Examination of Eyes

This involves visual function, pupils and accommodation and extraocular movements.

Important anatomical concepts:
The optic nerve histologically belongs to the central nervous system and is often involved in MS and neuromyelitis optica.

The retina receives its blood supply from the ophthalmic artery (as the central retinal artery) which is a branch off the internal carotid artery. This is the artery involved in amaurosis fugax.

Components of the visual pathway: optic nerve, optic chiasm, lateral geniculate body, optic tract, optic radiation (temporal pathway and parietal pathway) and visual cortex.

Blood supply: optic nerve- opthalmic artery, optic chiasm- anterior cerebral artery, optic tract- anterior choroidal artery, optic radiation- branches of internal carotid artery, visual cortex- posterior cerebral artery.

Recognition of visual objects is processed in two pathways:

1. Ventral pathway- “what pathway” projects from the occipital to the temporal cortex.
2. Dorsal pathway- “where pathway” projects from the occipital to the parietal cortex.

Nonvisual functions of the optic tract

3. Superior colliculus- eye-head coordination
4. Pretectal nucleus- afferent arc of the light reflex
5. Suprachiasmatic nucleus of hypothalamus- activated by bright light and activates the pineal gland to release melatonin

Visual Acuity

If patient wears glasses examine visual acuity with the glasses on.
Examine visual acuity of each eye separately.
Can check near and far vision.

In terms of a neurology examination you are thinking of optic nerve function. From the history you will already be formulating a differential diagnosis. If vision is completely lost from one eye and it occurred acutely then the likely underlying aetiolgy is vascular. If it happened over a few days and the visual loss is incomplete then optic neuritis is in the differential. So when you are testing visual acuity as a neurologist you are trying to establish if it's an optic nerve issue or is it the visual function. If it seems like the latter then a formal ophthalmology assessment will be indicated.

Visual Fields

Confrontation test- initally testing the visual fields of both eyes together. When testing both fields together include a test for neglect where you move both hands and see if the patient can see both hands together or is neglecting one. Then test each eye spearately. When doing this use

your own visual field as a control. This will pick up a gross abnormality of the visual field and is unlikely to pick up a subtle defect.

Patterns of visual field abnormalities:

1. Homonymous hemianopia- A hemifield defect of the same side in both eyes (ie temporal for one eye and nasal for the other eye) is caused by a lesion in the contralateral visual pathway after the optic chiasm- optic tract, lateral geniculate body, optic radiation or occipital cortex.
2. Homonymous superior quadrantanopia- A defect in the superior half of the visual field. Here the lesion lies in the contralateral temporal lobe.
3. Homonymous inferior quadrantanopia- A defect in the inferior half of the visual field. Here the lesion lies in the contralateral parietal lobe.
4. Bitemporal hemianopia- the lesion here lies in the optic chiasm and usually is caused by a pituitary adenoma compressing from below.
5. Scotoma- A localised field defect may be seen in the contralateral half of the central visual field of both eyes. This is caused by a lesion in the occipital pole.

Blind spot

The blind spot is the confluence of optic nerve fibres from the nasal and temporal portions of the retina that forms the optic disc. It is devoid of photoreceptors and is referred to as the blind spot. It is checked by slowly bringing a red topped pin across the field of vision and comparing to your own. There will be a split second where the image disappears. If

the image disappears for longer than it should then this is an enlarged blind spot and suggests inflammation of the optic nerve.

Fundoscopy

For the purposes of a neurological examination there is no need to dilate the pupils for the fundoscopic examination.

Identify the optic disc. At the initial stages of papilloedema the colour of the optic disc becomes more red than it should be and the surrounding vessels become engorged. As it progresses there is loss of the demarcation of the optic disc- it no longer has clear margins. When the disc looks pale this is suggestive of optic atrophy. This is commonly found in patients with multiple sclerosis or optic neuritis.
The fovea can be identified by asking the patient to look laterally or directly at the ophthalmoscope light.

Pupillary reflexes

The shape and diameter of the pupils should be observed. Asymmetry of the pupillary diameter is called anisocoria. A pathologically large pupil is called mydriasis. A pathologically small pupil is called miosis. For testing the light reflex- the penlight is brought in from the temporal side and both pupils are observed. For the convergence reflex an object is brought from a distance in front of the eyes to as close to the eyes that the object is still in focus. The you can measure the near-point. Accommodation is really tested in the same way but accommodation and convergence have different anatomical mechanisms. They are inter-related- in order to accommodate you have to converge the eyes also.

Light reflex- This is shrinkage of the pupillary diameter in response to a light stimulus. Afferent arc- optic nerve, efferent arc- autonomic fibres of oculomotor nerve. A light stimulus presented to one eye elicits the light relfex in both eyes. This is because of the partial cross over of optic nerve fibres at the optic chiasm and projection of the afferent fibres from the optic tract to the pretectal nucleus on both sides. So the light presented to one eye should cause constriction of both pupils.

Convergence reflex- This is a vergence adduction movement that increases the visual angle to permit single binocular vision during near viewing. This is manifested as shrinkage of the pupillary diameter when one looks at a near object placed right in front of the eyes. The efferent pathway of this relflex is the same as that for the light reflex- autonomic fibres of the oculomotor nerve- but the afferent pathway is not fully understood.

Accommodation – The near point of accommodation is the point closest to the eye at which a target is sharply focused on the retina. This is a function of the ciliary muscle which is innervated by the parasympathetic fibres of the occulomotor nerve. Its action is to focus the image on the fovea by controlling the thickness of the lens.

The pupils are dilated as a function of the sympathetic nervous system. The pupil dilator surrounds the pupil radially and its contraction dilates the pupil. Therefore, if the sympathetic nerves to the eye are interrupted this will cause the pupil to constrict- miosis. The sympathetic nerves also

supply the superior tarsal muscle which functions to maintain the elevated position of the upper eyelid. Therefore, when the sympathetic nerves are interrupted there will be a partial ptosis.

The pupils are constricted by the puillary sphincter which is part of the paraympathetic nervous system. When these nerve fibres are involved in a lesion the pupil becomes larger- mydriasis.

Aside from the optic nerve which receives light for the pupillary light relfex the oculomotor nerve is the primary nerve involved in mediating the reflexes of the eyes. Both parasympathetic and sympathetic fibres are carried on the oculomotor nerve. It also supplies most of the extraocular muscles (see below) and so these will constrict and relax in accordance with what is necessary to see objects in the distance and those much closer.

Patterns of pupillary abnormalities:
Marcus-Gunn pupil: This is elicited by performing the swinging flash light test. Here the penlight is moved from one eye to the other. When abnormal it elicits a relative afferent pupillary defect. This is explained as follows: the light is presented to the normal eye eliciting both a direct and consensual relflex. The consensual reflex occurs because the efferent limb of the light reflex is intact. Then the light is presented to the abnormal eye and because there is a problem with the afferent arc of the reflex (as in the optic nerve) the light is not perceived and so both pupils dilate (in contrast to remaining constricted which is what should

happen when light is presented to the eye). This indicates optic nerve dysfunction.

Argyll-Robertson pupil: The light reflex is lost but the convergence reflex remains intact. This pattern may be seen in neuro-syphilis and diabetic autonomic neuropathy.

Adie syndrome: Here the pupil is dilated and constricts very slowly to light and dark conditions. There is usually loss of ankle reflexes.

Horner syndrome: this is caused by compression of the sympathetic chain. It will cause the pupil to constrict (miosis) and there will be an associated partial ptosis due to loss of function of the superior tarsal muscle.

Eye Movements

Eye movements involve both smooth pursuit and saccades. Three cranial nerves are involved in eye movements- oculomotor, trochlear and abducens- and these supply the extraocular muscles. Then there is control of gaze which is a higher cortical function. The brainstem is involved in controlling movements of the eyes together most notably the medial longitudinal fasciculus (MLF) and paramedian pontine reticular formation (PPRF). The cerebellum and basal ganglia also play an important role in eye movements.

There are 6 extraocular muscles- superior and inferior recti, superior and inferior oblique and lateral and medial recti.

Inferior oblique, superior and inferior recti and medial rectus muscles all receive innervation from the oculomotor nerve. The oculomotor nerve

also supplies the levator muscle. The superior oblique is supplied by the trochlear nerve and the lateral rectus by the abducens nerve.

The oculomotor nerve- motor and autonomic nerves run together in the oculomotor nerve. The oculomotor nerve passes between the posterior cerebral artery and the superior cerebellar artery and so impairment of the oculomotor nerve is an important sign in tentorial herniation. The oculomotor nerve aslo runs through the cavernous sinus.

The trochlear nerve crosses in the brainstem. It is rarely compressed in isolation.

The abducens nerve runs the longest distance in the cranial cavity. In intracranial hypertension this nerve may be compressed unilaterally or bilaterally.

The frontal eye field and supplementary eye fields are the central cortical gaze centres. They send information to the contralateral PPRF which serves as the lateral gaze centre. Activation of the PPRF causes contraction of the ipsilateral lateral rectus and contralateral medial rectus allowing the gaze to move laterally (to the side of that PPRF). The abducens and oculomotor nuclei are connected by the MLF. The vertical gaze centre lies in the midbrain. The rostral intertitial nucleus of the MLF plays a role in control of vertical gaze. It receives input from the frontal eye fields, vestibular nucleus and superior colliculus. Compression of the quadrigeminal body by a pineal tumor could cause a vertical gaze palsy.

The vertical gaze paly seen in progressive supranuclear palsy is related to pathology in the superior colliculus.

The role of the cerebellum in eye movements is to control the amplitude of saccades. It also plays an important role in stabilising the visual image on the fovea of retina. Output from the basal ganglia is inhibitory and acts to select necessary movements and suppress unnecessary movements.

To examine: observe the eyes and look for any evidence of ptosis or pupillary asymmetries. Also look at the position of the eyes and observe if there is a strabismus present. Also observe for any involuntary movements of the eyes such as nystagmus in the primary position of gaze.

To test eye movements ask the patient to focus on your finger placed right in front of them. Then without moving the head ask them to follow your finger in each direction of gaze- to the right, left, outward and upward, outward and downward, inward and upward and inward and downward. Ask them to report any double (or multiple) vision. If they report double vision then complete all directions of gaze and then go back and focus more closely on the direction of gaze that triggered the double vision. Ask the patient to close each eye sequentially to establish that it is binocular rather than monocular diplopia. Also establish whether the images are separated horizontally or vertically.

After examining smooth pursuit movements then move to saccadic movements. Here you will hold your finger far right and then far left. Ask the patient to focus on your nose and then ask them to look either right or left. You are establishing that the saccade occurs in one movement. Two abnormalities may be observed. Firstly the saccade may overshoot and correct and secondly the saccade may occur in two distinct movements.

To examine gaze ask the patient to look up and down and right and left without following any visual target. The oculocephalic reflex can also be tested now. Here the head is passively rotated and if the eyes change their relative position toward the opposite side of passive head movement the oculocephalic reflex is judged to be present. If there is a gaze palsy and the oculocephalic reflex is present this is deemed to be due to a supranuclear gaze palsy (central gaze centres) and if it is absent then it is due to an infranuclear gaze palsy (PPRF or midbrain vertical gaze centre).

The eye movement examination will also allow an opportunity to assess for nystagmus. This is where the eyes are moving involuntarily. Most commonly you will observe gaze evoked nystagmus. This may be on horizontal or vertical gaze. Down-beat nystagmus on vertical gaze is seen in an Arnold-Chiari malformation and intoxication with lithium or carbamazepine. Horizontal nystagmus when pathological can be induced by cerebellar lesions. On the extremes of gaze there may be a physiological nystagmus.

A few important patterns to note:

Internuclear ophthalmoplegia- here the pathology lies in the MLF. The eye cannot adduct past the midline and the abducting eye then develops nystagmus as it keeps trying to correct for the other eye.

Oculomotor nerve palsy- This causes the eye to be deviated downward and outward with ptosis. What is important to note here is whether or not the pupil is involved. If the pupil is involved it will be dilated and this is seen in a compressive lesion (for example PICA aneurysm) of the oculomotor nerve as the parasympathetic fibres run along the outside of the nerve. This would be an indication for an urgent neurology review and imaging. In contrast, an oculomotor nerve palsy that occurs in the context of a diabetic microvascular ischaemia will spare the pupil.

Horner syndrome- In Horner syndrome there is compression along the sympathetic chain. Here the symptoms will be partial ptosis, miosis and anhidrosis on one half of the face. The sympathetic chain can be interrupted at various different points. For example, a horner syndrome forms part of the lateral medullary syndrome and it is here in this scenario that the sympathetic outflow to the eye is blocked. It can be interrupted more peripherally for example an apical lung tumour can compress the sympathetic chain peripherally also causing a Horners syndrome.

Abducens nerve palsy- this will cause horizontal diplopia in the direction of the abnormal muscle. The images will be separated horizontally and the further lateral you go the farther apart the images will be.

References:

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