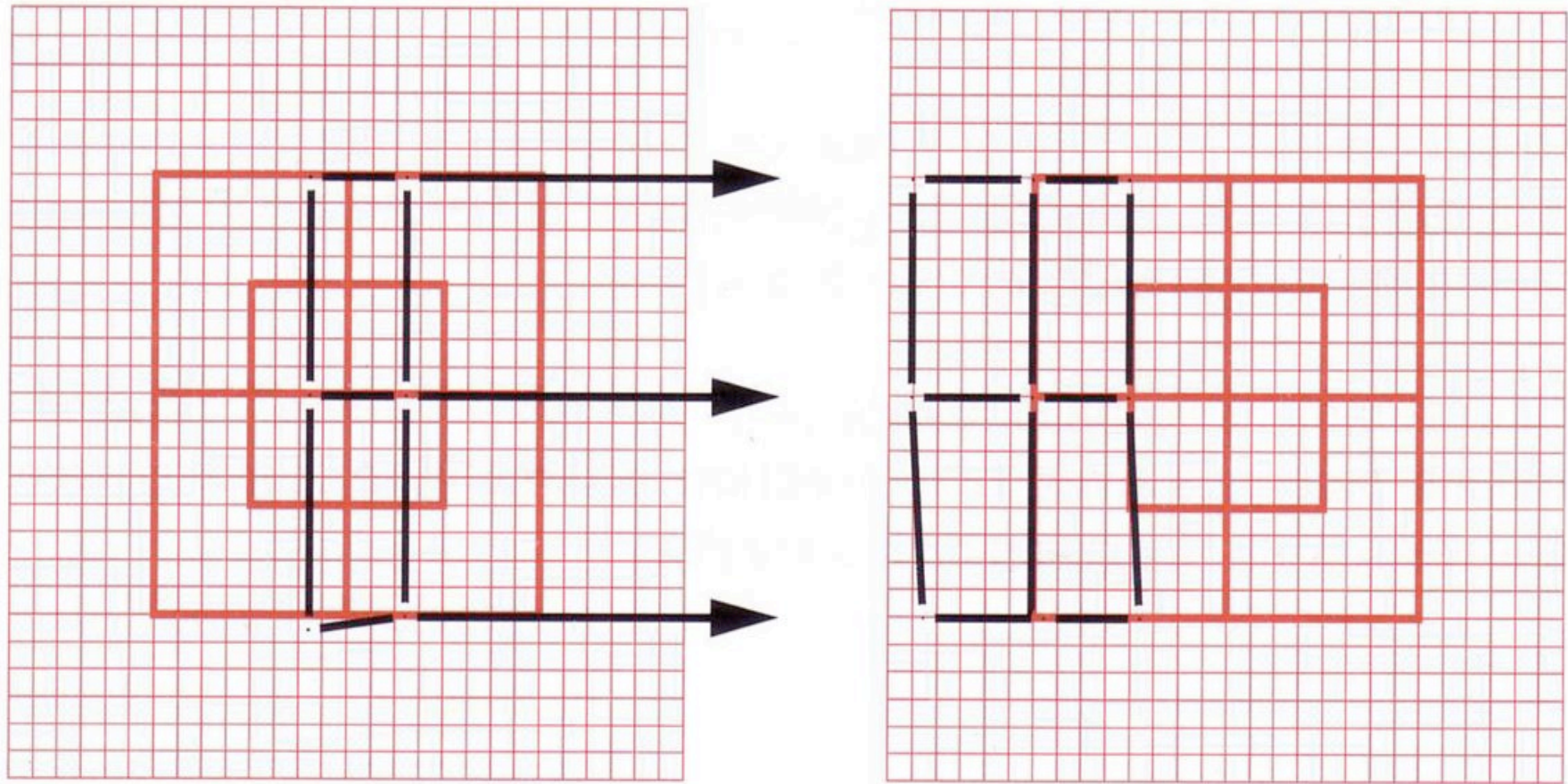


Clinical examination

- Objective: CT, ACT
- Subjective:
 - Maddox rod :even small incomitances will be seen
 - Hess Lancaster

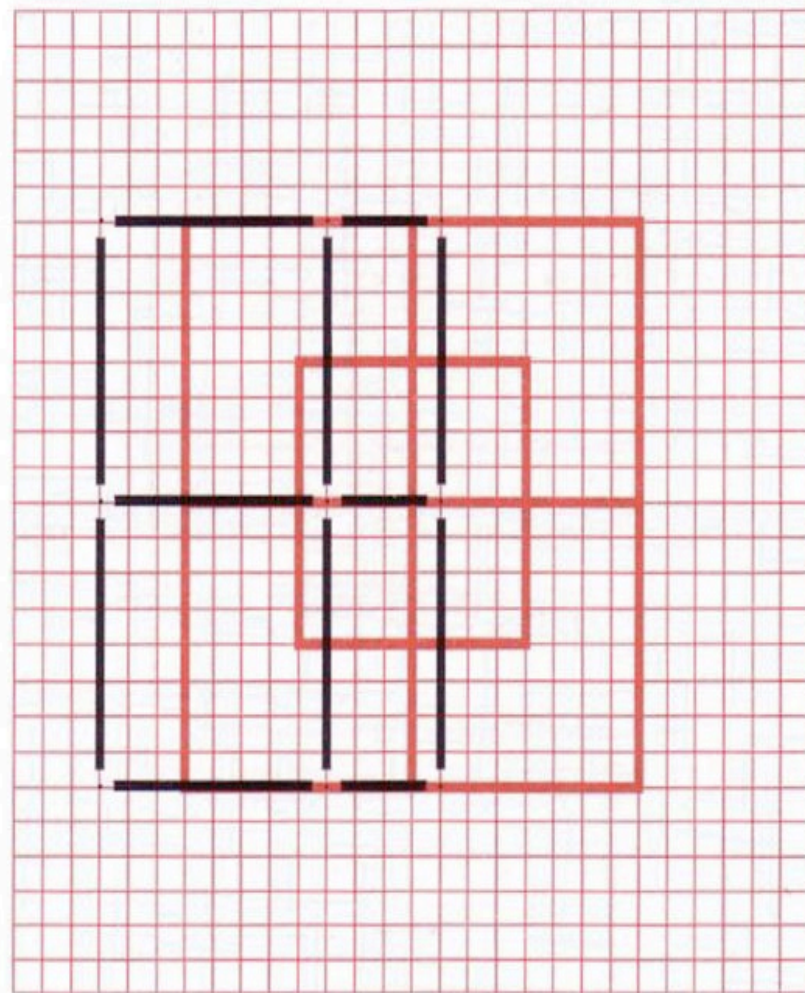
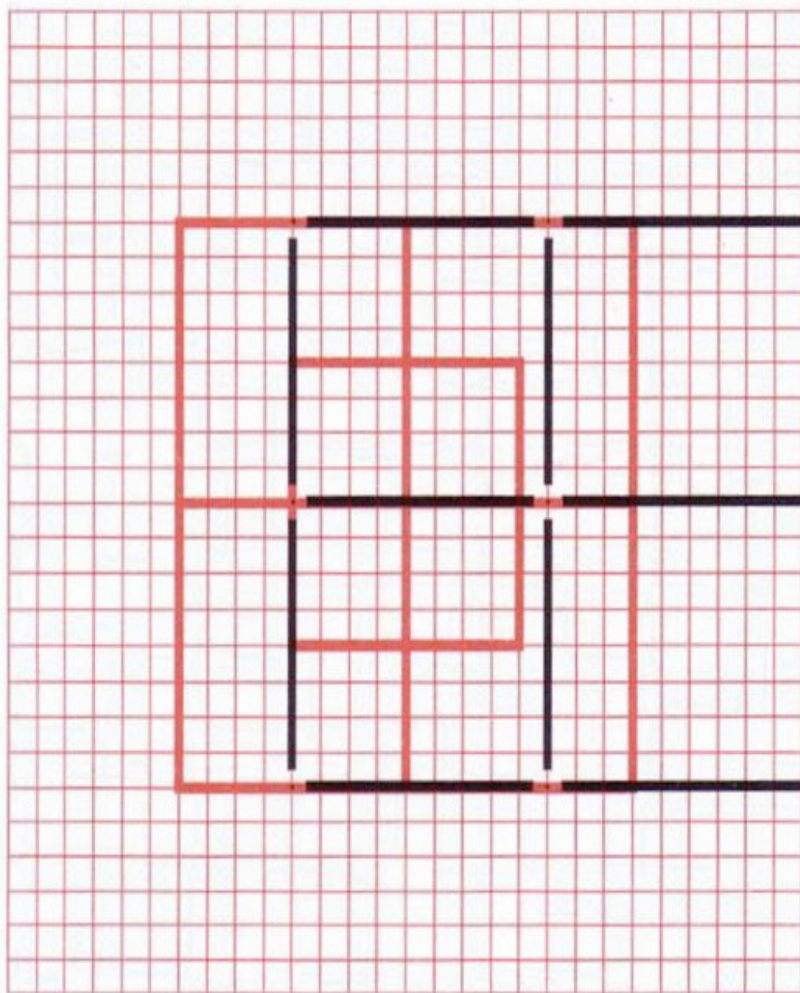


Hess - Lancaster





Hess Lancaster





Paresis versus complete palsy

Observation of abduction:

Abduction past midline = paresis

no abduction past midline : due to either
tight MR or true LR palsy
(in longstanding VI)

forced duction test to evaluate muscle
function



Differential Diagnosis of abduction deficits

- Graves' myopathy
- Myasthenia gravis (tensilon test)
- Orbital pseudotumor myositis
- Orbital trauma (medial rectus entrapment)
- Congenital defects (Duane)



Workup VI palsy

Exclude hypertension

Blood studies :

- diabetes
- lipids
- older than 55y: giant cell arteritis
(erythrocyte sedimentation rate)



Workup VI palsy

Radiologic investigation: CT, MRI, cerebral angiography

- Bilateral or multiple oculomotor paresis
- Other neurological signs (papiledema, nystagmus, hemiparesis)
- Isolated paresis:
 - observation monthly
 - if no recovery in 3 - 4 months



Recovery

- Spontaneous recovery depends on its cause
- Majority of isolated vascular VI palsy recover within 6 months
- Recurrences may occur, usually on the same side



Treatment : nonsurgical

Patching

- Occlusion of the good eye may lead to disorientation and vertigo
- Sectorocclusion: nasal part of the good eye or temporal part of the paretic eye



Treatment : nonsurgical

Fresnel add-on prisms

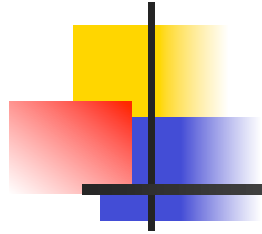
- Only for small deviations $< 15^\circ$
- Only if incomitances are small
- Best in front of the paretic eye
(secondary deviations)



Treatment : nonsurgical

Botulinum toxin injection in MR

- decision will depend on the degree of palsy
- Partial VI with area of binocular vision: no botulinum
- Complete VI : some will use botulinum within two weeks, other if no signs of improvement within a month



Study of botulinum toxin in acute unilateral VI palsy

(Graefes arc clin exp ophthalmol)

70% of patients who refused injection

10% of patients received botulinum
required surgery

Other studies showed no evidence of any difference in outcome between treated and untreated group

Disadvantage of botulinum: crossed diplopia in contralateral gaze, ptose, temporary contraction of the binocular single vision field



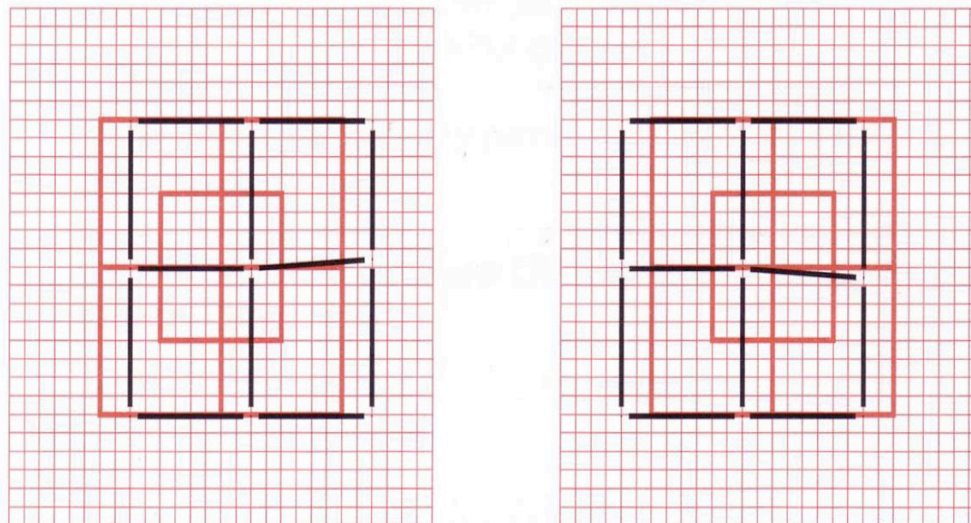
Treatment : Surgical

- Six months delay
- Good preoperative evaluation of the abduction, the incomitances, forced duction test
- Aim: correction of esodeviation, improvement of abduction, increase of size of the diplopia free binocular field

Treatment : Surgical

After recovery of the paresis only an esotropia can persist

- Hess Lancaster: concomitant
- Recession of both MR





Treatment : Surgical

Partial paresis remains

- Hess-Lancaster : incomitance
- Abduction past the midline
- Forced duction perop : MR contracture

Recession-resection of the horizontal muscles



Treatment : Surgical

Complete VI palsy

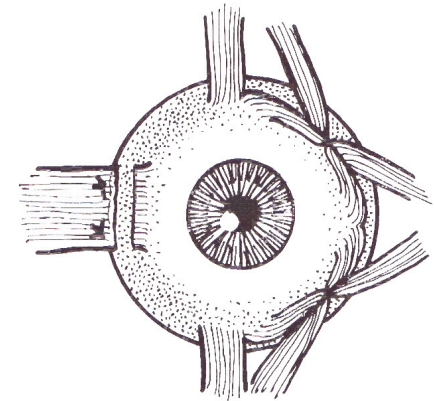
- Not done: Recession of the MR and resection of LR may have a transient mechanical result but a poor long-term alignment

Transposition of the vertical muscles with MR weakening

- Paretic muscle remains undisturbed to preserve blood supply to anterior segment

Transposition SR and IR and recession MR

- Improves the abduction postop
- Risk of anterior segment ischemia if 3 recti are operated in the same time
- Jensen procedure (muscle union)
or a partial muscle transposition
procedure may give undercorrection
- Sparing of the anterior ciliary vessels may be difficult because of the long distance of the transposition





Transposition SR and IR and recession MR

- At UZ Leuven we start with a recession MR and botulinum injection
- When the botulinum is worked out after a few weeks we do the full transposition SR and IR and repeat the botulinum if we observe again a MR contracture



Undercorrection

- Often when rec-res is performed when there was a complete VI palsy
- After transposition : reinjection of botulinum
or recession of the contralateral MR



Overcorrection

- Rare
- After Jenson procedure: difficult to repair
- Slipped MR



Induced vertical deviation

- Induced by the surgery
- Perop: take care of freeing the muscles : SR from superior oblique, IR from the capsulopalpebral attachments
- Some surgeons will reduce the incidence by placing SR and IR on adjustables
- Always be aware of an associated fourth nerve palsy or skew deviation



Conclusion

- Anamnesis (cardiovascular, neurological problems, malignance)
- Check other cranial nerves
- Observe monthly
- Appropriate surgical strategy after stabilisation (6 months)