OPHTHALMOLOGY

Diagnosing Acute Blindness

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Acute vision loss is an emergency and should be investigated and addressed promptly. While the likelihood that vision will return varies depending on the cause of the vision loss, in many cases, a delay in recognition and treatment worsens the prognosis.[1-4] This is particularly true of diseases involving the retina and optic nerve.[1-5]

When an acutely blind patient is presented, a thorough history is a necessary starting point. Owners should be queried on the onset and duration of the vision loss; whether they have noted a concurrent or antecedent change in the appearance of the eye(s); and the patient's medical history, including other signs of disease and medications that have been administered.

Following history taking, the clinician should perform vision assessments to confirm the visual deficit and a complete ophthalmic examination with an aim toward lesion localization (**BOX 1**).[6,7] Is the patient blind because:

- Something is obscuring the visual axis, such as corneal edema, cataract, or intraocular hemorrhage?
- The retina is not responding to light stimulation?
- The optic nerve and tracts cannot transmit the electrical responses of the retina?
- The central nervous system is unable to process the incoming visual information?

If vision is present but the patient's visual behavior is abnormal, consider neurologic or cognitive causes: Perform a complete neurologic examination and survey blood analysis, and query the owner about other behaviors (e.g., seizures, circling, stargazing, inappropriate vocalization).

While many of the ophthalmic conditions that result in acute blindness may be diagnosed strictly based on the appearance of the eye, others require additional testing for confirmation. A fundic examination should be attempted in all cases of acute vision loss. Inability to visualize the fundus when an anterior obscuring lesion is present is confirmation—if you cannot see "in," likely the patient cannot see "out." Also, posterior segment changes may be concurrent with optic nerve or central disease and may alter or inform the list of diagnostic differentials, etiologies, or prognoses. If the fundus cannot be visualized due to anterior segment changes or intraocular hemorrhage, ocular ultrasonography may facilitate understanding of the entire clinical picture (e.g., hyphema secondary to retinal detachment).

Pupil size and the direct and consensual pupillary light reflexes (PLRs) are critical pieces of the neuro-ophthalmic examination for lesion localization (**FIGURE 1**). Intact PLRs require integrity of the retinal neural cells, optic nerves, optic chiasm, optic tracts, midbrain, parasympathetic fibers accompanying the oculomotor nerve, ciliary ganglion, and iris sphincter muscle, but not the cerebral cortex. Therefore, they are a subcortical reflex and not a test of vision. The consensual PLR is the response that occurs in the contralateral, nonstimulated eye and in dogs and cats should be nearly equal to that of the direct PLR. These should be assessed with a bright light in a dimly lit room, and the rapidity and completeness of the response (extent of miosis) should be evaluated along with the ability of the eye to maintain miosis during constant light stimulation. If the contralateral pupil initially constricts (positive consensual PLR) but then dilates when directly stimulated (negative direct PLR), this suggests an afferent lesion (retina or optic nerve) of that eye. This is sometimes referred to as a swinging flashlight test.

BOX 1
Differential Diagnoses for Ocular Lesion Locations Manifesting With Acute-Onset Blindness

Cornea
- Corneal ulceration or perforation*
- Corneal edema
  - Endothelial decompensation*
  - Severe uveitis*
  - Uncontrolled glaucoma

Lens
- Rapid cataract development
  - Diabetes mellitus*
- Lens luxation (anterior); may be accompanied by elevated intraocular pressure*
  - Zonular dysplasia (genetic)
  - Glaucoma
  - Chronic uveitis

Anterior uvea
- Uveitis
  - Lens-induced
  - Systemic infectious/inflammatory disease
  - Generalized or metastatic neoplasia (e.g., lymphoma)
  - Trauma
- Uveal neoplasia
- Intraocular hemorrhage*
  - Any of the above
  - Systemic hypertension
  - Bleeding diathesis

Retina and posterior uvea
- Retinal detachment
  - Systemic hypertension
  - Systemic infectious/inflammatory disease
  - Local or systemic neoplasia
  - Secondary to congenital or developmental disorders (e.g., collie eye anomaly, vitreoretinal dysplasia)
  - Genetic predisposition (e.g., Shih Tzus, Boston terriers, bichon frises)
  - Trauma
- Retinal degeneration
  - Toxin exposure (e.g., enrofloxacin, ivermectin)
  - Sudden acquired retinal degeneration syndrome
- Chorioretinitis
  - Systemic infectious/inflammatory disease
  - Local or systemic neoplasia

Optic nerve
- Optic neuritis
- Glaucoma
- Optic nerve neoplasia
- Optic nerve trauma (e.g., proptosis)
- Compressive pituitary or hypothalamic mass (chiasmal)

Central nervous system
- Meningoencephalitis of unknown origin
- Infectious encephalitis
- Neoplasia of the visual cortex, optic chiasm
- Vascular event

**Lesion may cause acute blindness by obscuring the visual axis.*

In instances of bilateral blindness due to retinal or optic nerve disease or chiasmal lesions, both direct and consensual PLRs will be lacking (**FIGURE 2**). The approach to lesion localization should thereafter proceed as it would for a unilateral vision deficit.

Once the responsible structure(s) have been identified, the clinician can generate a list of potential etiologies (**BOX 1**), next-step diagnostics, and prognoses.[1-7]

FIGURE 1. Anisocoria in a dog secondary to unilateral iris atrophy. The right eye was visual although it lacked a direct pupillary light reflex. A consensual response in the left eye was normal.

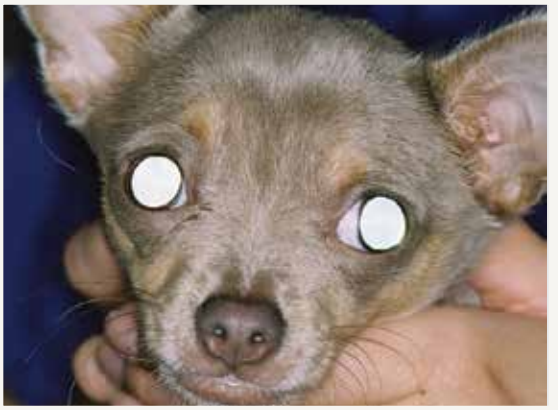

FIGURE 2. Acutely blind dog with mydriatic pupils. Direct and consensual pupillary light reflexes in both eyes were absent, localizing the lesion(s) to both retinas, both optic nerves, or the chiasm.

Confirm vision deficit

Multiple tests may be needed to confirm vision loss. Other tests include the cotton ball/laser pointer test, visual placing response, and maze/obstacle course. Each eye should be evaluated individually when vision testing is performed. Cover each in turn when assessing the menace response, the cotton ball test, and visual placing.

Test menace response[a]

- Present → Vision present
- Absent → Test palpebral reflex: Can the patient blink?
  - Yes → Vision deficit confirmed
  - No

- Evaluate for neurologic deficits (e.g., facial paresis/paralysis); if present, perform complete neurologic and otoscopic examinations.
- If concern persists for visual deficit, perform additional vision tests.

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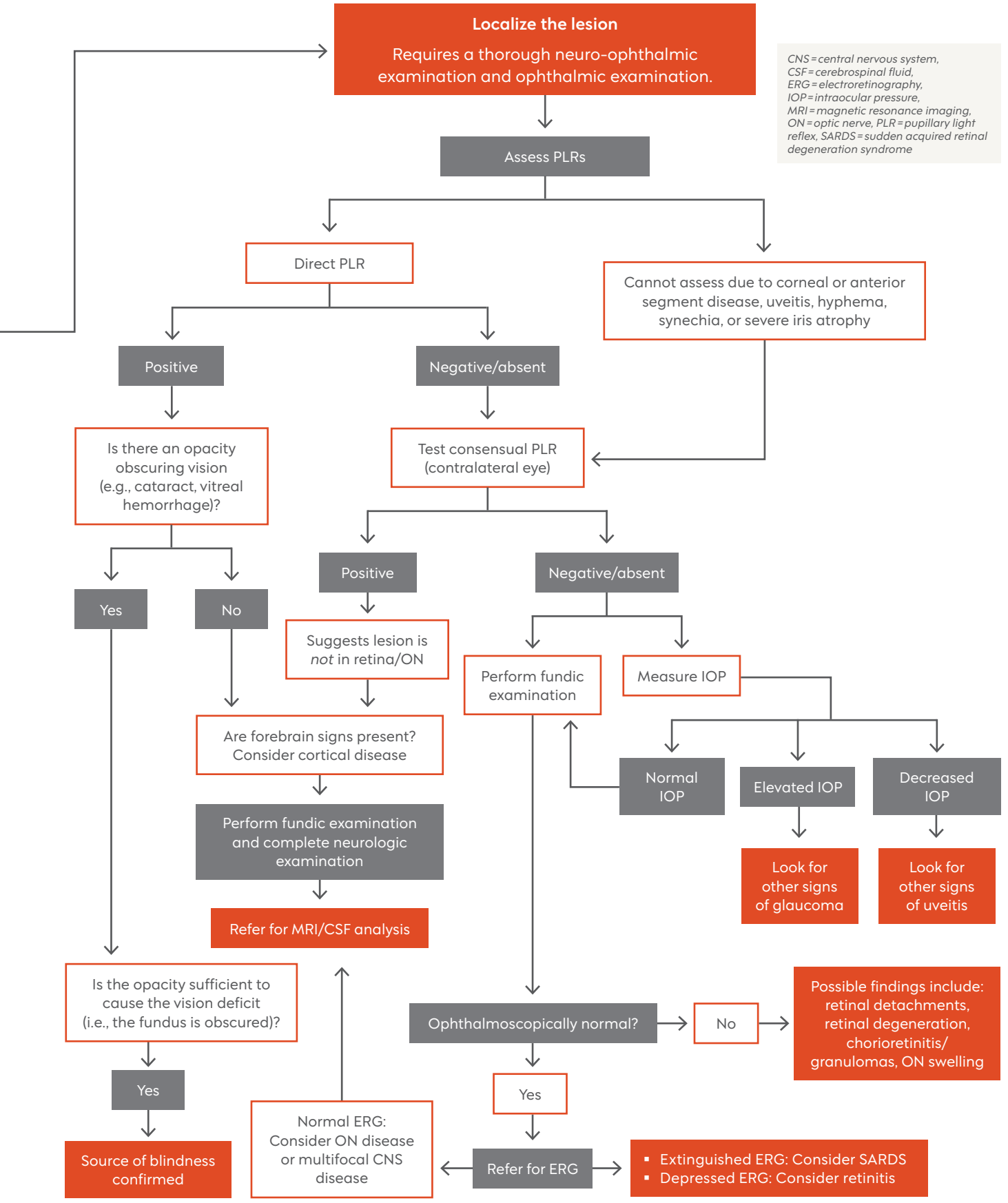
[a]Menace is a learned response and while it is a test of vision, a negative response does not always indicate that an animal is blind.


Localize the lesion
Requires a thorough neuro-ophthalmic examination and ophthalmic examination.
CNS = central nervous system, CSF = cerebrospinal fluid, ERG = electroretinography, IOP = intraocular pressure, MRI = magnetic resonance imaging, ON = optic nerve, PLR = pupillary light reflex, SARDS = sudden acquired retinal degeneration syndrome
Assess PLRs
Direct PLR
Cannot assess due to corneal or anterior segment disease, uveitis, hyphema, synechia, or severe iris atrophy
Positive
Negative/absent
Is there an opacity obscuring vision (e.g., cataract, vitreal hemorrhage)?
Test consensual PLR (contralateral eye)
Yes
No
Positive
Negative/absent
Suggests lesion is not in retina/ON
Perform fundic examination
Measure IOP
Are forebrain signs present? Consider cortical disease
Normal IOP
Elevated IOP
Decreased IOP
Perform fundic examination and complete neurologic examination
Look for other signs of glaucoma
Look for other signs of uveitis
Refer for MRI/CSF analysis
Is the opacity sufficient to cause the vision deficit (i.e., the fundus is obscured)?
Ophthalmoscopically normal?
No
Possible findings include: retinal detachments, retinal degeneration, chorioretinitis/ granulomas, ON swelling
Yes
Yes
Normal ERG: Consider ON disease or multifocal CNS disease
Refer for ERG
Extinguished ERG: Consider SARDS
Depressed ERG: Consider retinitis
Source of blindness confirmed

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Dr. Plummer is an honors graduate of the University of Florida College of Veterinary Medicine and a diplomate of the American College of Veterinary Ophthalmologists. After veterinary school, she completed a rotating internship at Michigan State University followed by a residency in comparative ophthalmology at the University of Florida (UF). After completing specialty training, Dr. Plummer joined the faculty at the UF College of Veterinary Medicine, where she is currently a tenured professor. Her research interests include corneal wound healing, immune-mediated uveitis, and glaucoma. She has lectured extensively both in the United States and abroad on many topics associated with clinical veterinary ophthalmology and animal models of ophthalmic disease, especially glaucoma.