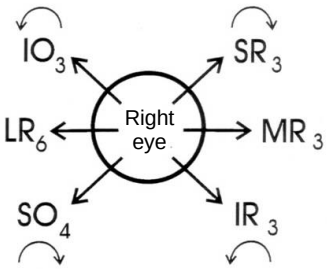
Diplopia and Cranial Nerve Lesions

Anatomy

There are a couple of points of confusion about the extra ocular muscle movements. These are related to the exact insertion points and direction of pull of the muscles related to the axis of the eye (so superior oblique counter-intuitively actually has a component of downward rotation of the eye and inferior oblique the opposite), and the fact that during gazing in most directions multiple muscles are involved and so the contribution of a muscle to the gaze direction may not match its action if anatomically isolated by cutting all the other muscles.

The actions (& innervation) **in isolation** of the extraocular muscles are:

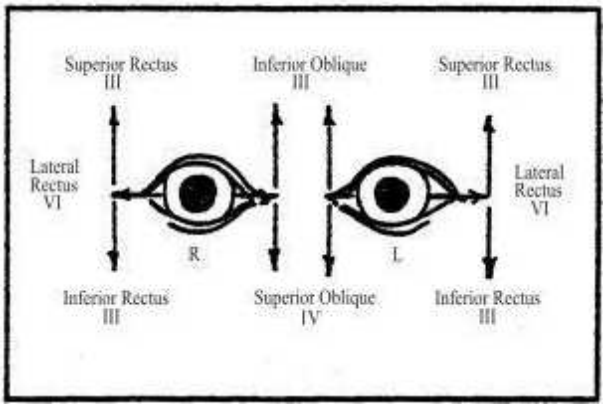


- Lateral rectus (VIn) → abduction.
- Medial rectus (IIIn) → adduction.
- Superior rectus (IIIn) → elevation, intorsion & adduction.
- Inferior rectus (IIIn) → depression, extorsion & adduction.
- Inferior oblique (IIIn) → elevation, extorsion & abduction.
- Superior oblique (IVn) → depression, intorsion & abduction.

So at rest: a IIIn palsy will have the eye abducted & sl depressed (i.e. so called "down & out"); a IVn palsy will have the affected eye elevated & head tilted to unaffected side with chin tucked in esp when looking in & downwards; and a VIn palsy will have the eye deviated medially.

However when testing using the standard 'H' pattern, the muscles responsible for most of the movement in the indicated directions are shown on the right:

Note how the actions of IR & SO and SR & IO have seemed to swap from their isolated actions. This is because the eye in abduction or adduction has rotated and changed the alignment of the muscles wrt the axis of the eye.



Aetiology

- Space occupying lesion in the orbit or skull
- Vascular lesion
- Trauma
- Neurological or myological disorder.

Symptoms

Double vision which may be dependent on direction of vision. It may be intermittent and associated with tiredness. A droopy eyelid may also be noticed. Is there pain in the eye?

Examination

- Ptosis is often the first sign of weakness and it suggests the affected side.
- Pupil sizes & reactions.
- Eye deviation. Squint.
- Test for diplopia in all visual fields.
- Fundoscopy
- Check the retina and ascertain that the disc is not obscured by papilloedema.
- Check the other cranial nerves as described in neurological history and examination.
- In children, use a pen torch for following and the *cover test*.

Differential Diagnosis

There are many possible causes of diplopia, including:

- The intracranial course of the abducent nerve (VI) is long and so it is vulnerable at many sites. It is a poor localising sign for a space occupying lesion.
- Myasthenia gravis. ~50 present with diplopia. Fluctuating weakness can also occur in encephalopathy and sepsis.
- Horner's syndrome is a good lateralising sign but no use to localise a lesion.
- Vertical gaze palsy, affects both eyes, pupils are often unequal but fixed and there is usually no diplopia. Think of Parkinson's disease and progressive supranuclear palsy. Inability to look down can lead to falling down stairs.
- MS - there will usually be nystagmus and an extensor plantar response too.
- Hysteria is a diagnosis of exclusion.
- *Rarer Causes*
    - Ophthalmoplegic migraine
    - Temporal arteritis, also called giant cell arteritis
    - Sarcoidosis
    - Post-refractive surgery.
    - Creutzfeldt-Jakob disease
    - Chronic progressive external ophthalmoplegia (CPEO)
    - Rare mitochondrial myopathies e.g. Kearns-Sayre syndrome (KSS)

Investigations

- Diagnosing which muscles are affected is fairly easy.
- Check for diabetes.
- Check blood pressure.
- MRI may show a tumour, an area of infarction or even an arterial aneurysm pressing on a nerve. It can also show demyelination.
- Chest x-ray may reveal malignancy or sarcoidosis with BHL.

Management

Depends on cause.

In childhood strabismus, surgery may be required but not always. An alternative that shows potential is botulinum toxin.

People with diplopia must not drive.

Prognosis

A sixth nerve palsy of vascular or unknown causes typically resolves within 6 to 8 weeks. If resolution does not occur within 2 to 3 months, the condition progresses, or if additional neurological signs or symptoms develop, imaging studies are required.