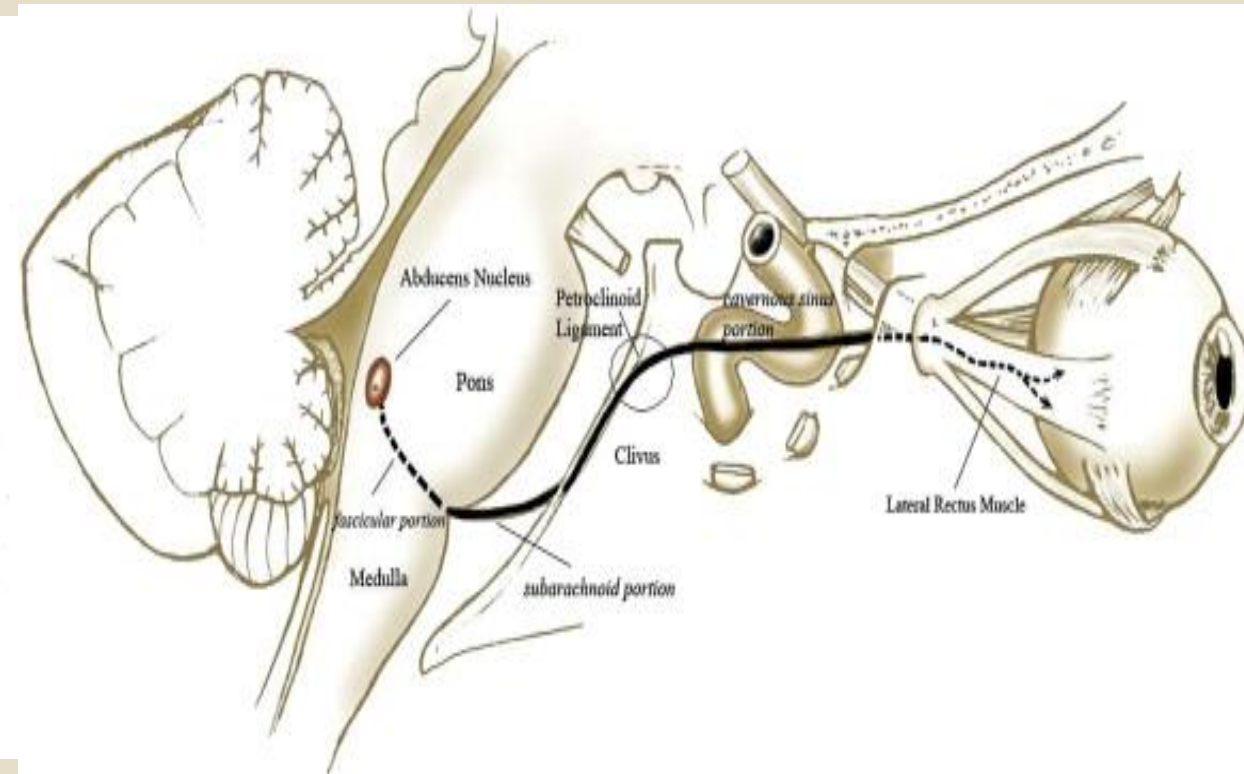
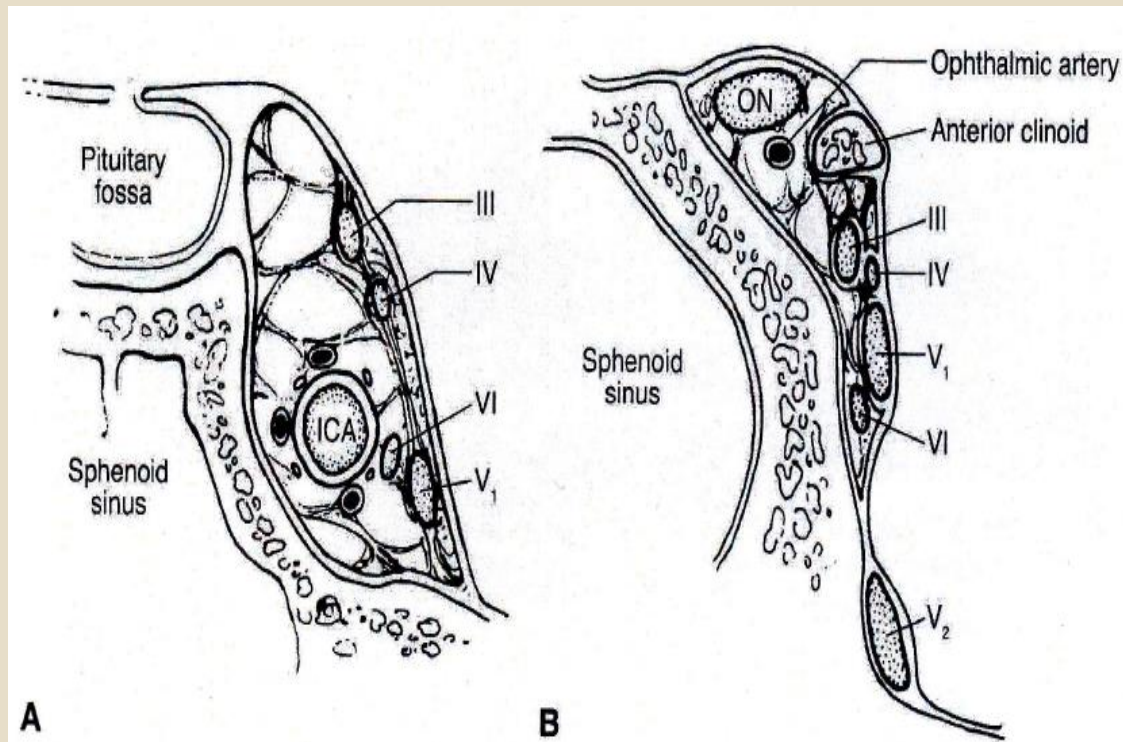




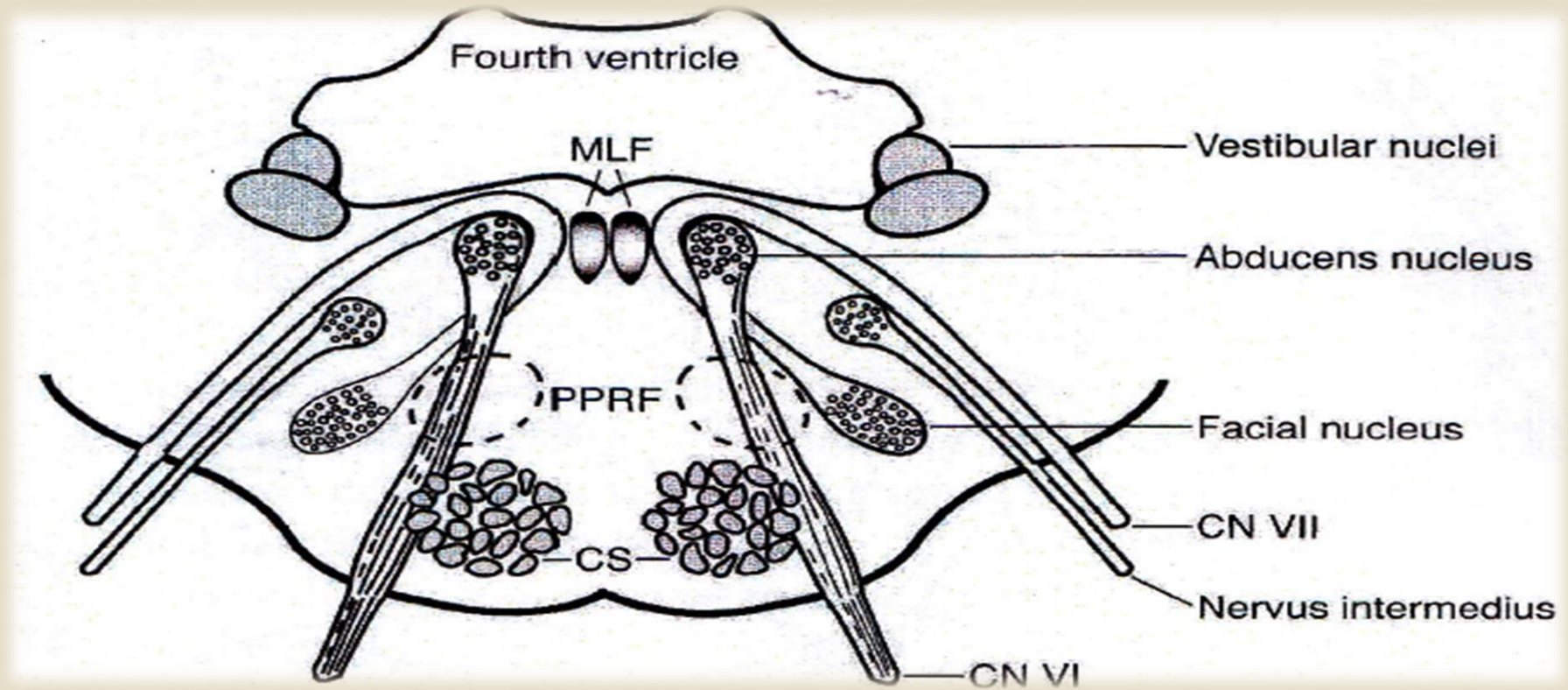
SIXTH NERVE PALSY

PRESENTER: DR.SHEIKHI

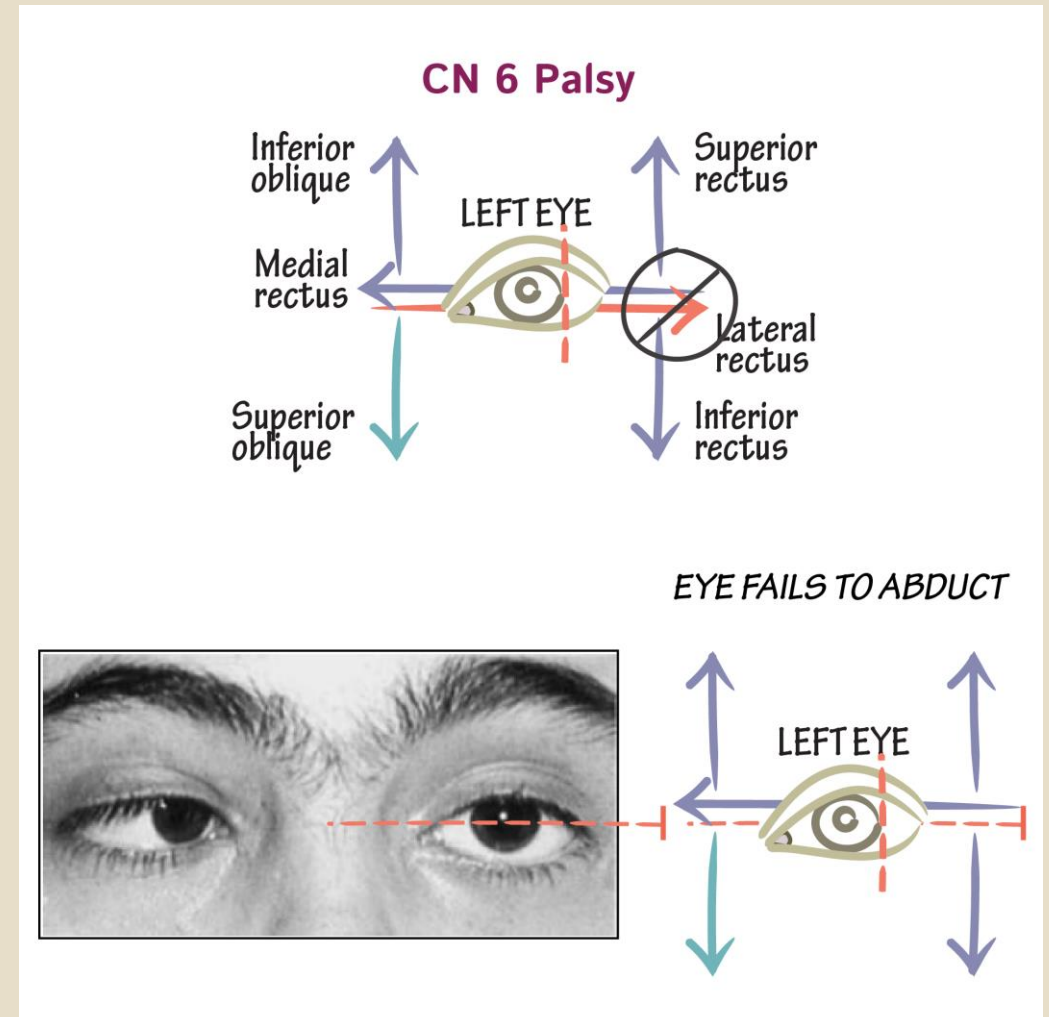
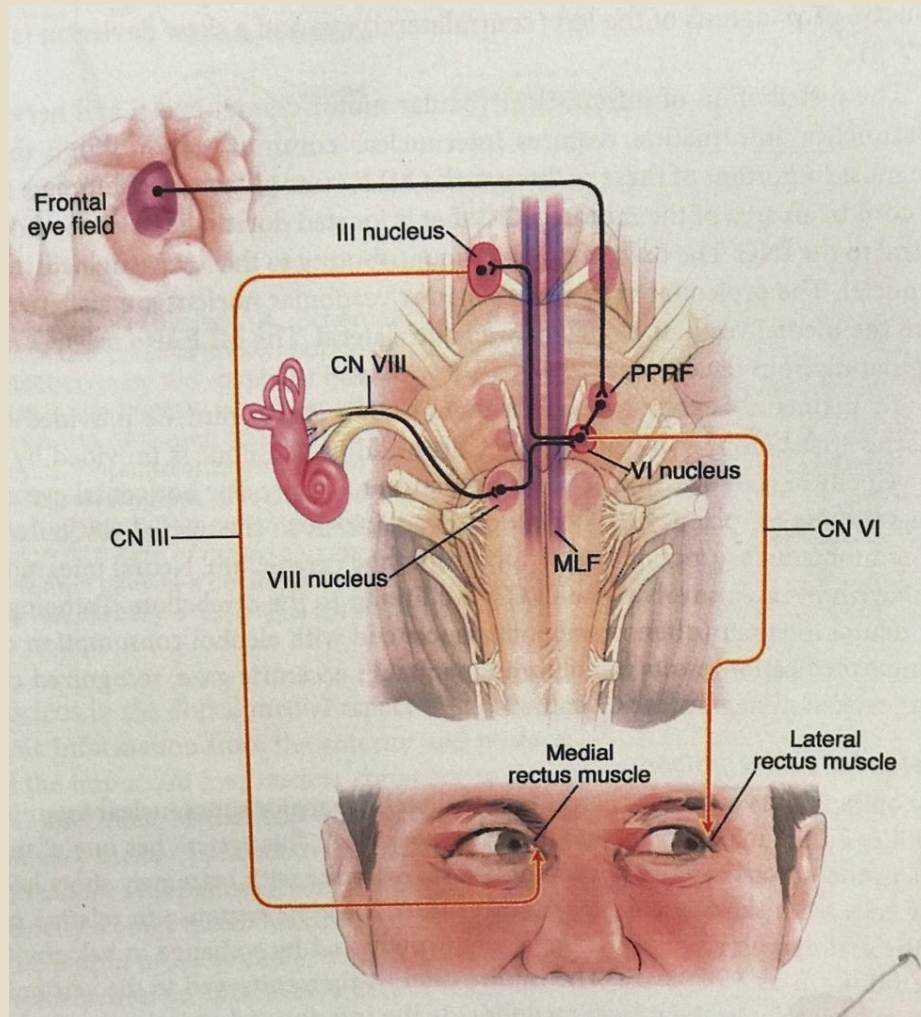
- The abducens nerve emerges from the **brainstem at the pontomedullary junction** to enter the subarachnoid space, coursing upward between the pons and clivus to enter the Dorello canal.
- At the petrous apex, it angulates to enter the cavernous sinus and travels in close proximity to the internal carotid artery.
- The abduces nerve then proceeds through the superior orbital fissure and innervates the lateral rectus muscle.



- Cranial nerve VI, also known as the **abducens** nerve, innervates the ipsilateral **lateral rectus (LR)**, which functions to **abduct** the ipsilateral eye.
- The sixth cranial nerve has a long subarachnoid course. The sixth nerve nucleus is located in the **pons**, just ventral to the floor of the fourth ventricle and just lateral to the medial longitudinal fasciculus (MLF).
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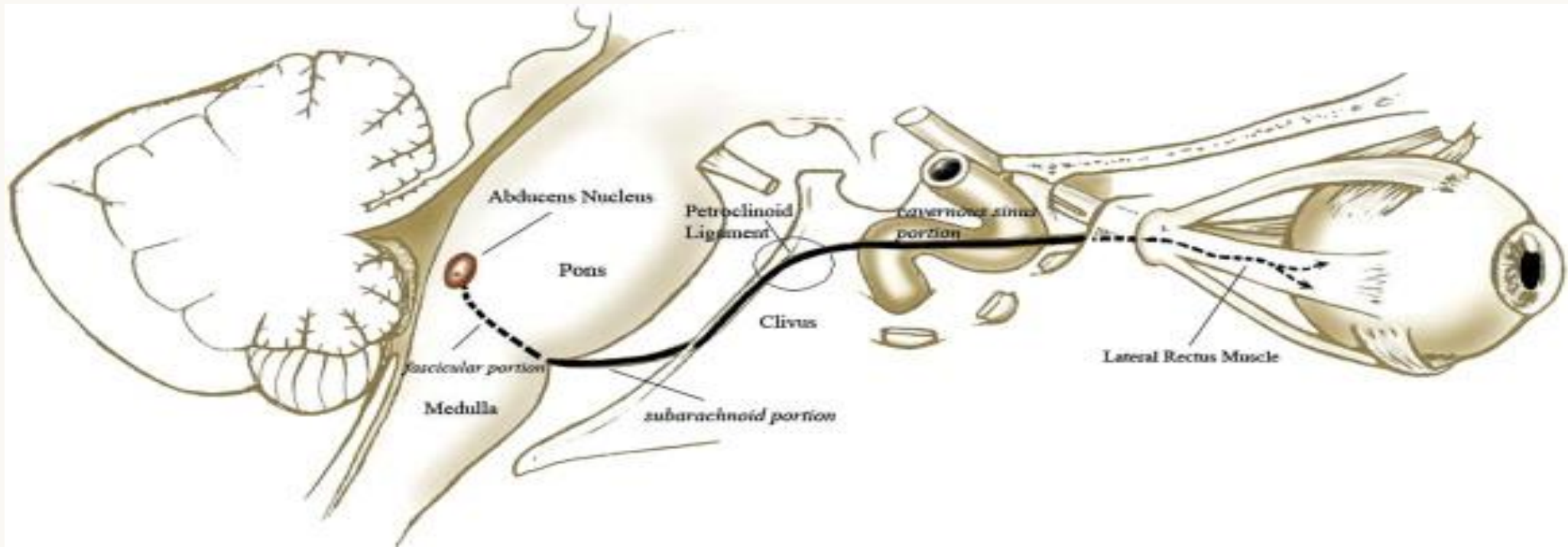


- Patients usually present with **binocular horizontal diplopia** (double vision producing a side-by-side image with both eyes open), worse in the distance, and **esotropia** in primary gaze. Patients also may present with a **head-turn** to maintain binocularity and binocular fusion and to minimize diplopia. Congenital sixth nerve palsy (Duane syndrome) is a well-recognized entity.

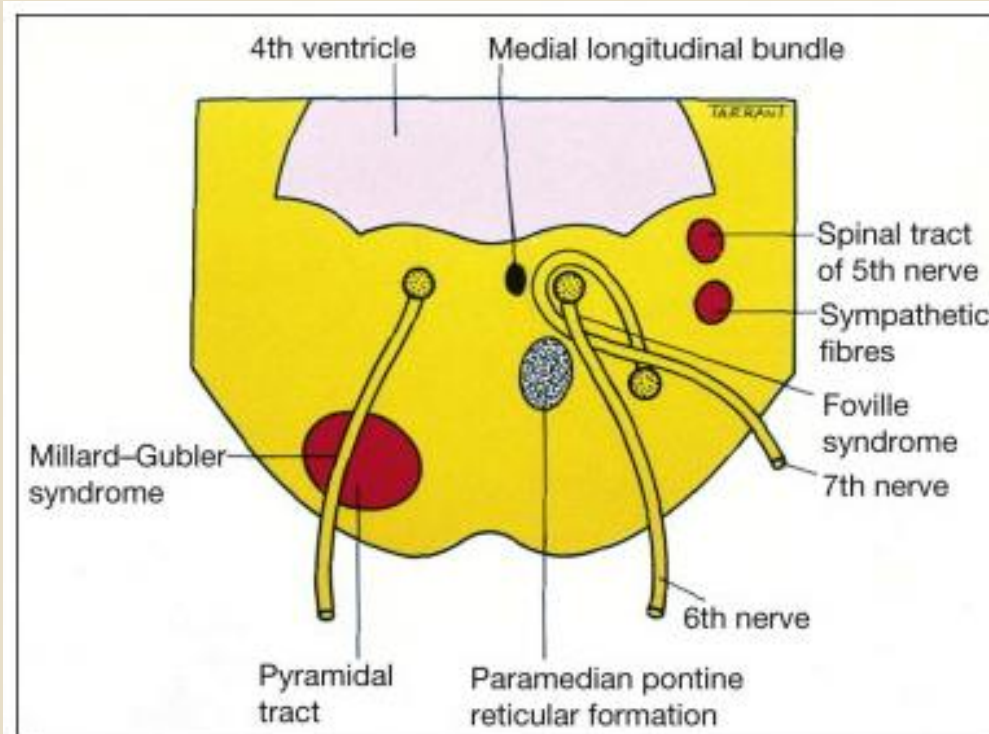


Pathophysiology:

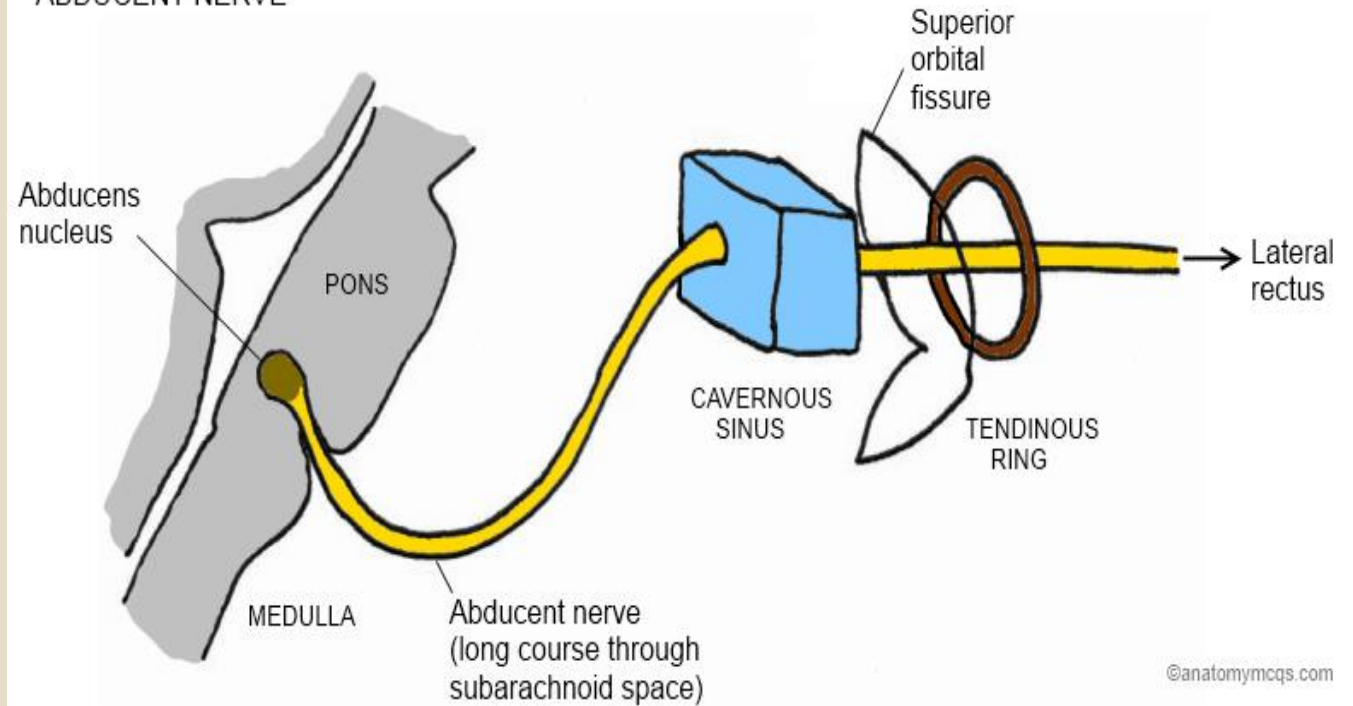
- Only the ipsilateral lateral rectus that is solely innervated by the involved peripheral sixth cranial nerve is affected; therefore, only deviations in the horizontal plane are produced. In isolated cases of peripheral nerve lesions, no vertical or torsional deviations are present.
- Central nervous system lesions of the abducens nerve tract are localized easily secondary to the typical findings associated with each kind of lesion.
- Damage to the sixth nerve nucleus results in an ipsilateral gaze palsy. The lack of a contralateral adduction defect makes it easy to differentiate a nuclear lesion from a fascicular or nonnuclear lesion.



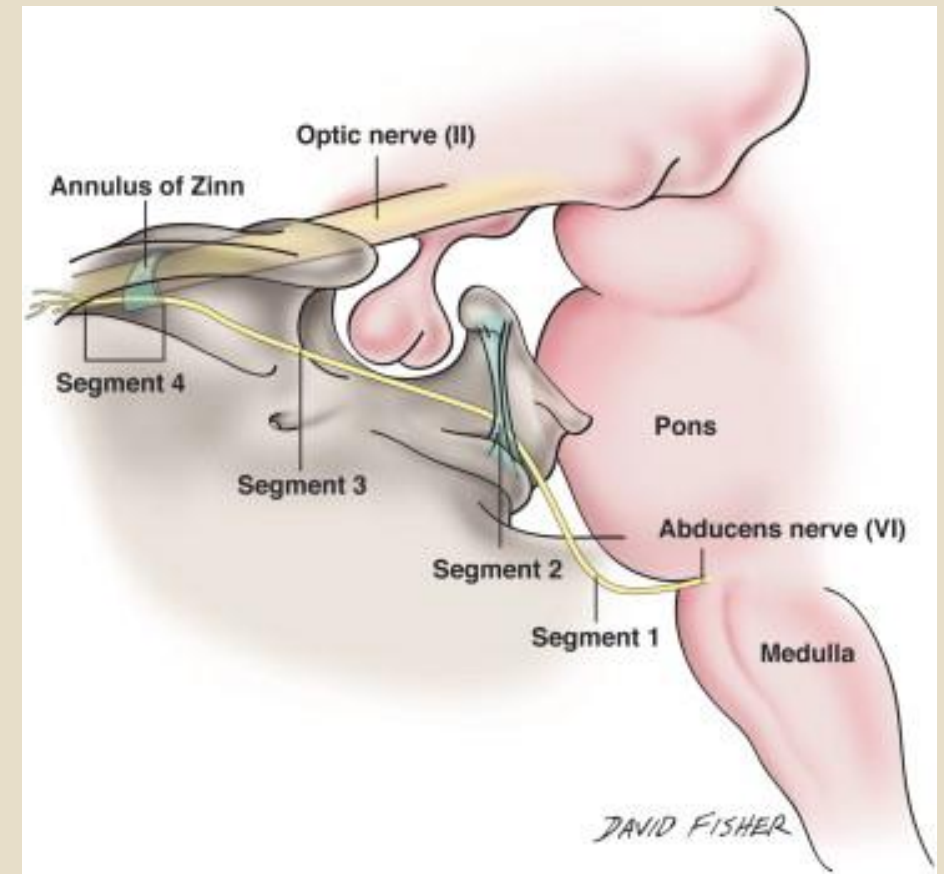
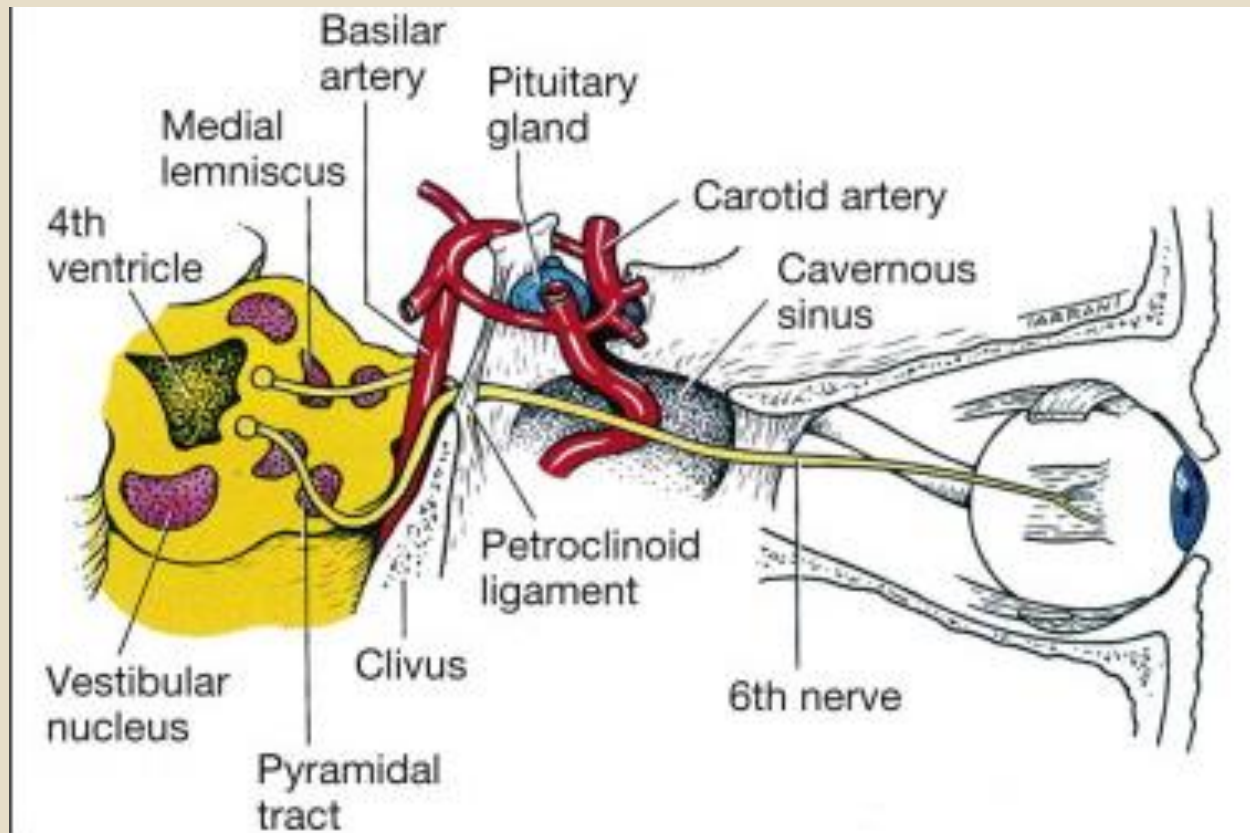
Causes:



ABDUCENT NERVE



- Elevated intracranial pressure can result in downward displacement of the brainstem, causing stretching of the sixth nerve secondary to its anatomic location within the Dorello canal. This is believed to be the reason that about 30% of patients with pseudotumor cerebri have an isolated abducens nerve palsy and explains how lesions remote from the sixth cranial nerve can cause abducens paresis (false localizing sign).
- Subarachnoid space lesions can be causes of abducens nerve palsy (eg, hemorrhage, infection, inflammation, space-occupying tumor, cavernous sinus mass). Inflammatory (eg, postviral, demyelinating, sarcoid, giant cell arteritis



- Vascular
- Metabolic (eg, vitamin B, Wernicke-Korsakoff syndrome)
- Neoplasm (children) - Pontine glioma
- Infectious (eg, Lyme disease, syphilis)
- Congenital absence of the sixth nerve (eg, Duane syndrome)
- Trauma, particularly if it results in a torsional head motion Post-lumbar tap

Epidemiology

Frequency:

- **United States**
- Sixth nerve palsies fall into the following categories: 3%-30% trauma, 0%-6% aneurysm, 0%-36% ischemic, 8%-30% idiopathic, and 10%-30% demyelination/miscellaneous.
- The sixth cranial nerve is the most commonly affected of the ocular motor nerves. In children, it is the second most common after the fourth nerve, with an incidence of 2.5 cases per 100,000 in the population.

Mortality/Morbidity:

A young patient should have an aggressive workup because of the greater likelihood of a neoplasm causing the palsy.

Patients older than 55 years with isolated sixth nerve palsies may require a less aggressive initial workup if they have predisposing microvascular ischemic risk factors, but no history of cancer.

Age

Cranial nerve VI palsy can occur in all age groups; however, the etiology varies depending on the age group.

- Clinical history of abducens nerve palsy includes the following:
 - Binocular diplopia (worse at distance or lateral gaze)
 - Esotropia
 - Head-turn
 - Vision loss
 - Headache, vomiting, pain, or facial numbness
 - Trauma
 - Symptoms of vasculitis, particularly giant cell arteritis
 - Hearing loss

■ Physical findings of abducens nerve palsy include the following:

- An esodeviation that increases on ipsilateral gaze and is often greater at a distance; prism measurements in different positions of gaze can reveal the magnitude of misalignment and its incomitance (asymmetry)
- An isolated abduction deficit
- Slowed ipsilateral saccades
- Papilledema (if increased intracranial pressure)
- Altered sensation in the V1 or V2 distribution with cavernous sinus lesions
- Nystagmus (usually in children, ie, secondary to pontine glioma)
- Otitis media
- Petrous bone fracture
- Tender, enlarged, nonpulsatile temporal arteries in giant cell arteritis
- Horner syndrome (Foville brainstem syndrome, carotid oculosympathetic plexus involvement in cavernous sinus)
- Contralateral hemiparesis may be seen in brainstem syndromes that involve the sixth cranial nerve (Millard-Gubler syndrome and Raymond syndrome)

Differential Diagnoses:

- Giant Cell Arteritis (Temporal Arteritis)
- Medial Wall Orbital Fracture
- Myasthenia Gravis
- Spasm of the near reflex
- Thyroid Associated Orbitopathy with medial rectus involvement

- Check history for diabetes mellitus, cancer, thyroid disease, and hypertension.
- Ask about history of recent trauma, ear infections (children).
- An otoscopic examination may be performed in children to rule out a complicated otitis media (consider an LP).
- Rule out other cranial nerve involvement.

Abducent nerve palsy



Laboratory Studies:

- See the list below:
- Complete blood cell (CBC) count
- Diabetes testing (glucose, glycosylated hemoglobin [HbA1C], glucose tolerance test)
- Erythrocyte sedimentation rate, C-reactive protein, in patients older than 50 years
- Acetylcholine receptor in the presence of variable strabismus or ptosis

- The following are not mainstream tests for abducens palsy but can be considered:

- Rapid plasma reagin test
- Fluorescent treponemal antibody-absorption test
- Lyme titer

- Procedures
- A temporal artery biopsy may be indicated in patients aged 50 years or older with findings and laboratory studies suggestive of giant cell arteritis.



Imaging Studies:

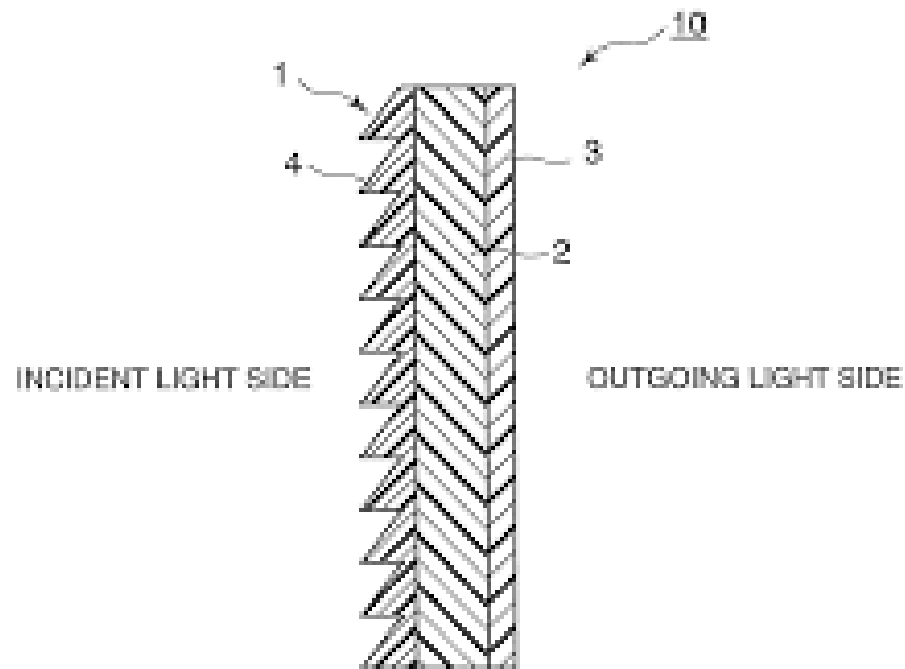
- MRI is indicated for the following:
 - Patients younger than 55 years with no vasculopathic history
 - Associated pain or other neurologic abnormality
 - History of cancer
 - Bilateral sixth nerve palsy
 - Papilledema
 - In the event no marked improvement is seen or other nerves become involved
 - An LP should be considered if MRI results are negative.
 - If a presumed microvascular ischemic sixth nerve palsy does not improve within 3-4 months or if other cranial nerves become involved, a full medical, neurologic, and imaging workup should be performed.

Treatment:

■ Medical Care

- Truly isolated cases of abducens nerve palsy are often benign. They can be followed with a serial examination, at least every 6 weeks, over a 6-month period to note decreasing symptoms (diplopia) and resolution of the paretic lateral rectus (increasing motility).
- Prism measurements are a simple objective method of documenting any changes in the esotropia.
- Children with sixth nerve palsy who are in the amblyopic age group can be treated with an alternating patching to decrease their chances of developing any amblyopia in the paretic eye.
- Additionally, prescribing the full amount of hyperopic correction helps to decrease the esodeviation by relaxing the child's accommodative effort.

- Adult patients and those children beyond the amblyopic age can be patched or have their lenses "fogged" with clear tape or nail polish to reduce their diplopia.
- Fresnel prisms also can be prescribed as an alternative.
- Older patients in whom giant cell arteritis is suspected should start the standard treatment with prednisone or intravenous methylprednisolone.

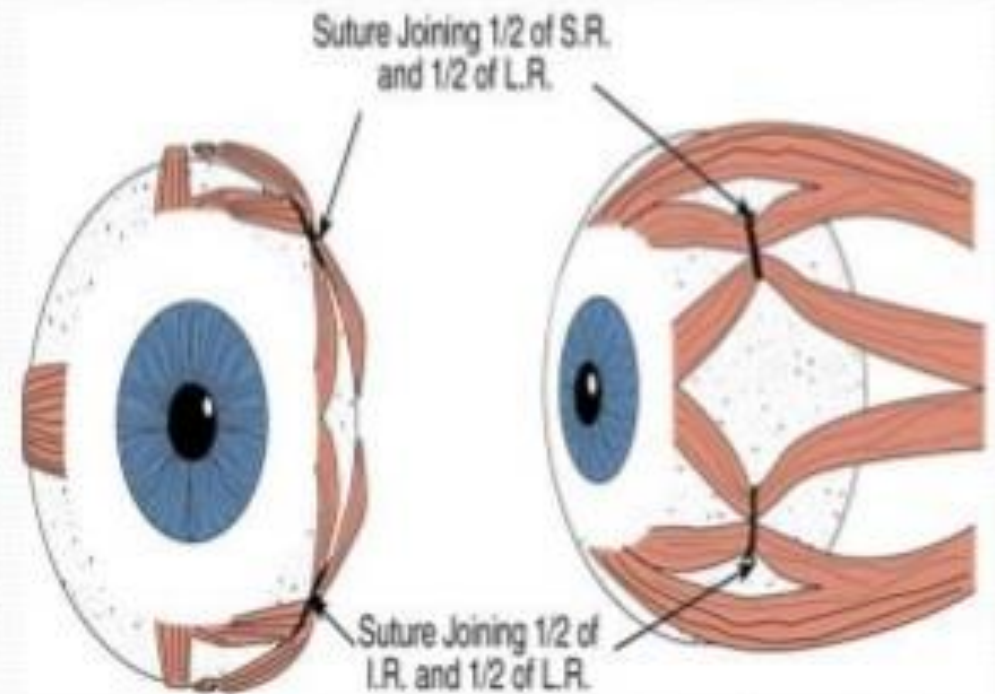


Surgical Care:

- If, after 9-12 months of follow-up care, the remaining deviation is still unacceptable and is too large to be corrected with prisms, surgical corrective options should be discussed with the patient. The procedure that is chosen depends on the remaining function of the lateral rectus and the experience of the surgeon.
- If some residual function exists in the lateral rectus, a graded recession of the medial rectus or botulinum toxin to the medial rectus, and resection of the lateral rectus.
- When little or no residual function is present, a transposition of the vertical recti toward the lateral rectus (eg, Hummelsheim, Jensen, or Nishida procedure), can be considered in conjunction with weakening of the ipsilateral medial rectus.

JENSEN 'S PROCEDURE

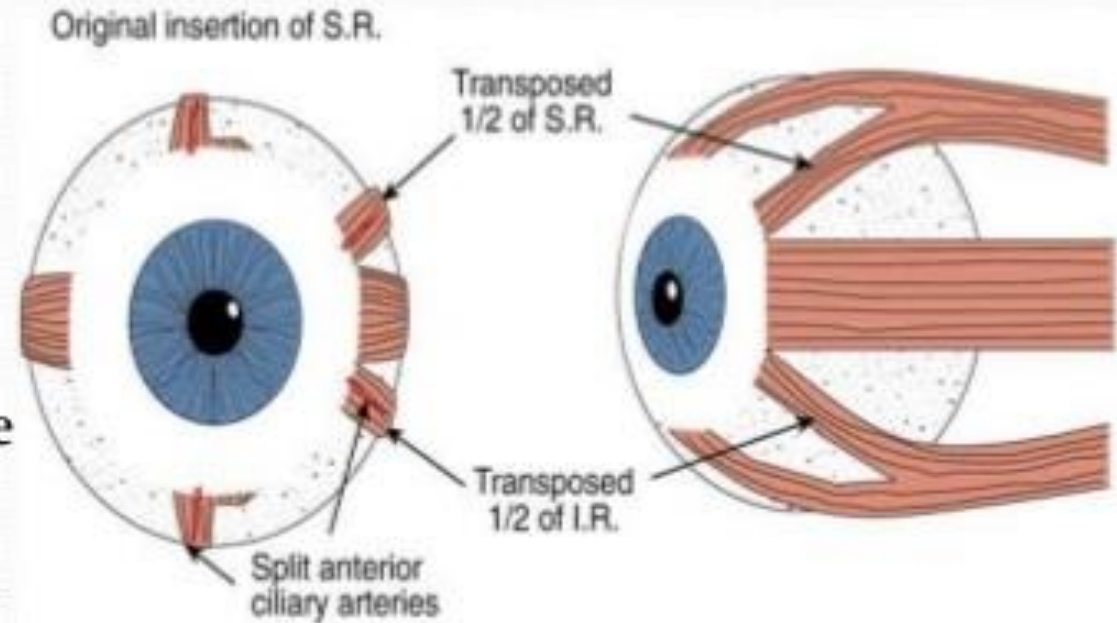
- Indications – Lateral rectus palsy
- Here the adjacent muscles are tied together 12 mm posteriorly, but not disinserted
- Lateral halves of SR and IR are dissected
- Upper and lower halves of LR are dissected
- Lateral half of SR and upper half of LR are sutured
- Lateral half of IR and lower half of LR are sutured



ADVANTAGE – Less chance of A/S ischemia

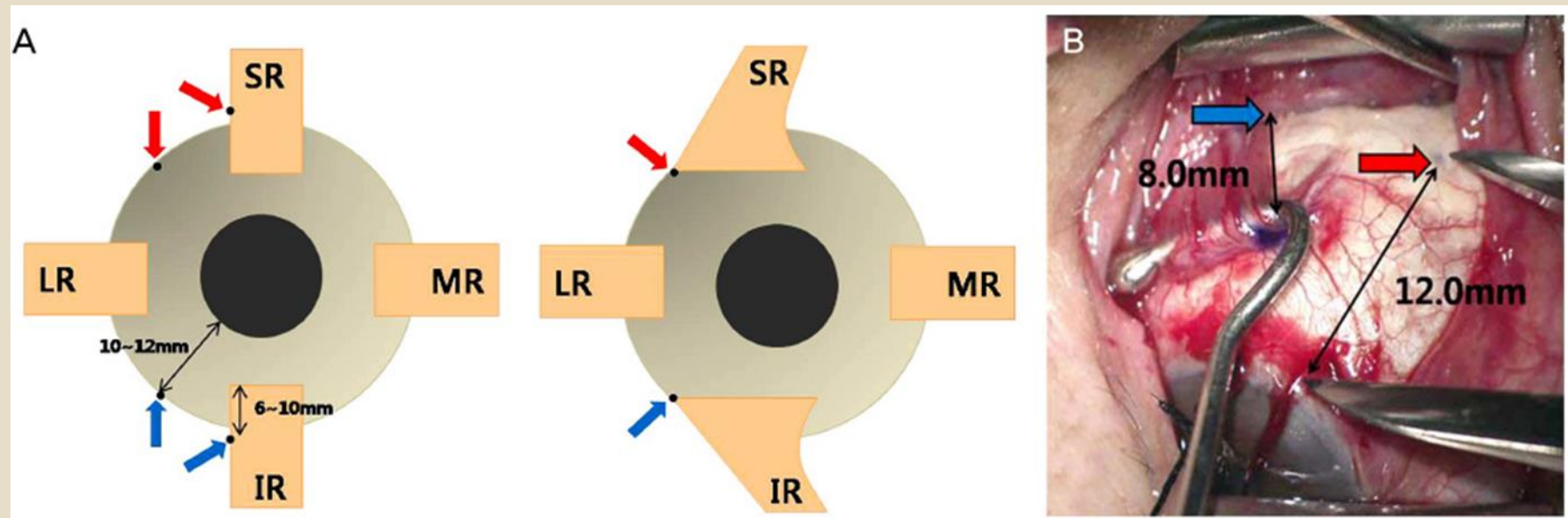
■ Hummelshein procedure:

- It is a split tendon transfer technique to preserve anterior ciliary artery perfusion
- Indications –
 - Lateral rectus palsy
 - Lost medial rectus muscle
- Lateral halves of SR and IR are dissected upto 14 mm from their insertion
- They are reinserted adjacent to LR insertion and they should touch the LR insertion



NISHIDA procedure:

- A 5-0 prolene suture was inserted through each temporal margin of vertical recti at approximately one third of the width from the edge at a distance of 8.0 mm behind the insertion.
- The same suture also was inserted through each scleral wall at a distance of 12.0 mm behind the limbus at the superotemporal or inferotemporal quadrant
- Then, the lateral margin of each vertical rectus muscle was transposed superotemporally or inferotemporally and sutured to the sclera.



Consultations:

- Patients with abducens palsy can benefit from consultation with a neurologist, ophthalmologist, or neuro-ophthalmologist, especially if the lesion does not resolve



■ Activity

- Patients who occlude an eye to alleviate diplopia should be warned that the resulting effects on depth perception may interfere with their ability to drive or perform certain occupations safely.



❑ Prognosis:

Prognosis depends on the underlying causes of the condition.

- SNP caused by viral illness generally goes away completely
- SNP caused by trauma may have residual symptoms
- The greatest improvement generally occurs in the first 6 months
- Most people with idiopathic SNP(of unknown case) completely recover
- Some people may experience permanent vision changes

