

Visual disturbances

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Abstract

The eyes, the visual pathways of the central nervous system, and the ocular motor system are frequently the seats of neurological disease. Patients with visual symptoms will often present first to an ophthalmologist or optometrist and it is as important for the eye specialist to have some knowledge of neurological disorders affecting vision as it is for the neurologist and general physician. The aim of this article is to outline the knowledge and skills required to carry out a basic assessment of a patient presenting with impaired vision either as a primary complaint or as part of a more generalized neurological disorder.

Keywords cortical blindness; diplopia; hemianopia; optic neuropathy; pupil; visual aura; visual disturbance; visual loss

Visual loss

In assessing a patient complaining of a deterioration in vision, the first task is to decide whether the impairment is monocular or binocular, whether the onset was gradual or abrupt and, if abrupt, whether transient or persistent. This is not always a straightforward exercise. A patient may not notice slowly progressive monocular visual loss until he or she happens to cover the unaffected eye giving a pseudo-abrupt onset. Patients with transient homonymous hemianopia will often report that the loss affected only the one eye ipsilateral to the field defect. Patients with gradually progressive or congenital homonymous hemianopia may be quite unaware of it until a perimetric examination is performed for some other reason.

Visual loss is most likely to be reported if abrupt in onset and if it affects central vision, especially if it affects both eyes. Extensive bilateral visual field loss which has no corresponding regions of loss in the two eyes (such as pure bitemporal hemianopia) may go unnoticed during habitual binocular viewing.

The examination in cases of visual failure

The first task is to decide whether one or both eyes are affected, and whether the problem is due to impaired optical quality, a disorder of the retina, the optic nerve, the chiasm or the post-chiasmal visual pathways. Inspection of the eye itself may reveal a paralytic strabismus or mechanical displacement of the globe

itself (e.g. proptosis). Examination will then continue with an assessment of visual acuity taking suitable precautions to ensure that neither refractive error nor lens opacity is a confounding factor.

Visual acuity will be impaired in most cases of optic neuropathy but will be spared in pure bitemporal and unilateral homonymous hemianopia. This is because visual acuity testing examines only central (foveal) vision and extensive peripheral field loss – including hemianopia – will spare acuity, although in hemianopia the patient may miss letters on one or other side of the chart.

Distance and near (reading) vision should routinely be tested with and without the patient's own glasses and with a pinhole (which will compensate, at least in part, for any additional refractive error). There are a number of advantages in testing near vision:

- it gives a practical indication of what problems the patient may have with reading
- it gives a clue that refractive error is playing a part (e.g. a myope will be better for near than far, *vice versa* for a presbyope)
- patients with central field defects may have trouble finding the letters on a distance chart whilst a reading chart presents them with a large area of text in which to pick out letters and words.

In cases of visual disturbance due to cerebral disease there are other reasons for specifically examining reading. Patients with right homonymous hemianopia have a particular difficulty with reading fluency (not acuity) because they cannot scan ahead. This occurs only when the macula is split and is referred to as 'hemianopic alexia' – reading speed is reduced a little and isn't affected by word length. Patients with posterior left hemisphere lesions may also have 'acquired alexia' and may no longer be able to read word shapes but read letter by letter – reading speed is reduced profoundly, getting slower for longer words. In neither of these situations is near visual acuity affected but the examination of reading performance is essential to diagnosis.

Colour vision testing is of particular importance in neurological visual disorders. This is because colour vision is disproportionately affected in optic neuropathy. This may be because in demyelinating and compressive optic neuropathy there is greater vulnerability of the axons of retinal ganglion cells processing colour information. The relative preservation of colour vision in retinal disease is also partly due to the way that the visual field is affected, so that a patient with, for example, maculopathy can use the paramacular visual field to read colour plates. The Ishihara pseudoisochromatic plates are used as standard. It is important to note that these plates are designed to distinguish the various degrees of Daltonism, although only protanopia ('red' deficiency) and deuteranopia ('green' deficiency), there is no plate to test for tritanopia ('blue' deficiency). Therefore the detail of the errors made is not as significant in optic neuropathy where various combinations of impaired colour vision may be found. However, it is clearly important to be able to recognise that a patient has Daltonism (the errors made will be identical in each eye) when interpreting the findings. It is usual to record whether or not the patient reads the first (control) plate: no colour vision is required to read this plate and, if the patient fails, there is no

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value in proceeding – the patient's visual acuity is not adequate to read the plates (it will be 6/60 or worse).

There are some types of error which are peculiar to neurological disorders and should be watched for while the patient is reading the Ishihara plates. Some of the plates have two digits and patients with hemianopia (which may be homonymous or heteronymous) will often miss the digit on the side of the hemianopia. Patients with cortical visual disorders may not be able to perform the 'Figure-Ground' nature of the task (i.e. the ability to group together the dots to perceive the number) and these patients will fail all of the plates – including the control – despite having adequate acuity and colour vision.

Colour vision is also assessed at the bedside by asking the patient to report any change in colour appearance of a coloured target. In the simplest form of this test the patient is asked to compare the appearance of a small coloured target (such as a red pin head) foveally, viewing first with one eye and then the other. A patient with unilateral optic neuropathy will usually report that the colour looks paler (less saturated or closer to white) with the affected eye: it might in some cases appear darker and appear maroon or brown. If both eyes are affected this interocular comparison is not possible but an advantage of this technique is that it can be extended across the visual field (see below).

Visual field testing is an extremely valuable skill which should be in the everyday practice of ophthalmologists, neurologists and other physicians. To be able to ascertain that the patient has, for example, a central scotoma, is a great advantage in the diagnosis of patients complaining of visual loss. Begin with both eyes open and test in all four quadrants for the ability to see a moving finger and to count fingers. This will exclude a gross homonymous hemianopia and it is also possible to look for visual inattention by testing simultaneously in the two hemifields. Then test monocularly in the same way in the four quadrants. If there is no abnormality a small coloured target (such as a 4 mm red pin head) can be used to look for evidence of colour desaturation across the visual field. Firstly the target is brought in from the periphery and the patient asked to report when the red colour is appreciated. Next, different regions of the field can be probed looking for relative or absolute scotomata. If a partial hemianopic defect is suspected (the patient is able to detect a moving finger or to count fingers but there is desaturation of colour) then the target can be presented moving across the vertical meridian where the sudden change in the appearance of the colour will be readily appreciated by the patient. See Figure 1 for some common patterns of visual field defect.

The pupil light reflex is of great importance and should be examined next. As bright a light source as possible should be used in dim surroundings, an additional light, directed towards the lateral aspect of the eye may be needed to visualize the pupil in the case of a dark iris. Firstly, it must be established that there is a direct and consensual response to light in each eye. If there is severe visual loss then it will be apparent that the amplitude of the pupil response to light is diminished, indeed it will be absent if there is no perception of light in the eye (care must be taken that stray light does not fall in the unaffected eye else a spurious consensual response will be seen). If there is partial loss of

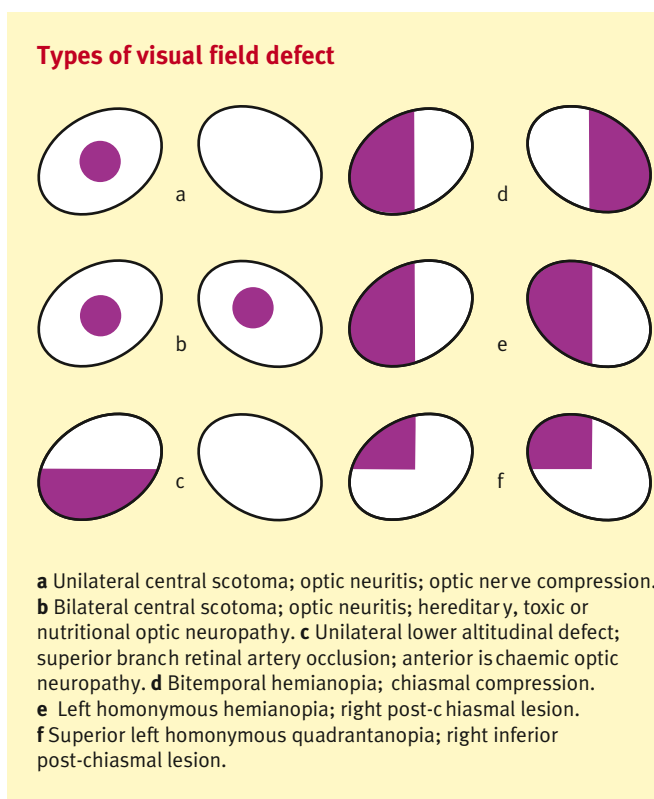


Figure 1

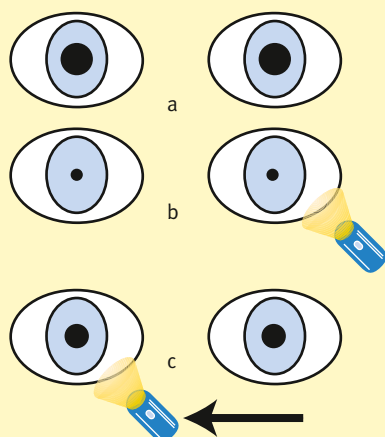
vision due to unilateral optic neuropathy the direct light reflex may appear symmetrical but if the light is swung from one eye to the other both pupils will dilate when the light is moved to the abnormal eye. This sign, known as the relative afferent pupillary defect (RAPD) is a very sensitive test of unilateral (or asymmetric bilateral) optic neuropathy. It is also reasonably specific because, as with colour vision, the pupil light reflex is affected in moderate optic neuropathy whereas retinal disease needs to be more severe before a RAPD will be detected. If both optic nerves are affected equally it will not be possible to demonstrate a RAPD. The pupil light reflex is not affected at all where the problem is optical or in cortical blindness. See Figure 2 for an illustration of the RAPD.

The fundus examination, using the direct ophthalmoscope, is also a most useful skill for a physician to acquire. The clinician must take the trouble to become accustomed to the range of normal appearances such as the difference between a myopic eye (a large eye with a large scleral opening – mistaken for optic atrophy) and a hypermetropic eye (small optic disc crowded with nerve fibres – mistaken for papilloedema). In acute visual loss the fundus examination may provide the diagnosis as in central retinal artery occlusion or may appear entirely normal as in retrolubar optic neuritis. The examination of the optic disc, the retina, retinal vessels and the macula is an essential part of the examination of any patient complaining of visual loss.

Causes of visual failure

It is best to take a syndromic approach to cases of visual failure, the following scheme is suggested.

A right relative afferent pupillary defect



a Observe the pupils in the ambient lighting. They are equal.
b The normal left eye is illuminated, both pupils constrict.
c The light source is moved quickly across to the right eye which has a damaged optic nerve, both pupils dilate. It is as if the light source is dimmer as it moves across to the right eye – but here it is the damaged optic nerve that signals a dimmer light.
 Note: if the right eye were stimulated first (move from **a** to **c** in the Figure) both pupils constrict, but less than moving from **a** to **b**. This difference is difficult to judge without carrying out the ‘swinging flash-light’ test.

Figure 2

Monocular visual loss

Acute and transient – a common clinical problem is the patient who notices transient loss of vision in one eye. The classic cause is a retinal embolus, in which case the onset is abrupt (often as if a curtain is descending in front of the eye) and the vision is lost into blackness for several minutes before recovering completely. Emboli may be visible in the retinal circulation, or there may be other evidence of vascular disease. However, a similar syndrome can occur as a result of retinal vasospasm in otherwise entirely fit young people, they may be more likely to complain of a ‘white out’ or ‘grey out’ of vision, or the loss occurs in patches which enlarge and coalesce reminiscent of the pattern of blood supply to the choroid rather than the retina.

Much briefer episodes of monocular visual loss, lasting seconds only, can occur if the eye is poorly perfused. Such episodes tend to occur only when standing. This can occur with combined disease of the external and internal carotid arteries but is also common in giant cell arteritis which is the most important cause of transient visual loss as prompt treatment with corticosteroids will prevent otherwise certain blindness. Transient monocular visual loss lasting for a few seconds also occurs in papilloedema due to raised intracranial pressure: here again the attacks are provoked by a change in posture but more usually standing after stooping – the mechanism is uncertain.

Acute and permanent – as stated above acute onset of visual loss can be spurious if the patient happened to cover the other eye. Where it is genuinely abrupt and not a result of trauma then there will usually have been an ischaemic event. Central and branch retinal artery occlusions produce visual field defects corresponding to the part of the retina that has undergone infarction

and will in most cases be due to embolism. Acutely the infarcted portion of the retina will demonstrate the appearance known as ‘cloudy swelling’, while the macula remains pink in a central retinal artery occlusion (the ‘cherry red spot’). The embolus responsible may be visible. Anterior ischaemic optic neuropathy (AION) is always associated with swelling of the optic disc in the acute phase and is very rarely due to emboli as it is poor perfusion in the posterior ciliary circulation that is responsible in most cases. The precise aetiology is unknown but patients with hypermetropic (crowded) discs are particularly at risk and, unfortunately, the second eye is often affected later for this reason. The field loss corresponds to the sector of the optic disc that has undergone infarction and is most commonly lower altitudinal but can be upper altitudinal or central. The most important cause of AION is giant cell arteritis because prompt treatment can prevent loss of vision in the other eye. Acutely the disc is swollen but looks pale very early and the loss of vision is more profound (usually total) than is usual in non-arteritic AION.

Subacute – in optic neuritis the visual loss tends to progress over a few days and is associated with pain on eye movement which is rarely severe. In cases associated with multiple sclerosis spontaneous recovery is the rule and there is no long term benefit in treating with corticosteroids. In other conditions (sarcoidosis or neuromyelitis optica for example) a prolonged course of corticosteroids may be mandatory for recovery to occur and longer term immunosuppression may be needed. Some compressive lesions can cause subacute and painful loss of vision, for example, mucocele of the paranasal sinuses or anterior communicating artery aneurysm. Leber hereditary optic neuropathy presents typically with unilateral optic neuropathy in a young male which progresses over a matter of weeks: the fellow eye will be involved similarly within a few months.

Progressive – if the loss of vision has progressed over a matter of weeks or months then a compressive cause is much more likely than inflammation. The list of causes of compressive optic neuropathy is a very long one but a clinical assessment can provide some important clues. If there is proptosis then there is likely to be a space-occupying lesion in the orbit which may be a tumour or compression of the nerve at the orbital apex by enlarged extraocular muscles as in dysthyroid eye disease. If there is ophthalmoplegia then the pathology is likely to be at the orbital apex because the ocular motor nerves do not pass close to the optic nerve intracranially but pass through the cavernous sinus and superior orbital fissure. If the optic disc is swollen then the ophthalmic vein must be compressed and the nerve must be compressed just behind the globe – optic nerve sheath meningioma will commonly present with this finding. If there is any form of temporal hemianopic defect in the fellow eye then the nerve must be compressed intracranially with involvement of the chiasm. Pituitary adenoma, suprasellar meningioma and craniopharyngioma can all cause chiasmal compression with signs of unilateral optic neuropathy: the patient may be unaware of the defect in the other eye.

Binocular visual loss

Acute and transient – isolated transient bilateral visual loss may occur as a result of a sudden drop in cerebral perfusion. Vision is particularly vulnerable in this respect and either the anterior or posterior visual pathways can be affected. Patients who

have suffered Stokes Adams attacks will often report that they suddenly lost vision just prior to losing consciousness and if the attacks are particularly brief then loss of vision for a few seconds can be the only manifestation. In the case of syncopal attacks patients will begin to feel faint and often lose vision prior to passing out. Therefore, if the episodes of loss of vision are brief – lasting seconds only – a sudden drop in cerebral perfusion should be considered as the likely cause. If the attacks only occur when standing then evidence of postural hypotension should be sought: if not related to posture then a dysrhythmia may be responsible. More prolonged attacks of lone bilateral blindness – lasting for several minutes – can occur as a result of posterior circulation embolism but often no cause is found for this complaint.

As mentioned above patients often report transient hemianopia as transient monocular visual loss and it can be difficult to be sure unless they covered one eye during the episode. A useful question is to ask the patient if they were looking at, for example, a face during the episode how it would appear. If the response is that they would see only a half of the face then you have the answer. Transient hemianopia is most likely to persist for some minutes and to be due to posterior circulation embolism; however, it is probably true that migraine can cause an isolated transient hemianopia without positive symptoms.

Acute and permanent – acute visual loss due to bilateral optic neuropathy is not common but can occur where infarction of the retina or optic nerve is provoked – for example as a result of acute blood loss. Pituitary apoplexy is a rare cause of sudden blindness due to chiasmal damage: ophthalmoplegia and pituitary failure are associated.

Isolated bilateral total visual loss usually results from bilateral occipital infarction due to an embolus to the bifurcation of the basilar artery and an isolated hemianopia follows occlusion of one posterior cerebral artery.

Progressive – bilateral progressive optic neuropathy will usually result from a toxic cause (such as treatment with ethambutol), a nutritional cause (such as B12 deficiency), a genetic cause (such as dominant optic atrophy), or, rarely, a paraneoplastic cause. Hemianopia of gradual onset will often indicate the presence of a space-occupying lesion of the post-chiasmal visual pathway (optic tract, optic radiation or occipital lobe). Inflammatory and infective lesions may also occur. Progressive multifocal leucoencephalopathy is particularly likely to cause homonymous visual field defects. Gradually progressive hemianopia may not be noticed by the patient and may be discovered incidentally. Similarly gradual onset of bitemporal hemianopia is often not recognized by the patient, or symptoms may be vague leading to delay in the diagnosis of compression of the chiasm. Pituitary adenoma, suprasellar meningioma and craniopharyngioma are the commonest causes of chiasmal compression.

The investigation of visual failure

Blood tests will in specific instances be of primary importance in establishing the diagnosis. For example:

- the erythrocyte sedimentation rate in a suspected case of giant cell arteritis
- serum B12 level in a case of bilateral progressive optic neuropathy
- genetic tests for Leber hereditary optic neuropathy or dominant optic atrophy.

Neuroimaging is required to investigate all cases where a space-occupying lesion may be responsible for progressive visual failure. Progressive uniocular visual loss, progressive chiasmal syndromes and progressive hemianopia are most likely to be in this category. In the case of anterior visual pathway pathology the protocols used are critical. Computerized tomography (CT) or magnetic resonance imaging (MRI) can be employed but in either case fine cuts through the orbits and chiasm must be requested and some lesions (for example small meningiomas) may be missed if contrast is not given. Neuroimaging is not helpful in the diagnosis of anterior ischaemic optic neuropathy but occipital infarction or haemorrhage can be diagnosed as for any other stroke. In optic neuritis MRI of the brain may establish MS as the likely aetiology if typical lesions are seen.

Electrophysiological testing is of major value in excluding retinal disease and it is therefore important that electrophysiological tests of the retina (electroretinogram: ERG) and optic nerve (visually evoked potential: VEP) should always be carried out together. If there is a retinal abnormality the VEP may be spuriously abnormal and it is unwise to rely on the VEP alone to confirm optic nerve disease. A cone dystrophy, for example, can mimic optic nerve disease because clinical examination reveals a central field defect and impaired colour vision. Most cases of retinopathy and optic neuropathy can be distinguished clinically, but in a few cases an electrophysiological assessment will prove helpful.

Positive visual symptoms

Patients may present with positive visual symptoms rather than loss of vision. Such cases are usually diagnosed on the history and it is the patient's description of the symptoms that is key. It is important to establish whether the symptoms are continuous or paroxysmal, whether the images are formed or unformed and whether there is an associated visual deficit.

Flashing lights: unformed and geometric visual phenomena

Retinal dysfunction is more likely to be associated with positive visual phenomena than is optic nerve disease. Patients report flickering lights and flashes which are constant but much more marked in the dark. In focal retinal disorders, the flickering may be localized to the visual field defect. Abrupt changes in ambient light levels may modify the symptoms.

Optic neuropathy does not often cause positive symptoms. Patients with acute optic neuritis may report brief flashes provoked by eye movement which may only be noticed in the dark.

Migraine aura is most commonly visual. This will usually precede headache but aura without headache (acephalgic migraine) is seen frequently and is a particularly common pattern in older people experiencing migraine for the first time: such cases are often misdiagnosed as transient ischaemic attacks. The descriptions are varied but the fortification spectrum (an expanding zig-zag) is frequently encountered as are descriptions of 'heat haze' or fractured vision as if looking through broken glass. An essential feature is the duration which is universally around 10–20 minutes.

Occipital epilepsy is rare but can be distinguished from migraine because the attacks last for seconds rather than minutes and tend to be coloured, circular images. Longer attacks

will progress to other epileptic phenomena such as generalised convulsions.

Visual hallucinations in visual loss

The Charles-Bonnet syndrome (visual hallucinations in the context of low vision) is common in patients with visual loss from any cause. The hallucinations are often very elaborate with images of unfamiliar people or patterns (e.g. “trellis work”) predominating. The images persist and evolve over long periods of time.

Visual hallucinations in acute hemianopia (usually following a stroke) are similar to the Charles-Bonnet hallucinations in that detailed images of people are common but they tend to be fleeting and are localised to the visual field defect.

Diplopia

This article is not concerned with congenital disorders, but the physician must be aware of the clinical features of the various forms of congenital strabismus in order to be able to distinguish them from acquired disease. It is also essential to have an understanding of the actions of the six extraocular muscles; of how they are innervated; and of the brain stem and cerebral mechanisms involved in the control of eye movements.

In the history it is first necessary to decide if the diplopia is binocular or monocular. Most patients complaining of diplopia will have misalignment of the two eyes, and the double vision will be relieved by covering either eye. However, there are some situations where diplopia can be monocular (mostly refractive error such as astigmatism) and on further enquiry the patients will usually report more ‘ghosting’ than true diplopia and covering the affected eye, but not the fellow eye, will relieve the symptom. Patients will usually be able to tell if the separation of images is vertical or horizontal but should also be asked if it is torsional, that is to say that the two images are not parallel but are oriented obliquely. It is also necessary to establish whether the symptom is continuous or paroxysmal and whether it shows diurnal variation as is often the case in myasthenia gravis.

The examination in cases of diplopia

The examination should begin with an assessment of visual acuity – it is not possible to interpret an ocular motility examination without knowing this. Once again acuity should be tested with and without glasses, monocular diplopia due to refractive error or lens opacity will be relieved by viewing through a pin-hole. The examiner should then ask the patient to look at a pen torch and observe the reflection of the torch in each cornea. If it falls in the centre of the pupil of each eye then the patient is fixating the target with either eye and there is no misalignment. Note should be taken of any displacement of the globe or ptosis, and any abnormality of the pupils.

The range of eye movements should be examined by asking the patient to follow a target such as a moving finger. For straight-forward third or sixth nerve palsies it is usually sufficient to ask the patient to follow the target to the extremes of gaze, vertically and horizontally, and observe which muscle or muscles are underacting. In a patient with an ophthalmoplegia what is seen will depend upon which eye is used to fix and follow the target, hence it is often useful to re-examine testing monocularly, each eye in turn. It is also necessary to check vergence movements by

asking the patient to look from a distant to a near target: the near target should have a good stimulus to accommodation such as a small picture.

Cover testing is invaluable in sorting out minor degrees of strabismus or complex situations. The patient is first asked to fix on a distant target and each eye is covered and uncovered in turn using an occluder. If a tropia is present then the affected eye will be seen to take up fixation when the fellow eye is covered. If a phoria is present then the affected eye will deviate under cover and be seen to take up fixation again when the cover is removed. Note that the abnormal position of the eye must be inferred from the movement that is seen when the eye takes up fixation. The cover test can be repeated with the patient fixating a near target and with the eyes in various positions of gaze. See Figure 3 for an illustration of abnormalities on cover testing.

Saccadic eye movements are examined by asking the patient to look from one stationary target to another. Saccades are rapid conjugate eye movements, the lid also has a saccadic movement in upgaze. In cases of ophthalmoplegia which are neurogenic (such as a nerve palsy) or myogenic (ocular myopathy) the muscle cannot generate the high velocities required and saccades will be slow. In ocular myasthenia the *initial* movement may still be fast, best seen in the lid (Cogan sign) and the presence of a rapid movement – however brief – excludes a neurogenic or myopathic problem. In restrictive ophthalmoplegia (such as dysthyroid eye disease or muscle entrapment in orbital fractures) the saccadic velocity is normal but comes to an abrupt stop when the mechanical restriction is reached. The examination of saccades is important in the diagnosis of brain stem and cerebral disorders but these do not commonly cause diplopia because the

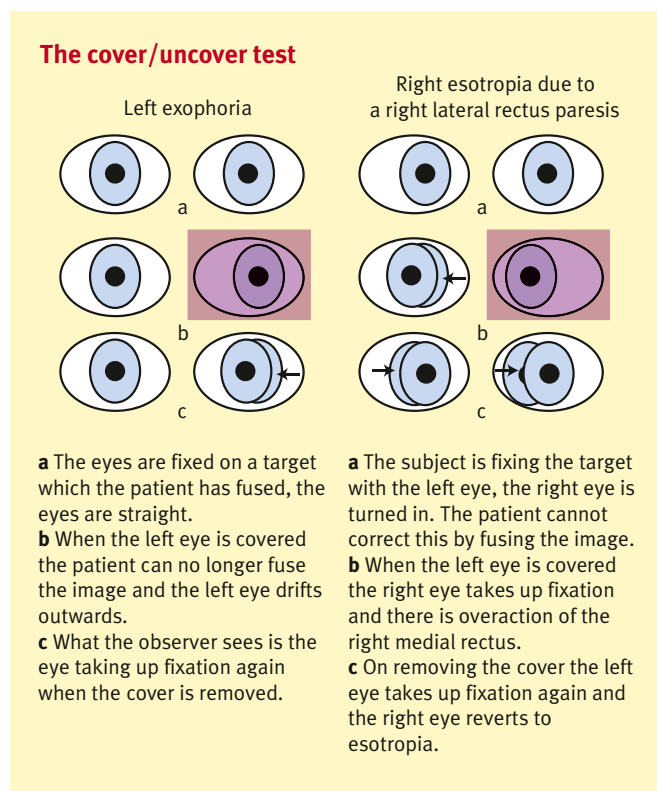


Figure 3

movements of the eyes remains conjugate apart from internuclear ophthalmoplegia where the velocity of the saccade is slow but for the adducting eye only.

Other cranial nerves should be examined as co-localization often gives a clue as to the location of the pathology causing the ophthalmoplegia: such as damage to the ophthalmic division of the fifth cranial nerve in the cavernous sinus; or the seventh and sixth nerves affected together when there is disease of the petrous bone.

Some causes of diplopia

Lateral rectus or sixth nerve palsy will give rise to horizontal diplopia worse in the distance (because the eyes cannot fully diverge). Microvascular ischaemic sixth nerve palsy is very common, especially in diabetes, but always recovers within three months. Persistent lateral rectus paresis should always be investigated thoroughly until a cause is found. Very high quality imaging may be required.

Third nerve palsy, if complete, is not associated with diplopia because of the complete ptosis. If partial then horizontal diplopia is usual. A major challenge in acute cases is excluding compression of the nerve by a posterior communicating artery aneurysm: typically there will be severe pain and a dilated pupil while in microvascular ischaemia, third nerve lesion pain is less common and involvement of the pupil less prominent.

Fourth nerve palsy causes torsional diplopia because the superior oblique muscle acts principally to intort the eye. The patient will often tilt the head away from the affected eye to correct this. Fourth nerve palsies are most commonly congenital or result from trauma. An isolated unilateral fourth nerve palsy will rarely be caused by a structural lesion.

Myasthenia gravis very commonly causes diplopia and ptosis. Any pattern of ophthalmoplegia can result but the diagnosis can be made if fatigue of a muscle action is demonstrated or a brief, rapid movement is witnessed at the onset of a saccade in

a weak muscle. Check for anti-acetyl choline receptor antibodies and image the chest for thymoma.

Dysthyroid eye disease does not cause muscle weakness but the affected muscles are enlarged and stiff. Therefore when the inferior rectus is involved it is elevation of the eye that is affected and the saccadic movement is not slowed. There may be proptosis and lid retraction. Check for anti-thyroid antibodies (the patient may or may not be euthyroid) and image the orbits to confirm muscle enlargement.

Giant cell arteritis is not often considered as a cause of diplopia but it is surprisingly common. It is a very important diagnosis to consider because if untreated blindness or other complications will follow. Any pattern of ophthalmoplegia is possible, the mechanism may be ischaemia of the extraocular muscles themselves.

Conclusion

Treating a patient with visual failure or diplopia is challenging. An understanding of the anatomy and physiology of the visual pathways and cranial nerves is essential and the physician must be aware of how to exclude non-neurological conditions. The highest quality imaging studies are required. The benefits to the patients of acquiring the necessary knowledge and skills are self evident. ◆

FURTHER READING

Kidd DP, Newman NJ, Bioussé V, eds. *Neuro-ophthalmology*. Philadelphia: Butterworth: Heinemann, 2008.

Levin LA, Arnold AC, eds. *Neuro-ophthalmology. The practical guide*. New York: Thieme, 2005.

Schiefer U, Wilhem H, Hart W, eds. *Clinical neuro-ophthalmology and practical guide*. Berlin; Springer, 2007.