Approach to the adult with acute persistent visual loss

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INTRODUCTION

Acute vision loss is a frightening experience for patients and has the potential for long-term consequences. The many causes of acute vision loss and the time-sensitive need for evaluation and treatment pose diagnostic and therapeutic challenges [1].

A careful history is key to narrowing the differential diagnosis and will allow for a more focused yet systematic physical examination. Prompt diagnosis and treatment may influence the visual outcome.

This topic will present an overview of the approach to patients with acute persistent visual loss. The differential diagnosis of the red eye and specific causes of acute vision loss are discussed separately. Transient visual loss, also known as amaurosis fugax, should be treated as a transient ischemic attack. Most transient ischemic attacks last less than one hour. (See "Amaurosis fugax (transient monocular or binocular visual loss)" and "The red eye: Evaluation and management".)

DEFINITION OF TERMS

Acute persistent visual loss is defined as lasting at least 24 hours and is typically not caused by transient ischemia [2]. By contrast, acute transient visual loss is defined as a sudden deficit in visual function in one or both eyes lasting less than 24 hours. It is caused by a temporary

vascular occlusion in the circulation to the eye or visual cortex, or by neuronal depression after a seizure or migraine [3]. (See "Amaurosis fugax (transient monocular or binocular visual loss)".)

ETIOLOGY AND CLINICAL MANIFESTATIONS

Visual pathway anatomy — Alterations in function of any of the structures along the visual pathway may cause vision loss. Pathology can be broadly divided into three major anatomic categories: media, retina, and neural visual pathway (table 1).

To achieve clear vision, light must follow an unhindered path from the front to the back of the eye, traveling through the cornea, aqueous humor, lens, and vitreous humor to the retina (figure 1). Refracted by the cornea and lens (and perhaps also by glasses or contact lenses), light is focused onto the retina where it is transformed into an electrochemical signal by photoreceptors and supporting cells. The signal is transmitted via the optic nerve through the visual pathways to the occipital lobes.

Media problems

Corneal abrasion and keratitis — A corneal abrasion is the result of mechanical trauma to the cornea resulting in loss of epithelial cells. Keratitis is inflammation of the cornea due to trauma, abrasive exposure, allergy, or infection. It is marked by cloudiness, irregularity, or loss of epithelial or sub-epithelial corneal tissues. Typically the eye is tearing, red, and painful or irritated, and there is often a foreign-body sensation. (See "Corneal abrasions and corneal foreign bodies: Management", section on 'Diagnosis'.)

On physical examination, loss of epithelial cells is demonstrated by corneal uptake of fluorescein dye, creating a focal or diffuse green glow under a cobalt blue light. Deeper corneal disease may be visible on ophthalmoscopic examination as a focal or diffuse white opacity, or by dulling of the usually distinct reflection of light off of the cornea (corneal light reflex). Evaluation of abnormal findings requires ophthalmologic expertise in order to arrive at a precise diagnosis.

The most common causes of keratitis are infection (viral, bacterial, fungal, or parasitic) and trauma. Bacterial keratitis is usually associated with contact lens wear. Corneal abrasion and keratitis can also cause corneal edema. Patients suspected of having infectious keratitis should be evaluated by an ophthalmologist within 24 hours. (See "Herpes simplex keratitis" and "Complications of contact lenses", section on 'Infectious keratitis' and "The red eye: Evaluation and management".)

Patients with a simple corneal abrasion without history of associated occult infection or embedded corneal foreign body can typically be treated by the initial provider, and symptoms should resolve in 24 hours. (See "Corneal abrasions and corneal foreign bodies: Management".)

Corneal edema — Corneal swelling results in loss of corneal clarity. It most commonly occurs secondary to corneal abrasion, epithelial or stromal keratitis, or acute glaucoma. Physical examination may reveal dulling of the corneal light reflex or a frank grey or white color to the substance of the cornea.

A serious cause of sudden corneal edema is acute angle-closure glaucoma. The patient will have severely elevated intraocular pressure (IOP), typically associated with nausea and vomiting and may see colored halos around lights. The eye is tearing, red, and extremely painful, often with ipsilateral brow ache. Patients may have throbbing, intermittent, or constant unilateral headache, which rarely may simulate migraine [4]. In angle closure, the pupil may be fixed in a mid-dilated position, and biomicroscopic (slit lamp) examination may reveal a shallow anterior chamber. IOP is often dangerously elevated (usually 40 to 80 mmHg). If formal tonometry is not available, severely elevated IOP may be suspected by noting hardness to palpation in comparison to the fellow eye (tested sequentially rather than together). Patients suspected of having acute angle-closure glaucoma should be triaged immediately to the emergency department. Treatment is discussed elsewhere. (See "Angle-closure glaucoma".)

Hyphema — A hyphema is blood in the anterior chamber. It may result from blunt trauma or may occur spontaneously in a handful of conditions marked by abnormal growth or fragility of iris blood vessels (eg, poorly controlled diabetes mellitus).

Biomicroscopic examination reveals red blood cells circulating and/or layered in the anterior chamber (see "Traumatic hyphema: Clinical features and diagnosis"). Hyphema can induce dangerously high eye pressure and possible permanent corneal blood staining. Patients with hyphema should be evaluated by an ophthalmologist within 24 hours. Management requires detailed, often daily, examination to ensure that rebleeding does not occur. (See "Traumatic hyphema: Management".)

Lens changes — Changes in the size, clarity, or positioning of the crystalline lens may alter the focus of light onto the retina, resulting in visual disturbance. Trauma or a variety of congenital conditions can lead to lens dislocation and resultant vision loss. Lens clouding (cataract) is painless and does not typically occur acutely, except in the setting of trauma. (See "Cataract in adults" and "Cataract in children" and "Ectopia lentis (dislocated lens) in children".)

Elevated blood glucose can cause increased lens tumescence, altering the refractive error. If the change is great enough, patients may perceive painless vision loss. Vision impairment typically

resolves within days to weeks of normalization of blood glucose.

Lens changes can best be seen after pupillary dilation with a slit lamp biomicroscope.

Vitreous hemorrhage — Bleeding into the vitreous humor can occur in the setting of trauma, spontaneous retinal tear, spontaneous vitreous detachment, or any condition with retinal neovascularization such as poorly controlled diabetes mellitus.

Typical symptoms include painless floaters and sometimes flashes of light (photopsias).

The reduction in vision is directly proportional to the amount of blood in the vitreous. If the hemorrhage is dense enough, there may be a decreased red reflex (the reddish orange reflection off the subretinal layers when examining the eye with an ophthalmoscope), or the retina may not be visible on fundoscopic examination. Patients suspected of having vitreous hemorrhage should be evaluated by an ophthalmologist within 48 hours.

Uveitis — Uveitis is the general term for inflammation inside the eye generally caused by autoimmunity or infection. Trauma may also cause inflammation of the anterior structures of the eye. Inflammation of the anterior structures of the eye is typically associated with redness and painful light sensitivity, whereas isolated inflammation of intermediate and posterior structures may be associated with normal general appearance of the eye, a decrease in the red reflex, and/or patient complaint of new floaters and sometimes photopsias. Biomicroscopic examination reveals white blood cells in the anterior chamber, vitreous space, or both. Posterior uveitis may manifest as whitening of the retina or choroid. A hypopyon (white blood cell accumulation) may form in the anterior chamber. Corneal edema and a decreased red reflex may also be present. (See "Uveitis: Etiology, clinical manifestations, and diagnosis".)

- **Endophthalmitis** – The most important cause of acute persistent visual loss in this category is acute endophthalmitis. Endophthalmitis is a serious bacterial or fungal infection of all intraocular tissues, caused by surface organisms (usually in the setting of recent ocular surgery) or bloodborne pathogens. Patients suspected of having endophthalmitis should be evaluated by an ophthalmologist within 24 hours. (See "Bacterial endophthalmitis" and "Treatment of endogenous endophthalmitis and chorioretinitis due to Candida species" and "Treatment of exogenous endophthalmitis due to Candida species" and "Treatment of endophthalmitis due to molds".)

- **Acute retinal necrosis** – Acute retinal necrosis (also known as acute viral panuveitis) is a rare and serious cause of visual loss. It is usually caused by herpesviruses and is discussed elsewhere. Patients suspected of having acute retinal necrosis should be evaluated by an ophthalmologist within 24 hours. (See "Epidemiology, clinical manifestations, and

diagnosis of herpes zoster", section on 'Acute retinal necrosis' and "Retinal vasculitis associated with systemic disorders and infections", section on 'Infectious causes'.)

Other etiologies for uveitis are discussed elsewhere. (See "Uveitis: Etiology, clinical manifestations, and diagnosis", section on 'Etiology'.)

Retinal problems

Central retinal artery occlusion — The central retinal artery is the first branch of the ophthalmic artery and provides blood supply to the inner retina. Interruption in perfusion results in ischemic retinal damage leading to severe, sudden, painless, central or paracentral visual loss. A central retinal artery occlusion (CRAO) is the retinal equivalent of a cerebral vascular accident (stroke) and is typically caused by thromboembolism seen in patients with cardiovascular risk factors. Other etiologies such as hypercoagulability, hypotension, and, importantly, giant cell arteritis (GCA; also known as temporal arteritis) must be considered [5]. It is typically unilateral but may be bilateral in cases of a cardiac source of thromboembolism. (See "Central and branch retinal artery occlusion".)

Within minutes to hours of the occlusion, the only abnormality noted on funduscopic examination may be vascular narrowing. An embolus is visible in about 20 percent of patients with CRAO [5]. After several hours, the inner layer of the retina becomes ischemic, turning milky white except in the fovea, which appears as a cherry-red spot. An afferent pupillary defect is typically present. (See 'Physical examination' below.)

Patients suspected of having CRAO should be triaged immediately to the emergency department for a stroke workup (including head/neck imaging) and possible ocular interventions. Proposed interventions are numerous, but most include an immediate attempt to lower the IOP with digital massage, paracentesis, or pharmacologic agents. Treatment is discussed elsewhere. (See "Central and branch retinal artery occlusion".)

The most common cause of CRAO is cardiovascular disease leading to a thromboembolic event. The most important alternative cause is GCA. Patients with GCA are at risk for any type of ischemic ophthalmic event, most notably retinal artery occlusions, and ischemic optic neuropathies. GCA is discussed below and elsewhere. (See 'Optic nerve lesions' below and "Diagnosis of giant cell arteritis".)

Central retinal vein occlusion — Patients with central retinal vein occlusion (CRVO) usually complain of the acute onset of painless blurred vision in one eye. The thrombosis results in venous stasis, leading to disc swelling, diffuse nerve fiber layer/preretinal hemorrhages, and cotton wool spots that create a dramatic appearance on funduscopic examination, often called

"the blood and thunder" fundus (picture 1). (See "Retinal vein occlusion: Epidemiology, clinical manifestations, and diagnosis" and "Retinal vein occlusion: Treatment".)

While vision loss may be severe, the onset is typically subacute in contrast to the sudden visual loss typical of CRAO. When venous stasis is severe, infarction may occur due to slowed retinal blood flow on the arterial side. In this setting, a relative afferent pupillary defect is often present. Patients suspected of having CRVO should be evaluated by an ophthalmologist within 48 hours.

Retinal detachment — Detachment of the neurosensory retina may occur spontaneously or in the setting of trauma. The most common form is related to a tear or break in the retina in patients with a preexisting area of retinal thinning (usually highly myopic, or nearsighted, individuals). Patients may describe sudden onset of new floaters or black dots in their vision, often accompanied by photopsias. Symptoms do not include pain. In its early stages, a detachment may present as a persistent missing portion of the monocular visual field. Once the macula (central retina) has become involved, visual acuity will be severely compromised. Patients suspected of having retinal detachment should be evaluated by an ophthalmologist within 24 hours. (See "Retinal detachment".)

Retinal detachment does not cause a red eye. There may be a dulling of the red reflex, and funduscopic examination may reveal the retina to be elevated with folds or cause the retina to be difficult to visualize with a direct ophthalmoscope. If the detachment is extensive, there may be a relative afferent pupillary defect.

Acute maculopathy — Conditions that affect the macula are associated with a central blind spot (scotoma), blurred vision, or visual distortion. Alteration of the macula due to fluid leakage, bleeding, infection, inflammation or other causes can occur de novo or as an acute worsening of a chronic disease (eg, new edema in previously dry diabetic retinopathy, or new bleeding in previously dry macular degeneration). Diagnosis usually requires detailed ophthalmoscopy with a high magnification lens through pharmacologically dilated pupils and specialized ophthalmic imaging such as optical coherence tomography (OCT). Patients suspected of having acute maculopathy should be evaluated by an ophthalmologist within 48 hours.

Neural visual pathway problems — Patients with neural pathway visual loss must be evaluated immediately for additional neurologic symptoms, with a low threshold for additional studies as described below.

Optic nerve lesions — Optic nerve lesions usually produce monocular visual loss. Optic neuritis is the most common cause of optic nerve disease in younger adults, while ischemic optic neuropathy is the most common etiology in older patients. Acute visual loss can also occur

when orbital cellulitis spreads to infect and damage the optic nerve. (See "Optic neuropathies" and "Orbital cellulitis", section on 'Clinical manifestations'.)

- **Ischemic optic neuropathy** – Ischemic optic neuropathy refers to ischemic damage to the optic nerve due to an interruption in its blood supply, leading to severe, sudden, painless vision loss. It is the optic nerve equivalent of a stroke. Vision loss may be diffuse, but infarction occurring at the optic disc may result in a superior or inferior altitudinal defect.

  The occlusion and resultant damage may be anterior (affecting the optic disc) or posterior (retrobulbar), and etiology may be due to GCA (arteritic) or due to other causes of ischemia (non-arteritic). GCA must be ruled out or treated in order to reduce risk of contralateral vision loss. Most embolic ischemic optic neuropathy occurs in patients with cardiovascular risk factors. Nonarteritic anterior ischemic optic neuropathy (NAION) is the most common form of ischemic optic neuropathy and is discussed in detail elsewhere. (See "Nonarteritic anterior ischemic optic neuropathy: Clinical features and diagnosis".)

  An afferent pupillary defect is typically present (see 'Physical examination' below). In anterior optic neuropathy, the optic disc is swollen, often accompanied by splinter hemorrhages (picture 2 and picture 3). If nerve damage occurs posterior to the globe, no abnormalities may be seen on initial ophthalmoscopic examination.

  GCA as the cause of CRAO or ischemic optic neuropathy is more common in older patients (>50 years), women, and persons of Scandinavian descent. Patients may present with a temporal headache, scalp tenderness, jaw claudication, fever, weight loss, fatigue, and/or night sweats. The reported incidence of ocular involvement ranges between 14 and 70 percent [6]. An elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) may suggest the diagnosis. Imaging studies such as vascular ultrasound, magnetic resonance imaging (MRI), and positron emission tomography/computed tomography (PET/CT) play an increasingly important role in diagnosis; the gold standard is a temporal artery biopsy [7]. (See "Diagnosis of giant cell arteritis".)

  If GCA is suspected, high-dose IV solumedrol or high-dose oral prednisone should be initiated emergently. Steroid therapy should never be delayed for biopsy confirmation, which may take many days to schedule. Histological abnormalities will persist for weeks after initiation of treatment [8-10]. In the absence of GCA, and in the absence of other neurologic deficits, patients should be referred to an ophthalmologist or neuro-ophthalmologist within 24 hours. (See "Treatment of giant cell arteritis".)

- **Optic neuritis** – Optic neuritis is the most common cause of optic nerve disease in younger adults. Inflammation of the optic nerve may be associated with a variety of

conditions, most notably multiple sclerosis. Optic neuritis is the presenting feature in 15 to 20 percent of patients with multiple sclerosis, and it occurs at some time during the course of the disease in 50 percent of patients [11]. (See "Optic neuritis: Pathophysiology, clinical features, and diagnosis".)

Affected patients note pain on eye movement, reduced visual acuity, and washed-out color vision. An afferent pupillary defect is typically present (see 'Physical examination' below), and the optic disc is either normal or swollen on ophthalmoscopic examination. Noninfectious, demyelinating optic neuritis is typically managed with intravenous solumedrol given within 24 to 48 hours of onset of symptoms. Patients should be referred to an ophthalmologist or neuro-ophthalmologist within 24 to 48 hours.

Distinguishing isolated optic neuritis and ischemic optic neuropathy may be difficult at initial presentation. Diffusion-weighted MRI and post-contrast enhancement characteristics of the intraorbital segment of the optic nerve may help differentiate the two [12].

- **Papilledema** – Papilledema is bilateral, sometimes asymmetric, optic nerve head swelling due to elevated intracranial pressure. Patients with newly discovered papilledema require a brain MRI with and without contrast and thorough neurologic examination to exclude the presence of a mass lesion. Patients with accompanying severe headache/nausea vomiting should be sent to the emergency department for immediate evaluation. Chronic elevated intracranial pressure can lead to transient visual obscurations or mild persistent blurred vision, often accompanied by positional headaches and pulsatile tinnitus or whooshing. Examination reveals bilateral optic nerve swelling without relative afferent pupillary defect. In the absence of a mass lesion on imaging, stable, non-toxic patients require non-emergency lumbar puncture with assessment of opening pressure and cerebral spinal fluid analysis (see "Overview and differential diagnosis of papilledema"). Papilledema may be subtle on examination, often prompting second opinion examination by a trained ophthalmologist.

Chiasmal and retrochiasmal disorders — Disorders of the optic pathways posterior to the optic nerve include chiasmal and retrochiasmal disorders. Chiasmal disorders are typically associated with either a monocular visual field defect or a bitemporal hemianopia:

- **Homonymous hemianopia** – Brain lesions in the region of the optic tract, the lateral geniculate body, the optic radiations, or the visual cortex produce a loss of vision on one side of both visual fields (figure 2). The visual field defect is relatively symmetric and respects the vertical midline. No other abnormalities are seen on ophthalmic examination.

The most common cause is stroke or hemorrhage into a brain tumor. These patients often interpret the field cut as an “eye problem” (ipsilateral to the field cut). They often complain of bumping into things or being surprised by objects as they emerge from the side of decreased vision (see "Homonymous hemianopia"). A bitemporal hemianopia is typical of a chiasmal lesion, and a right- or left-sided homonymous hemianopia is typical of a retrochiasmal lesion.

Any acute vision loss associated with a chiasmal or retrochiasmal disorder requires an emergency neurologic examination and brain MRI with and without contrast to rule out stroke or brain tumor.

Compressive chiasmal lesions typically cause gradual decline in vision, as they impinge upon the chiasm, optic nerve, or optic tract. Peripheral vision loss is often asymptomatic until visual acuity is compromised; therefore, most visual complaints are of gradual blurring or dimming of vision. Sudden chiasmal vision loss is less common and implies a rapidly expanding mass or an infectious, vascular, or inflammatory cause.

Chiasmal visual field defects classically involve the temporal fields and respect the vertical midline in one or both eyes. Defects of ocular motility, due to palsies of the third, fourth, or sixth cranial nerve, may accompany chiasmal lesions that extend into the cavernous sinus. Ophthalmoscopic examination may reveal optic disc pallor if the process is longstanding. Optic disc swelling may result from papilledema (with third ventricle obstruction causing elevated intracranial pressure) or from an infiltrative or inflammatory process.

Pituitary apoplexy is relatively rare, usually occurring in patients with preexisting (but often undiagnosed) pituitary adenomas that undergo hemorrhage or infarction. Emergency steroid and other hormonal replacement, and/or transsphenoidal surgery, is often indicated. The clinical syndrome is characterized by the acute onset of visual loss, sudden-onset severe headache, ophthalmoplegia, and altered mental status due to hemorrhage or infarction of the pituitary gland, resulting in rapid expansion into the suprasellar space and cavernous sinuses. Clinical suspicion mandates emergency MRI, as CT may miss the lesion [13]. (See "Causes of hypopituitarism", section on 'Pituitary apoplexy'.)

Retrochiasmal disorders can result in isolated vision loss or may be associated with other neurologic deficits corresponding to the location of the brain lesion.

Cortical blindness — Extensive bilateral damage to the cerebral visual pathways may result in complete loss of vision. Rarely, patients may confabulate and deny blindness, exhibiting a condition known as Anton syndrome. Other than blindness, no other abnormalities are seen on ophthalmic examination.

Psychogenic problems — Patients describing visual loss without an organic basis are said to have "functional" visual loss. Patients who are purposely feigning blindness are malingerers, whereas those who truly perceive blindness have a functional neurological symptom disorder (conversion disorder). (See "Functional neurological symptom disorder (conversion disorder) in adults: Terminology, diagnosis, and differential diagnosis", section on 'Malingering' and "Functional neurological symptom disorder (conversion disorder) in adults: Clinical features, assessment, and comorbidity", section on 'Visual symptoms'.)

The visual loss may be monocular or binocular, total, or subtotal. Malingerers have been known to pharmacologically alter pupillary function.

Once organic disease has been ruled out, sophisticated ophthalmic techniques and slights of hand may be required for proper diagnosis. As a general rule, clinicians should be cautious about diagnosing a psychogenic problem without a thorough evaluation and specialist consultation to exclude treatable organic causes.

APPROACH TO THE PATIENT

Health care practitioners in the primary care or urgent care setting most often do not have the equipment or expertise to perform a comprehensive ophthalmic examination. However, key history and physical examination findings can help determine the general category, if not the specific etiology, of vision loss.

A symptomatic approach to the differential diagnosis is presented in a table (table 2) and in an algorithm (algorithm 1).

An algorithmic approach — Key steps in the algorithm are listed below and can help clinicians make an initial determination of the most likely diagnostic entities, or referral strategies (algorithm 1):

- Three questions in the initial evaluation can help narrow down the diagnostic possibilities and referral plan:
  - Recent ocular surgery in the affected eye – These patients should see an ophthalmologist immediately for evaluation
  - Contact lens users – If there is a red, painful eye, the first consideration is corneal abrasion, keratitis, or a corneal ulcer
  - History of trauma – See algorithm for limited differential diagnosis of likely considerations

- Evaluation for pain in the affected eye:
    - If there is no pain, the next step is to evaluate for homonymous field loss and refer to algorithm for further details of the differential diagnoses
    - If there is pain, evaluate for ocular erythema, and follow algorithm

History — The history of present illness should include inquiry about the following:

- **Verification of acuity** – A distinction must be made between acute visual loss and the sudden discovery of preexisting visual loss. For example, if the normal eye is inadvertently covered, a longstanding or gradual loss of vision in the other eye may be mistaken for acute loss. Importantly, clinicians should approach patients who are symptomatic at the time of presentation within the first 24 hours as acute persistent visual loss, even if the duration is less than 24 hours.

- **Laterality** – Bilateral acute vision loss often suggests a retrochiasmal visual pathway disorder.

- **Quality** – Monocular loss of a portion of peripheral vision suggests subtotal retinal detachment, ischemic optic neuropathy, or branch retinal vascular occlusion. However, patients with a homonymous visual field defect often perceive vision loss only in the eye ipsilateral to the visual field cut.

- **Pain** – Keratitis produces a sharp superficial pain, acute glaucoma produces a deep brow ache with nausea and vomiting, endophthalmitis and certain uveitic conditions may also produce a deep boring pain, and optic neuritis produces pain worse with eye movement. Giant cell arteritis (GCA) is often associated with headache. Other causes of vision loss are typically painless.

- **Redness** – Patients with keratitis, acute glaucoma, and uveitis usually present with conjunctival hyperemia (red eye).

- **Associated symptoms** – Neurologic deficits can accompany a stroke (homonymous defect) or multiple sclerosis (optic neuritis). Patients with GCA may have shoulder and hip pain of associated polymyalgia rheumatica, fever, fatigue, weight loss, jaw claudication, and scalp tenderness. Nausea and vomiting accompany severely elevated intraocular pressure (IOP). Rhinorrhea is common when tearing is present. Some patients with monocular or binocular vision loss complain of a vague sense of disorientation or difficulty with balance and depth perception.

- **Ocular trauma** – Mild trauma can cause corneal abrasion, keratitis or uveitis; more severe trauma can cause hyphema, vitreous hemorrhage, traumatic cataract, traumatic lens dislocations, retinal detachment, or traumatic optic neuropathy. Trauma with a sharp or blunt object may produce a ruptured globe. (See "Overview of eye injuries in the emergency department" and "Approach to diagnosis and initial treatment of eye injuries in the emergency department" and "Open globe injuries: Emergency evaluation and initial management".)

- **Preexisting visual problems** – Patients who have preexisting visual loss on the contralateral side to the new symptoms should be approached with a great deal of urgency to exclude or treat a new vision-threatening problem of their "good eye."

Elements of the past medical history should focus on the following:

- **Vascular disease** – Patients with diabetes mellitus, coronary artery disease, hypertension, hypercoagulability, or vascular risk factors are at increased risk for retinal vascular occlusion, ischemic optic neuropathy, or visual pathway stroke. Patients with preexisting diabetic retinopathy can experience a vitreous hemorrhage or acute maculopathy.

- **Refractive status** – Highly nearsighted (myopic) eyes have a higher incidence of retinal tears, leading to retinal detachment [14]. Patients who have had refractive surgery to correct for myopia still have increased risk. Highly farsighted (hyperopic) eyes have a higher incidence of acute angle-closure glaucoma.

- **Contact lens wear** – Contact lens wear is a risk factor for corneal abrasion or microbial keratitis, especially with soft lenses and when lenses are used improperly (poor hygiene, overnight wear, contaminated solutions) [15]. (See "Complications of contact lenses".)

- **Eye surgery** – Previous procedures may put certain patients at increased risk for uveitis, corneal edema, acute glaucoma, maculopathy, or retinal detachment. Cataract surgery, the most commonly performed ophthalmic procedure in Americans over age 65, can lead to retinal detachment in 0.36 to 2.9 percent of cases within 10 years of surgery [16]. Rates of cataract-surgery associated endophthalmitis have been decreasing over the last decade, with an overall reported incidence of 0.04 percent [17].

- **Medications** – Many systemic medications are associated with ocular side effects. Most produce visual symptoms gradually with high dosages and/or with prolonged use. Medications that are associated with acute visual loss include:

    - Anticholinergics – Loss of accommodation, angle-closure glaucoma

- Bisphosphonates – Uveitis
- Digoxin – Yellow vision
- Rifabutin – Uveitis
- Sildenafil – Blue vision, ischemic optic neuropathy
- Sulfonamides – Myopia
- Topiramate – Angle-closure glaucoma [18]
- Oral Contraceptives – Ischemic, retinal, or optic nerve events [19]
- Fingolimod – Macular edema [20]
- A variety of cancer therapy drugs (biologics, small molecule inhibitors chemotherapies) – Retinopathy, uveitis, acute dry eye [21]

Physical examination — A systematic eye examination will help to localize the problem and therefore inform the differential diagnosis. This examination should include the following elements:

- General inspection – Noting erythema, tearing, light sensitivity, proptosis, ptosis, and temporal artery tenderness or nodularity.

- Visual acuity – This must be formally tested in every patient with a visual complaint, with glasses, one eye at the time. If glasses are not available and the patient has a refractive error, a pinhole occluder can be used to correct visual acuity. If one is not available, simply punching a tiny hole in an index card or other piece of stiff cardboard or paper will suffice. Patients with macular disease will have no improvement in vision. (See "Visual impairment in adults: Refractive disorders and presbyopia", section on 'Screening and diagnostic tests'.)

- Evaluation of extraocular movement.

- Confrontation visual fields, as follows:

  - With the patient seated approximately 1 to 2 feet in front of the examiner, the examiner instructs the patient to cover their left eye with the left hand. The patient fixates their right eye to look straight ahead at the examiner's left eye. This fixation of gaze is to be maintained by the patient and any movement of the eye away from the target should be corrected by the examiner. The examiner closes their right eye and says: "I will present a wiggling or moving finger on the edge of your vision and slowly bring it towards the center. As soon as you see movement, say 'yes.'" The presentation of a wiggling finger slowly from the periphery to the center, repeated in each quadrant, will give a sense of the exact location and shape of the visual field.

- Abnormalities in the visual pathways posterior to the optic chiasm can be distinguished by the presence of a homonymous visual field defect, often detectable with confrontation field testing (figure 2).

- Pupils – Symmetry, reactivity to light, pupillary reflex, carefully assessing for an afferent pupil defect. An afferent pupillary defect is demonstrated by shining a light alternately in one eye and then the other and finding that the direct response to light is more sluggish or absent in the affected eye. If swung quickly between the two eyes, the pupil of the affected eye may paradoxically dilate to the light being shone on it. The room should be dark, and the patient should fixate on a distant target to prevent miosis due to accommodation (figure 3 and picture 4). (See "The detailed neurologic examination in adults".)

  The presence of an afferent pupillary defect is fairly specific for unilateral optic nerve pathology and generally does not occur with media or retinal problems

- Fluorescein application. Fluorescein staining is seen in keratitis, corneal abrasion, and corneal edema.

- IOP testing (by tonometry or palpation).

- Pen light or slit lamp examination (with testing for red-reflex symmetry) (see "Slit lamp examination"). Alterations in the red reflex are seen in most media opacities and in retinal detachment.

- Ophthalmoscopic examination. Subtle retinal findings are best visualized with a detailed examination following pupil dilation with mydriatic eyedrops. The fundus can be viewed in a variety of ways. The direct ophthalmoscope and the pan-ophthalmoscope are handheld viewing devices typically used during a routine physical examination. These techniques provide a high magnification monocular, non-stereoscopic view of the optic nerve and macula. Indirect ophthalmoscopy is performed with a head-mounted lamp and a handheld lens but may not be available or familiar to the untrained. It provides a binocular stereoscopic wide-angle view best for examining the peripheral retina. Biomicroscopy of the fundus with the slit lamp and a handheld lens provides a binocular, stereoscopic, magnified view best for fine details on the retina.

- Ocular ultrasound is increasingly being used by emergency and critical care clinicians both to diagnose patients with visual symptoms and to estimate intracranial pressure [22,23]. In one multicenter study of 225 patients, accuracy for detecting retinal detachment (96.9 percent sensitive and 88.1 percent specific) and vitreous hemorrhage (81.9 percent

sensitive and 82.3 percent specific) as compared with the gold standard of the ophthalmologists' diagnosis was excellent [24]. Point-of-care ocular ultrasound can also measure optic nerve sheath diameter as a proxy for intracranial pressure. In a meta-analysis comparing ultrasound with computed tomography (CT; n = 478 patients from 12 studies), the performance characteristics of optic nerve sheath diameter were excellent (96.5 percent sensitivity and 92.3 percent specificity) [25].

- Retrochiasmal tumors may also cause palsy of the sixth cranial nerve.

SUMMARY AND RECOMMENDATIONS

- **Definition** – Acute persistent visual loss, defined as a sudden deficit in visual function in one or both eyes lasting more than 24 hours, is distinguished from acute transient visual loss (amaurosis fugax). (See 'Definition of terms' above.)
- **Etiology** – Conditions that cause acute persistent visual loss can be divided into three categories: media problems, retinal problems, or neural visual pathway problems (table 1).
  - Media problems include corneal abrasion and keratitis, corneal edema, hyphema, lens changes, vitreous hemorrhage, and uveitis.
  - Retinal problems include vascular occlusion, retinal detachment, and acute maculopathy.
  - Neural visual pathway malfunction may be subdivided into optic nerve lesions, chiasmal, and retrochiasmal visual pathway pathology. (See 'Etiology and clinical manifestations' above.)
- **Approach to the patient** – Health care practitioners in the primary care or urgent care setting most often do not have the equipment or expertise to perform a comprehensive ophthalmic examination. However, a thorough history and eye examination can help determine the general category, if not the specific cause, of vision loss (table 2 and algorithm 1). (See 'Approach to the patient' above.)

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Topic 6902 Version 46.0

GRAPHICS

Examples of acute persistent visual loss by anatomy

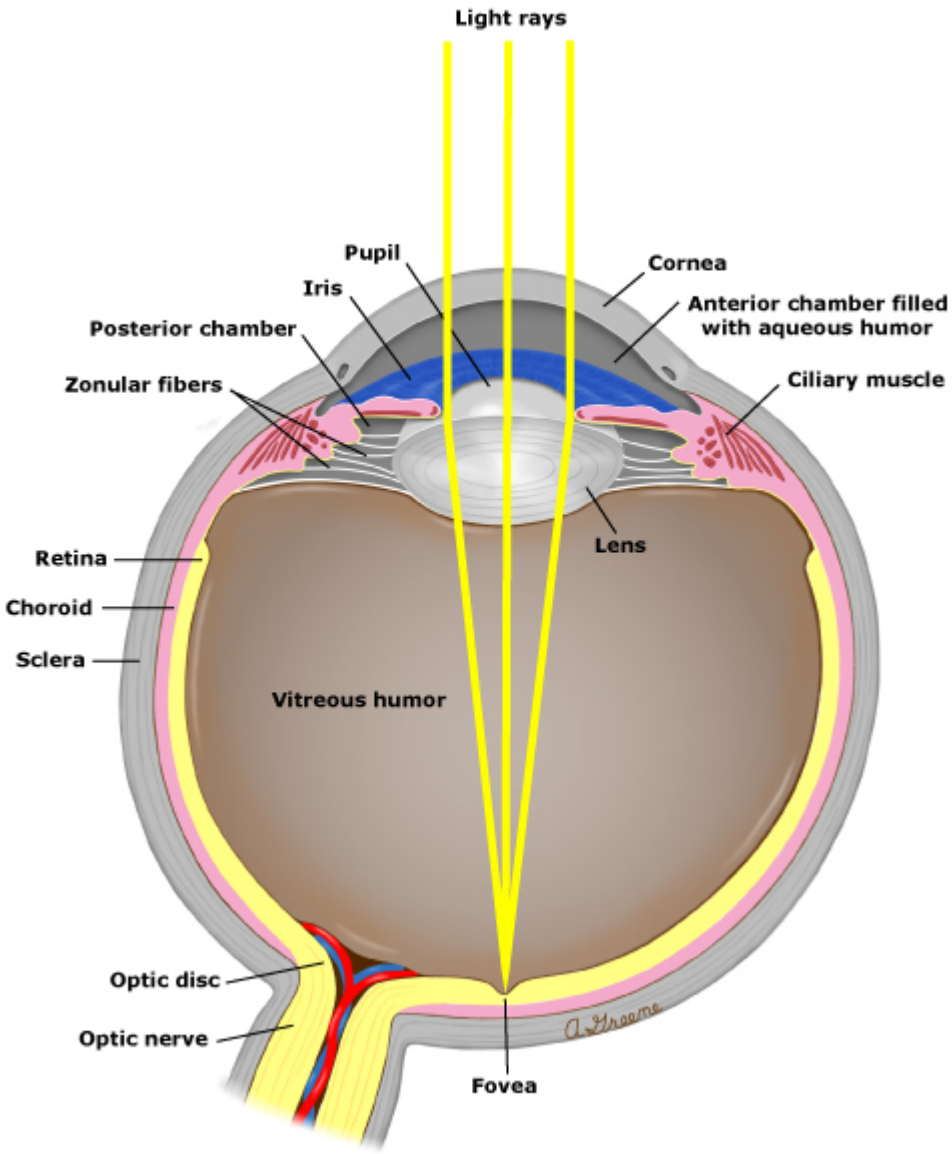
Media problems
Keratitis
• Infectious or noninfectious
Corneal edema
• Acute glaucoma
Hyphema
• Spontaneous or traumatic
Alterations in crystalline lens
• Thickening, clouding, or dislocation
Uveitis
Vitreous hemorrhage
• Spontaneous or traumatic
Retina problems
Retinal vascular occlusion (CRAO, CRVO)
Retinal detachment
Acute maculopathy
Problems of the neural visual pathway
Optic neuropathy
• Ischemia, inflammatory, or infectious process
Papilledema
• Elevated intracranial pressure
Chiasmal disorders
• Pituitary apoplexy
Retrochiasmal disorders
• Brain lesions in the visual pathway
• Posterior reversible leukoencephalopathy (PRES)
Other considerations

Trauma as a cause of many of the above
Acute glaucoma
• Vision loss due to corneal edema and/or optic nerve ischemia in the setting of severely elevated intraocular pressure
Functional visual loss

CRAO: central retinal artery occlusion; CRVO: central retinal vein occlusion; PRES: posterior reversible encephalopathy syndrome.

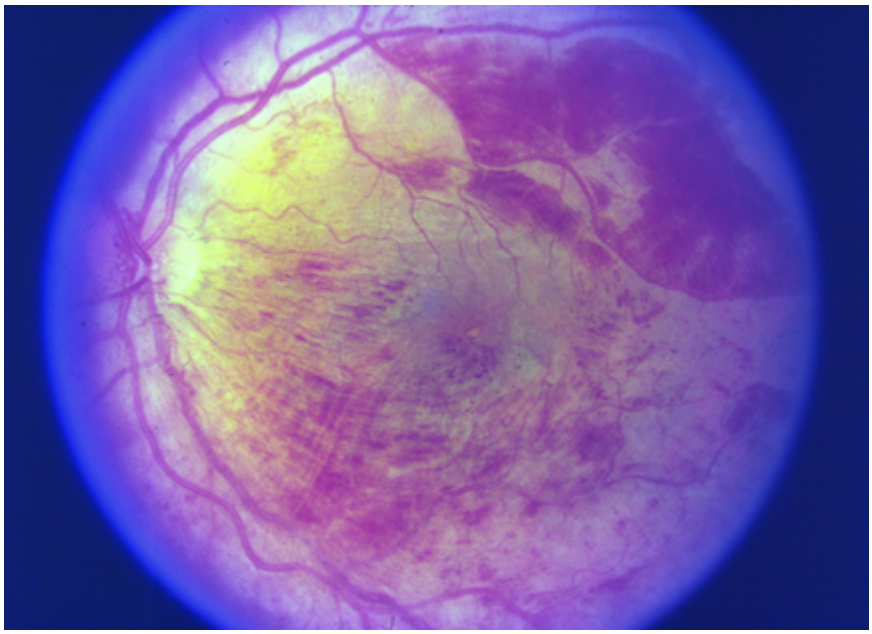
Graphic 61855 Version 11.0

Anatomy of the eye



Graphic 67844 Version 2.0

Retinal vein occlusion

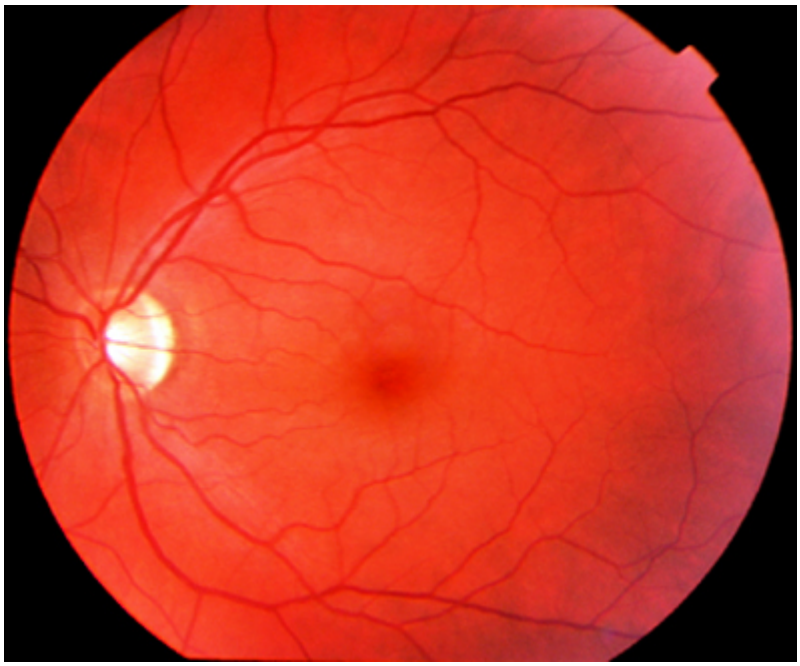


Thrombosis of the central retinal vein results in venous stasis, leading to disc swelling, diffuse nerve fiber layer and preretinal hemorrhage, and cotton wool spots that create a dramatic appearance, often called "the blood and thunder" fundus.

Courtesy of Don C Bienfang, MD.

Graphic 72976 Version 2.0

Normal fundus appearance



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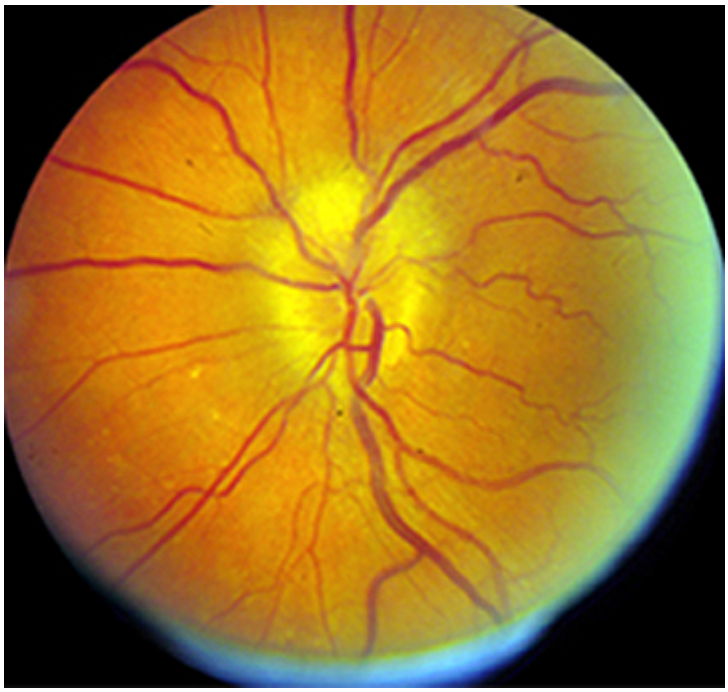
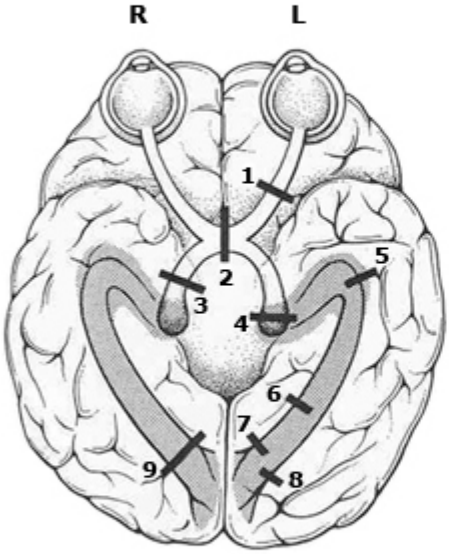
Optic neuritis fundus



Papillitis: Note the absence of hemorrhages and exudates in the setting of optic neuritis.

Graphic 50320 Version 1.0

Anatomy of the visual pathways and visual field correlation (view of underside of brain)



Location		Field defect		
		Left eye	Right eye	Comment
1	Left optic nerve			No light perception left eye
2	Chiasm			Bitemporal hemianopsia
3	Right optic tract			Incongruous left homonymo hemianopia
4	Left lateral geniculate nucleus			Right homonymous sectorar (lateral choroidal artery)
				OR
				Incongruous right homonym hemianopia
5	Left temporal lobe			Right homonymous upper quadrant defect ("pie in the
6	Left parietal lobe			Right homonymous defect, denser inferiorly
7	Left occipital lobe (upper bank)			Right homonymous lower quadrananopsia (macular sp
8	Left occipital lobe (lower bank)			Right homonymous upper quadrananopsia (macular sp
9	Right occipital lobe			Left homonymous hemianop (macular sparing)

Graphic 74068 Version 3.0

Causes of acute persistent visual loss by presentation

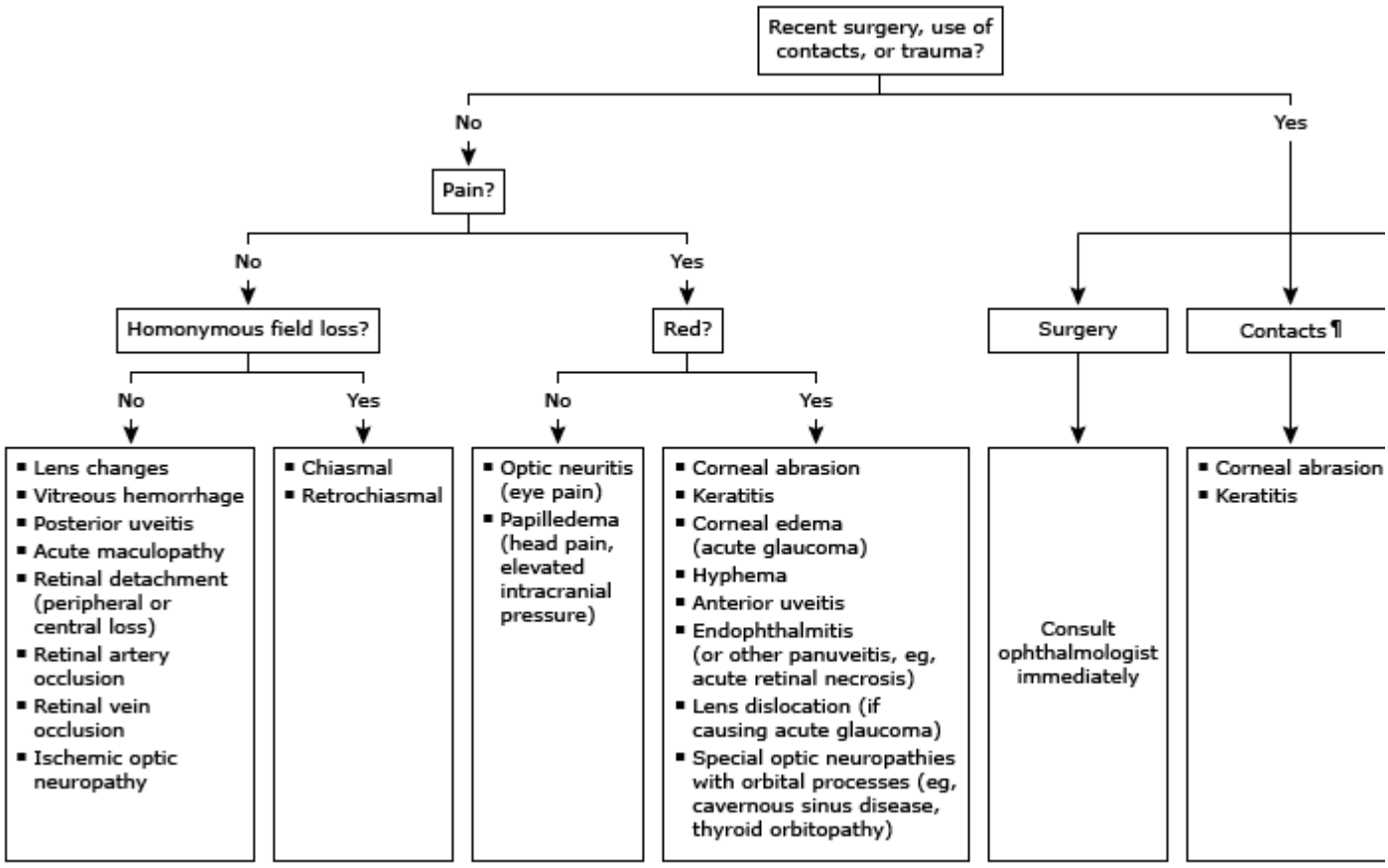
Presentation	Differential diagnosis
Unilateral painless	Lens dislocation
	Vitreous hemorrhage
	Acute maculopathy
	Retinal detachment
	Retinal artery occlusion
	Retinal vein occlusion
	Ischemic optic neuropathy
Unilateral painful	Corneal abrasion
	Keratitis
	Acute glaucoma
	Hyphema
	Endophthalmitis
	Anterior uveitis
	Optic neuritis
Bilateral painless	Pseudotumor cerebri (variable symptoms)
	Metabolic or toxic (hyperglycemia, methanol toxicity)
	Homonymous field loss (chiasmal or retrochiasmal etiology)
Bilateral painful	Keratitis from bilateral exposure (eg, contact lens, UV light, chemical, etc)

UV: ultraviolet.

Adapted from Guluma, K. An Evidence-Based Approach to Abnormal Vision. Emergency Medicine Practice 2007; 9:1.

Graphic 72615 Version 3.0

Considerations in diagnosing acute persistent visual loss*

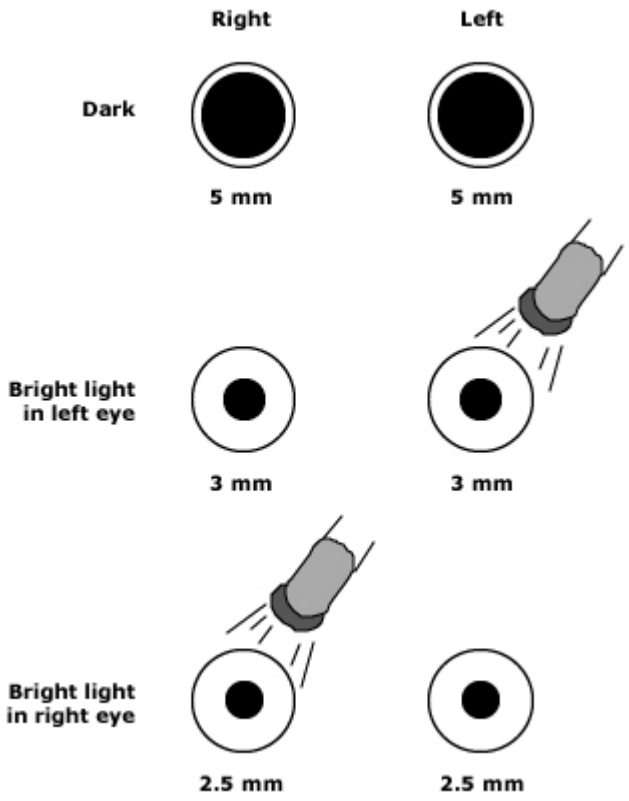


* These are considerations only. Clinical picture must otherwise fit.

¶ While corneal abrasion and keratitis are most common in patients who wear contact lenses, other sources of acute persistent visual loss should be considered if findings do not confirm abrasion or keratitis.

Graphic 50345 Version 4.0

Left afferent pupillary defect

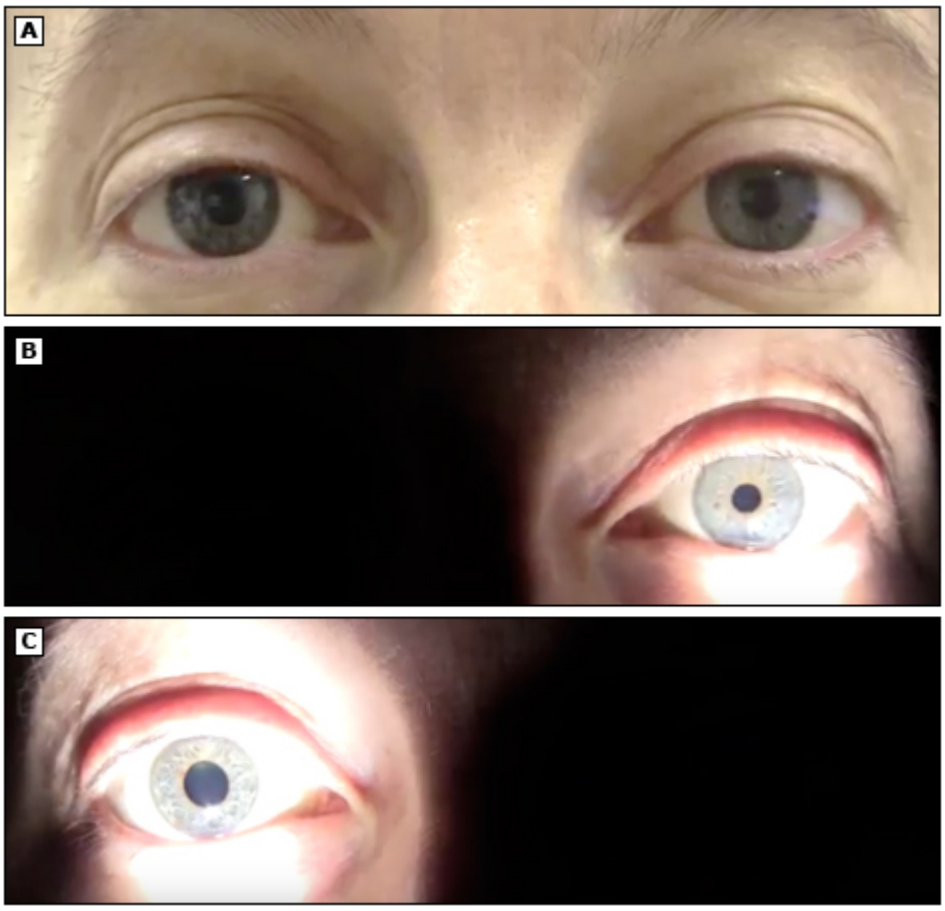


The left pupil is the same size as the right under all conditions of illumination because the efferent pathways are intact, but both pupils are smaller when light is directed at the right eye than when it is directed at the left eye, because light is detected better by the right eye. Alternate swinging of the light between the two eyes therefore produces dilation each time the light is directed to the left eye.

With permission from: Gelb DJ. The Neurologic Examination. In: Introduction to Clinical Neurology. Woburn, MA, Butterworth-Heinemann 2000.

Graphic 71937 Version 4.0

Afferent pupillary defect of the right eye, demonstrated by the swinging flashlight test



An afferent pupillary defect occurs in optic neuritis if the other eye is uninvolved and otherwise healthy. This is demonstrated by shining a light alternately in one eye and then the other and finding that the direct response to light is more sluggish in the affected eye. Alternate swinging of the light between the two eyes therefore produces dilation each time the light is directed to the affected eye.

(Image A) With both eyes exposed to the same amount of light, the pupils are equal because the efferent pathways are intact.

(Image B) When the light is directed to the unaffected (left) eye, both eyes constrict normally because light is detected normally by the left eye.

(Image C) When the light is directed to the right eye, it perceives little or no light because the afferent pathway on that side is damaged, and both pupils dilate.

Graphic 119490 Version 1.0