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AN EVIDENCE-BASED APPROACH TO EMERGENCY MEDICINE

An Evidence-Based Approach To Abnormal Vision

It is late in the evening, and the ED is packed. You head towards your next patient who is in her gurney in the hallway; she's a pleasant, elderly female with all the sweetness of the perfect grandmother.

"So, what brings you here today?" She smiles sheepishly. "Well, Doctor… earlier this evening I suddenly noticed that I kept bumping into my furniture. That's really it."

You have a long conversation with her, and she has absolutely no other symptoms, except she comments that the run-ins with her furniture were more frequent on her right side than on her left. She has normal pupils, visual acuity, fundoscopy, extraocular eye movements, facial and tongue movements, motor strength, sensation, coordination, balance, reflexes, and gait. Heart, lungs, abdomen, extremities, face…everything is normal.

Nothing tells you that you are dealing with anything other than a normal grandmother, until you think of one last exam element to check…

An acute onset of abnormal vision is distressing to the patient and a challenge to the emergency physician. The problem can result from any component in the visual process, from light transmission through the cornea to cortical perception and fixation. It is important for the emergency physician to rapidly determine the nature of the abnormality, localize the cause, administer interventions that are required emergently, and then to arrange either an immediate or deferred evaluation by the appropriate specialist, if needed. This issue of *Emergency Medicine Practice* concentrates on the symptom-based development of a differential diagnosis for an acute non-traumatic visual disturbance and the management of various etiologies prioritized by those that require time-critical interventions.

September 2007
Volume 9, Number 9

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CME Objectives

Upon completion of this article, you should be able to:
1. Understand the various manifestations of visual disturbances.
2. Establish a focused differential diagnosis for an acute visual disturbance based on symptoms, involvement of one or both eyes, and the presence or absence of pain.
3. Determine the most likely etiologies of an abnormal ocular examination finding.
4. Distinguish those etiologies of a visual disturbance that require emergent intervention from those that do not.
5. Evaluate various neuro-ophthalmological causes of abnormal vision using a rational strategy.
6. Appreciate the degree of evidence behind treatment strategies for various causes of visual disturbance.

Date of original release: September 1, 2007
Date of most recent review: August 2, 2007
Termination date: September 1, 2010
Time to complete activity: 4 hours
See "Physician CME Information" on back page.

Faculty Disclosure: Dr. Guluma, Dr. Sharma, and Dr. Jagoda report no significant financial interest or other relationship with the manufacturer(s) of any commercial product(s) discussed in this educational presentation.
Commercial Support: *Emergency Medicine Practice* does not accept any commercial support.

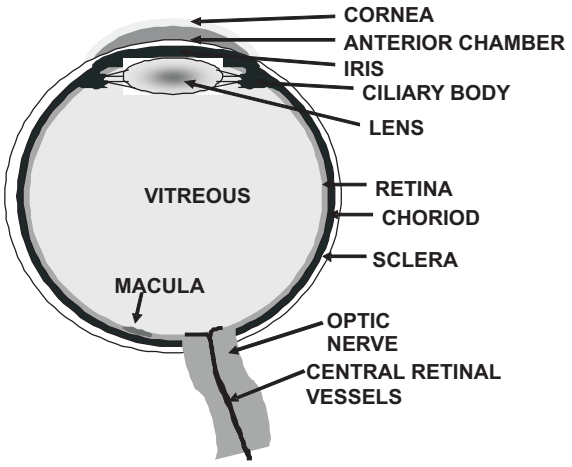
Critical Appraisal Of The Literature

The literature regarding the management of chronic eye processes such as open angle glaucoma, macular degeneration, amblyopia, cataracts, and diabetic retinopathy is rich with large, prospective, randomized, controlled trials and practice guidelines. On the other hand, the literature regarding the diagnostic and therapeutic issues in addressing acute visual disturbances, especially emergency conditions presenting to the ED, is limited and primarily characterized by observational case series and small trials. In some instances, the only "evidence" are either case reports or expert opinion.

Anatomy, Epidemiology, And Pathophysiology

The etiology and pathophysiology of abnormal vision is best described by using the anatomic framework of the visual pathway. The process of visual perception starts as light reflected off ambient objects is refracted as it is transmitted through the cornea and lens and travels through the vitreous to impact the retina, see **Figure 1**. Light paths cross prior to reaching the retina, and, therefore, objects in the temporal field of vision are detected on the nasal portion of the retina, while those in the nasal field of vision are detected on the temporal portion of the retina. The central macula is the area of most acute vision. Photoreceptors in the retina generate electrical impulses which travel centrally through the optic nerve and then decussate at the optic chiasm such that information from one side of the visual field is conducted in the contralateral side of the post-chiasmic neural tracts. The impulses travel backwards through each optic tract to the lateral geniculate bodies, and travel from there via the optic radiations to the visual cortex in the ipsilateral occipital lobe. Impulses also leave the lateral geniculate bodies to synapse in the midbrain pre-tectal nucleus, divide again in the posterior commissure, and then pass to the Edinger-Westphal nuclei anteriorly, from which emerge the efferent pathways that innervate the peripheral oculomotor nerves and ciliary muscles.[1]

Figure 1. Anatomy Of The Eye



To view color version of the figures in this article, subscribers can go to "Topics" on the left side of the page at ebmedicine.net/redirect.

Abnormalities In The Ocular Media

The ocular media refers to those transparent constituents with a primary purpose of refraction and light transmission to the retina and includes the cornea, iris, lenses, and vitreous.

Corneal Ulcerations And Abnormalities

Corneal abnormalities can arise from trauma (in some cases relatively innocuous), chemical or ultraviolet exposures, infections, or autoimmune disorders; they will typically be associated with a notable amount of patient discomfort due to the afferent sensory innervation of the cornea. Corneal ulcerations from bacterial infections or herpetic viral infections are especially critical to diagnose in a timely fashion.[2]

On occasion, contact lenses can result in peripheral corneal infiltrates[3] as well as infected corneal ulcers and keratitis.[4]

Hyphema, Hypopyon And Problems With The Iris

A hyphema is a collection of red-blood cells in the anterior chamber of the eye. A hypopyon is a collection of white-blood cells in the anterior chamber typically caused by adjacent inflammation such as iritis, uveitis, or keratitis. Both hyphemas and hypopyons present with an obscuration of visual clarity.

Non-traumatic iritis (inflammation of the iris) is largely idiopathic, or due to entities such as vasculitis, sarcoid, collagen vascular disease, or tuberculosis.[5] It typically presents with visual blurring and photophobia.

The uvea of the eye is made up of three tissues that are continuous with one another: the iris anteriorly, the choroid posteriorly, and the ciliary body in between them, see **Figure 1**.[6] Uveitis, which is any inflammatory process involving the tissues of the eye, most commonly afflicts patients 20–50 years of age and is the etiology in 10% of visual loss cases and up to 20% of legal blindness cases.[7] It is a typical ocular manifestation of autoimmune disease,

affecting the anterior uvea (iris) in up to 90% of cases.[8-9]

Glaucoma

Glaucoma is the second most common cause of blindness in the United States. Open angle glaucoma is the most common form of this disease, is related to chronically-elevated intraocular pressure, and is largely treated medically on a chronic basis.

Angle-closure glaucoma occurs when the lens and iris are excessively apposed, narrowing the angle in the anterior chamber and obstructing aqueous outflow from the anterior chamber via the canal of Schlemm. An acute rise in intraocular pressure occurs, resulting in eye pain, corneal edema, and resultant visual blurring. This form of glaucoma accounts for 10% of cases and may present more acutely.[1]

Lens Subluxations

A lens subluxation is a typically painless condition that may be traumatic or atraumatic (especially in patients with connective tissue disorders such as Marfan's disease). It presents with an acute change in vision and will usually be evident on ophthalmoscopy.

Vitreous Processes

The pathway of light transmission through the vitreous may be obscured by hemorrhages or infection. Hemorrhages may be spontaneous (especially in diabetic patients) or post traumatic.[10] Infection in the globe (endophthalmitis) causes pain and visual loss.

Retinal And Vitreous Detachment

Retinal detachment affects 1 in 10,000 people per year.[11] It is a separation of the neurosensory retina from the underlying retinal pigment epithelium, two structures normally held in direct apposition by a variety of mechanical and oncotic factors.[12] There are three types: rhegmatogenous, tractional, and exudative.

Rhegmatogenous retinal detachment is the most common. The vitreous is a hydrated gel that shrinks with age, exerting increased vitreoretinal traction, eventually resulting in partial detachment from the retina.[13] A rhegmatogenous retinal detachment occurs when vitreous fluid dissects through this tear into the space between the neurosensory retina and the retinal pigment epithelium.[14] One in four adults between the ages of 61 and 70 have a posterior vitreous detachment (a prevalence that rises significantly with higher age).[15]

The second type of retinal detachment, a tractional retinal detachment, forms when a centripetal force (usually from relatively innocuous trauma) acts on the retina through a post-surgical, neoplastic, or post-inflammatory adhesion.

The third type, an exudative retinal detachment, results from the accumulation of serous or exudative fluid in the subretinal space from a hydrostatic (e.g., severe acute hypertension), inflammatory (e.g., sarcoid), neoplastic, or infectious process.[14]

Retinal Artery Occlusion And Acute Retinal Ischemia

Acute retinal ischemia may develop from an embolic, thrombotic, vasculitic or vasospastic occlusion of the retinal blood supply and may involve a branch of the retinal artery (a branch retinal artery occlusion [BRAO]) or the central retinal artery itself (a central retinal artery occlusion [CRAO]). It generally has a poor visual prognosis, with spontaneous resolution occurring in only 1–8% of cases.[16] Etiologies include emboli,[24] hyperviscosity[23] and vasculitis.

Certain patients may have chronic ocular hypoperfusion at baseline which predisposes them to transient monocular visual loss. This may occur when there is a postural blood pressure change or increased retinal oxygen consumption from exposure to bright light.[17-19] The retina can tolerate up to 105 minutes of ischemia before permanent damage occurs,[31] but it sustains profound irreversible damage by about four hours.[20]

Retinal Migraine

In young patients with amaurosis fugax and no cardiovascular risk factors, the etiology underlying the majority of presentations is thought to be due to a transient, reversible vasospasm of the retinal artery, likely as part of a migraine phenomenon (retinal migraine)[19] or possibly from retinal spreading depression (a depolarization phenomenon).[21] Fortunately, these patients rarely develop subsequent central nervous system (CNS) ischemia.[22]

Retinal Vein Occlusion

In addition to being susceptible to ischemia from arterial occlusion upstream, the microvascular perfusion of the retina can also be compromised by a problem with an obstruction of venous outflow. A central retinal vein occlusion (CVRO), which is a prominent cause of visual loss from retinal vasculopathy,[23] results from intraluminal thrombus formation at or just posterior to the lamina cribrosa[24] that

leads to venous congestion, capillary stasis, and intraretinal hemorrhage; it is typically associated with a poor prognosis for recovery of vision in those presenting with poor visual acuity.[25-27] There is a non-ischemic subtype (called papillophlebitis) that is found in younger (less than 50 years of age), healthier patients and is thought to be from venous congestion related to optic nerve head swelling; it carries a very good prognosis.[28-29]

Disorders Of The Optic Nerve

Ischemia, edema, inflammation, or compression of the optic nerve may cause abnormalities in vision.[19,30-31]

Optic neuritis is a primary inflammatory process of the optic nerve, sometimes associated with systemic autoimmune disorders. The most common form, acute demyelinating optic neuritis, is associated with multiple sclerosis (MS);[32] it develops at some point in 50% of patients with MS, is the presenting feature in 15-20% of MS cases, and is associated with a generally increased risk of subsequently developing MS in those without evidence of MS on MRI.[32-36] It typically develops in young patients and presents with a decrease in visual acuity – sometimes profound – and pain with eye movement.[2,32,37-38]

Another condition that may be hard to distinguish from optic neuritis is anterior ischemic optic neuropathy (AION). This is an acute and irreversible ischemic event affecting the anterior optic nerve that typically occurs in patients over the age of 50.[39]

Idiopathic intracranial hypertension (IIH), formerly called pseudotumor cerebri, is a condition in which increased intracranial pressure leads to optic nerve edema and significant visual abnormalities; it has a predilection for young, obese females. The female:male ratio is anywhere from 4.3:1 to 15:1. The annual incidence is about 3 per 100,000 in the general population, but rises to 21 per 100,000 in women of childbearing age, 70% of whom are obese (compared to 36% of women in the general population).[40-45] The condition is idiopathic, but a variety of etiologies have been suggested, including excessive CSF production, compromised CSF resorption, venous outflow obstruction in the cerebral circulation, and increased cerebral blood volume.[40-41] Visual symptoms are common and can progress to permanent blindness in 10% of patients.[40]

Neuro-ophthalmologic And Other Retrobulbar Etiologies Of Acute Visual Disturbances

Neuro-ophthalmological processes causing visual disturbances tend to fall into two broad categories: 1) Those that affect transmission of visual input from the retina to the occipital cortex (typically presenting with stereotyped visual field defects); and 2) Those that affect ocular motility (typically presenting with diplopia).

Lesions that compromise the transmission of visual input from the retina to the occipital cortex vary from parasellar masses (such as pituitary adenomas, craniopharyngiomas, meningiomas, or aneurysms) that compress the optic chiasm to more general neoplastic, inflammatory, ischemic, or infectious processes and manifest with somewhat stereotypical visual field defects, see **Figure 2**.

Diplopia, or the visual perception of two separate images, may be monocular or binocular and may manifest in a variety of directions from a variety of causes. Monocular diplopia, or double vision that persists in an affected eye even with the other one closed, is typically due to corneal distortion and refractive errors.[48-49] Binocular diplopia, or double vision that resolves when one eye is closed, can result from oculomotor muscle dysfunction, cranial nerve (CN) dysfunction, or supranuclear lesions in the brainstem or above. Processes such as myasthenia gravis, thyroid disease, and trauma may cause oculomotor muscle dysfunction and misalignment of the visual axes, leading to diplopia.

A cranial palsy of either the oculomotor nerve (CN III), trochlear nerve (CN IV) or abducens nerve (CN VI) can cause diplopia. The abducens is the most commonly affected, followed by the oculomotor, and then the trochlear.[50-51] Palsies in any of these nerves may present as an isolated mononeuropathy from a demyelinating process (such as MS) or from hypertensive or diabetic vasculopathy. They may also be affected together in a polyneuropathy caused by inflammation, neurotoxins (e.g., botulism), infectious processes,[52] or Guillain-Barre syndrome.[51,53] The abducens, which innervates the lateral rectus muscle, is the most common nerve to be affected by tumor and elevated intracranial pressure;[54] an isolated abducens palsy should be considered a sign of elevated intracranial pressure until proven otherwise.[51]

The oculomotor nerve (CN III) innervates the medial, inferior, and superior recti muscles, as well as the inferior oblique muscle. It also innervates the lev-

ator palpebrae superioris muscle which lifts the upper eyelid and provides parasympathetic innervation to two intrinsic ocular muscles (the ciliary and constrictor pupillae muscles) which constrict the pupil. It is commonly affected by diabetic or hypertensive vasculopathy, presenting as a pupil-sparing palsy. In addition, a third nerve palsy is the most common neurologic sign accompanying posterior circulation aneurysm, due to compression, and presents with impairment of adduction and vertical movement as well as ptosis and mydriasis in the affected eye,[55] although the pupil is occasionally spared.[56]

The trochlear nerve (CN IV) innervates the superior oblique muscle and is most commonly affected by trauma from abutment against the tentorium. A palsy in this nerve, usually due to a process in the subarachnoid space, is rare and presents with difficulty looking downward and inward that is frequently associated with compensatory head tilt.[51]

A particularly ominous but rare retro-orbital etiology of abnormal vision is cavernous sinus thrombosis. Intimately interlaced in the cavernous sinus are cranial nerves III, IV, and VI and the ophthalmic and maxillary branches of CN V. The trabeculated sinuses act like sieves, trapping bacteria and thrombus from infection in the medial third of the face, nose, teeth, and the ethmoid and sphenoid sinuses (the most common source of infection); these may become secondarily infected, leading to a septic thrombophlebitis[57-58] that is complicated by impaired vascular drainage and associated meningitis, subdural empyema, pituitary necrosis, and carotid thrombosis.[57-59] The patient with cavernous sinus thrombosis presents with symptoms from cranial nerve palsies, infection, and venous congestion.

Pharmacological, Toxic, And Metabolic Etiologies Of Acute Visual Disturbances

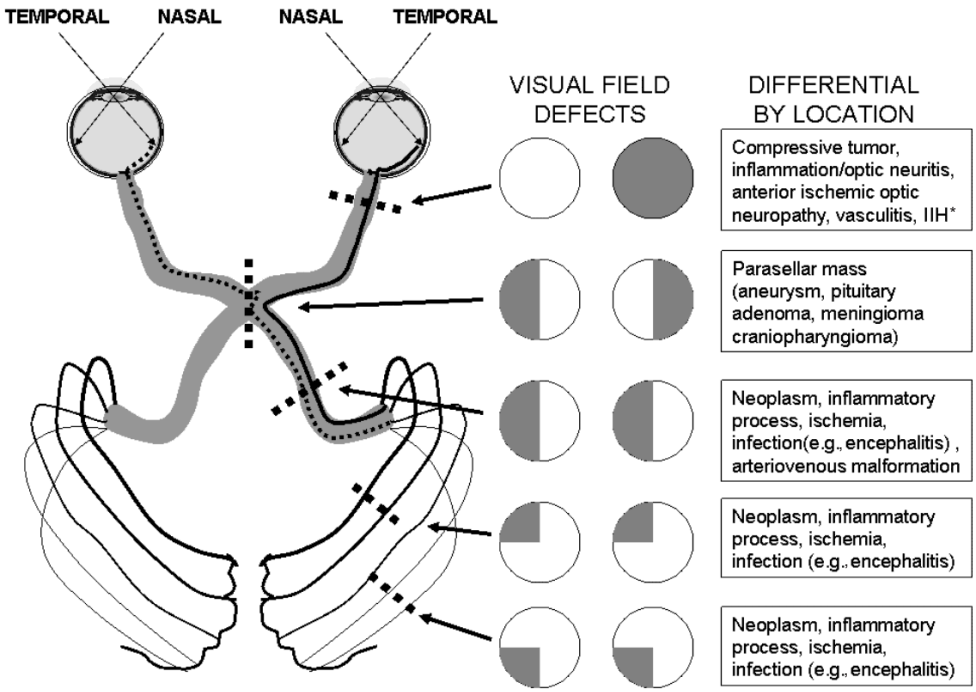
The variety of pharmacological **(Table 1 on page 6)**, toxicological, and metabolic etiologies of acute visual disturbance create a wide range of visual disruption ranging form corneal edema, to cataract formation in the lens, to neurosensory retinal abnormalities.

Toxic And Metabolic

Any rapid physiological change which results in rapid osmolar shifts can theoretically lead to visual changes. The most representative is acute hyperglycemia, in which a rapid elevation of intracellular glucose levels in the lens overwhelms metabolic pathways resulting in an acute hyperosmolar state that causes stromal swelling and lens fiber separation and leads to an acute change in refraction, typically an acute myopia.[60-63] The process typically reverses when hyperglycemia is treated.

Perhaps the most characterized toxidrome presenting with acute visual change is methanol

Figure 2. The Visual Field Defects Associated With The Various Possible Locations Of A Pathological Lesion



** IIH, idiopathic intracranial hypertension.*

Table 1. A Partial List Of Medications That Can Cause Abnormalities Of Vision

Medication	Typical use	Toxic effect and symptoms
cidofovir (Vistide®)	Treatment of CMV*	25% incidence of iritis and ciliary body hypotony in HIV* patients
sildenafil (Viagra®), tadalafil (Cialis®), and vardenafil (Levitra®)	Erectile dysfunction	inhibition of retinal phototransduction leading to changes in retinal color perception or transient decreased vision
chloroquine (Aralen®)	Anti-malarial	Reversible corneal deposits and irreversible maculopathy
hydroxychloroquine (Plaquenil®)	Rheumatological disease treatment	Reversible corneal deposits and irreversible maculopathy
amiodarone (Cordarone®)	Cardiac dysrhythmias	Blue-green colored rings or halœs around lights
digitalis (Digoxin®)	Atrial fibrillation; heart failure	Chromatopsia (yellow-green tint to vision)

*CMV, cytomegalovirus; HIV, Human Immunodeficiency Virus infection

toxicity.[64] Orally ingested methanol, which is a colorless and clear alcohol used in industrial solvents, is metabolized by alcohol dehydrogenase to formaldehyde and formic acid. Formic acid accumulates in the optic nerve, inhibiting cytochrome oxidase; the histotoxic effect leads to edema, compromised axoplasmic flow,[65-66] and widespread electrophysiological dysfunction that also affects photoreceptors in the retina,[67] leading to visual loss.

Differential Diagnosis

In order to help focus the evaluation etiologically, the varied causes of an acute visual disturbance can be separated into six groups based on presentation: 1) unilateral painless visual disturbance; 2) unilateral painful visual disturbance; 3) bilateral painless visual disturbance; 4) bilateral painful visual disturbance; 5) diplopia; and 6) gradual visual disturbance, see **Table 2**.

Prehospital Care

In general, the most important and time-sensitive ophthalmological prehospital intervention is ocular lavage for chemical burns to the eye, as time-to-lavage significantly affects visual prognosis. Patients may also access Emergency Medical Services for care of acute, non-traumatic visual complaints. When it comes to an acute visual disturbance, one of the most important aspects of prehospital care is the timely and efficient transportation of a patient with an underlying cause that may require specialized treatment to an appropriate facility (e.g., a stroke center). As a general rule of thumb, painless visual disturbances of relatively acute onset tend to portend a CNS pathology whether they are unilateral (e.g., vision loss in one eye due to amaurosis fugax, central retinal artery occlusion, or central retinal vein occlusion) or bilateral (e.g., a visual field cut due to an occipital stroke, vertebrobasilar insufficiency, or tumor) or associated with diplopia (e.g., brain stem tumor or stroke). The presence of associated neurological deficits such as speech or language disturbances or motor or sensory deficits further increases this likelihood.

Table 2. Differential Diagnosis Of Various Presentations Of Visual Disturbance

Presentation	Differential
Unilateral painless visual disturbance	Retinal detachment Posterior vitreous detachment or hemorrhage/floaters Amaurosis fugax Retinal artery occlusion Retinal vein occlusion Non-arteritic anterior ischemic neuropathy, giant cell (temporal) arteritis Retinal migraine Vitreous hemorrhage Lens dislocation Optic nerve sheath tumor
Unilateral painful visual disturbance	Corneal abrasion or infection Acute angle-closure glaucoma Iritis/uveitis Optic neuritis Endophthalmitis Cavernous sinus thrombosis
Bilateral painless visual disturbance	Metabolic or toxic derangement (hyperglycemia, methanol toxicity) Idiopathic intracranial hypertension (pseudotumor cerebri) Medication effect Ischemic stroke Brain tumor Migraine headache Vertebrobasilar insufficiency
Bilateral painful visual disturbance	Chemical exposure Photokeratitis from a UV light exposure Welders exposure Tanning beds Sunlight (e.g., snow covered mountain)
Diplopia	Thyroid disease Cranial neuropathy (CN III, IV, VI) Neuromuscular disease (myasthenia gravis) Botulism, Miller-Fisher syndrome Encephalitis, basilar meningitis Brainstem stroke
Gradual visual disturbance	Progressive refractive error Macular degeneration Brain tumor

ED Evaluation

History

Getting To the Root Of The Problem

The first challenge facing the emergency physician is to define the complaint (e.g., "I'm having trouble seeing"). A detailed and careful history is the most important first diagnostic step. Information regarding the acuity of onset of the visual abnormality, the nature of the visual abnormality (e.g., whether it is blurring, double vision, sense of movement), and associated negative or positive associated symptoms (e.g., floaters, flashing lights, pain) should be carefully elicited. Keep in mind that patients may not notice a problem in one eye as long as the other functions normally; the abnormality in vision in the problem eye may only be unmasked when a related or unrelated problem finally develops in what was the normally functioning eye. In cases in which the abnormality in vision was transient, it is helpful to ask if the patient checked their vision with one or the other eye closed during the episode, so as to reveal evidence regarding whether the disturbance was binocular or monocular. In addition, a patient may refer to what is actually diplopia as "blurred" vision or vice versa. When the primary complaint is diplopia, the patient should be asked whether it is horizontal, vertical, and/or oblique, whether it is gaze-evoked or not, whether it is transient or constant, and whether there are any exacerbating factors or associated neurological symptoms such as vertigo, slurred speech, or problems with coordination. Diplopia that is worsened or triggered by fatigue or sunlight suggests a neuromuscular etiology (such as myasthenia gravis) whereas diplopia that is worse in the morning suggests an ocular muscle problem (such as thyroid myopathy) which is presumably worsened due to the muscular venous congestion associated with being supine.[68] The patient with diplopia should be specifically questioned about other symptoms, such as proximal muscle weakness (e.g., difficulty holding arms above the head or climbing stairs), shortness of breath, or difficulty swallowing, which would suggest a systemic neuromuscular disease.

Red Flags In The History

The etiologies of abnormal vision range from the relatively benign to the very serious. Some of the serious pathologies may present somewhat subtly, and the practitioner should be aware of red flags that prompt their consideration. Frank loss of vision has a relatively straight-forward implication of a serious etiology. Other red flag symptoms are photophobia (which may suggest infection or inflammation), deep eye pain (as opposed to corneal discomfort or a foreign body sensation), pain with eye movement, a decrease in visual acuity, associated headache, and diplopia. Constitutional symptoms, such as myalgias or fever, also suggest a more complicated process, such as inflammation or infection. In addition, remember that the eye is the window into (and out of) the body and may be the first indicator of systemic disease; in one study, over 8% of patients presenting primarily with ocular complaints had an occult, underlying contributory systemic disease.[69]

The Patient's Background

Any visual complaint should be taken in the context of the patient's optometric or ophthalmological background. Make inquiries as to contact lens wear (and the type of contact. It is important to know about a history of prior eye disease, eye surgery or procedures (especially if recent), and baseline visual status such as near-sightedness (myopia), far-sightedness (hyperopia), or the need for reading glasses (presbyopia).

Physical Examination

The eye examination can be divided into six parts: visual acuity, visual field, pupillary examination, extraocular muscle movement, anterior segment, and posterior segment. The examination should entail a systematic progression through all six elements, as the true cause of the presenting complaint may be either unapparent or unexpected.

The Face And External Eye

A careful external inspection of the face and eyes can easily be overlooked in the setting of an acute visual disturbance but should be performed. Subtleties in the external appearance of the face or eye (such as proptosis, ptosis, and mild periorbital swelling) can suggest orbital disease where intraocular examination findings may be unremarkable.

Visual Acuity

The visual acuity is an important vital sign when it comes to visual complaints and should be measured with the patient wearing their corrective lenses at a distance of 20 ft from either a Snellen eye chart, an illiterate E chart, or an Allen card of objects for chil-

dren.[1] The ratio of what the patient is able to see compared to what a normal person should see is recorded, such that "20/40" means that the patient can only see at 20 ft what a person with normal vision would be able to see at 40 ft.

Visual Fields

The patient's visual fields should be assessed by confrontation. Defects due to lesions in the chiasm or beyond typically start abruptly at the vertical midline, whereas those from lesions isolated to the optic nerve or retina do not respect the midline.[1] The locations of visual field defects may offer a localization of the pathology and narrow the differential, see **Figure 2 on page 5**.

Pupils

Pupils should be black, round, equally sized, and reactive to light. A non-black pupil suggests opacification of the refracting media. A misshapen or eccentric pupil suggests a pathological process such as synechiae (adhesions) from iritis.[9,70] The swinging flashlight test can reveal an afferent pupillary defect (APD) which may be a sign of optic nerve or retinal disease, see **Figure 3**.[71] With physiological anisocoria, both pupils will change in response to a change in the lighting conditions, but one will always be smaller than the other.[1]

An Adie's pupil is an idiopathic denervation pupillary abnormality that typically affects young women; the abnormal pupil is tonically dilated, with no direct or consensual light reflex, and constricts slowly to accommodation, but constricts robustly on administration of pilocarpine 0.1% due to denervation cholinergic hypersensitivity.

Extraocular Muscle Movement

Test the six cardinal gaze positions by having the patient look up-and-down and right-to-left with the help of fixation on a moving penlight. The eyes should be aligned in all positions. Alignment can be further tested by having the patient look at a distant object, shining a light on the eyes from a distance of approximately 18 inches, and assessing for symmetrical placement of the light reflex on the corneas.[1] The corneal light reflex, which is the reflection of light off the cornea when the eyes are viewed straight-on with a light source such as a hand-held ophthalmoscope centered over both pupils, can be helpful in revealing subtle abnormalities in alignment.

Anterior Segment

The anterior segment consists of the sclera, conjunctiva, cornea, anterior chamber, iris, and the lens. The sclera and conjunctiva should be examined for discharge, swelling, and vascular injection. The instillation of topical fluorescein, illuminated with a cobalt-blue light, can be used to check for corneal irregularities such as ulcerations or abrasions. Examine the anterior chamber for hyphema and hypopyon, and examine the iris for signs of iritis such as pupillary dilatation or constriction, irregularity, or direct and consensual photophobia. Examine the lens for opacity (which suggests a cataract) or frank dislocation.

The Slit Lamp

The slit lamp (often referred to as a biomicroscope because it can allow microscopy of living tissue) is a useful tool in the evaluation of acute visual complaints. Using a mechanism in which a binocular microscope with a swiveling light source can be positioned anywhere in the immediate three-dimensional space between the examiner and the patient, it enables binocular microscopy of the cornea and anterior chamber, contains specific light filters that enable inspection for corneal abrasions, and has the capability to project beams of light of various widths and lengths at various angles, allowing a detailed morphological inspection and measurement of the structures in the anterior chamber and beyond. The slit lamp is useful, if not indispensable, for inspection of the anterior chamber and the assessment of conditions such as iritis, scleritis, uveitis, corneal abrasions, or ulcerations, and for searching for and removing corneal foreign bodies.

Posterior Segment

The posterior segment consists of the vitreous, retina, and the optic nerve — structures best examined under direct ophthalmoscopy, aided with pupillary dilatation with agents such as a combination of 1% tropicamide and 2.5% phenylephrine.[2] The retina should be free of hemorrhages, exudates, and ischemic cotton-wool spots, and the margins of the optic discs should be clear and without papilledema. Pseudopapilledema, which has the appearance of papilledema but without the venous engorgement, is most commonly caused by optic disk drusen[2] (collections of hyaline bodies which distort the architecture of the head of the optic nerve). At times, the view obtained through an undilated pupil for inspection of the posterior segment may be sufficient. At other

times, an unobstructed and panoramic view of the retina is necessary (e.g., in evaluating for a retinal detachment or if the cause of the visual complaint is relatively undifferentiated and not seen on undilated exam), and dilation of the pupil may be extremely helpful if not indispensable.

Measuring Intraocular Pressure

Intraocular pressure can be measured by a Schiotz tonometer which is a device with an attached 5.5 g weight (which can be increased) that rests on the cornea, a Tonopen which is a digital device that utilizes a pressure gauge directly applied to the cornea, or a Goldman applanation tonometer which is attached to the slit lamp and utilizes the matching of the semi-circles of a fluorescein-highlighted tear film meniscus to determine the amount of force required to flatten the cornea.[71] Important considerations with all three techniques are that: 1) The cornea be topically anesthesized prior to measurements; and 2) To maximize accuracy, measurements should be made in the center of the cornea, before any administration of mydriatic drops or ocular massage.

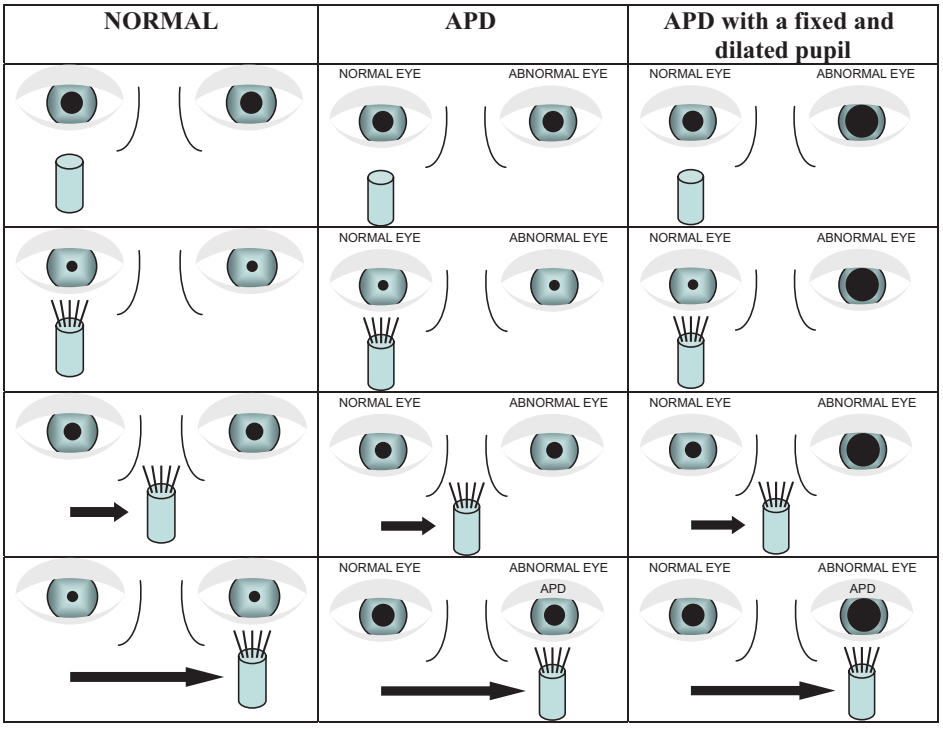
"You can't depend on your eyes when your imagination is out of focus." - Mark Twain

Diagnostic Testing

The most common causes of acute visual loss, apart from trauma, are due to impairments at the neurosensory portion of the visual process (e.g., retinal detachment, retinal vascular occlusion, and optic neuritis).[1] The majority may be evident after a history and physical with ophthalmoscopy. The evaluation of other causes, however, may require laboratory and imaging tests. The laboratory test most germane to acute visual disturbance are the erythrocyte sedimentation rate (ESR) and C-reactive protein (CSR), both of which can help differentiate an inflammatory process such as giant cell (temporal) arteritis. A urine or serum toxicology screen may be useful in presentations of acute vision loss that are consistent with drug-induced vasospasm or methanol toxicity. Lumbar puncture with CSF analysis may be used to evaluate intracranial pressure in cases where idiopathic intracranial hypertension is suspected or to assess for inflammatory or infectious causes of diplopia, such as meningitis or encephalitis.

Most of the neuro-ophthalmological and orbital etiologies of an acute visual disturbance will need imaging. CT with contrast is an option;[72] however, an MRI of the brain and/or orbit with and without contrast is listed in a 2006 imaging guideline from the American College of Radiology as the optimal imaging

Figure 3. Afferent Pupillary Defect (APD) As Detected With The "Swinging Flashlight Test"



In the normal condition, both pupils constrict regardless of which eye is illuminated, due to intact direct and consensual light reflexes. With an APD, the pupil in the normal eye dilates upon illumination of the pathological eye because of a lack of stimulus for the consensual light reflex.

modality to assess adults with acute visual loss, ophthalmoplegia, or orbital disease.

Ultrasound has demonstrated utility in the diagnosis of various pathologies in the eye, including retinal detachment, vitreous hemorrhage, foreign body detection, and ocular tumors[73-74] and is used as a clinic- and office-based diagnostic tool by ophthalmologists. It has been shown to be of similar utility in the ED[75] and results in relatively high accuracy in the hands of emergency physicians.[76] It is most commonly performed using a high-frequency (at least 7.5 MHz) linear probe, insonating through water-soluble gel placed on top of the closed eyelid. An abnormal increase in the diameter of the optic nerve sheath to greater than 5 mm on ultrasound correlates with increased intracranial pressure[77] and has been shown to have utility as a screening tool for raised intracranial pressure in ED patients with intracranial masses and traumatic brain injury;[78-79] however, no studies have been published that firmly establish its use as a diagnostic tool for idiopathic intracranial hypertension (pseudotumor cerebri), specifically as something that would obviate the need for a lumbar puncture.

Unilateral Painless Visual Disturbance

Floaters And Retinal Detachment

Presentation And Examination Findings

A floater will typically present with a sensation of something floating in the visual field of one eye, possibly with photopsia (flashing lights); it is typically painless.[80] The differential diagnosis of floaters includes posterior vitreous detachment, a retinal tear related to this (which can progress to a detachment), and vitreous hemorrhage.

The first symptom of a retinal detachment may be light flashes (photopsia), a positive visual symptom due to tractional stimulation of photoreceptors. When the neurosensory retina separates from the retinal pigment epithelium, the outer retina becomes ischemic due to the loss of its blood supply from the choroid[12] and visual deficit develops rapidly, typically spreading from the periphery towards the central axis over hours to days, sometimes described by the patient as a shadow or "curtain" progressing over the visual field.[14] What starts out as a relatively subtle peripheral visual field disturbance may develop into profound visual loss once the detachment reaches the macula. Ophthalmoscopy may reveal a billowing detached retina, especially if performed through a dilated pupil,[2] see **Figure 4**.

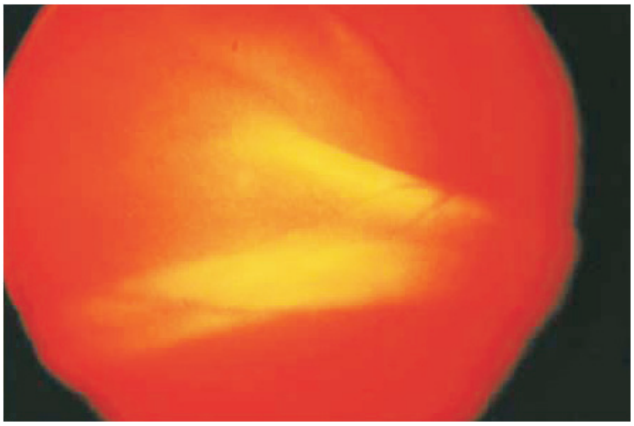
Diagnostic Workup And Management

Visualization of the processes that lead to floater symptoms on routine fundoscopy using equipment routinely available in the emergency department may be challenging and will typically require a diagnostic evaluation by an ophthalmologist, typically on an urgent basis due to the association between vitreous detachments and retinal detachments. There is little in the literature to make an evidence-based recommendation as to whether or not this needs to be done emergently; however,[81] symptoms consistent with a retinal detachment require a more emergent evaluation.

Imaging may reveal a retinal detachment in cases where ophthalmoscopy is limited. Ophthalmic ultrasonography is superior to MRI or CT for the detection of an occult detachment;[14] it is a Level II recommendation from the American Academy of Ophthalmology[81B] and is a useful modality in the ED.[76] Once an acute symptomatic detachment is diagnosed, the main goal of the ED management is prompt evaluation and treatment by an ophthalmologist (a Level II recommendation from an American Academy of Ophthalmology guideline).[81B]

The chances of preserving normal vision are very high (expected) if the detachment has not reached the macula, but the detachment becomes problematic once the macula is involved. Surgical repair is indicated more urgently in patients with preserved central acuity, less urgently in patients whose macula detached in the previous hours to days, and routinely in those whose macula has been detached for several days or weeks.[14,82-83]

Figure 4. Retinal Detachment



A retinal detachment can be seen as a billowing abnormality in the central field of view.

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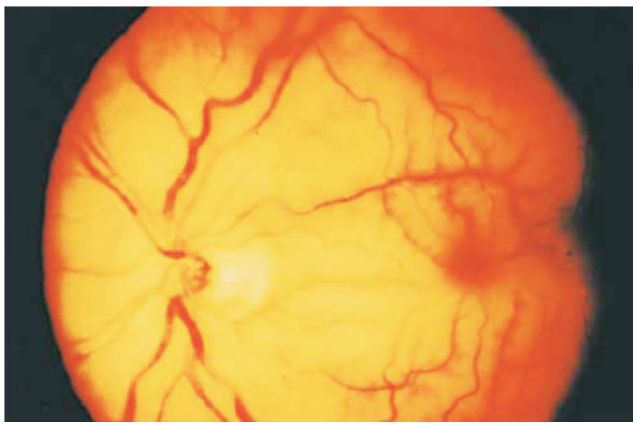
Amaurosis Fugax & Central Retinal Artery Occlusion

Presentation And Examination Findings

Amaurosis fugax and central retinal artery occlusion would appear to be two extremes of one continuous entity, with amaurosis fugax being a fleeting loss of vision from transiently compromised retinal blood flow. The visual loss associated with amaurosis fugax may present with a camera-diaphragm effect at onset and typically lasts on the order of minutes to hours.[19] Medically treated patients with carotid artery disease and amaurosis fugax have a risk of ipsilateral ischemic stroke within three years that is approximately half that associated with other types of transient ischemic attacks. However, in patients with three or more risk factors for stroke, the risk of stroke after amaurosis fugax approaches 24%.[84-85]

A retinal artery occlusion typically presents with sudden vision loss in the affected eye, to a degree concordant with the location of the vascular occlusion; a branch retinal artery occlusion (BRAO) may present with a sudden peripheral or segmental visual field cut, while a central retinal artery occlusion (CRAO) may present with sudden, painless, complete loss of vision. On examination, the patient may have an afferent pupillary defect; on fundoscopy, the patient will have a pale-appearing retina with attenuated arterioles in the vascular distribution affected and may have a "cherry red" spot in the macula due to preserved perfusion,[1-2] see **Figure 5.**

Figure 5. Central Retinal Artery Occlusion



Note the attenuation of retinal arteries, the generally pale retina, and the "cherry red" spot in the location of the macula.

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Diagnostic Workup And Management

The management of amaurosis fugax is largely one of prophylaxis, risk stratification, and referral; that of retinal artery occlusion is somewhat different. The literature regarding treatment for an acute central retinal artery occlusion is extensive but lacks robust, randomized, controlled clinical trials. There are a variety of treatments for central retinal artery occlusion that have been described in the literature, and reviews have highlighted them in detail.[86-87] They include dilation of the artery via administration of sublingual isosorbide dinitrite, rebreathing expired CO_2, or breathing a mixture of 95% oxygen and 5% carbon dioxide (Carbogen). Increasing ocular perfusion pressure via an acute reduction in intraocular pressure with an anterior chamber paracentesis, intravenous acetazolamide or mannitol, or surgical trabeculotomy can be used to create an aqueous fistula between the anterior chamber and the subconjunctival space. Other described treatments include the use of pentoxyfilline to increase red blood cell deformability (the presumption is that this will allow better rheology for flow past an obstruction), systemic steroids (since vascular endothelial edema has been suggested as being contributory to tissue damage following retinal artery occlusion), and ocular massage in an attempt to break up an acute occlusion to enable it to flow downstream (though this is not a definitive treatment).[86] Unfortunately, the literature regarding these treatments is primarily one of case reports and series, at times with conflicting results, and none of these treatments have been evaluated in a randomized, controlled trial.

In a 2002 review of the literature for the Cochrane Database, Fraser and Siriwardena could not recommend any of these treatments due to a lack of robust evidence.[86] A retrospective evaluation of ocular massage, acetazolamide, hemodilution, aspirin, and paracentesis on retinal artery occlusion using best-corrected visual acuity as the outcome measure found no statistically significant improvement in outcome compared to the natural history.[88] However, one treatment, thrombolysis via superselective administration of an agent via a catheter in the ophthalmic artery (local intra-arterial fibrinolysis [LIF]), was suggested in a meta-analysis to have potential benefit.[89] A recently published small, retrospective, case–control study appeared to show statistically significant benefit with LIF compared to conventional treatment,[90] and a multicenter, randomized controlled study is planned.[91]

Temporal (Giant Cell) Arteritis

Presentation And Examination Findings

Temporal arteritis is classically described as a clinical syndrome of temporal headache with a tender temporal artery in an older patient with headache, polymyalgia rheumatica symptoms, and fever. The majority of the available literature on the ocular manifestations of this rare condition appears to be based on a single prospective cohort of 170 patients with biopsy-proven temporal arteritis.[24] Hayreh noted that, of these 170 patients, 50% presented with visual loss of varying intensity involving one or both eyes. Patients with ocular involvement tended to be older, have a lower CRP, and have less headache, myalgia, and fever than those without visual involvement. A vague symptom of associated eye pain was relatively rare (9% of those with visual symptoms), whereas amaurosis fugax was relatively common (31%).[30] Interestingly, 21% of patients presented with visual symptoms in the absence of other systemic symptoms of temporal arteritis (i.e., with occult arteritis).[92] While fluorescein angiography of the retina and eye performed by an ophthalmologist will reveal varying degrees of ischemia, most commonly anterior ischemic optic neuropathy, regular fundoscopy in the ED will likely be relatively equivocal, except in instances of central retinal artery occlusion (which may be seen in 6.5% of cases); in which case, the typical findings of retinal artery occlusion may be evident.[30]

Diagnostic Workup And Management

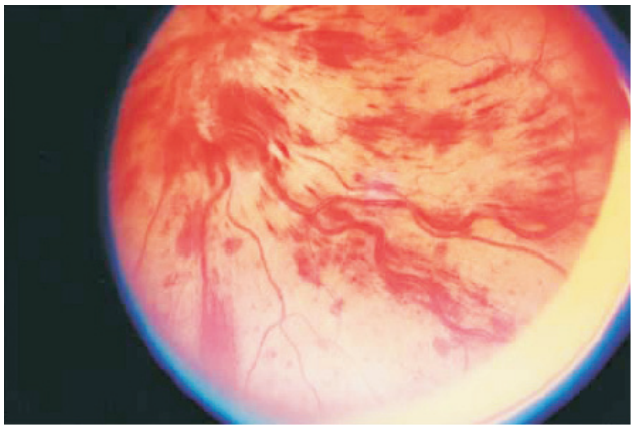
The possibility of temporal arteritis should be considered in any patient over the age of 55 presenting with amaurosis fugax, diplopia, or acute visual loss and findings of ocular ischemia on examination, especially with concurrent symptoms of headache, myalgias, and fever. An ESR and CRP are helpful laboratory analyses to obtain as they will typically be elevated with temporal arteritis, although a normal ESR does not rule out temporal arteritis. A combination of ESR and CRP give the best specificity for ruling out temporal arteritis[92B] and also result in a sensitivity approaching 99%.[93] Therapy is largely geared at preventing further visual loss, as opposed to reversing it; even high-dose steroids will have little effect on reversal.[94-95] In the setting of ocular symptoms, the treatment regimen consists of methylprednisolone 1 gram intravenously per day, with an eventual taper to a lower maintenance dose around 40–80 mg of oral prednisone per day, titrated to symptoms.[94-95] The initial high-dose steroid treatment may require admission.

Central Retinal Vein Occlusion

Presentation And Examination Findings

Retinal vein occlusion, which may be in a branch or in the central vein, typically presents with sudden painless visual loss. On fundoscopy, the presence of venous congestion, tortuosity of the retinal veins, and a "blood and thunder" appearance to the retina from intraretinal hemorrhages may be evident,[25-27] see **Figure 6.** An impending central retinal vein occlusion can cause transient monocular visual loss.[96]

Figure 6. Central Retinal Vein Occlusion With Venous Engorgement And Retinal Hemorrhages



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Diagnostic Workup And Management

The ED diagnosis of retinal vein occlusion is largely examination-based, although the ophthalmologist will also have the option of performing fluorescein retinal angiography to further delineate the extent and nature of the occlusion and to differentiate between ischemic and non-ischemic subtypes. While its presence may herald the existence of underlying thromboembolic disease or hypercoaguable state, there is no effective therapy for a retinal vein occlusion itself, and outpatient therapy is focused on preventing the neovascular complications that appear as sequelae.[23,29]

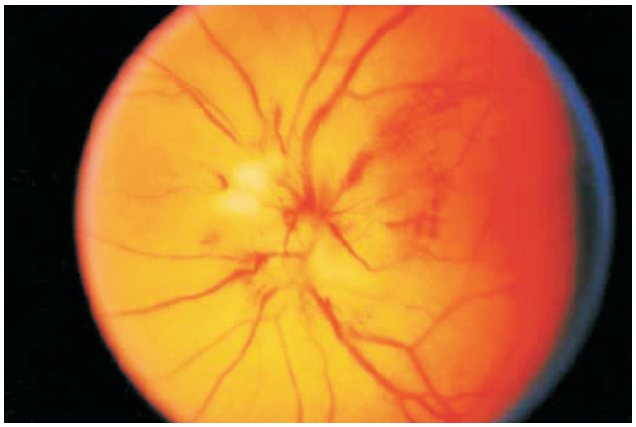
Non-Arteritic Anterior Ischemic Optic Neuropathy (NAION)

Presentation And Examination Findings

The elderly patient with NAION will typically pres-

ent with sudden painless monocular vision loss. However, 10% of patients will experience ocular pain or headache.[97] On examination, the visual acuity may vary from 20/20 on one extreme to no light perception on the other; an afferent pupillary defect and visual field defect are typical, with the visual field defect usually being altitudinal (i.e., affecting either the upper half or lower half of the visual field) due to the segmented microvascular anatomy of the optic nerve head.[98] Altitudinal optic nerve head swelling in the affected eye may be visible on examination, see **Figure 7**.

Figure 7. Anterior Ischemic Optic Neuropathy With Visible Swelling Of The Optic Disc



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Diagnostic Workup And Management

The diagnosis of ischemic optic neuropathy is based on historical features and the appearance of the optic disc on fundscopy.[99] Magnetic resonance imaging (MRI) of the orbits may be useful to further distinguish this condition (which is ischemic) from a clinically similar condition: optic neuritis (which is inflammatory); in one study, 31/32 patients with optic neuritis (compared with only 5/32 patients with ischemic optic neuropathy) had abnormalities of the optic nerve visible on MRI.[100]

Unfortunately, there is no known treatment for NAION. Multiple treatments have been tried in clinical trials, including corticosteroids, anticoagulants, and hyperbaric oxygen,[101] as well as surgical decompression; [102-103] though none have been successful. Recovery is variable, with improvement attributed to adaptation to the visual field deficit as well as improvement in visual acuity.[39,104]

Unilateral Painful Visual Disturbance

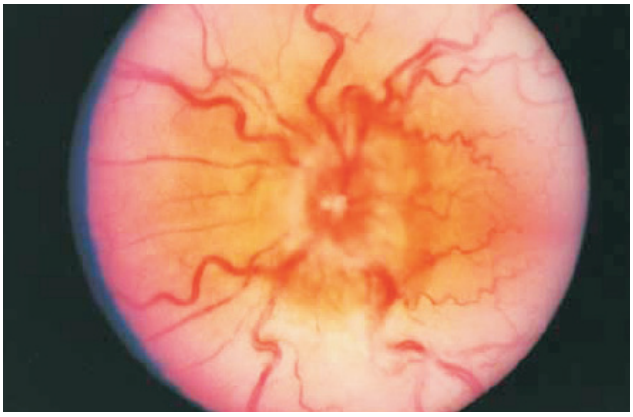
The areas of the eye that are sensitive to pain are the cornea, iris, and ciliary body, as well as the periorbital tissues. Any inflammatory or infectious process that involves these areas will result in some discomfort or pain.[2] The retina, optic nerve, and vitreous are relatively insensitive to pain.

Optic Neuritis

Presentation And Examination Findings

Optic neuritis is typically heralded by a dull retro-orbital ache, followed in the next day or so with monocular blurred vision. It is usually subacute in onset, with a nadir over hours to days, and is associated with pain on eye movement in 92% of patients.[2,32-33,37-38,105] On examination, there may be varying degrees of compromised visual acuity, and a central visual field defect is typical; an afferent papillary defect may be present, and papilledema or swelling may be visible on fundoscopy if the nerve is affected anteriorly (**Figure 8**) but is absent initially in about 70% of patients because the process is primarily retrobulbar.[2,32,37-38]

Figure 8. Optic Nerve Swelling (Papillitis) From Optic Neuritis



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Diagnostic Workup And Management

The most comprehensive work on the clinical assessment and management of optic neuritis comes from the Optic Neuritis Treatment Trial, a multicenter, randomized, controlled trial funded by the National Eye Institute.[33] The diagnosis of optic neuritis is a clinical one based on history and fundoscopic examination and will likely be significantly aided by ophthalmo-

logical consultation. Gadolinium-enhanced MRI of the orbits (with fat saturation) is useful in cases in which the diagnosis is suspected but uncertain, as it will highlight the inflammation and swelling of the optic nerve and can delineate high-risk multiple sclerosis lesions if imaging of the brain is also included.[32,106] Other diagnostic modalities, such as searching for oligoclonal bands in CSF and assessing visual evoked potentials,[32] are impractical in the ED setting.

The prognosis of optic neuritis is generally good but return of visual function is almost never complete.[32,107-108] Current guidelines refer to treatment with high-dose steroids.[109-110] A meta-analysis of 12 randomized, controlled, clinical trials found that steroids resulted in early but non-sustained improvement in visual acuity.[111] It has also been associated with a decrease in the incidence of subsequent development of multiple sclerosis at two years, although this does not appear to be a sustained effect beyond that time point.[32,34,108]

Acute Glaucoma

Presentation And Examination Findings

The patient with an acute elevation in intraocular pressure from glaucoma typically presents with a boring, ocular, or peri-ocular pain with nausea and/or vomiting and blurred vision in the context of a history of intermittent blurring of vision with halos. Examination findings will typically be notable for corneal injection, corneal epithelial edema, elevated intraocular pressure (IOP) over 21 mmHg, and - with acute angle-closure glaucoma - a mid-dilated unreactive pupil and a shallow anterior chamber.[112-113] With significantly elevated IOP, the globe may feel firmer than the normal eye when digitally palpated.[114-115]

Table 3. Medical Therapy Of Acute Glaucoma In The ED

Agent	Concentration	Dosing	Mechanism
Timolol	0.5%	1 drop twice daily, topically	Reduces aqueous humor secretion
Latanoprost	0.005%	1 drop, topically	Prostaglandin analogue; improves uveoscleral outflow
Pilocarpine	1% to 2%	1 drop topically every 15 minutes until pupillary constriction occurs	Constricts the pupil and allows optimized drainage of aqueous humor secretions
Acetazolamide	N/A	500 mg IV	Carbonic anhydrase inhibitor; decreases aqueous humor secretion
Mannitol	20%	2-7 mL/kg IV	Osmotic agent; reduces intraocular pressure oncotically
Glycerol	50% solution	1.5 to 4 mL/kg PO	Osmotic agent; reduces intraocular pressure oncotically
Narcotic analgesia	variable	variable	Analgesia; improves patient comfort

Diagnostic Workup And Management

While open-angle and closed-angle glaucoma have slightly different etiologies, their acute treatment in the ED is initially the same and is centered on medical treatment as a temporizing measure until definitive ophthalmological treatment can be arranged. In the case of open-angle glaucoma, the patient can be symptomatically stabilized using a medical regimen of topical and systemic medications, see **Table 3**.

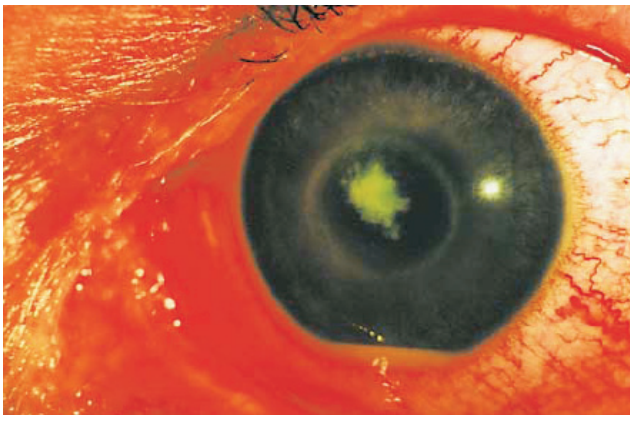
In the case of acute closed-angle glaucoma, definitive treatment will require evaluation by an ophthalmologist emergently, unless initial medical therapy results in acute and complete improvement. A recent evidence-based review of the literature regarding available therapies for closed-angle glaucoma found mostly class III evidence. Laser peripheral iridotomy was a Level A recommendation, supplemented by topical administration of timolol and latanoprost which was a Level B recommendation.[113]

Corneal Infections, Ulcers, And Abrasions

Presentation And Examination Findings

Corneal processes such as abrasion, infection, and ulceration typically present with eye pain and a red eye and may have obscured vision.[2] The presence of flare (from hypopyon) or iritis suggests infection over abrasion and a defect, with "heaped up" edges on fluorescein staining suggesting an acute infectious ulceration (keratitis), see **Figure 9**. Herpes simplex keratitis (the most common viral keratitis) may have a dendritic or geographic appearance on fluorescein staining, see **Figure 10**.

Figure 9. A Fungal Corneal Ulcer



Note the associated hypopyon.

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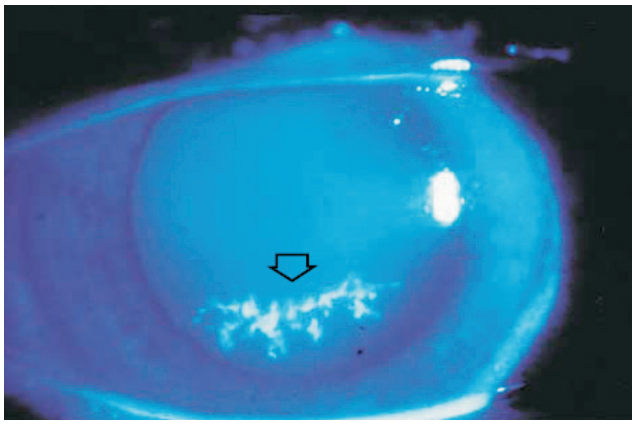
Figure 10. Acute Herpes Keratitis



Acute herpes keratitis, with a dendritic pattern visible on cobalt blue illumination after fluorescein staining.

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Diagnostic Workup And Management

The diagnosis of the process that is causing an acute corneal abnormality is essentially achieved on examination, and the management implemented is based on whether the examination findings suggest a corneal abrasion, an infectious corneal ulcer, or a herpetic viral infection.

A recent meta-analysis of randomized, controlled trials found that topical non-steroidals reduce self-reported pain from corneal abrasions,[116] and a review for the Cochrane Database found that the use of eye patches actually slows corneal healing in the first day and does not reduce patient discomfort over the subsequent days.[117] Simple corneal abrasions can be discharged to follow-up with an ophthalmologist with topical antibiotic prophylaxis to prevent complications from bacterial superinfection and topical non-steroidals; they should avoid patching.

A recent Cochrane Database review found that the use of topical antibiotics for simple bacterial conjunctivitis improves rates of clinical and bacterial remission but has a marginal impact on actual outcome of the infection itself (since bacterial conjunctivitis is generally a self-limited problem).[118B] On the other hand, an ulcerative bacterial keratitis represents a more fulminant infection in which topical antibiotics have a significant impact on outcome; patients with this finding should be urgently referred to an ophthalmologist and treated emergently. Contact lens wearers should be treated with topical antibiotics with coverage for *Pseudomonas*, as this pathogen has been shown to be present in about 50-60% of corneal infections in this patient population.[118-119] For herpes simplex keratitis, treatment with topical antivirals (such as acyclovir) has been clearly shown to improve corneal epithelial healing in clinical trials, as confirmed in a review for the Cochrane Database;[120] institute treatment immediately, and refer the patient urgently to an ophthalmologist.

Endophthalmitis

Presentation And Examination Findings

The patient with endophthalmitis presents with pain out of proportion to the clinical examination, photophobia, and visual loss, typically about six days or so after routine eye surgery such as a cataract removal,[121-122] or after trauma.[123] There may be no precipitant in endogenous endophthalmitis. The examination may be subtle or may be notable for lid edema, hypopyon, conjunctival erythema and edema, corneal edema, and obscuration of the fundoscopic view.[123]

Diagnostic Workup And Management

The mainstay of treatment of endophthalmitis is the immediate administration of intravitreal antibiotics and vitrectomy; therefore, emergent consultation with an ophthalmologist is necessary. The bacterial species in the majority of post-surgical and post-traumatic cases of endophthalmitis is staphylococcal[121-123] so a regimen of intravenous antibiotics that includes staphylococcal coverage, such as ceftazidime 1 gram IV every 12 hours and vancomycin 1 gram IV every 12 hours, should also be administered.[123]

Cavernous Sinus Thrombosis

Presentation And Examination Findings

The most common signs of cavernous sinus thrombosis are due to fulminant infection and compression of the nerves that run through the sinus and are progressive, involving fever, ptosis, proptosis, chemosis, and cranial nerve palsies in 80% of patients; lethargy, periorbital edema, headache, papilledema, and venous engorgement in 50–80% of patients; decreased visual acuity, sluggish or dilated pupil (due to affected parasympathetic fibers), periorbital sensory loss, decreased corneal reflex (due to CN V involvement), and nuchal rigidity in less than 50% of patients; and diplopia, seizures, and hemiparesis in less than 20% of patients.[57] Involvement of the contralateral eye is typical by about 12-24 hours.

Diagnostic Workup And Management

Contrast CT with 3 mm slices may reveal bulging

cavernous sinus with lateral wall flattening or convexity rather than normal concavity or filling defects (thrombosis),[57,124] but an MRI venogram is considered a more sensitive imaging modality[59,125] (although no distinct head-to-head trials have been performed) and has the added benefit of highlighting carotid artery involvement and dural sinus thrombosis.[57-58] Blood cultures may be positive in 80% of cases (especially fulminant cases), and a lumbar puncture will reveal CSF with elevated protein and WBC in most patients but will be culture positive in only 20%.[57]

Staphylococcus aureus is the causative organism in 60–70% of cases of septic cavernous sinus thrombosis with organisms such as *streptococcus pneumoniae*, gram-negative bacilli, and anaerobes being less common.[57-58] Anticoagulation with warfarin or heparin started within seven days of onset may theoretically improve morbidity,[57,125B-126] but no randomized trials have been conducted. The use of steroids, though with some suggestion of benefit in improving cranial nerve dysfunction and persistent orbital congestion, is unsupported.[57]

Uveitis Or Iritis

Presentation And Examination Findings

A patient with anterior uveitis (or iritis) may present with photophobia, eye pain, and slightly blurred vision. On examination, the patient may have the proverbial "red eye," with signs of conjunctivitis or scleritis and may have a hypopyon[9,70] and pain on direct and consensual light reflex. The pupil may be miotic, presumably due to inflammatory synechiae, but a normal pupil does not exclude an acute iritis.[127]

Diagnostic Workup And Management

The diagnosis of iritis is a clinical one based on examination. The goals of acute treatment are twofold. The first goal is provision of comfort to the patient and prevention of complications from synechiae, achieved by administering cyclopegics (such as atropine) or mydriatics (such as phenylephrine), one or two drops three to six times a day until inflammation is completely controlled.[9] Sight-threatening complications from anterior uveitis include keratopathy, synechiae, and glaucoma (from debris);[70] therefore, the second goal of treatment is to reduce inflammation, usually with topical corticosteroids such as betamethasone, dexamethasone, or prednisolone, given every one to two hours until inflammation is under control.[9,128]

Bilateral Painless Visual Disturbance

While sudden bilateral painless visual disturbances can, on very rare occasions, be caused by a transient ischemic attack to the occipital visual cortex[129] or transient vertebrobasilar insufficiency, the etiology to an acute painless visual disturbance affecting both eyes is likely to be from physiological, metabolic, toxic, or pharmacologic etiologies. Acute methanol toxicity may present with ocular manifestations of blurred vision, photophobia, painful eye movements, and reduced visual acuity. On examination, the patient may have optic disc edema with engorged retinal veins.[130]

Idiopathic Intracranial Hypertension (Pseudotumor Cerebri)

Presentation And Examination Findings

Patients with idiopathic intracranial hypertension (IIH) typically present with chronic daily headaches (the major cause of morbidity)[40] and visual abnormalities – either monocular or binocular transitory visual obscurations varying from slight blurring to total loss of light perception - which are seen in up to 72% of the patients.[131] The headache, sometimes associated with nausea and pulsatile tinnitus, is generally worse in the morning and worsened by valsalva.[41] On examination, papilledema with blurring of the optic disc border, absent spontaneous venous pulsations, distention of the retinal veins, visual field deficits, and possibly even protrusion of the optic disc with hemorrhages and exudates may be present; a horizontal diplopia from an associated sixth nerve palsy may rarely be present.[41]

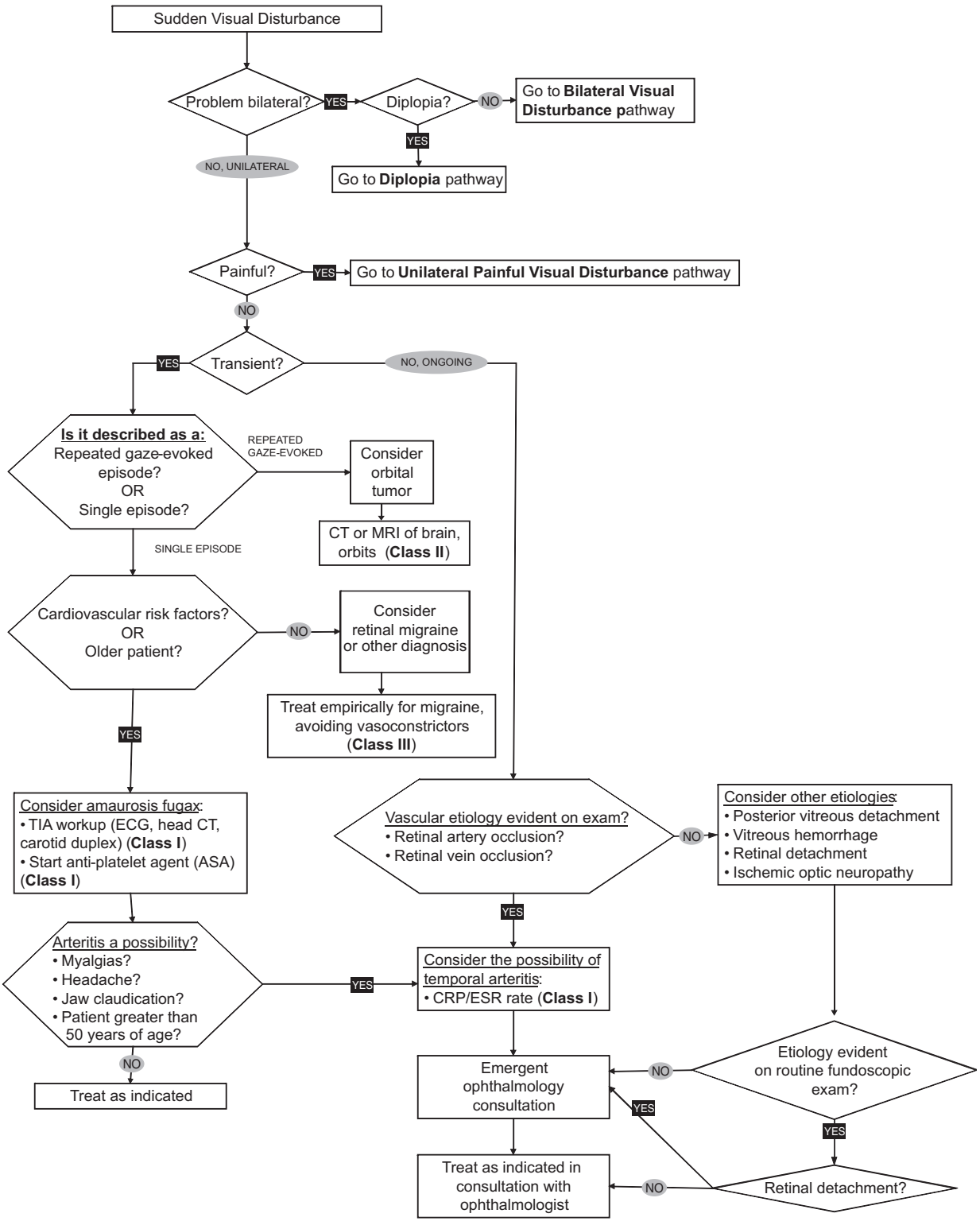
Diagnostic Workup And Management

IIH should be strongly suspected in any young obese female presenting with chronic headaches, blurred vision or visual disturbance, and papilledema on examination. The diagnosis of IIH requires four diagnostic criteria:

1) Increased ICP
2) Normal ventricles on neuroimaging
3) No intracranial mass
4) Normal CSF

A lumbar puncture with measurement of opening pressure, CSF analysis, and head CT are indicated to establish the diagnosis and rule out intracranial mass, hydrocephalus, or other etiologies of headache and papilledema.[40-41] The CSF pressure will usually

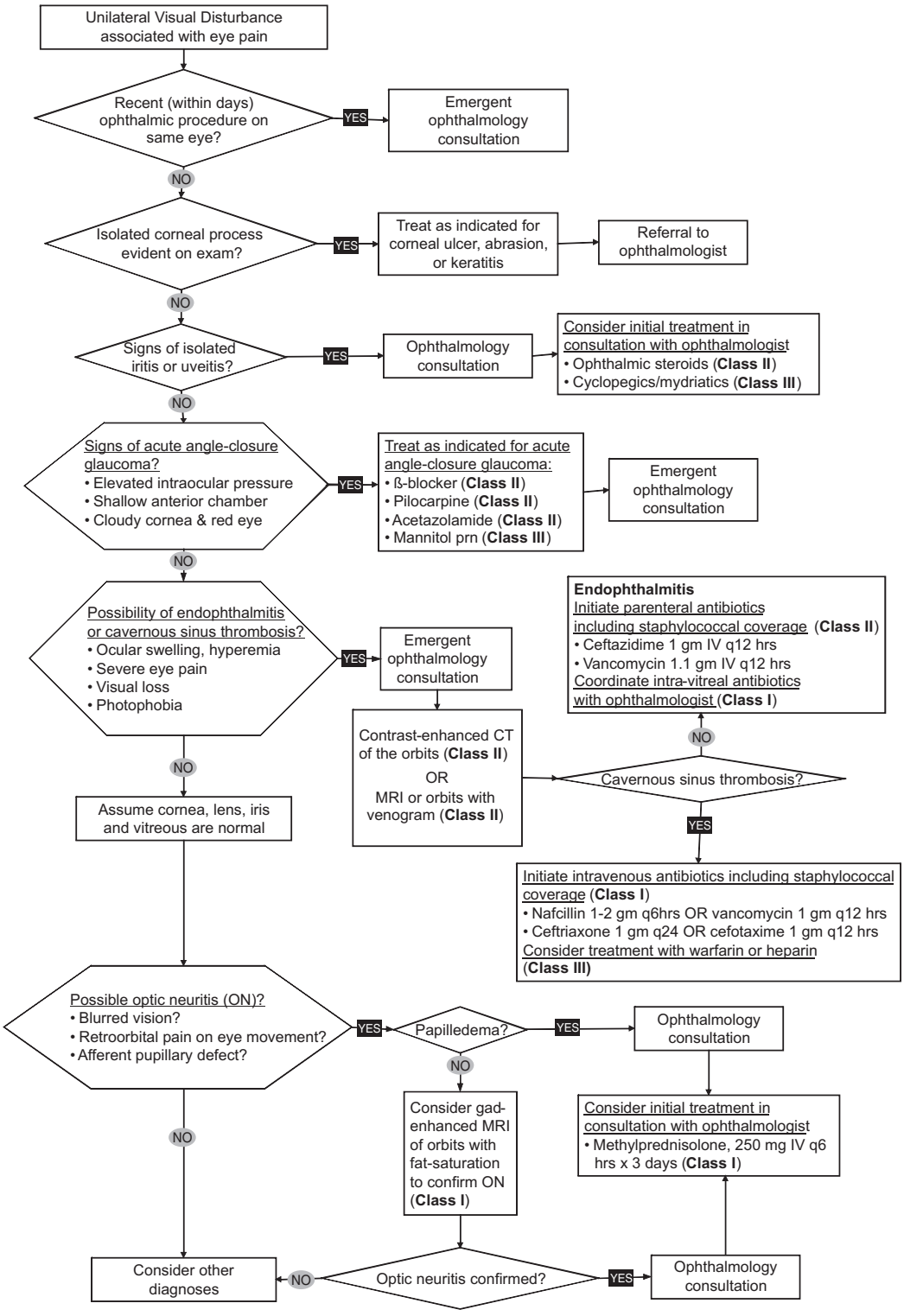
Clinical Pathway: Abnormal Vision



The **evidence for recommendations** is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II:** Acceptable and useful. Good evidence provides support. **Class III:** May be acceptable, possibly useful. Fair-to-good evidence provides support. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

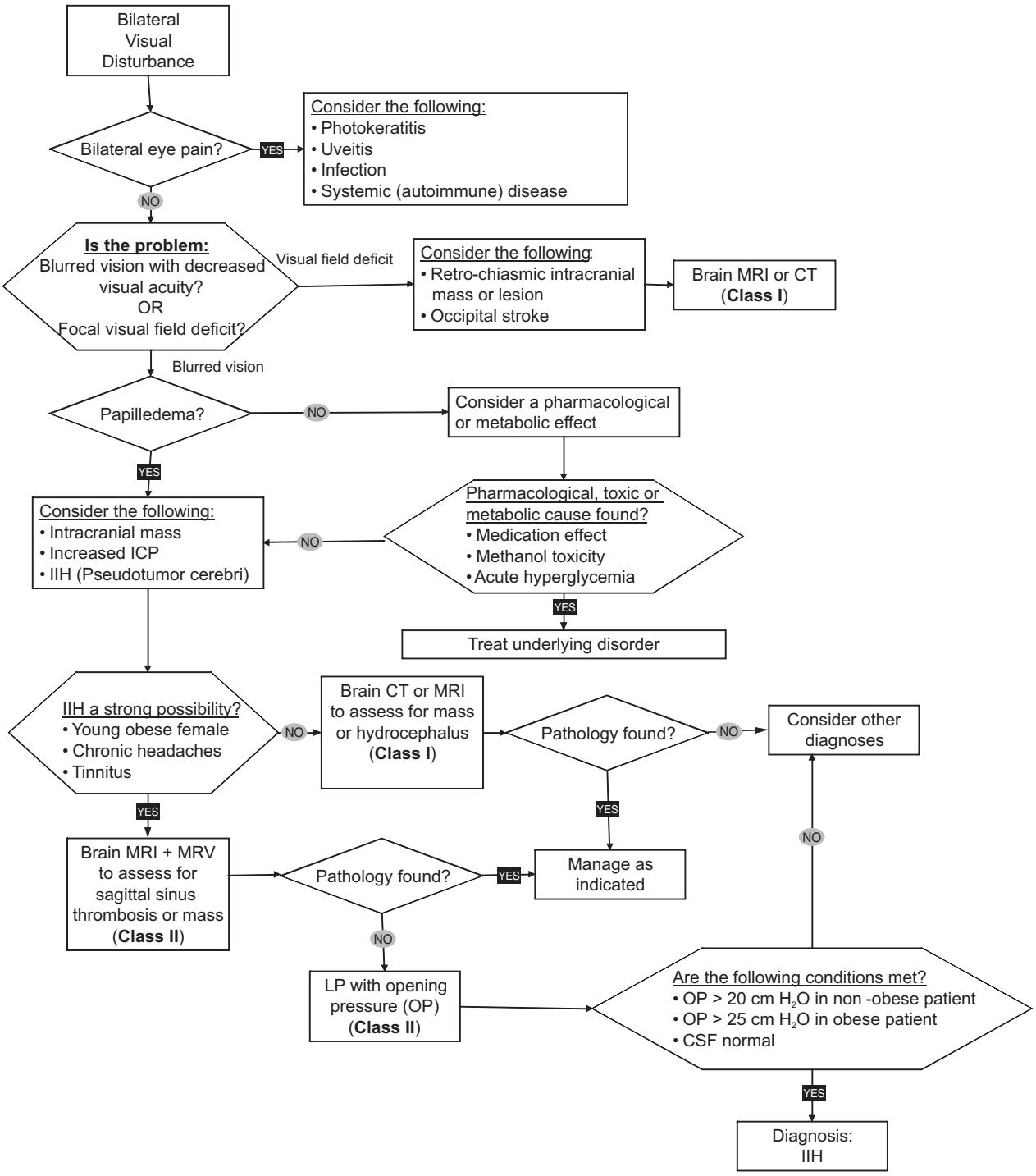
Clinical Pathway: Unilateral Painful Visual Disturbance



The **evidence for recommendations** is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II:** Acceptable and useful. Good evidence provides support. **Class III:** May be acceptable, possibly useful. Fair-to-good evidence provides support. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

Clinical Pathway: Bilateral Visual Disturbance



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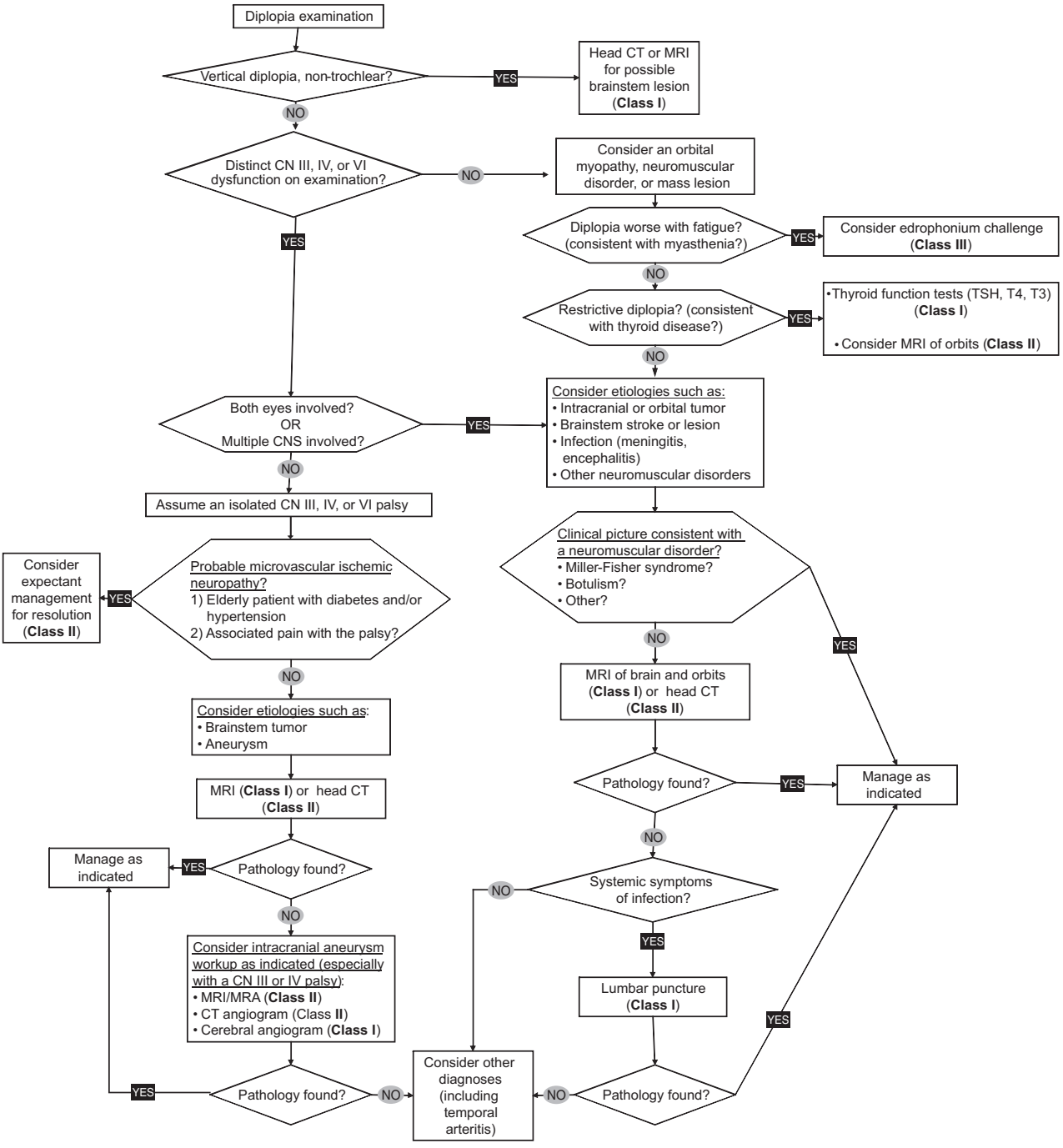
Clinical Pathway: Diplopia



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be elevated above the normal value of 20 cm H_2O but is prone to natural variations and even transient normalcy.[41,132] A pressure of 20 cm H_2O or below is considered normal, a value between 20 and 25 cm H_2O is non-diagnostic, and a pressure of over 25 cm H_2O is diagnostic of IIH.[133-134] A young female presenting with headache and papilledema may raise the specter of sagittal sinus thrombosis; an MRI and MRV can be used to evaluate for this and may reveal additional signs of elevated CSF pressure related to IIH, such as a partially empty sella, dilation or tortuosity of the optic nerve sheet, or gadolinium enhancement of the optic disc.[135]

A variety of treatments for IIH have been described in the literature, including repeated lumbar punctures, weight loss, a variety of drugs (such as acetazolamide, diuretics, oral glycerol, corticosteroids, and cardiac glycosides), hyperbaric oxygen, vasopressin, and a variety of CSF shunting approaches. A 2005 Cochrane Database review of the available literature found no evidence that any of these work in a sustained fashion.[40] In the ED, the crux of management revolves around symptomatic relief and appropriate referral.

Sudden Bilateral Painful Visual Disturbance

The most common nontraumatic etiologies of a bilateral painful visual disturbance include chemically-induced and ultraviolet light-induced corneal injuries (such as from tanning beds, skiing, and arc welding). These patients can be treated with topical antibiotics and analgesics pending referral.

Diplopia

The approach to diplopia in the ED entails sorting out the etiologies which may result in rapid or profound morbidity from those which can be followed up as an outpatient with specialty referral.

Cranial Nerve (CN) Dysfunction

Presentation And Examination Findings

The cranial nerves responsible for ocular motility are the oculomotor nerve (CN III), the trochlear nerve (CN IV), and the abducens nerve (CN VI). Palsies in each present with typical findings, see **Table 4**. An oculomotor (CN III) palsy results in impaired elevation, depression and adduction of the eye; when complete, it results in an eye that is deviated "down and out," with a dilated pupil and ptosis.[68] A trochlear (CN IV) palsy results in paresis of the superior oblique muscle leading to vertical diplopia with a torsional component that is worse on downward gaze and makes descending stairs, reading, and watching television in bed difficult. A compensatory head tilt may be the first indication of trochlear lesion.[51,68] An abducens (CN VI) palsy results in paresis of the lateral rectus muscle and impairment of abduction of the ipsilateral eye and esotropia.[51,68]

Diplopia due to microvascular ischemia, which is typically seen in older patients with vascular risk factors such as diabetes and hypertension, may present with an isolated palsy associated with pain in one of the cranial nerves. An oculomotor palsy from this process classically spares the pupil. An oculomotor palsy that develops from compression, such as that from an aneurysm, typically involves the pupil, due to compression of parasympathetic fibers in the exterior third of the nerve.[55]

Involvement of multiple cranial nerves suggests an orbital, cavernous sinus, or brainstem process. Vertical diplopia without the torsional component seen with trochlear palsy (called a vertical skew deviation) suggests a brain stem lesion. An internuclear ophthalmoplegia (the ability to adduct the eye on one side in the contralateral direction during lateral gaze that resolves during convergence) implicates a lesion in the medial longitudinal fasciculus (MLF) that is commonly found in patients with MS.[51]

Diagnostic Workup And Management

Given the etiologies of diplopia, most cases will need imaging. The possibilities suggested by the history and physical guide the workup. A CN III palsy is perhaps the most critical to assess, since an aneurysm in the basilar, superior cerebellar, posterior cerebral, posterior communicating arteries, or the cavernous portion of the internal carotid artery can produce the palsy.[51] Choose imaging and management standard for that required to assess for an aneurysm. Magnetic resonance angiography (MRA) and CT angiogram have risen to the forefront as alternatives to traditional cerebral angiogram.[136] Three-dimensional MRA has a 97% sensitivity for detecting aneurysms greater than 5 mm in diameter, but only a 54% sensitivity for aneurysms less than 5 mm in diameter.[137] Ninety-two percent of the posterior communicating artery aneurysms which cause a third nerve palsy are greater than 5 mm in diameter,[68,137] which makes MRA a useful modality. However, MRA will overlook 1.5%

of third nerve palsy-causing intracranial aneurysms that, if left untreated, will rupture in the subsequent eight years.[137] MRA should therefore be the single diagnostic test only in cases in which the likelihood of aneurysm is low or the likelihood of having a complication from cerebral angiography is high.

One caveat for CN III palsies is the elderly diabetic patient presenting with an acute pupil-sparing CN III palsy associated with eye discomfort. In such a patient, microvascular ischemic neuropathy is such a strong possibility that imaging in the ED can probably be deferred in favor of discharge with close outpatient followup to assess for spontaneous resolution. This type of vascular neuropathy, which is typically seen in older patients with risk factors for microvascular disease (such as diabetes), is generally self limited; the pain usually resolves after a few days,[55] and complete, spontaneous resolution is the norm, occurring in 86% of patients in one study[138] and in 95% by 12 months in another.[139] This same consideration can be given to a similarly presenting acute CN VI (abducens) palsy, but not to a CN IV palsy since the trochlear nerve is rarely affected by this condition.[68]

Palsies in CN IV and CN VI presenting without other deficits require less emergent imaging than those of CN III. The most optimal study is MRI of the brain with gadolinium, high-resolution cuts through the brainstem, and fat-suppressed orbital imaging to assess for inflammation, neoplasm, or demyelination along the course of the nerves.[68,72] If dysfunction of cranial nerves III, IV, and VI occurs in combination without any other signs of bulbar disease, strongly suspect an infiltrative, inflammatory, or compressive lesion of the cavernous sinus or orbital apex, and obtain an MRI of the brain and orbits with gadolinium.[68]

Dysfunction of multiple oculomotor nerves due to neuromuscular or infectious syndromes such as botulism and Miller-Fisher syndrome should be managed as indicated for each disease process.

Table 4. Symptoms, Examination Findings, And Differential Diagnoses In CN III, IV, And VI Palsies

Nerve palsy	Symptoms	Typical examination findings	Differential diagnosis
Normal	N/A		N/A
Oculomotor (CN III) palsy	Diplopia, horizontal and vertical		Posterior circulation aneurysm Brainstem lesion Microvascular ischemia (if pupil spared) Cavernous sinus disease
Trochlear (CN IV) palsy	Torsional ("tilted") diplopia worse on downward gaze		Brainstem lesion Posterior circulation aneurysm Cavernous sinus disease
Abducens (CN VI) palsy	Horizontal diplopia on lateral gaze to the ipsilateral side		Brainstem lesion Elevated intracranial pressure Cavernous sinus disease

Extraocular Muscle Dysfunction

Presentation And Examination Findings

Diplopia due to extraocular muscle dysfunction presents in a somewhat variable fashion that is typical for myopathy. Diplopia that is worse in the morning suggests an extraocular muscle etiology,[68] and diplopia or ptosis that are more prominent when fatigued suggests myasthenia gravis.[51] The diplopia may be just one of a constellation of symptoms of neuromuscular disease that includes proximal muscle weakness, difficulty breathing, or difficulty swallowing. The examination may reveal stigmata of the underlying disease process, such as lid lag in thyroid disease, muscle atrophy or weakness in neuromuscular disease, or facial swelling, proptosis, or ocular protrusion in structural orbital disease. Thyroid eye disease classically affects the inferior and medial recti muscles first, leading to restriction of elevation and abduction of the eye.[68]

Diagnostic Workup And Management

Since diplopia from extraocular muscle dysfunction is caused by either neuromuscular disease or structural orbitopathy, the diagnostic workup is primarily focused on screening for neuromuscular disease and/or imaging of the orbit and brain where indicated. MRI of the brain with gadolinium with high-resolution cuts through the brainstem and fat-suppressed orbital imaging can allow an assessment for enlargement or enhancement in extraocular muscles, orbital structures, and central nervous system structures.[68] An edrophonium (Tensilon) challenge test can be preformed for patients in whom myasthenia gravis is suspected. It has a reported sensitivity and specificity for ocular myasthenia of 92% and 97%, respectively, based on the single trial in which it was comparatively evaluated.[140-141] Once the etiology of diplopia has been determined, disease-specific treatment (a comprehensive review of which is beyond the scope of this article) can be initiated.

A Special Consideration: Functional Vision Loss

Functional vision loss, sometimes called factitious visual loss, has a prevalence of 5-12% in ophthalmology practices[142-143] and can present with vision loss in one or both eyes, ptosis, blepharospasm, and diplopia.[143] The prevalence of a history of psychiatric disease is relatively high (almost 40%),[144] and psychosocial stress may underlie the presentation.[143,145] Over 90% of complaints are afferent symptoms, such as vision loss, visual field deficit, or decreased acuity.[145] The patient with FVL may "bump" into objects in the room in an exaggerated fashion, compared to the truly blind patient who moves very cautiously.[146]

While the task of distinguishing functional vision loss (FVL) from true organic disease may be best left to a consultant ophthalmologist, neuro-ophthalmologist, or neurologist, there are a few tests that can be performed in the emergency department which will go a long way towards confirming a functional overlay. The most common presentation of FVL is decreased visual acuity[143-144] so the following examination techniques may be used:

1) **The optokinetic nystagmus test**: For the patient with complete vision loss, check for optokinetic nystagmus. Place a cylinder with alternating black and white longitudinal lines in front of the patient. Upon spinning the cylinder in its longitudinal axis, the patient with intact vision will develop horizontal nystagmus.
2) **The hand-held mirror test**: Place a mirror in front of the eye (or eyes) in question, and slowly rock it back and forth. The patient with normal vision will find it extremely difficult to keep his or her eyes from moving.
3) **The secret proprioception test**: Have the patient sit upright, with both index fingers extended but flexed medially, and instruct him or her to touch the right finger tip with the left. Even the truly blind patient will be able to perform this task, since proprioception is intact, but the patient with FVL will often feign an inability to do this, missing the target.[143,146]
4) **The blink-to-visual-threat test**: Upon a rapid forward motion of the examiners hand towards the eye in question (while covering the normal eye if vision loss is monocular), the patient with true visual loss will not blink, while the patient with FVL will be unable to suppress his or her blink reflex. Care must be taken not to illicit a corneal blink reflex with air movement thereby created.
5) **The APD test**: The patient with true complete monocular vision loss of a retinal or optic nerve etiology will have an afferent pupillary defect in the eye in question, while the patient with FVL will not.
6) **The surprise test**: As per Egan, "*Surprise can be elicited from a patient who has poor vision. Using a card or piece of paper, a common word such as "house" is displayed to the patient's bad eye or eyes. When the patient professes that he cannot see the word, the card is turned over, revealing an expletive in small print. A look of surprise may suggest that the patient can indeed read the small print.*"[143] The ability of an expletive to elicit surprise in a cohort of malingering ED patients presenting during a night shift has not been prospectively evaluated, however.

Controversies/Cutting Edge

Intra-arterial Thrombolytics For Central Retinal Artery Occlusion

In addressing the wide range of causes of an acute visual disturbance, the emergency physician is faced with specific dilemmas. Central retinal artery occlusion results in a sudden, profound, and usually permanent loss of vision in the affected eye, and the variety of treatments that have been suggested lack any robust evidence of efficacy. Superselective thrombolysis via ophthalmic artery (local intra-arterial fibrinolysis [LIF]) has been suggested to have some benefit[89-90,147] and is being used in certain centers. The approach is not without issue, however, as the best results seem to occur if thrombolysis is performed within six to eight hours of onset,[147] and the lack of expedient availability of an interventional radiologist may prevent some patients from receiving this therapy in time to benefit from it.[90] In addition, while there are no reports in the literature clearly describing a significant rate of lytic-induced hemorrhage, it is a complication that is theoretically possible. There is also a small catheter-related risk of transient ischemic attack and stroke associated with the procedure itself.[90] The results of a multicenter, randomized, controlled study currently being planned[91] should help clarify the efficacy and safety of local intra-arterial fibrinolysis as a treatment for retinal artery occlusions.

Deferring Neuroimaging In Patients With Suspected Microvascular Ischemic Neuropathy

The literature shows that with microvascular ischemic neuropathy, which typically results in a CN III palsy in elderly patients with diabetes and/or hypertension, resolution is typically spontaneous and complete and warrants no acute intervention.[138-139] While the implication and recommendation in the literature is that no acute imaging workup is necessary (reserving such a workup to cases in which resolution does not occur) in the elderly patient with diabetes or hypertension presenting with a pupil-sparing CN III palsy and eye discomfort typical of microvascular ischemic neuropathy, there are no published studies that clearly support a contention that clinical criteria alone have enough specificity to reliably differentiate this process from more ominous potential causes of an isolated pupil-sparing CN III palsy, such as an aneurysm or an orbital or brainstem tumor. The emergency physician is therefore left with the dilemma of obtaining an expensive imaging study for a benign, self-limited condition on one hand versus missing the diagnosis of a more serious pathological condition on the other and has to use clinical judgment in the clinical decision-making.

Summary

An acute disturbance in vision may be a distressing development for the ED patient. The underlying etiology may range from a simple, innocuous ocular problem to a complex or serious vascular, central nervous system, or systemic process; as such, the ED evaluation has to be systematic. A methodical progression through the examination, starting with a general inspection of the face and orbital tissues, visual acuity, visual fields, and structures of the eye, as well as measurement of intraocular pressure when indicated will go a long way in revealing sometimes unexpected contributory pathology. The differential can be narrowed down based on the presence or absence of diplopia, involvement of one or both eyes, the presence or absence of pain, and, of course, the examination. In most cases, the examination will be diagnostic; though neuroimaging is indicated in select cases.

Case Conclusion

The smiling grandmother who kept bumping into furniture had a right-sided visual field defect when you examined her. A non-contrast CT scan of the brain revealed a stroke to her left occipital lobe, involving the visual cortex. She was outside the eligibility time window for any reper-

Cost-Effective Strategies For The Management Of Abnormal Vision

1. Think about topical anesthetics as both a diagnostic and examination aid.

In the patient with a painful eye, a few drops of a topical ophthalmic anesthetic at the start of the examination may offer two distinct benefits. First, if the cause of the problem is due to a corneal abrasion or superficial process, the anesthetic may significantly reduce pain and suggest this as the etiology. Secondly, the pain reduction thereby attained will facilitate improved cooperation and result in a faster, more thorough examination.

2. Use a pin-hole card in assessing visual acuity when needed.

Patients frequently present to the ED with a visual complaint and may leave their corrective lenses at home. Rays of light passing through a pin-hole are parallel and do not need to be focused by the cornea and lens. Using a pin-hole to assess visual acuity in this circumstance can go a long way in ruling out a significant pathological aberration in visual acuity.

3. Utilize specialty resources for detailed examination when available.

The ophthalmologist will typically have an entire suite of specialized examination equipment and techniques at his or her disposal. In cases in which the examination is difficult or limited (such as with visual obscuration on fundoscopy), utilize this resource early – if available - in order to facilitate and expedite the evaluation of the patient.

Risk Management Pitfalls For Abnormal Vision

"I believe in looking reality straight in the eye and denying it." - Garrison Keillor

1) "…But the patient told me she had a headache, not an eye ache."

This patient had acute closed-angle glaucoma in her left eye; she was treated for a "migraine headache" on that side and subsequently developed complications from the misdiagnosed glaucoma. Due to the systemic complaints that frequently accompany an acute elevation in intraocular pressure, such as headache, nausea and vomiting, the physician can easily be misled and the diagnosis easily missed.[147B] A missed diagnosis of glaucoma is a common source of physician liability.[148]

2) "He begged me for the anesthetic drops, and they did make him feel so much better in the ED…so I gave them to him and told him to use them sparingly."

While, in the acute setting, instilling topical anesthetics into the eye of a patient with a corneal abrasion, ulcer, or inflammation may result in a marked amount of relief from the pain that is gratifying to the physician (and is useful in the examination setting), the use of topical anesthetics for even a limited amount of time after that has been associated with corneal breakdown. Topical ophthalmic anesthetics inhibit the rate of corneal epithelial cell migration (which impairs healing of corneal epithelial abrasions), have direct toxic effects on keratocytes,[149-150] and are prone to misuse in the hands of patients.[150-152] In addition, topical anesthetics can mask the pain of a tarsal (eyelid) foreign body.

3) "He looked like he had simple conjunctivitis, and blurred vision wasn't one of his complaints, so I assumed that checking a visual acuity would be of low yield."

Oops, this patient had much more than a simple conjunctivitis. Visual acuity is considered a vital sign of the eye, and, if significantly decreased below baseline, may be an indicator of a more ominous pathological process. A distinct abnormality in corrected visual acuity is not consistent with a simple conjunctivitis.

4) "She said she'd had a reaction to the oral form of that antibiotic, not the formulation they put in the eye drops."

An allergy is an allergy, and a molecule is a molecule. Medication errors, especially antibiotic-related ones, are the most common cause of litigation in the ophthalmology literature.[153] Be wary of the ophthalmic preparation of an oral antibiotic that is on a patient's allergy list.

5) "My tonometry device was broken… but his eyes felt like they had the same turgor to me, so I didn't think his intraocular pressure was elevated."

Digital palpation, while usually revealing in cases of IOP significantly elevated over 30 mmHg, is an unreliable measure of IOP as a whole[115] and should never be used to exclude the diagnosis of acute glaucoma in cases in which it is suspected.

6) "The patient didn't tell me that something got into her eye."

Eversion of the eyelids, especially the upper one, is an important part of the ophthalmological examination in a patient presenting with a corneal abrasion without a history of trauma. The patient may not be aware that a foreign body got into the eye.[154] If missed, a tarsal foreign body will continue to inflict injury on the corneal epithelium.

7) "The patient had a completely normal visual acuity examination… 20/20 in both eyes. It just didn't seem consistent with a retinal detachment at the time."

Visual acuity is a task for the macula, which is a relatively small part of the retina. In a patient with pathology not immediately or directly compromising it, such as a branch retinal artery occlusion or a retinal detachment in the periphery that hasn't yet reached the macula, visual acuity may be deceptively normal.

8) "The patient should have known better and *not* tried to drive with his blurred vision after I put those dilating drops in."

Mydriatic drops are very useful in obtaining an unencumbered view of the retina through a dilated pupil. The pupillary dilation does, however, result in blurring of vision. This effect, though transient, can be incapacitating, especially if both eyes are dilated in the same setting. A first layer of safety is to only dilate one eye unless it is necessary to dilate both, and a second layer of safety is to advise the patient of the side-effect and the limitations it will place upon him or her.

9) "Yes, she did complain about a headache and problems with her vision, but she said both eyes were involved and couldn't describe the problem well - and her visual acuity was normal, as was her examination on fundoscopy. I just didn't find any conclusive evidence that would have led me to suspect the brain tumor over a migraine."

That's because visual field testing wasn't performed. This patient had a pituitary tumor with a visual field deficit that was missed and had a bad outcome from pituitary apoplexy – a type of diagnostic error and complication that has been a source of litigation on several, albeit rare, occasions.[155] In a patient with abnormal vision, the visual complaint has to be fully delineated, and visual field testing should be performed unless the etiology is obvious without it.

10) "I thought that she just had eye strain which caused the pain when she moved her eye; after all, she was young and healthy and the eye examination was normal."

Yes, the eye examination was normal - except for the swinging flashlight test that was never done. If it had been, you would have discovered the afferent pupillary defect (APD), realized there was more to the story, and made the diagnosis of optic neuritis, which was the presenting finding of this young woman who was subsequently diagnosed with multiple sclerosis.

fusion therapy, so you started her on aspirin and admitted her for further workup.

You reflected on how you arrived at the diagnosis... You first went at the exact nature of her complaint. Her lack of double vision took you out of the diplopia pathway. There was also nothing that pointed towards a cranial nerve problem. The subtlety of the vision complaint took out of the equation the majority etiologies of a unilateral loss of vision and problems with the eye itself, such as amaurosis fugax, central retinal artery occlusion, retinal detachment, or even lens dislocation.

You were fortunate to have remembered to perform the visual field exam, because you discovered a homonymous hemianopia with macular sparing. This indicated to you that her pathological lesion was intracranial, possibly chiasmic, but likely in the contralateral occipital lobe (you remembered that occipital lesions typically present with a congruous hemianopsia, occasionally with macular sparing owing to the dual blood supply of the occipital poles that results in a peripheral field cut with central sparing). Given the rapidity of the onset, you suspected an ischemic event, which was confirmed with a non-contrast head CT scan.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available.

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139. Comer RM, Dawson E, Plant G, Acheson JF, Lee JP. Causes and outcomes for patients presenting with diplopia to an eye casualty department. *Eye*. 2007;21(3):413-418. (**Retrospective; 165 patients, of whom 59% had microvascular ischemic neuropathy)**
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149. Bisla K, Tanelian DL. Concentration-dependent effects of lidocaine on corneal epithelial wound healing. *Invest Ophthalmol Vis Sci*. 1992;33(11):3029-3033. (**Study using an animal model of corneal toxicity**)
150. Fraunfelder FW. Visual side effects associated with erectile dysfunction agents. *Am J Ophthalmol*. 2005;140(4):723-724. (**Retrospective case series and review of the literature; 892 cases**)
151. Rocha G, Brunette I, Le Francois M. Severe toxic keratopathy secondary to topical anesthetic abuse. *Can J Ophthalmol*. 1995 Jun;30(4):198-202. (**Report of three cases**)
152. Pharmakakis NM, Katsimpris JM, Melachrinou MP, et al. Corneal complications following abuse of topical anesthetics. *Eur J Ophthalmol*. 2002;12:373–378. (**Case series; 3 patients**)
153. Brick DC. Medication errors result in costly claims for ophthalmologists. *Surv Ophthalmol*. 1995;40(3):232-236. (**Review of closed legal claims**)
154. Khaw PT, Shah P, Elkington AR. Injury to the eye. *BMJ*. 2004;328(7430):36-38. (**Review**)
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CME Questions

The CME six-month print semester starts with the January issue and restarts with the July issue. Subscribers will receive a print answer form for the previous six months with the June and December issues. Alternatively, current subscribers can take the tests online monthly; correct answers and CME certificates are stored online permanently.

1. Patients with optic neuritis are at an increased risk for developing which of the following diseases?

a. Systemic lupus erythematosus
b. Multiple sclerosis
c. Open-angle glaucoma
d. Fibromyalgia

2. A patient presenting with a restrictive horizontal diplopia and mild proptosis bilaterally has symptoms that are most consistent with which of the following as the cause?

a. Thyroid disease
b. Oculomotor nerve palsy
c. Brainstem tumor
d. Myasthenia gravis

3. A palsy in which of the following cranial nerves is classically suggestive of elevated intracranial pressure?

a. Abducens (CN VI)
b. Oculomotor (CN III)
c. Trochlear (CN IV)
d. Facial (CN VII)

4. In a patient presenting with an acute monocular visual abnormality characterized as a shimmering, curtain-like disturbance associated with flashes of light, which of the following is the most likely etiology?

a. Amaurosis fugax
b. Retinal detachment
c. Acute angle-closure glaucoma
d. Lens dislocation

5. Ocular ultrasound can be used to most reliably diagnose which of the following etiologies of acute abnormal vision?

a. Amaurosis fugax
b. Retinal detachment
c. Acute angle-closure glaucoma
d. Optic neuritis

6. For which patient with a retinal detachment is an evaluation by an ophthalmologist most urgent?

a. The patient with six hours of macular involvement with complete visual loss
b. The patient with over two days of macular involvement with complete visual loss
c. The patient with a symptomatic retinal detachment but with preserved macular vision
d. All of the above

7. The amount of time before an ischemic human retina suffers profound, irreversible damage is approximately:

a. 15 minutes
b. 105 minutes
c. Four hours
d. Eight hours

8. An elderly male presents with acute painless unilateral vision loss lasting about 30 minutes in the setting of a week of malaise, myalgias, and headache. Which of the following is the most important diagnostic test to obtain in this patient?

a. MRI of the brain with gadolinium
b. Duplex of the carotid arteries
c. Contrast CT of the orbit
d. Erythrocyte sedimentation rate and C-reactive protein (ESR and CRP)

9. A 65-year-old female presents after a few hours of decreased visual acuity associated with severe diffuse left eye pain. She had an uncomplicated cataract replacement in the same eye three days prior. Which of the following should be earnestly considered in the evaluation of the complaint?

a. Retinal detachment
b. Acute endophthalmitis
c. Open-angle glaucoma

10. Which of the following is the standard initial treatment for inflammatory optic neuritis?

a. Aspirin
b. High-dose steroids
c. Plasmapheresis
d. Intravenous immunoglobulin

Coming In Future Issues:

Shoulder Fractures

Neuroimaging

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case-control studies
- Less robust RCTs
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. JAMA 1992;268(16):2289-2295.

Physician CME Information

Accreditation: This activity has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of Mount Sinai School of Medicine and *Emergency Medicine Practice.* The Mount Sinai School of Medicine is accredited by the ACCME to provide continuing medical education for physicians.

Credit Designation: The Mount Sinai School of Medicine designates this educational activity for a maximum of 48 *AMA PRA Category 1 Credit(s)™* per year. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Credit may be obtained by reading each issue and completing the printed post-tests administered in December and June or online single-issue post-tests administered at EBMedicine.net.

Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

Date of Original Release: This issue of *Emergency Medicine Practice* was published September 1, 2007. **This activity is eligible for CME credit through September 1, 2010.** The latest review of this material was August 2, 2007.

Discussion of Investigational Information: As part of the newsletter, faculty may be presenting investigational information about pharmaceutical products that is outside Food and Drug Administration approved labeling. Information presented as part of this activity is intended solely as continuing medical education and is not intended to promote off-label use of any pharmaceutical product. Disclosure of Off-Label Usage: This issue of *Emergency Medicine Practice* discusses no off-label use of any pharmaceutical product.

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In compliance with all ACCME Essentials, Standards, and Guidelines, all faculty for this CME activity were asked to complete a full disclosure statement. The information received is as follows: Dr. Guluma, Dr. Sharma, and Dr. Jagoda report no significant financial interest or other relationship with the manufacturer(s) of any commercial product(s) discussed in this educational presentation.

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Emergency Medicine Practice (ISSN Print: 1524-1971, ISSN Online: 1559-3908) is published monthly (12 times per year) by EB Practice, LLC, 5550 Triangle Parkway, Suite 150, Norcross, GA 30092. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. This publication is intended as a general guide and is intended to supplement, rather than substitute, professional judgment. It covers a highly technical and complex subject and should not be used for making specific medical decisions. The materials contained herein are not intended to establish policy, procedure, or standard of care. *Emergency Medicine Practice* is a trademark of EB Practice, LLC.