

# Eye Signs in Stroke

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## Abstract

A large part of the central nervous system is involved in the normal functioning of the vision, and hence vision can be affected in a stroke patient. Transient visual symptoms can likewise be a harbinger of stroke and prompt rapid evaluation for the prevention of recurrent stroke. A carotid artery disease can manifest as transient monocular visual loss (TMVL), central retinal artery occlusion (CRAO), anterior ischemic optic neuropathy or ocular ischemic syndrome (OIS). Stroke posterior to the optic chiasm can cause sectoranopias, quadrantanopias, or hemianopias, which can be either congruous or incongruous. Any stroke involving the dorsal stream (occipito-parietal lobe), or ventral stream (occipito-temporal lobe) can manifest with visuospatial perception deficits. Similarly, different ocular motility abnormalities can result from a stroke affecting the cerebrum, cerebellum, or brainstem. Among these deficits, vision and perception disorders are more difficult to overcome. Clinical, experimental, and neuroimaging studies have helped us to understand the anatomical basis, physiological dysfunction, and the underlying mechanisms of these neuro-ophthalmic signs.

**Keywords:** Dorsal stream, neuro-ophthalmic signs, quadrantanopias, vision

## INTRODUCTION

Neurologists or physicians may be the first point of medical contact for patients presenting with neuro-ophthalmic disorders. The eye is a “window to the brain,” and a thorough ocular examination can assist clinicians to understand the mechanism of acute neurologic deficits. Poststroke visual impairment is common. It is seen in 48% stroke admissions and 60% of stroke survivors with 56% presenting impaired central vision, 40% eye movement abnormalities, 28% visual field loss, 27% visual inattention, and 5% with visual perceptual disorders.<sup>[1]</sup>

### Purpose of review

The objective of this review was to help the clinician to identify common neuro-ophthalmic signs in stroke patients, which will aid in ordering appropriate investigations and making a therapeutic and rehabilitation plan.

## METHOD

We searched the literature for studies describing “Eye signs and stroke” from Jan 2002 to March 2022 in PubMed databases. PubMed search was done using (‘Eye signs in stroke’[Title/Abstract]) OR (‘Eye signs and stroke’[Title/Abstract]) OR (‘Neuro ophthalmic manifestations of stroke’[Title/Abstract]) OR (‘Neuro ophthalmic manifestations and stroke’[Title/Abstract]) OR (‘Higher cortical visual disorders’[Title/Abstract]). We included review articles, original articles, case series, and case reports. Conference presentations were excluded. A search with the keywords mentioned above led to 1,924 articles from PubMed for “Eye signs and stroke”, 1026 articles for ‘Higher cortical visual disorders’ and 129 articles for ‘Neuro ophthalmic

manifestations of stroke’ The abstracts were reviewed by the authors and articles relevant for this review were selected. Thirty-five articles and three textbook references were included for this review.

## RESULTS

This review will cover common eye signs in stroke patients, anatomical localization, underlying neural mechanisms and how to elicit them. The algorithm for examining eye signs in a patient with stroke has been depicted in Figure 1.

### Eye signs have been divided into the following categories

- Eye signs related to afferent visual system
- Eye signs related to higher cortical function
- Eye signs related to efferent visual system

### I. EYE SIGNS RELATED TO AFFERENT VISUAL SYSTEM

Ophthalmic symptoms and signs may occasionally be the only manifestation of a stroke/transient ischemic attack (TIA)

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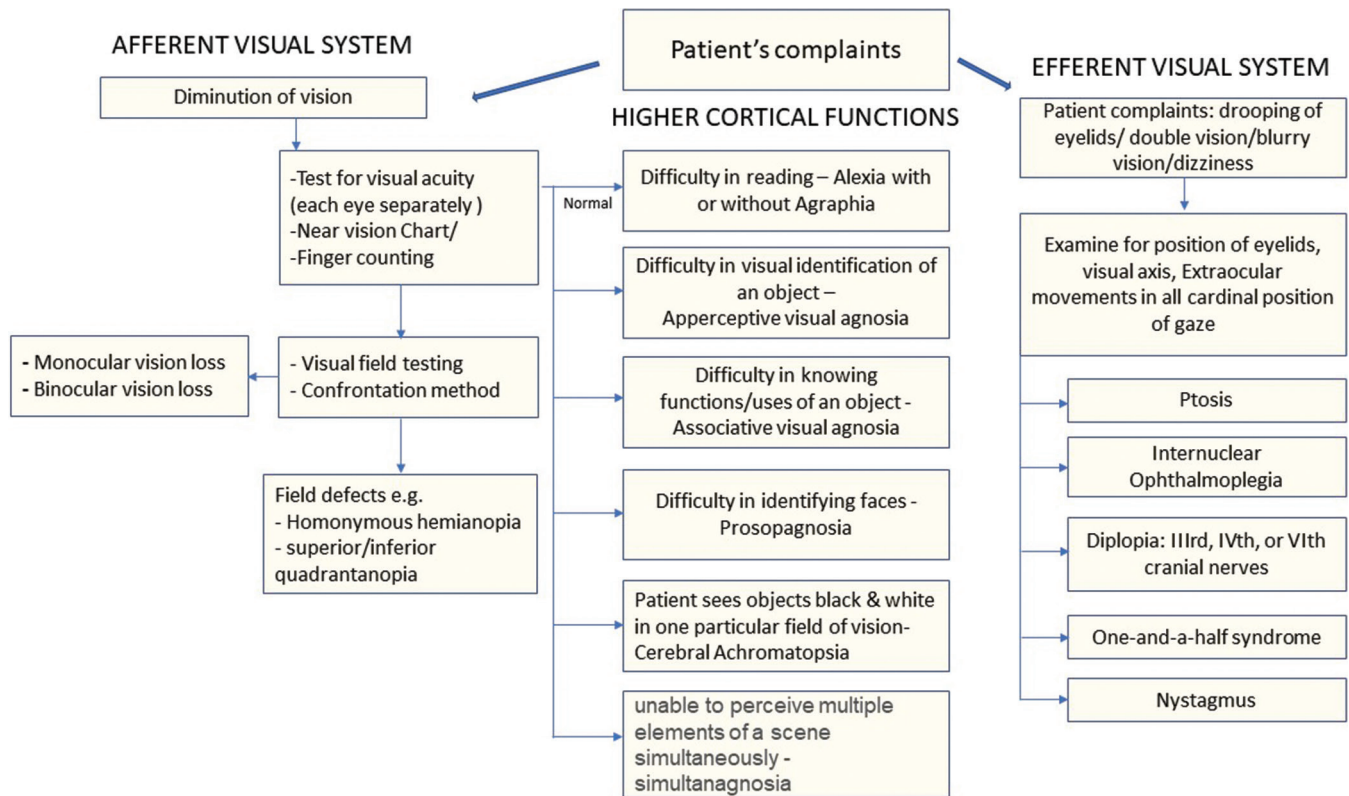
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**Figure 1:** Algorithm for examining eye signs in a patient with stroke

due to carotid artery disease. Hemianopia may be a clue to a stroke involving the retro chiasmal visual pathway and rarely the chiasm.

### Transient monocular visual loss or amaurosis fugax

Emboli originating in the carotid artery can enter the ophthalmic artery and can cause either a transient monocular visual loss (TMVL) or amaurosis fugax. A “curtain” or “shade” dropping over the vision is the traditional description.<sup>[2]</sup> This is because the arteries supplying the inferior retina are more susceptible to ischemia. The duration of these episodes ranges from a few seconds to a few minutes, and they rarely exceed 15 minutes.

### Iris diaphragm type of TMVL

It should be noted that the visual symptoms experienced with internal carotid artery (ICA) hypoperfusion unlike in amaurosis fugax results in the constriction of the visual fields start from the periphery and moving toward the centre.<sup>[2]</sup>

Retinal hypoperfusion due to internal carotid stenosis can also cause Bright light amaurosis or transient amaurosis after exposure to bright light (retinal claudication).<sup>[3]</sup> Transient amaurosis brought on by a change in head position, a heavy meal or exercise may be other features.<sup>[4]</sup>

### Examination of the face

**Prominent pulsation of the superficial temporal artery** can occur due to compensatory hyper-perfusion of the ECA with

a subjective bruit over the ipsilateral ear. It can be reduced by compressing the temporal artery (**Olivares’ external carotid sign**).<sup>[5]</sup> **Dilated episcleral arteries** may be seen due to retrograde filling of the ophthalmic arteries.<sup>[6]</sup>

### Visual field examination

#### Paracentral Scotoma

Central retinal artery branch occlusions (CRABO) where only the upper, lower, nasal, or temporal portion of the retina may be involved, and a small central or paracentral scotoma may be the only manifestation of a carotid disease.<sup>[7]</sup>

**Contralateral homonymous superior quadrantanopia**, (pie-in-the-sky) along with seizure, memory deficits and sensory aphasia can occur with inferior temporal lobe infarction (Meyer’s Loop) [Figure 2, lesion D].

**Homonymous inferior quadrantanopia** (pie-on-the-floor) can occur on the contralateral side along with hemineglect with superior parietal lobe infarction [Figure 2, lesion E].

**Bitemporal hemianopia.** Strokes involving the optic chiasma (supplied by branches from the Circle of Willis) are rare and can cause bitemporal hemianopia [Figure 2, lesion B].

**Incongruous hemianopias** can arise from the optic tract and lateral geniculate body (LGB) ischemia, and they become more congruous with more posteriorly placed lesions since the fibers of the visual pathway converge in the occipital lobe.<sup>[8]</sup>



**Visual agnosia**

It is defined as a neuropsychological disorder characterized by failure to recognize familiar objects in patients who apparently have adequate sensory functions (e.g., normal vision and normal sensitivity to luminance in their visual fields), and this disorder cannot be explained by other cognitive deficits like aphasia, poor semantic memory, or low intellectual capacity.<sup>[16]</sup>

Visual Agnosia has been divided into 1) **Apperceptive visual agnosia** 2) **Associative visual agnosia**

Differentiating two forms of agnosia is shown in **Table 1**

**Testing visual agnosia**

Visual agnosia is diagnosed by examining the patient’s capacity to name an object, describe its use, and pantomime the usage of visually presented objects.

**Matching Test:**

**(a) View-matching task:** In this test, the patient has to match the sample to the target image, which is presented along with similar-looking objects. Patients with apperceptive agnosia cannot complete this task correctly as they are not able to generate a coherent visual perception of an object [**Figure 4**].

**(b) Function-match task:** The subject has to match those two pictures that share a common function. Deficits in performing this task are distinguishing features of associative agnostic patients [**Figure 4**].

**Cerebral Achromatopsia**

These deficits arise when a lesion disrupts area V4 in the inferior occipital cortex, which is specialized for color vision processing in the contralateral hemifield [**Figure 5a**]. The area V4 also plays an important role in processing forms, such as the aspect ratio and spatial frequencies of a visual stimulus.<sup>[18]</sup>

**Clinical Presentation:** Patients with hemiachromatopsia presents with altered color perception in which the deficit is limited to one homonymous visual field. It is an isolated disorder of color perception, without other visual deficits (visual acuity, brightness, etc.) [**Figure 5b**].

**Testing:** Hemiachromatopsia is tested by moving a colored object (a pin with a red ball at the top) from one visual field to the other and asking the patients if there are any changes in the color of the object.<sup>[19]</sup>

**Prosopagnosia**

It is a specific form of visual agnosia in which face perception is impaired while elementary aspects of vision, such as acuity and visual field, remain intact. More recently, studies using Positron-emission tomography (PET) and functional magnetic resonance imaging (fMRI) have demonstrated that face recognition areas are located in the fusiform face area, which is part of the fusiform (occipitotemporal) gyrus.<sup>[20]</sup>

**Etiology:** 1) Bilateral posterior cerebral infarcts 2) head trauma 3) herpes encephalitis, particularly if bilateral 4) tumors 5) lobar hemorrhages, and 6) surgical resections for epilepsy. Progressive forms can occur with focal temporal atrophy from frontotemporal degeneration.

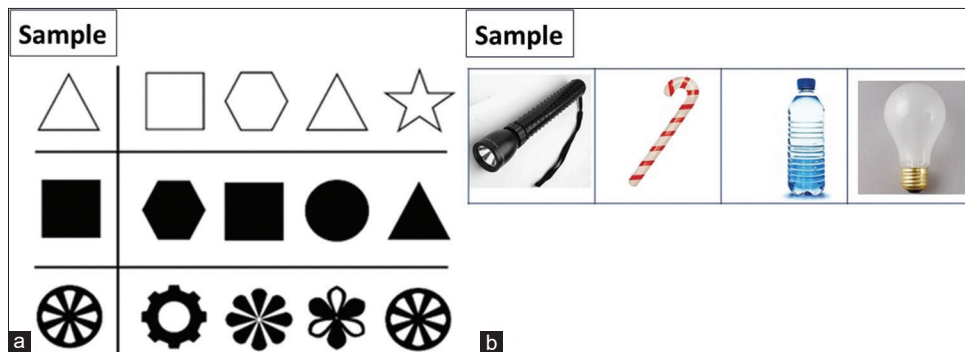
**Testing of Prosopagnosia**

To diagnose prosopagnosia, the clinician should first confirm that the patient does not have a general visual agnosia, by administering a basic object recognition test. The Boston Naming Test is commonly used for this purpose. Patients are presented with images of celebrities and unknown people and asked to indicate which faces look familiar. A patient with prosopagnosia has decreased ability to make this distinction.<sup>[21]</sup>

**Table 1: Differentiating the forms of visual agnosia**

	Apperceptive	Associative
Object feature description	×	✓
Visual identification	×	×
Copying line drawings	×	✓
Object matching	×	✓
Object knowledge (from name)	✓	×
Tactile naming	✓	×

(Adapted from John R. Hodges. Testing cognitive function at the bedside. In: Cognitive Assessment for Clinicians. 3rd Ed. Oxford University Press; Oct 2017)<sup>[17]</sup>



**Figure 4:** Illustration of matching tasks in which agnostic patients depending on type of agnosia typically show deficits. (a) View-matching task; (b) Function-match task

## Bálint syndrome

Diagnosis of the Bálint syndrome requires the presence of all three core symptoms: simultanagnosia, optic ataxia, and ocular apraxia. Each symptom can be found in patients without the others, indicating that they result from damage to independent processing networks that have in common a spatial analysis of vision. Clinically, it is recognized by deficits in simultaneously processing multiple items resulting in the poor interpretation of complex visual scenes, deficits in visually guided limb movements, eye movements, and sensory inattention.<sup>[22]</sup>

**Tests for simultanagnosia:** Navon letter and lady cooking on chullah can be used to test simultanagnosia. A patient with simultanagnosia see only small letter and ignore large letter when asked to describe a Navon letter, and similarly, the interpretation of the number of people present is slow and often inadequate when they are asked to describe a lady cooking on chullah [See Figure 6].<sup>[21,23]</sup>

## Optic ataxia

It is an inability to accurately point to or clench objects under visual guidance. The lesions that cause optic ataxia are present in the occipitoparietal area involving the junction between the superior occipital cortex and the inferior parietal lobule.<sup>[21]</sup>

## Acquired Ocular Motor Apraxia

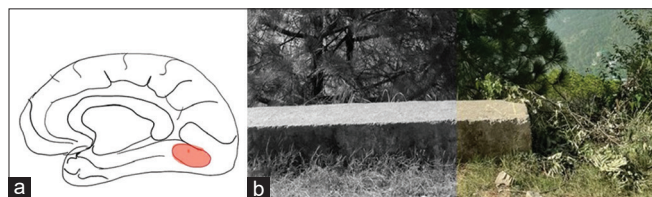
Bilateral lesions can cause a defect in the initiation and guidance of saccades to visual targets. Patients with severe acquired ocular motor apraxia may have trouble generating any saccades in response to a visual stimulus on command, however saccades can sometimes be helped by blinking; this difficulty is also known as psychic paralysis of gaze.<sup>[21]</sup>

## Cerebral Akinetopsia

Akinetopsia is a condition in which a moving item appears to “jump” from one stationary position to another due to an inability to detect motion. Cerebral akinetopsia is a relatively unusual condition caused by bilateral lesions in the human visual motion area (V5/MT).

## III. EYE SIGNS RELATED TO EFFERENT VISUAL SYSTEM

Stroke can affect the efferent visual system, including the extraocular muscles, cranial nerves or the supranuclear pathways, either individually or in combination producing a spectrum of ophthalmological symptoms.



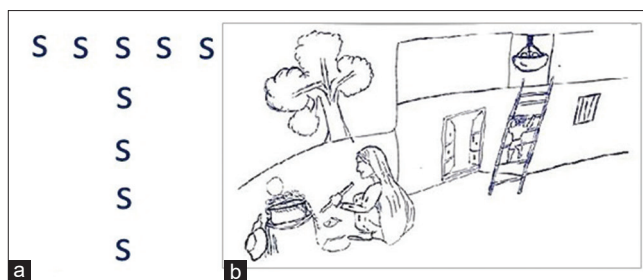
**Figure 5:** A lesion circumscribed to area V4 in the inferior occipital cortex [area highlighted in red]. (5a) Loss of color vision perception in the contralateral homonymous visual field. (5b)

## 1. Ptosis:

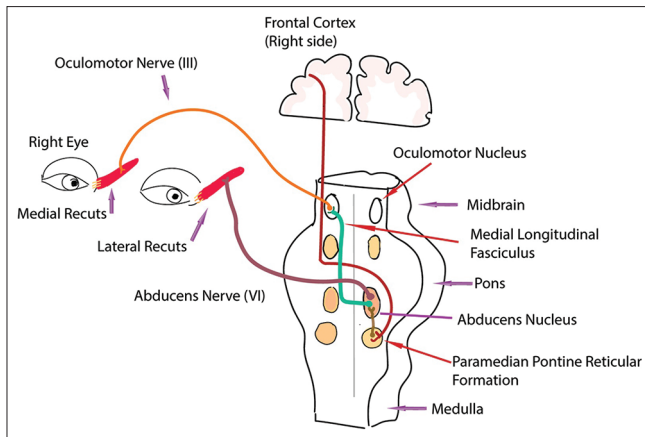
Pure midbrain strokes constitute about 0.6-2.3% of total strokes.<sup>[24]</sup> Infarction of the paramedian midbrain or the oculomotor fasciculus can manifest with isolated unilateral ptosis without ophthalmoplegia or pupillary involvement.

## 2. Oculomotor dysmotility:

- i. **Oculomotor nerve (CN III) palsy** was observed to have the highest rate of occurrence in patients with stroke, according to a retrospective review.<sup>[25]</sup> Ptosis, diplopia, mydriasis and gaze palsies, along with hemiparesis or cerebellar signs may occur. Midbrain infarction results in a constellation of ipsilateral third nerve palsy with contralateral hemiparesis called Weber's syndrome. An aneurysm of the posterior communicating artery (PCOM), which lies in close proximity to the CN III can result in external compression resulting in mydriasis and ophthalmoparesis.
- ii. **Diplopia** is a common presenting complaint after stroke. According to a prospective study, diplopia was seen in 16.5% of patients after stroke.<sup>[26]</sup> Involvement of the IIIrd, IVth, or VIth cranial nerves can result in horizontal or vertical ocular misalignment presenting as diplopia. The rate of occurrence of diplopia is equal with the involvement of either cerebral hemisphere.
- iii. **Internuclear Ophthalmoplegia:** INO is characterized by ipsilateral impaired adduction, partial or complete, along with a contralateral horizontal nystagmus of the abducting eye. It occurs due to a lesion of the medial longitudinal fasciculus, which connects the ipsilateral CN III nucleus to the contralateral paramedian pontine reticular formation (PPRF) and the adjacent CN VI nucleus [Figure 7].<sup>[27]</sup> Infarction of the MLF is the most common etiology of INO in the elderly.<sup>[26]</sup>
- iv. **Gaze Palsies:** The most common ocular motility dysfunction seen after a stroke is gaze palsy reported in 18-44% of patients.<sup>[28]</sup> Horizontal gaze palsy is more common than vertical gaze palsy, and complete palsy is more than partial.<sup>[29]</sup> A stroke involving the PPRF at the pons can result in an ipsilateral horizontal gaze palsy, whereas that involving the frontal cortex rostral to the precentral cortex can result in an interruption of input from the frontal eye fields with a subsequent contralateral gaze palsy.



**Figure 6:** (a) Navon letter (b) lady cooking on chullah. (Adapted from Prasad K, Dash D, Kumar A. Validation of the Hindi version of National Institute of Health Stroke Scale. *Neurol India*. 2012 Jan-Feb; 60 (1):40-4.)<sup>[23]</sup>



**Figure 7:** Neural pathway of horizontal eye movements. (Adapted from Alberstone CD, Benzel EC, Najm IM, Steinmetz MP, editors. *Anatomic Basis of Neurologic Diagnosis*. 1<sup>st</sup> ed. New York, NY: Thieme Medical Publishers; 2009. p. 427.)<sup>[27]</sup>

The Rostral interstitial nuclei of the medial longitudinal fasciculus (riMLF) are the final center for the integrated neural pathways for vertical gaze. Midbrain and thalamic infarcts can result in vertical gaze defects.<sup>[29]</sup> The constellation of impaired upgaze, lid retraction (Collier sign), convergence-retraction nystagmus, downward gaze preference (setting-sun sign), light-near dissociation (poor response of pupils to light but better to accommodation) is called Parinaud syndrome or dorsal midbrain syndrome. It occurs usually due to a pineal tumor compressing the midbrain, or an infarct or tumor of the midbrain preectum. Complete vertical gaze palsy is indicative of bilateral riMLF lesions.

**v. One-and-a-half syndrome:**

The combination of an ipsilateral horizontal gaze palsy (one) with ipsilateral INO (a half) is termed one and a half syndrome. This in combination with ipsilateral facial nerve involvement is called eight and a half syndrome.<sup>[30]</sup> Brainstem infarction followed by demyelinating disorders like multiple sclerosis are among the most common causes.

**vi. Saccades:** Saccades are rapid, conjugate, voluntary eye movements that help shift your gaze toward an object of interest. A right frontal lobe lesion will result in a transient left gaze palsy with a right gaze preference, which resolves when the PPRF regains saccadic control. Brainstem lesions can also cause saccadic defects. Ipsilateral hypermetric saccades with contralateral hypometric saccades can occur with infarctions of the brainstem and cerebellum.

**vii. Pursuits:** The pursuit system allows the eyes to smoothly follow a moving object. The neural circuit of the smooth pursuit is a subject of debate. There is some evidence to suggest that descending projections from the primary visual cortex, frontal eye fields, temporo-parieto-occipital region, are likely involved in the initiation of pursuit,<sup>[31]</sup> with inputs from pons and cerebellum. Ipsilateral

occipitoparietal lesions can result in ipsilateral impaired smooth pursuit. Cerebellar infarctions involving the vermis have also been reported to inhibit smooth pursuits.<sup>[32]</sup>

**viii. Nystagmus:** Cerebellar stroke is reported to have the highest association with acute nystagmus.<sup>[33]</sup> One study reported a 24% prevalence of nystagmus in patients with posterior circulation stroke.<sup>[34]</sup> Posteroinferior cerebellum infarction can result in bidirectional horizontal nystagmus, whereas the involvement of the superior vermis causes an upbeat nystagmus.

**ix. Strabismus and Vergence:** Strabismus has been reported to occur in 16.5- 50% of patients<sup>[35,36]</sup> with stroke, with a higher prevalence (69%) in cortical strokes. Exotropia is the most common (74%) type of strabismus noted.<sup>[37]</sup> Convergence insufficiency has been reported in almost 55% patients after stroke.<sup>[38]</sup>

## SUMMARY

The first contact physician needs to be vigilant as visual symptoms and signs may be the only manifestation of stroke. Visual symptoms in stroke patients can occur as vision loss, visual perceptual disorders, or disorders of ocular motility. Elicitation of different eye signs in stroke patients not only helps in anatomical localization and ascertaining etiology but also guide in making a diagnostic and therapeutic plan.

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## Conflicts of interest

There are no conflicts of interest.

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