EYE - GENERAL

STEPS OF EYE EXAMINATION

Step 1: Approach the patient
- Read the instructions carefully for clues
- Shake hands, introduce yourself
- Ask few questions "could you tell me your name please? Are you right- or left-handed? Are you quite comfortable"
- Ask permission to examine him "I am just going to test your eye; is that alright?"
- Ask the patient to sit upright on the edge of the bed facing you, while you position yourself one arm's length from the patient at the same level.

Step 2: General inspection
- **Bedside:** walking stick, shoes-callipers, built-up heels
- Nutritional status (under/average built or overweight)
- Intention tremors (cerebellar syndrome – MS)
- Long, lean look of myotonic dystrophy
- Myasthenic facies
- Tabetic facies
- Facial asymmetry in hemiparesis
- Face and hands of acromegaly
- Foot ulcers in diabetes
- Pes cavus in Friedreich's ataxia
- Foot ulcer or necrobiosis lipoidica in DM

Step 3: Look at the eyes:
- Ptosis
- Exophthalmos
- Strabismus (divergent or convergent)
- Skew deviation (vertical misalignment of the eyes)
- Nystagmus (consider cerebellar syndrome – MS)
- Xanthelasma, arcus senilis
- Kayser-Fleischer rings (brownish yellow ring in the outer rim of the cornea, diagnostic of Wilson's disease)
- Corneal calcifications
- Blue sclera
- False (glass) eye.

Step 4: Look at the pupils:
- Size (dilatation, constriction)
- Equality and regularity
- Iris abnormalities (e.g. Lisch nodules in neurofibromatosis)
- Foreign bodies in the anterior chamber (e.g. lens implants)

Step 5: Visual Acuity:
- Ask the patient "can you see from each eye? Do you use eye glasses? Put it on please. Cover your left eye please".
- Hold a pocket Snellen's chart ***2 m*** from the patient's eyes and ask him to read sections of the print.
- Record the smallest print size read (e.g. No. 9 → visual acuity of 6/9).
- Repeat in the other eye.

Step 6: Visual Fields:

- **Visual attention**:
  - Tell the patient "Keep looking at my eyes and point to the finger which moves" Place your index fingers out on both sides approximately 50 cm apart and approximately 30 cm above eye level. Your fingers should now be in the patient's upper temporal fields on both sides.
  - Move your fingers in turn, and then both at the same time.
  - Repeat with fingers approximately 30 cm below eye level.
  - If one finger is seen when moved by itself but is ignored when both fingers are moved together, then there is visual inattention → **sensory** lesion above the thalamus
- **Peripheral visual fields**:
  - Tell the patient "Cover your right eye with your right palm and keep looking at my right eye" Now you should shut your left eye and keep looking at the patient's left eye.
  - Hold up the index finger of your right hand (or white hat pin) midway between you and the patient, at almost a full arm's length to the side.
  - Tell the patient "Tell me when you see my finger move" Move your wagging finger slowly from the upper then the lower temporal periphery towards the center (to test his upper and lower temporal quadrants against yours).
  - Then test the upper and lower nasal quadrants using the index finger of your left hand in a similar manner.
  - Then test the visual fields of his right eye in a similar manner.
  - When there is a homonymous hemianopia, the macula needs to be tested. Bring your wagging finger horizontally from the side with the defect towards the point of fixation. If your finger is seen before it gets to the midline, there is macular sparing, while if your finger is only seen once it crosses the midline, there is no macular sparing.
  - **N.B**. you may ask the patient to slightly raise his head up while testing the upper temporal fields to remove his eyebrows from the field, and ask him to slightly tilt his head down while testing the lower nasal fields to remove his nose from the field. This applies to you also.
- **Central scotoma** is tested for with a red hat pin (unless you have already found a field defect which does not require further examination). It is useful to mount the pin on the rubber eraser of a pencil, in order to be sure your hand is out of the patient's visual field:
  - Tell the patient "Cover your right eye with your right hand and keep looking at my right eye" Shut your left eye and keep looking at the patient's left eye.
  - Hold up the pin at the eye level midway between you and the patient just inside the outer limits of your temporal fields.
  - Tell the patient "Can you see the head of the pin? What color is it? Tell me if it disappears or changes color" Move the pin slowly from the temporal periphery through the central field to the nasal periphery. Patients with optic nerve neuropathy may report altered color vision even if there is no absolute central loss of vision.
  - If there is no scotoma, examine the size of the **blind spot**: Tell the patient "Tell me if the head of the pin disappears" Move the red hat pin from the point of fixation temporally and horizontally slowly until you find your own blind spot. Now, compare the size of the patient's blind spot to yours. The blind spot is situated 30 degrees to the temporal side of the point of visual fixation. Its size should correspond exactly with your own blind spot when the red pin is held equidistant between your eye and the patient's eye. The blind spot may be enlarged in chronic papilloedema or consecutive optic atrophy.
  - Then test the right eye for central scotoma and size of the blind spot in a similar manner.

Step 7: Eye movements:

- **Pursuit eye movement:**
  - Tell the patient "Look at my finger (or the white hat pin); follow it with your eyes without moving your head and tell me if you see double".
  - Move your finger slowly, like a ***letter H***: from side to side (lateral and medial recti), then up and down at the extreme of lateral gaze (superior and inferior recti and superior and inferior oblique), thus allowing the ***action of each muscle*** to be assessed.
  - If the patient reports he sees double; ask "Are the images side by side, up and down or at an angle? Where the images are widest apart? ". Diplopia is maximal at the direction of action of the paretic muscle. In this position cover one eye and ask "which image disappears: the inner or outer". Repeat this by covering the other eye. The outer (false) image disappears when covering the affected eye. If diplopia persists with one eye covered it may be lens dislocation, astigmatism or factitious. If the pattern of loss is complex and cannot be easily explained, always consider Grave's disease or myasthenia
  - If there is ***internuclear ophthalmoplegia***, on attempted gaze to one side;
    - the adducting eye (ipsilateral to the lesion) is slow or fails to adduct (*unable to get closer*), and
    - the abducting eye (contralateral to the lesion) shows ataxic nystagmus (*in hurry saying "come on come on"*).
    - Normal convergence differentiates INO from a medial rectus or III nerve lesion, and intact abduction in the other eye differentiate INO from lateral gaze palsy (nuclear [VI nerve nucleus] lesion, or supra nuclear [PPRF or frontal lobe] lesion)
- **Nystagmus**:
  - Ask the patient to follow your finger with both eyes. Move the finger in turn up, down and to each side. Hold the finger at a point where the finger can be easily seen by both eyes (in the range of binocular vision) and wait for at least 5 seconds in each position.
  - Describe the nystagmus in terms of the direction of fast phase and direction of gaze at which nystagmus is most marked (right, left, up, down, or greater in the abducting > adducting eye), and notice if it is central (sustained) or peripheral (fatigues, improves by fixation, aggravated by head movement and associated with vertigo, deafness and tinnitus).
  - In ***cerebellar nystagmus***, the fast-phase direction is towards the side of the lesion, and is maximal on looking towards the lesion, whereas in ***vestibular nucleus/VIII nerve lesion***, the fast-phase direction is away from the side of the lesion, and is maximal on looking away from the lesion.
  - In ***ataxic (dissociated) nystagmus***, there is nystagmus of abducting eye >> adducting eye, with weakness of adduction, a characteristic sign of INO, commonly due to MS (associated with cerebellar syndrome)
- **Observe for fatigability on holding upwards gaze** (myasthenia gravis).
- **Saccadic eye movements:** tell the patient "look to the right, to the left, up, down" Observe the eye movements. Look particularly at the speed of adduction.
- **Vestibulo-ocular reflex (doll's eye manoeuver):**
  - This test is used in patients with limited eye movement on command or pursuit to demonstrate preserved eye movement on vestibulo-positional stimulation, indicating a supranuclear eye movement abnormality. Otherwise, the test is most commonly used in unconscious patients when it provides a way of testing eye movements
  - Ask the patient to look into the distance at a fixed point, and then turn the patient's head to the left, then the right and flex and extend the neck. The eyes should move within the orbits, maintaining forward gaze.
- **Lid lag**: Also while testing eye movement note any lid lag: the upper lid fails to cover the sclera above the iris when the patient looks down. This is a cardinal feature of thyroid eye disease and its absence, in the presence of proptosis, should make one suspicious of another orbital cause of proptosis (e.g. orbital cellulites, caroticocavernous fistula, orbital metastasis, orbital haematoma, Wegener's granulomatosis, or pseudoproptosis)

Step 8: Light reflex:

- Tell the patient "look into the distance".
- Shine a bright light in the right eye. Look at the reaction of that eye (the direct reflex)
- Shine the light again in the right eye and look at the reaction in the left eye (the consensual reflex).
- Repeat the test for the left eye (shining the light twice, and looking at the reaction in the same eye in the first time, and in the other eye in the second time)
- **Afferent***:* **II** (sensory) → optic tract → LGG → **efferent***:* parasympathetic EWN of **III** (parasympathetic) → fibres to ciliary muscle

Step 9: Swinging light test:

- Shine a bright light into one eye and then the other at about 1 second-intervals.
- Swing the light repeatedly between the two eyes. Observe the pupillary response as the light is shone into the eye.
- Shining the light in the healthy eye causes rapid constriction to both eyes.
- In **relative pupillary afferent defect** (RAPD), the pupil on the abnormal side dilates when the light is shone into it (Marcus Gunn pupil), as the signal carried by **II** on the abnormal side is weaker than that from the contralateral side. Lesion is always unilateral, anterior to the optic chiasm, commonly due to optic neuritis, and rarely due to compression of the optic nerve or retinal degeneration.

Step 10: Accommodation-convergence reflex:

- Place your finger 10 cm in front of the patient's nose.
- Tell the patient "Look into the distance. Now look at my finger".
- Observe the eyes and pupils for their reaction to accommodation (the eyes should adduct, intort and the pupils should constrict)
- **Afferent**: frontal cortex → **efferent***:* parasympathetic EWN of **III** → fibres to ciliary muscle)

Step 11: Optic disc: Fundoscopy (see *Ch 19. Eye - Fundus*)

Step 12: Additional signs:

- Thyroidectomy scar, thyroid acropachy or pretibial myxoedema (in patient with exophthalmos)
- Acromegaly
- Cerebellar signs (in patient with nystagmus)
- Sympathectomy scar over the clavicle (in patient with Horner's syndrome)
- Absent limb reflexes (in Holmes-Adie pupil)
- Hemiparesis

Step 13: Thank the patient and cover him (her)

THEORETICAL NOTES

Visual acuity:

- The pocket Snellen's chart gives only an approximation because 6 m is the least distance at which the effects of accommodation can be ignored; hence visual acuity is normally tested at 6 m with a Snellen's chart three times as big as this.
- Remember that the commonest cause of diminished visual acuity is a refractive error so that to gain information about other pathology in the eye (e.g. diabetic maculopathy), the corrected visual acuity needs to be assessed (with glasses on or through a pinhole). In the UK you need a visual acuity of 6/10 or better to drive a car.

Abnormalities of the pupils:

Large pupil (mydriasis)	Causes
Normally reacting	**Anisocoria:** pupils are unequal but normally reacting (normal variant)
Doesn't react to light but **a**ccommodation reflex **p**reserved	**Afferent pupillary defect:** lesion anterior to the optic chiasma. Common cause: optic neuritis. Rarer causes: compression of the optic nerve, retinal degeneration
Doesn't react to light and reaction to accommodation is slow	**Adie's (tonic) pupil:** poor reaction to light and slow reaction to accommodation. Causes: **idi**opathic degeneration of ciliary ganglion; may be associated with loss of tendon reflexes (Holmes- Adie pupil)
Doesn't react to light or accommodation	**1)** with ptosis → **Surgical III palsy** **2)** No ptosis → **Mydriatic drugs**

Small pupil (miosis)	Causes
Normally reacting	▪ **Senile miosis:** normal age-related change ▪ **Horner's syndrome:** miosis, partial ptosis, enophthalmos, and hemi-facial anhydrosis. Causes: lesion to sympathetic fibres either: o Centrally in the hypothalamus, the medulla or the upper cervical cord (exits at T1). Common causes: stroke (lateral medullary syndrome) or demyelination. Rare causes: trauma or syringomyelia o Peripherally in the sympathetic chain, the superior cervical ganglion or along the carotid artery. Common causes: Pancoast's tumour (apical bronchial carcinoma) or trauma. Rare causes: carotid dissection. Sometimes no cause is found.
Doesn't react to light but **a**ccommodation **r**eflex **p**reserved	**Argyll-Robertson pupil:** lesion in the rostral (upper) midbrain near the Sylvian aqueduct, in which the light reflex fibres are interfered with, but the more ventral accommodation reflex fibres are spared. Causes: syphilis, DM, MS
Doesn't react to light or accommodation	**Miotic drugs**

Types of eye movement and site of control:

Type of eye movement	Site of control
Pursuit: slow eye movement to maintain fixation on a moving object (looking at the person moving across the room)	Occipital lobe
Saccadic (command): rapid movement form one point of fixation to another (looking from the page to someone in the room)	Frontal lobe
Vestibular-positional (vestibule-ocular reflex): eye movement that compensate for movement of the head to maintain fixation (looking at fixed point from moving car)	Cerebellar vestibular nuclei
Convergence: movement that maintain fixation as an object is brought close to the face (rarely affected in clinical practice)	Midbrain

Anatomical sites of abnormal eye movements:

Without diplopia	**Supranculear** (above the nuclei) → conjugate disorder of eye movement (both eyes move together and are equally impaired)
	Internuclear (connections between nuclei; MLF) → disconjugate eye movement
	Nuclear → disconjugate eye movement (except for lesion of VI nerve nucleus, which produces ipsilateral conjugate horizontal gaze palsy; however can be overcome by doll's eye manoeuver)
With diplopia	**Nerve** → disconjugate eye movement
	Neuromuscular junction → disconjugate eye movement
	Muscle → disconjugate eye movement

Concept of supranuclear gaze palsy:

- Supranuclear gaze centres control the individual cranial nerve nuclei to ensure that both eyes move together in conjugate fashion.
- Lateral gaze is controlled by the **PPRF** in the pons, which controls the ipsilateral VI nerve nucleus and, via the MLF, the contralateral III nerve nucleus.
- Vertical gaze is controlled by **accessory oculomotor nuclei** in the upper midbrain, which induce the III nerve nuclei to turn the eyes upward or downward in coordinated fashion.

Examples of abnormalities of eye movements:

Lesion	Features and Causes
III nerve palsy	**Features:** – Ptosis – Eye deviated down and out (divergent squint) because of the unopposed action of the SO and LR – Loss of upward, downward and medial movements (If the IV nerve is intact → intorsion of the eyes upon attempted downward gaze due to unopposed action of SO muscle) (if VI nerve is intact → lateral gaze is preserved) – Angulated diplopia – Dilated unreactive pupil → extrinsic compression, commonly by aneurysm of posterior communicating or internal carotid artery (painful) **Syndromes**: – Nothnagel's syndrome: injury to the superior cerebellar peduncle causes ipsilateral oculomotor palsy and contralateral cerebellar ataxia – Benedikt's syndrome: injury to the red nucleus results in ipsilateral oculomotor palsy and contralateral tremor, chorea, and athetosis – Claude's syndrome incorporates features of both Nothnagel's syndrome and Benedikt's syndrome, by injury to both the red nucleus and the superior cerebellar peduncle. – Weber's syndrome: injury to the cerebral peduncle causes ipsilateral oculomotor palsy with contralateral hemiparesis
IV nerve palsy	**Features:** – Head is tilted away from the side of lesion – Adducted eye cannot look downwards – Diplopia is maximal on looking downward and medially and images are at angle
VI nerve palsy	**Features:** – Convergent squint at rest – Impaired lateral movement – Diplopia is maximal on lateral gaze and images are side by side **Syndromes**: – Foville's syndrome: dorsal pontine injury results in lateral gaze palsy, ipsilateral facial palsy, and contralateral hemiparesis – Millard-Gubler syndrome: ventral pontine injury results in picture similar to Foville's syndrome, except for the eye findings. There is lateral rectus weakness only, instead of gaze palsy, because the abducens fascicle is injured rather than the nucleus – Gradenigo's syndrome*:* mastoiditis at the petrous apex produces deafness, pain, and ipsilateral abducens palsy

Internuclear ophthalmoplegia	**Features**: – Impaired adduction of the eye Ipsilateral to the lesion – Ataxic nystagmus in the abducting eye contralateral to the lesion: adducting eye is slow, and abducting eye is in hurry saying "come on come on" – Normal convergence (differentiate INO from a medial rectus lesion) **Causes**: Lesion in the MLF: – Gradual onset of INO which is usually bilateral is highly suggestive of MS (look for cerebellar signs, pyramidal signs, and pale discs). – A sudden onset of INO with facial numbness is more in keeping with stroke – INO with a contralateral hemiparesis and ipsilateral ataxia is indicative of brainstem disease (medial pons – lesion of basilar artery or paramedian branches)
Dysthyroid eye disease	**Features:** – Complex eye signs due to involvement of the eye muscles (exophthalmic ophthalmoplegia) – Exophthalmos, thyroid scar or goitre
Myasthenia gravis	**Features:** – Complex eye signs due to involvement of the eye muscles (diplopia worsen on sustained gaze) – Bilateral ptosis
Cavernous sinus and superior orbital fissure syndromes	**Features:** – Total or subtotal painful ophthalmoplegia – Sensory loss over the first division of the V nerve – Absent corneal reflex **Causes of cavernous sinus syndrome:** – Cavernous sinus thrombosis, is the most frequent cause; often secondary to infection from orbital cellulitis (frequently *S.aureus*), a cutaneous source on the face, or sinusitis (especially with mucormycosis in diabetic patients) – Aneurysm of the carotid artery – Carotid-cavernous fistula (orbital bruit may be present) – Meningioma, nasopharyngeal carcinoma or other tumour – Idiopathic granulomatous disorder (Tolosa-Hunt syndrome)
Ocular myopathy	**Oculopharyngeal muscular dystrophy** – Eye signs begin with ptosis (often complete), then ophthalmoplegia – Dysphagia is usually the most prominent symptom – Face and neck muscles are often mildly involved **Chronic progressive external ophthalmoplegia (CPEO)** – Absence of soft tissue in the lids and periorbital region – Ophthalmoplegia and mild facial and neck weakness
Horizontal (lateral) gaze palsy	**Causes**: – Injury to one of the frontal lobes (acute stroke) may result in deviation of the eyes toward the injured side due to the unopposed output from the intact frontal lobe (since the voluntary movement towards one side is initiated in the frontal eye fields on the other side). This sign is not always present; even when it is present initially, it may resolve within a short time. Conversely, focal seizure activity may induce deviation of the eyes to the contralateral side. – Lesion of the PPRF (supranuclear lesion) → ipsilateral horizontal conjugate gaze palsy (can be overcome by doll's eye manoeuver) – Lesion of the VI nerve nucleus (nuclear lesion) → ipsilateral horizontal conjugate gaze palsy (cannot be overcome by doll's eye manoeuver) – One-and-a half syndrome is due to a combined lesion of the medial longitudinal fasciculus and the VI nerve nucleus on the same side. The patient's only horizontal eye movement is abduction of the eye on the other side.

Vertical gaze palsy	**Features** (that distinguish vertical gaze palsy from third, fourth or sixth nerve palsies): – Both eyes are affected – Pupils are often unequal but fixed – Generally there is no diplopia – Intact vestibule-ocular reflexes (e.g. On extending or flexing the neck) **Causes**: – Lesions of the rostral interstitial nucleus of the MLF and the interstitial nucleus of Cajal (distal basilar artery ischemia is the most common aetiology) → supranuclear paresis of upgaze, downgaze, or all vertical eye movements. – Parinaud's Syndrome (Also known as dorsal midbrain syndrome) is a distinct supranuclear vertical gaze disorder from damage to the posterior commissure. o Features: ➢ Loss of upgaze (and sometimes downgaze) ➢ Convergence-retraction nystagmus on attempted upgaze ➢ Downwards ocular deviation ("setting sun" sign) ➢ Lid retraction (Collier's sign) ➢ Skew deviation ➢ Pseudoabducens palsy ➢ Light-near dissociation of the pupils (absent light reflex but intact convergence reflex) o Causes: ➢ Hydrocephalus from aqueductal stenosis ➢ Pineal region tumours (germinoma, pineoblastoma) ➢ Cysticercosis ➢ Stroke – Steele-Richardson syndrome or progressive supra-nuclear palsy (PSP): akinetic-rigid syndrome associated with progressive supra-nuclear palsy. It is characterized disorders of vertical gaze, especially downwards saccades in early stage (can be overcome by vestibulo-ocular reflex). Smooth pursuit is affected later in the course of the disease – Parkinson's disease – Huntington's chorea – Olivopontocerebellar degeneration o Myasthenia gravis o Thyroid ophthalmopathy o Miller-Fisher

Skew deviation refers to a vertical misalignment of the eyes, usually constant in all positions of gaze. It has poor localizing value as it has been reported after lesions in widespread regions of the brainstem and cerebellum (in cerebellar lesion, the ipsilateral eye deviates down and in, and the contralateral up and out).

Congenital squint (concomitant; non paralytic):

- Ocular misalignment equal in all directions of gaze (concomitant deviation)
- Eye movements are full
- Vision is suppressed from the nonfixating eye → **No diplopia**

Causes of nystagmus:

- **Physiological (end point nystagmus) (nystagmoid reaction) (Oculokinetic nystagmus):** nystagmus only at extremes of lateral gaze, as seen in people looking out of the windows of trains (usually physiological)
- **Ocular (retinal) (fixation nystagmus) (pendular nystagmus):** due to inability to fixate, usually congenital and may also occur when the vision is poor as in severe refractive error or macular disease.
- **Vestibular nystagmus**: the fast way is **a**way from the side of the lesion
  - **Peripheral:** lesion in the vestibular apparatus or VIII nerve. Produces unidirectional nystagmus away from the affected side irrespective of the direction of the gaze. It fatigues, improves by fixation, aggravated by head movement and associated with vertigo, deafness and tinnitus. Common causes: vestibular neuronitis, Meniere's disease, vascular lesions
  - **Central (central vestibular):** lesion in the vestibular nuclei in the brain stem. Produces bidirectional nystagmus and usually has vertical or rotatory component. It is sustained (not adaptable).Common causes: MS (young patients), vascular disease (older patients)
  - **Cerebellar nystagmus (central cerebellar)**: the fast way is **t**owards the side of the lesion. Common causes: cerebellar syndrome due to drugs, alcohol or MS. Rarer causes: cerebellar degeneration, cerebellar tumour
  - **Ataxic (dissociated) nystagmus**: nystagmus of abducting eye >> adducting eye, with weakness of adduction, associated with INO. Common causes: MS, CVA

Types of nystagmus:

- **Oculokinetic (end point nystagmus) (nystagmoid reaction) (physiological nystagmus)**: nystagmus only at extremes of lateral gaze, as seen in people looking out of the windows of trains (physiological)
- **Pendular (fixation nystagmus) (ocular/retinal nystagmus):** inability to fixate; the speed and amplitude are equal in all directions, usually congenital and may also occur when the vision is poor as in severe refractive error or macular disease.
- **Jerk nystagmus:** nystagmus with distinct fast and slow phases. The fast phase represents reflex correction of a slower deviation in the opposite direction
  - **Horizontal**
    - **First degree**: occurs only when looking to direction of fast phase, causes may be central or peripheral
    - **Second degree**: occurs in primary position of gaze, causes are usually central
    - **Third degree**: occurs even when looking in opposite direction to fast phase, causes are usually central
    - **Multidirectional (bidirectional) gaze evoked nystagmus**: direction of nystagmus changes with the direction of gaze (always in the direction of gaze); causes are always central.
    - **Ataxic (dissociated) nystagmus**: nystagmus of abducting eye >> adducting eye, with weakness of adduction, a characteristic sign of INO, commonly due to MS and CVA
  - **Vertical**
    - **Upbeat:** Indicates upper brainstem lesion. Common causes are demyelination, stroke, Wernicke's encephalopathy
    - **Downbeat**: Indicates medullary-cervical junction lesion. Common causes are Arnold-Chiari malformation, syringobulbia, demyelination
  - **Rotatory (rotary):** combination of vertical and horizontal nystagmus. Pure rotatory nystagmus is always central; however peripheral horizontal nystagmus usually has a rotatory component

Causes of bilateral sudden blindness:

- Bilateral occipital lobe infarction or trauma
- Bilateral optic nerve damage, e.g. methyl alcohol damage,
- Hysteria

Causes of unilateral sudden blindness:

- Retinal artery emboli
- Retinal vein thrombosis
- Retinal detachment
- Temporal arteritis
- Occasionally optic neuritis and migraine

Causes of unilateral/bilateral gradual blindness: cataract, glaucoma, macular degeneration, diabetic retinopathy, optic nerve or chiasmal compression, nerve damage, e.g. tobacco amblyopia