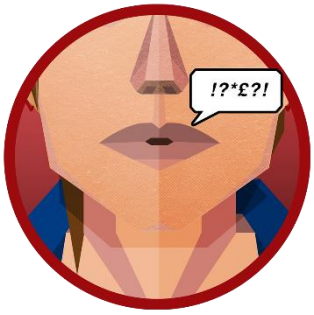

V-FAST

Training Manual

The V-FAST tool has been developed by the VISION Research Unit, University of Liverpool in collaboration with the North West Ambulance Service.

The purpose of the V-FAST tool is to screen for visual impairment during paramedic call-outs to suspected stroke. It is not a diagnostic tool but serves to screen for visual impairment and thus highlighting potential need for specialist visual assessment.

Contents

This manual includes detailed instructions for each section plus some additional background information on anatomy and physiology of stroke-related visual impairment. Key instructions are included in the V-FAST 1-page assessment tool, these instructions along with a form to record your results is available in the V-FAST instructions and checklist document. Video of the V-FAST assessment is also available.

Department of Health Services Research

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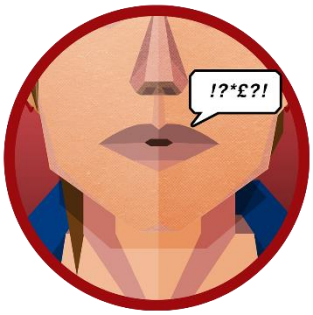
Liverpool, L69 3GL

VISION Assessment outlined in this manual

FACE Has their face fallen on one side?
Can they smile?

ARM Can they raise both arms and keep them there?



SPEECH Is their speech slurred?

TIME Time is of the essence

Visual impairment due to stroke and acquired brain injury

Visual impairment following stroke is common and estimated to affect two thirds of all stroke survivors[1]. There is currently no standardised protocol for referral and a considerable proportion of patients who have visual problems go unrecognised, thus receiving no advice or management[2]. There are various visual treatment options which can have a beneficial effect on vision and to general rehabilitation[3-4].

Visual impairment can have a considerable impact to quality of life including loss of confidence, impaired mobility, inability to judge distances and increased risk of falls[3]. There is a known link between poor vision, quality of life and depression in older persons[4]. For the above reasons it is important that patients with visual impairment are identified by the stroke multidisciplinary team (MDT) and appropriate referral made for vision assessment. It is equally important that the effects of visual impairment on functional mobility are established and information is provided regarding the use of residual vision to facilitate general rehabilitation. These issues have been recognised as research priorities in an NIHR James Lind Alliance sight loss prioritisation process[5] in which screening and assessment of stroke survivors for visual problems is listed as a top ten priority for research.

One particular aspect of screening and assessment of stroke survivors for visual problems is the input by the paramedic team during the pre-hospital call-out for suspected stroke. There is currently no standardised screening for visual impairment by paramedic services. Usual care involves screening for stroke using the FAST test but this does not incorporate any visual screen. The AVVV test, as currently used by some ambulance Trusts, may be used for patients with possible posterior circulation stroke in which there is a screen for acute sudden onset of ataxia and/or acute sudden onset of visual field loss along with association of vertigo and/or vomiting. Additional tools, such as the BE-FAST test, screen for balance and eyes in addition to the FAST test with the eye screen considering visual field loss and horizontal gaze. A particular issue is posterior circulation stroke in which visual impairment is common such as visual field loss, visual inattention and eye movement disorders[2]. As there is limited paramedic screening provided for such visual problems, there is the potential for misdiagnosis or missed diagnosis. Past research has shown that the FAST test can miss up to 40% of posterior circulation strokes[6-8]. Furthermore, patients with posterior circulation stroke have significantly longer door-to-needle durations[9].

Where strokes affect the occipital lobe only, about 90% of patients will have only visual complaints. It is therefore important to assess specifically for this. Furthermore one quarter of stroke survivors are of working age and often easily misdiagnosed where their primary complaint is visual, typically diagnosed as migraine.

Mis- or missed diagnosis means that patients are not treated within the thrombolysis time window that is important for the 85% of strokes that are due to infarction. As a result visual impairment can be permanent with life changing disability and impact to daily life including loss of confidence, impaired mobility, inability to judge distances and increased risk of falls[3]. Patient stories recount the impact to their lives of permanent visual impairment: JC was FAST positive due to posterior circulation infarct but his primary symptom was visual field loss (hemianopia). This was neither addressed or highlighted in his care plan for over 5 years causing huge distress and disability.

By increasing the identification rate of these cases by the paramedic service, diagnostic accuracy will improve ensuring appropriate onward referral. Recent hospital-based acute assessment of visual impairment in stroke survivors show that common visual impairments include reduced central vision, eye movement abnormalities, visual field loss, visual inattention/neglect and visual perceptual disorders[10]. Recent data[11] shows that where paramedics detect a visual problem in stroke survivors, often doctors also detect the visual problem during A&E assessment. However, paramedics and A&E doctor assessment frequently fail to detect visual problems because the stroke screening tests (typically NIHSS on admission) are not targeted enough for the range of visual problems that may occur.

To our knowledge, there is no paramedic screen that broadly considers all categories of visual impairment in a time-considerate screen. This V-FAST vision screening tool uses simple questioning skills and validated assessments of visual function coupled with detailed instructions. The intention is to increase accurate detection of visual problems and, in turn, increase accurate detection of stroke within the time window required to access hyperacute stroke management. The target population are patients with stroke causing visual impairment who are atypical of typically recognised stroke onset.

Screening Instructions

Section 1: History

Using the visual symptom guide, please ask the person about their past ocular history, what their current visual symptoms (if any) are, and whether their visual symptoms are new or preceded their brain injury. Ask the person to cover each of their eyes in turn and to ask if they note any difference in their vision from either eye and whether this is new.

If the person is unable to provide information due to cognitive or communication difficulties, please ask family members/carers for further information about past history and any changes they may have noted since the brain injury. It is useful to ask family members/carers about their observations even if the person has also been able to provide information.

It is important to document your own observations about whether the eyes look different to each other and your opinion of how well the patient can see and/or read.

Visual Symptoms

Ask the patient and their family/carers about the following.

Remember that the patient may not recognise that anything is wrong with their vision – even if the vision problem is severe.

Ask family or carers if they have any concerns about the patient's visual behaviour.

History of eye care

- Does the person need glasses for near and/or distance vision? These must be worn for vision testing, e.g. reading glasses for near and TV glasses for distance.
- Are these broken or lost?
- Does the person have any previous eye history other than glasses? e.g. attending eye clinic for glaucoma, cataract, diabetes, macular degeneration, problems as a child, etc.
- Ask patient cover each eye in turn to check if there are any differences in vision between the two eyes and whether they have noticed anything new or unusual.

Symptoms

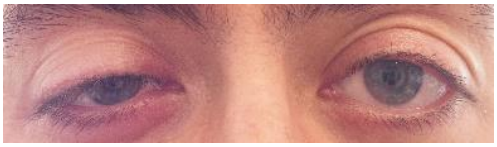
Questions to ask:

- Have you any problems with your eyes/vision?
- Is anything different with your vision?
- Do you have blurred/altered/missing bits of vision?
- Do you have jumbled or double vision?
- Do you have any dizziness or are unsteady?
- How quickly did symptoms come on?
- Have vision problems (if noted) lasted for longer than one hour without any change?
- Over recent days/weeks, have you noticed black outs of vision in one/both eyes?

Visual Observations

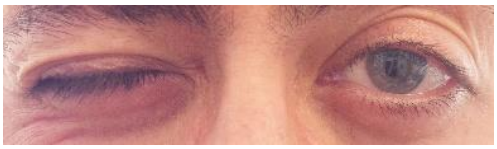
Do you notice altered appearance between right and left sides, or both eyes, for?:

- Lids – one lid lower or higher than the other or both very low or very high



- Pupils – one pupil smaller or larger than the other or both very small or large

- Squint – one eye turned in, out, up or down while other eye is straight (see eye alignment section)
- Eye movements – both eyes not moving by the same amount or to same extent (see eye movement section)
- Turning the head – turns head to one side when trying to look at things
- Closing one eye to see better



- Wobbling eyes

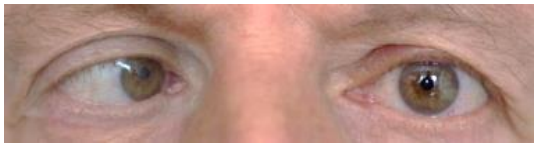
Section 2: Eye alignment and movement

Alignment

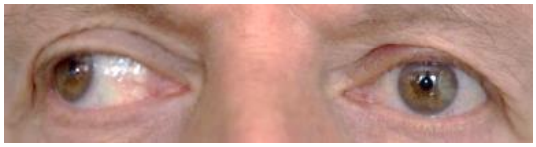
Normally, eyes are aligned such that each eye points towards the target they are looking at. This allows single vision. Remove the person's glasses (if worn). Using a pen torch or your finger, ask the person to look at the light/finger held at arm's length from them.

Observe the position of both eyes and determine if both eyes point to the target or whether one or both eyes point elsewhere. Using the pictures compare what you see for the person's eye positions versus the normal position and various abnormal positions indicated in the pictures.

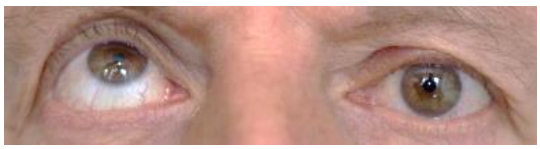
Straight eyes



Turn inwards



Turn outwards



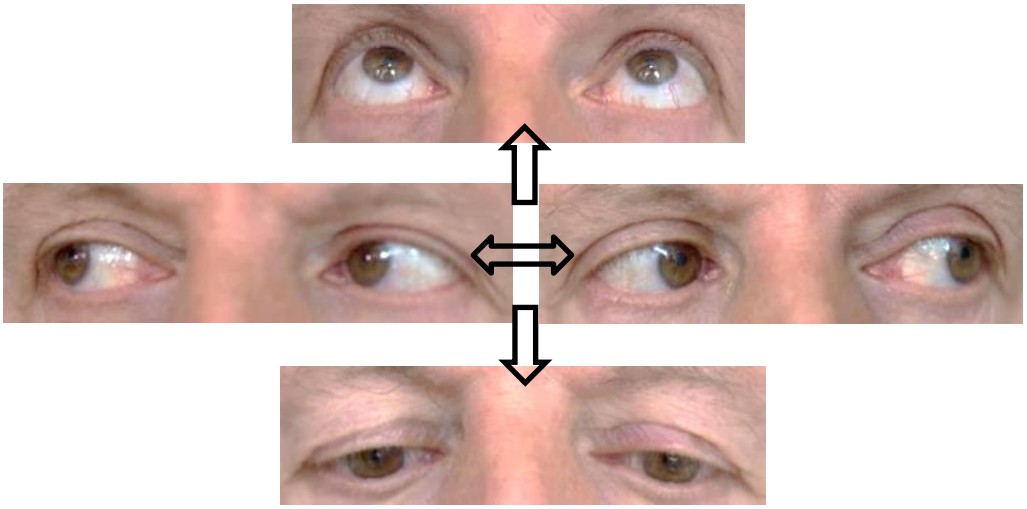
Turns upwards

Turns downwards

Movement

Normally, both eyes move symmetrically and evenly together when looking sideways and up or down. Using a pen torch or your finger, keep the person's head still and ask them to follow the pen torch/finger as you move it slowly to the right side, left side, upwards and downwards.

Observe the movements of each eye and determine whether both eyes move together and fully over to the skin margin (as per the pictures below), whether one or both eyes fail to move fully or one eye does not move the same as the other eye.



Using the pictures in the *Eye alignment and movement* section, compare what you see for the person's eye positions versus the normal position and various abnormal positions indicated in the pictures.

Observe whether the eyes are steady when looking straight ahead and when looking to either side or up and downwards. If the eyes 'wobble' this may indicate nystagmus.

Pupils

Ask the patient to look across the room/ambulance and note any differences in pupil size between the two eyes. Note which pupil appears bigger/smaller.

Then ask the patient to look from the distance and back to a pen held within arm's length of their face. Observe whether both pupils constrict (get smaller) as they focus on the near target.

Section 3: Reading

Ask the person to read the sentence on the assessment sheet, with their usual glasses for reading on and at their chosen distance if they are able to hold the sheet. If they are unable to hold the sheet hold it in a normal reading position.

Section 4: Visual Fields

Normally it is possible to look straight ahead and be aware of the peripheral surroundings detected by out peripheral field of vision.

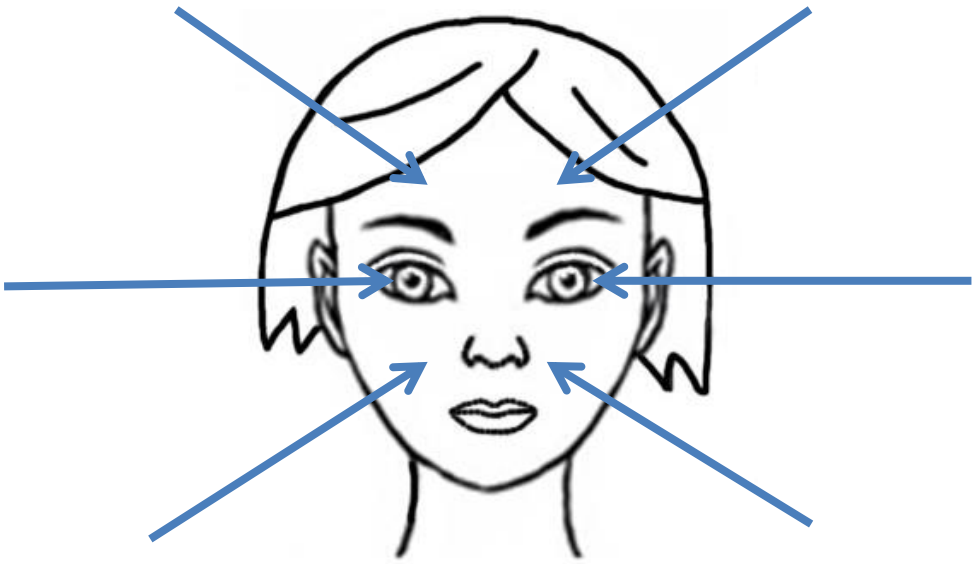
To screen a person's field of vision, the person should be seated straight in front of the examiner at a distance of 100cms and at eye level.

First remove the patient's glasses (if worn). Perform the following assessments with both eyes open. The examiner should hold both arms out when doing the assessment.

Peripheral boundary

The person is asked to continue looking at the examiner's nose and to say 'yes' or nod their head if unable to communicate when they are aware of a target moving in their outer vision.

Hold both of your arms out to each side. Pointing your index finger of the moving hand, slowly move this in from the periphery. In random order; 3 positions from the right side and three from the left side with one along the horizontal and one in each of the upper and lower quadrants in random order (see illustration below)



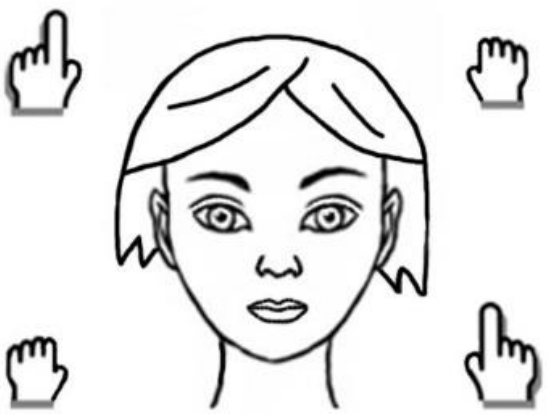
[The arrows indicate the direction from which the target should be moved from the periphery]

Central field – if time permits

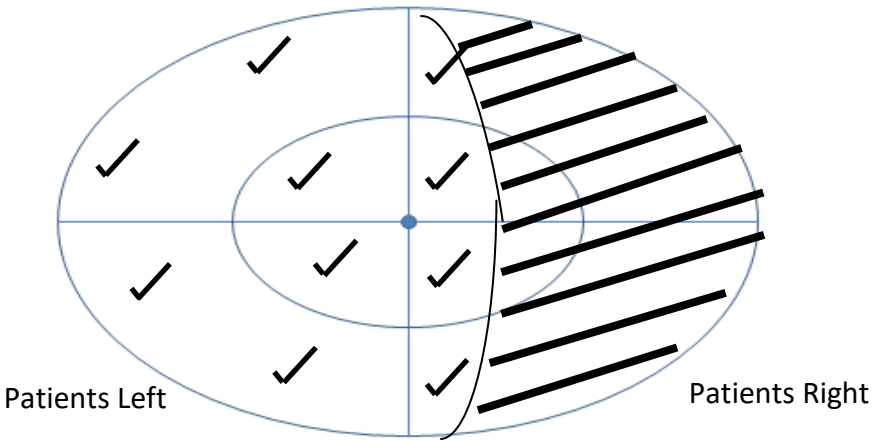
The person continues to look at the examiner's nose and is asked whether they can see all parts of the examiners face or whether part or one side of the face appears more blurred or faded than the rest.

The person should consider whether right versus left eyes, mouth versus forehead, right versus left ears are seen equally well to further qualify their responses.

Next, compare finger counting in each quadrant. Hold both hands up (with fingers closed), one hand positioned to the outer side of each of the patient's eyes. Briefly raise one or two fingers from one hand and ask the person to say whether fingers were raised and if so, how many were seen. Repeat this with both hands held in the lower quadrants below each cheek level. Next, briefly raise fingers on both hands and ask the person how many are seen in total.



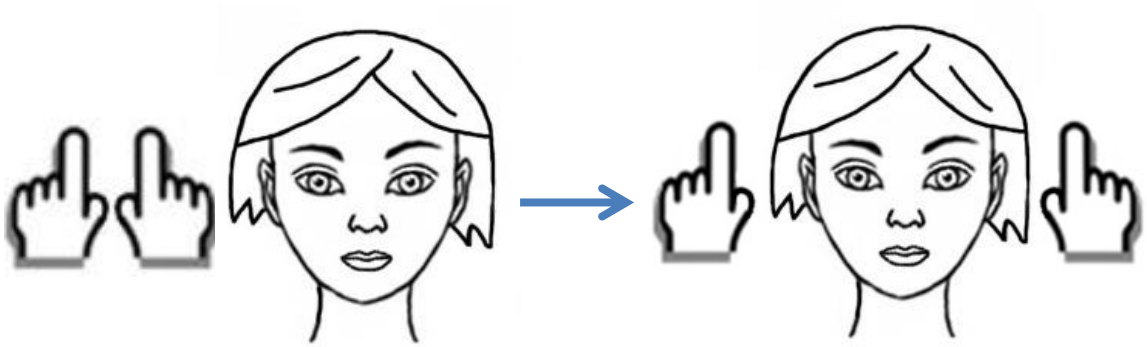
The chart below can be completed on the V-FAST instructions and checklist document. Hashed lines can be used to indicate areas where there is visual field loss and tick (✓) marks can be used to indicate areas where visual field appears normal (as per below).



Section 5: Visual inattention/extinction

In order to assess a person's spatial visual attention, observe the patient to determine if they are consistently failing to look to one side and fail to respond to people or objects on one side. Note, left side neglect is more common than right side. Extinction may help with assessment. Ask the patient to look straight ahead at the examiner's nose. Hold two fingers up (one finger from each hand) to the right side of the patient and ask them how many fingers they can see. Then repeat when fingers held to the left side of the patient. With extinction the patient may not see the finger to their affected side.

If unsure, hold up both fingers to the right side and slowly move one finger across to the left side – the other finger stays stationary. With extinction, the patient should initially see both fingers but will ignore the moving finger once it crosses to the affected side. (Note; this is an example for left sided inattention/extinction).



Visual/stroke mimics and TIA

Transient Ischaemic attack (TIA)

TIA is a mini stroke in which stroke signs and symptoms resolve within 24 hours. TIA can include visual symptoms and, although TIA may occur in isolation, it often precedes stroke.

Amaurosis fugax

Amaurosis fugax is classically described as a temporary loss of vision in one or both eyes. This may include a monocular blindness, dimming, fogging, or blurring of vision. Total or sectorial loss of vision generally lasts only a few seconds, but may last minutes or even hours. If the cause relates to cardiovascular events, this transient monocular visual loss occurs due to a temporary reduction in the retinal, ophthalmic or ciliary artery blood flow, or indeed the carotid or anterior cerebral arteries, leading to a decrease in retinal circulation which, in turn, causes retinal hypoxia.

One mimic is that of amaurosis fugax in which temporary vasospasm leads to decreased blood flow. Typically this type of temporary visual loss last no longer than five minutes and often follow exercise.

Migraine

Migraine occurs in many forms. Where vision is affected, migraine may be in the form of retinal migraine or migraine with visual aura.

Retinal migraine

This type of migraine typically affects just one eye and causes brief losses of vision or visual flashes of light. After a short time, vision goes back to normal. Loss of vision in one eye can be partial or total and can last for up to 10-20 minutes. Partial loss includes vision that has become blurred or dimmer than usual or there may be flashes of light in one eye. Retinal migraine may also start with a random pattern of blank spots in the field of vision which get larger to eventually cause total visual loss. Migraine headache may start before, during or after the visual problems.

Where vision loss last longer than one hour, there should be a strong suspicion that the cause is stroke and not migraine, particularly in individuals who have no prior history of such vision loss.

Migraine with visual aura

Migraine may occur in which there are visual aura occurring before or during the headache; or visual aura occurring without the migraine headache.

Visual aura are typically in both eyes and to the same side. These may be flashing or shimmering lights, they may be in zig-zag patterns, and they can start centrally in a small area before gradually getting larger and moving towards the periphery of vision; or vice versa starting in the periphery and moving towards the central area of vision. Often, the field of vision alongside the area of flashing lights is lost or 'missing'.

Visual aura develop gradually over 5-10 minutes and can last for 30-60 minutes. Where vision loss last longer than one hour, there should be a strong suspicion that the cause is stroke and not migraine, particularly in individuals who have no prior history of such vision loss.

Vestibular conditions

The vestibular system involved parts of the inner ear and brainstem and process information that help control balance and eye movements. Typical examples of vestibular disorders are inner ear infection, Meniere's disease and benign paroxysmal positional vertigo (BPPV).

Symptoms of vestibular disorders include vertigo, dizziness and the feeling of being pulled in one direction. Patients may have poor balance and coordination and struggle to walk straight or when turning. Visual symptoms may include difficulty with slow tracking eye movements, double vision and juddering vision because of uncontrolled eye movements. There may be some hearing impairment in which hearing is distorted or lost on one side.

Combined symptoms of vertigo, vomiting, visual disturbance (vertical eye deviation), head motion intolerance and nystagmus are indicative of brainstem involvement due to stroke rather than a vestibular disorder.

Visual anatomy and physiology

Visual pathway (cranial nerve II) and visual field loss

[Adapted from 'Visual fields via the visual pathway textbook'[12]]

The normal monocular visual field extends 50-60 degrees superiorly, 60 nasally, 70-75 inferiorly and 90-100 temporally. The extent of visual field will vary with object/target size. As the optic disc has no retinal photoreceptors it forms the blind spot of the visual field.

The visual field is produced by retinal stimulation of each eye and relates to what is seen by the individual whilst maintaining steady fixation, i.e. the perceived vision of an individual. Retinal images are projected to a position opposite the area of retina stimulated, for example, objects that stimulate nasal retina are situated in the temporal visual field and objects that stimulate inferior retina are situated in the superior visual field.

A high percentage of nerve fibres arise from the macular area of the retina and pass directly to the optic disc (papillomacular bundle). Nerve fibres located further temporally in the peripheral retina (nasal field of vision) must arc above and below the macular fibres to enter the optic disc superiorly and inferiorly. Nerve fibres on the nasal side of the optic disc (temporal field of vision) pass directly to the nasal border.

Once in the optic nerve the macular fibres move to a central position with superior retinal fibres above and inferior retinal fibres below. Temporal and nasal nerve fibres retain their temporal and nasal location within the optic nerve.

On reaching the optic chiasm the temporal nerve fibres maintain their temporal position whilst nasal nerve fibres (both central and peripheral) decussate. Ipsilateral temporal nerve fibres and contralateral nasal nerve fibres regroup in the optic tracts but again with superior fibres retaining a more superior location to the inferior fibres.

Nerve fibres are distributed in a complicated multi-layered arrangement in the lateral geniculate nucleus of the lateral geniculate body with macular fibres distributed throughout the nucleus. Ipsilateral and contralateral peripheral nerve fibres are located in different layers

of the nucleus. There is a synapse of nerve fibres in the lateral geniculate body. Thus the entire visual pathway is a two-neurone pathway.

Fibres leaving the lateral geniculate body fan out to form the optic radiations, many of which pass directly posterior to the visual cortex. A proportion, however, initially pass anteriorly and laterally before turning posterior towards the visual cortex.

Within the striate visual cortex (area V1) the macular fibres terminate on the tip of the occipital lobe (occipital pole) whilst the more peripheral fibres terminate more anteriorly. The most peripheral fibres relating to the monocular crescent of each eye are the most anteriorly represented. Superior fibres are on the upper lip of the calcarine fissure whilst inferior fibres are on the lower lip. Figure 1 represents the afferent visual pathway and figure 2 outlines the different types of visual field loss that occur because of damage at different parts of the visual pathway.

Visual field loss

Visual field loss types that can occur following stroke affecting the visual pathway include hemianopia, quadrantanopia, altitudinal, sector defects and scotomas.

Altitudinal visual field defects involve two quadrants of either the superior or inferior visual field and is typically seen in an ocular stroke in which ischaemia has affected the central retinal artery which is a branch from the anterior brain circulation. The defect precisely respects the horizontal meridian. They may also be due to bilateral symmetric involvement at a cortical level including bilateral lesions affecting the occipital lobe. Hemianopia is a complete defect involving one half of the visual field. Homonymous hemianopia involves the same side of the visual field in each eye (e.g. lesions of the post-chiasmal pathways typically produce homonymous hemianopias).

Quadrantanopia is a complete defect involving a quadrant of each visual field. Homonymous quadrantanopias involve the same side of the visual field in each eye and either superior or inferior quadrants. These may be produced by temporal, parietal or occipital lobe lesions. Scotoma is an absolute or relative area of depressed visual sensitivity surrounded by normal

vision. Scotomas can be caused by retro-chiasmal visual pathway lesions in which bilateral homonymous scotomas occur in the left or right visual field and in the central, paracentral or peripheral visual field. Sector-shaped (wedge) visual field defect may be seen as a type of partial hemianopia in which a ‘wedge’ of the visual field loss extends to the very central area of vision.

Figure 1 Afferent visual pathway

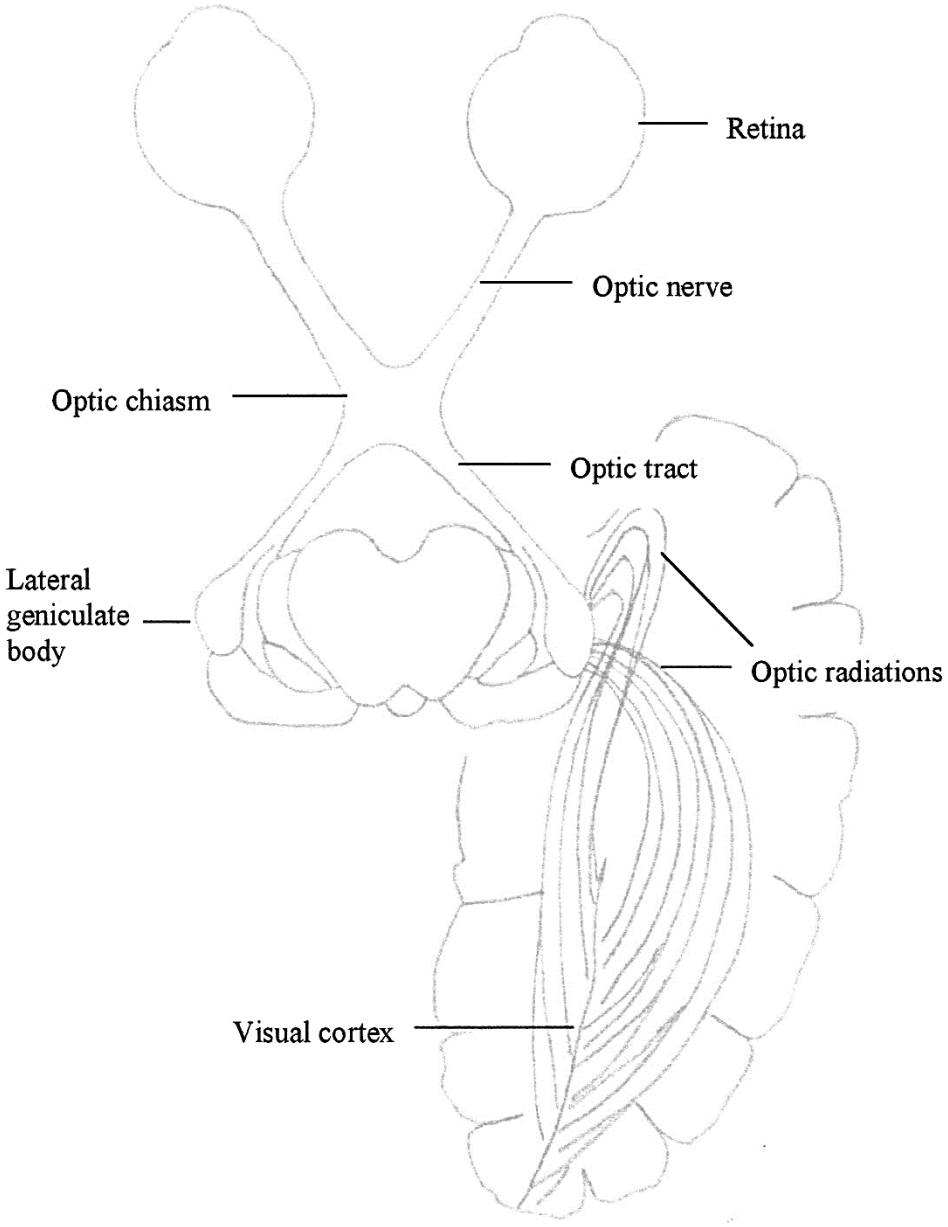
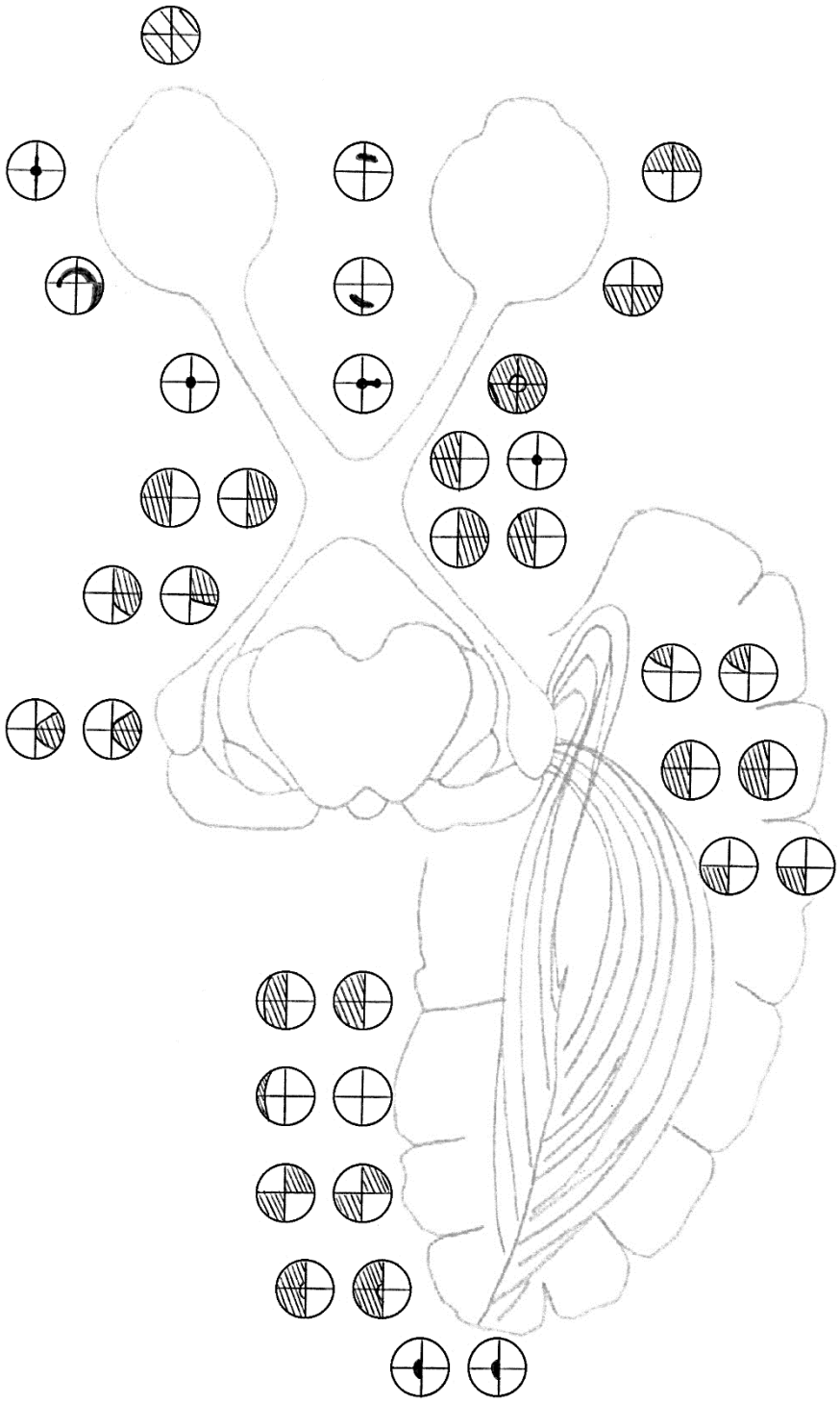


Figure 2 Visual field loss types



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Lids and Pupils

[Adapted from 'Clinical Orthoptics' textbook'[13]]

Lids

The levator muscle is the primary elevator of the upper lid. Its failure to function properly produces a ptosis (lid droop). The levator muscle is supplied by the III cranial nerve – see next section for detail.

Pupils

The sphincter pupillae receives parasympathetic nerve supply and constricts the pupil (miosis). This pupillary light reflex is a four neurone arc. The first order neurone passes from the retina to the pretectal nucleus. The second neurone passes from the pretectal nucleus to the Edinger-Westphal nucleus and the third neurone passes from there to the ciliary ganglion via the third cranial nerve. The fourth order neurone passes from the ciliary ganglion to the sphincter pupillae via the short ciliary nerves.

The dilator pupillae receives sympathetic nerve supply and dilates the pupil (mydriasis). This is a three neurone pathway. The first order neurone passes from the posterior hypothalamus to the cilio-spinal centre of Budge, situated from C8 to T2 in the spinal cord. The second neurone (pre ganglionic pathway) passes from the spinal cord to the superior cervical ganglion via the vertebral sympathetic chain. The third order neurone (post ganglionic pathway) passes from the superior cervical ganglion via the internal carotid artery, through the cavernous sinus to the short posterior ciliary nerves and on to the dilator pupillae.

Lid disorders

Ptosis may be present from birth or childhood, or it may have developed gradually in older adult years; but this will be known to the patient/family. Where ptosis is acquired in relation to a stroke, it may be linked to a facial palsy, to III nerve palsy (see next section) or associated with a pupil problem.

Pupil disorders

Pupil disorders may occur in one or both eyes. Where pupils are dilated (large), this is termed mydriasis. Where they are constricted (small), this is termed miosis. Where only the pupils are affected, it can be difficult to specify which is the affected eye; the bigger or the smaller pupil? In such instances, the term anisocoria should be used which merely indicates that on pupil is bigger/smaller than the other. Anisocoria can be seen in cases of intracranial haemorrhage. Acquired pupil disorders related to stroke may be linked to III nerve palsy (large pupil on affected side) and Horner's syndrome (small pupil on affected side along with a small ptosis).

Ocular motor pathways (cranial nerves III, IV and VI) and eye movement disorders

[Adapted from 'Clinical Orthoptics' textbook'[13]]

The extraocular muscles are innervated by the III, IV, and VI nerves.

III nerve

The III nerve (third/oculomotor) supplies four eye muscles in each eye; the superior rectus, inferior rectus, medial rectus, inferior oblique and levator muscles. Its visceral fibres innervate the ciliary muscle and sphincter pupillary muscle which synapse in the ciliary ganglion. These fibres cause the pupil to constrict (get smaller) and allow accommodation (increased focussing when looking at near objects).

The nuclei are in the upper midbrain the level of the superior colliculus. The nerve fibres emerge ventrally where they are closely associated with the posterior cerebellar and superior cerebral arteries. The nerve courses forward through the subarachnoid space to pierce the dura mater at the posterior clinoid process and enter the cavernous sinus.

IV nerve

The IV nerve (fourth/trochlear) supplies the superior oblique eye muscle. The nucleus lies in the midbrain at the level of the inferior colliculus. The nerve fibres cross over and emerge from the brainstem dorsally. The nerves curve around the brainstem and course forward through the subarachnoid space to pierce the dura mater and enter the cavernous sinus.

VI nerve

The VI nerve (sixth/abducens) supplies the lateral rectus. The nucleus is situated in the pons in the floor of the IV ventricle near the midline. The nerve fibres emerge from the brainstem ventrally and course forward and laterally over the petrous tip of the temporal bone and under the petrosphenoid ligament. The nerve pierces the dura mater to enter cavernous sinus. The nerve divides into two distinct trunks along its pathway between the brainstem and the lateral rectus muscle.

Common nerve pathways

The III, IV and VI nerves course forward together in the lateral aspect of the cavernous sinus entering the orbit through the superior orbital fissure. The III and VI nerves enter within the muscle cone.

VII nerve

The VII nerve (seventh/facial) serves sensory and motor functions. The VII nerve has central connections to the motor face area of the cerebral cortex and the nuclei are divided into upper and lower halves. Corticobulbar fibres double decussate for the upper face but there is single decussation for lower face fibres.

Sensory fibres: Ganglion cells supply taste buds in the palate and tongue and sensory fibres are also present in the skin, in and around the external acoustic meatus. Fibres pass to the geniculate ganglion situated in the internal auditory meatus and pass back to the pons.

Motor fibres: The nuclei are located in the lateral part of the pons and fibres loop around the abducens nuclei, forming the facial colliculus, before leaving the pons ventrally. Fibres pass anteriorly and enter the internal auditory meatus. The nerve enters a narrow bony canal above the labyrinth and descends to the stylomastoid foramen where a branch supplies the stapedius muscle. It leaves the skull and supplies the facial muscles (frontal, zygomatic, buccal, mandibular marginal and cervical branches).

VIII nerve

The VIII nerve (eight/auditory) serves the sensory functions of hearing and balance.

For the Vestibular nerve (balance), receptor cells are hair cells in the utricles, saccules and semicircular canals of the ears. Fibres pass from Scarpa's ganglion along the Vestibular nerve through the internal auditory meatus to the cisterna pontis and to the vestibular nuclei in the pons/medulla. Within the internal auditory meatus, the vestibular and cochlear nerves are in close association with the facial nerve. Within the acoustic foramen and intracranial cavity, these nerves are closely associated with both the sixth and facial nerves.

Cranial nerve palsies and nystagmus

Ocular cranial nerve palsies can include third, fourth, sixth and seventh nerve palsies. As the III nerve supplies the levator, superior rectus, inferior rectus, medial rectus and inferior oblique muscles along with carrying the parasympathetic efferent pupil fibres, a complete third nerve palsy will cause the signs of ptosis (lid droop), strabismus (an eye that is turned downwards and outwards) because of limited eye movements in upgaze and inward gaze, and pupil dilation. The IV nerve supplies the superior oblique muscle and will cause strabismus (an eye that is turned upwards) because of limited downgaze. The VI nerve supplies the lateral rectus muscle and will cause strabismus (an eye that is turned inwards) because of limited eye movements on outward gaze. The VII nerve supplies the facial muscles and is assessed as the 'face' part of the FAST test. The VIII nerve is closely involved in the fine control of eye movements. Damage to the vestibular pathway typically causes nystagmus – a condition in which the eyes continually move in a symmetrical but uncontrolled manner. There are multiple types of nystagmus which can include horizontal, vertical and/or rotary nystagmus and nystagmus that is present in all eye gaze positions or only when looking in one particular direction.

Saccades – fast eye movements

There are strong interconnections for saccades between the frontal and parietal lobes. The frontal eye fields in the precentral sulcus are involved with volitional, visually guided, purposive saccades and the parietal lobe is involved with a shift in attention to new targets appearing in the visual field. The inferior parietal lobe is involved with planning saccades and

the frontal eye fields with intentional visual exploration. Other areas involved in the saccadic system include the dorsomedial supplementary motor area which is important in learned ocular motor behaviour and the dorsolateral frontal cortex which is involved in the programming of saccades and attention shifts to remembered target positions.

Fibres pass from cortical areas down through the internal capsule. Below this level there are different pathways including basal ganglia (caudate nuclei), thalamic nuclei, superior colliculi, substantia nigra, vestibular and pontine nuclei and the pedunculopontine pathway to the cerebellum (vermis and fastigial nuclei) and to brainstem nuclei. There is probable decussation at the level of the third nerve nuclei in the midbrain. The superior colliculi (superficial and intermediate layers) are key structures in the generation of saccades. The basal ganglia also aid control of saccades.

Specific nuclei to generate horizontal and vertical saccades are located in the pons and upper midbrain. Horizontal saccades to the left direction are made by nuclei on the left side of the pons and vice versa for saccades to the right direction. Vertical saccades – upwards or downwards – are generated from both sides of the upper midbrain nuclei.

Smooth pursuits – slow, tracking eye movements

The brain requires information regarding target movement relative to the position of the head. Motion processing areas of the brain are therefore important in the production of a smooth pursuit eye movement. Visual information is received in the visual cortex and passed to areas 17 to 19 (occipital and parietal cortex) including the middle temporal visual area in the superior temporal sulcus, the medial superior temporal visual area and the posterior parietal area where speed and direction of moving stimuli are encoded. There is a link with the frontal eye fields which contain neurones which discharge during smooth pursuit and aid programming of predictive pursuit movements, initiation and maintenance. Input from the basal ganglia and thalamus aid smooth pursuit control.

Efferent fibres from cortical areas pass through the internal capsule under the superior colliculi at the midbrain with a probable double decussation at the level of pons, medulla and cerebellum, on to the dorsolateral pontine nuclei and the cerebellum (flocculus, paraflocculus and vermis) via the cerebral peduncles. The visual information that is passed to the

cerebellum is synthesised for pursuit signal and the cerebellum outputs to the vestibular nuclei which integrate the signal and passes this to the oculomotor nerves.

For horizontal smooth pursuit the signal is directed via the dorsolateral pontine nuclei, cerebellum, nucleus reticularis tegmenti pons and vestibular nuclei to the sixth nerve nucleus. For vertical smooth pursuit the signal is directed to the third and fourth nuclei. Medial vestibular nuclei and nucleus prepositus hypogloss link for velocity and position integration of horizontal smooth pursuit eye movements. The interstitial nucleus of Cajal encodes neural integration predominantly for vertical and cyclorotatory smooth pursuit.

Gaze palsies

Gaze palsies can be horizontal, vertical or combined dependent on the location and extent of stroke lesion. The sixth nuclei control horizontal conjugate gaze and govern conjugate movement of both the ipsilateral lateral rectus and contralateral medial rectus. The nuclei house two neurones; abducens motoneurones to the lateral rectus, and abducens internuclear neurones via the medial longitudinal fasciculus (MLF) to the contralateral medial rectus motoneurones in the third nuclei. In horizontal gaze palsy, both eyes cannot move from the centre position (looking straight ahead) to the right if a right brainstem stroke; or to the left side if a left brainstem stroke.

Vertical gaze is under bilateral control from the cerebral cortex. Ocular motoneurones are in the third and fourth nerve nuclei. For vertical saccadic eye movement saccadic generation is from rostral interstitial nucleus of the MLF. Neural signals for vertical vestibular and smooth pursuit eye movements ascend from the cerebellum, medulla and pons to midbrain oculo motor nuclei via the medial longitudinal fasciculus but also the superior cerebellar peduncle. In vertical gaze palsy, both eyes cannot look upwards (upgaze palsy) or downwards (downgaze palsy) or both up and downwards (complete vertical gaze palsy).

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