

OPHTHALMOLOGY

Diagnosing Acute Blindness

Caryn E. Plummer, DVM, DACVO

University of Florida College of Veterinary Medicine, Gainesville, Florida

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.

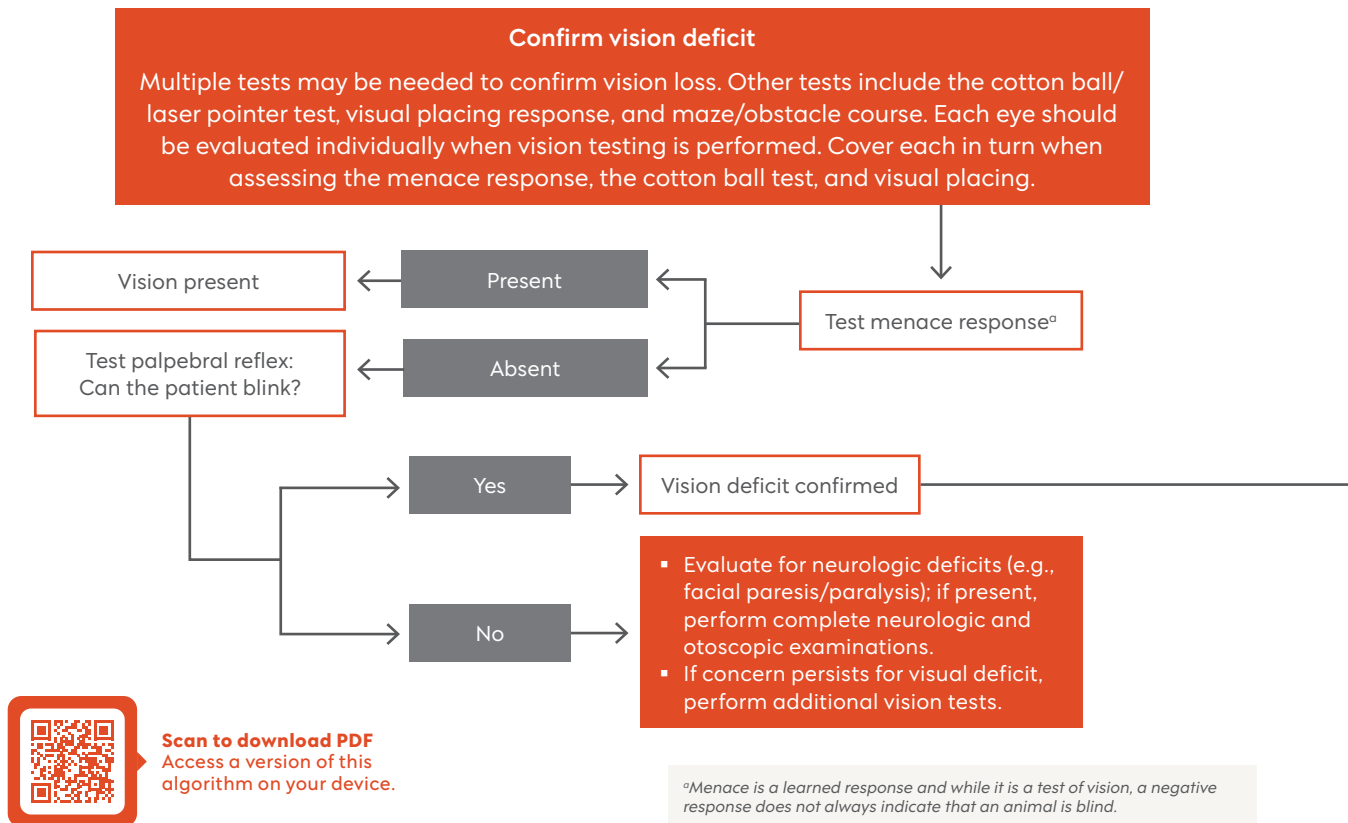
Once the responsible structure(s) have been identified, the clinician can generate a list of potential etiologies (**BOX 1**), next-step diagnostics, and prognoses.¹⁻⁷



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.



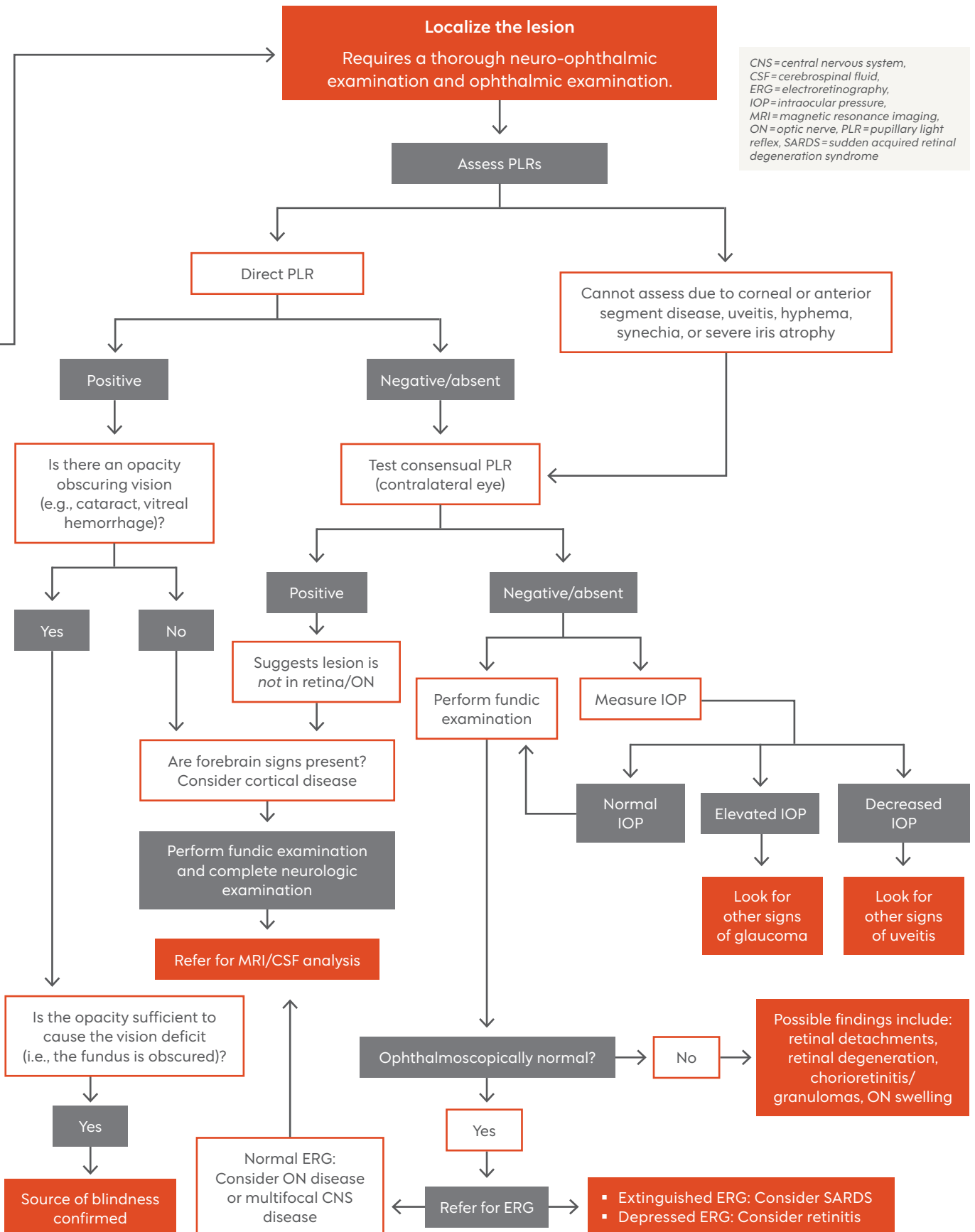
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Zenalpha®

(medetomidine and vatinoxan hydrochlorides injection)
Sedative, Analgesic
For Use in Dogs Only

CAUTION: Federal (USA) law restricts this drug to use by or on the order of a licensed veterinarian.

BRIEF SUMMARY: (for full prescribing information, see package insert)

DESCRIPTION: Zenalpha is a combination of medetomidine and vatinoxan hydrochlorides. Each mL of Zenalpha contains 0.5 mg medetomidine hydrochloride, 10 mg vatinoxan hydrochloride, 32.5 mg mannitol (USP), 4.16 mg citric acid monohydrate (USP), 1.8 mg methylparaben (NF), 0.2 mg propylparaben (NF).

INDICATION: Zenalpha is indicated for use as a sedative and analgesic in dogs to facilitate clinical examination, clinical procedures and minor surgical procedures.

CONTRAINDICATIONS: Do not use Zenalpha in dogs with cardiac disease, respiratory disorders, shock, severe debilitation, that have hypoglycemia or are at risk of developing hypoglycemia, or are stressed due to extreme heat, cold or fatigue.

Zenalpha is contraindicated in dogs with a known sensitivity to medetomidine or vatinoxan.

WARNINGS:

Human User Safety Warnings

Not for use in humans. Keep this and all medications out of reach of children and pets.

Avoid skin, eye or mucosal contact. Use caution while handling and using filled syringes. Absorption of the active ingredients is possible following exposure via the skin, eye or mucosa. In case of accidental eye exposure, flush eyes with water for 15 minutes, remove contact lenses then continue to flush. In case of accidental skin exposure, wash with soap and water and remove contaminated clothing. If symptoms occur, seek the advice of a physician.

In case of accidental oral intake or self-injection, seek medical advice immediately and show the package insert to the physician. DO NOT DRIVE as sedation, loss of consciousness, and changes in blood pressure may occur.

Persons with cardiovascular disease (for example, hypertension or ischemic heart disease) should take special precautions to avoid any exposure to this product.

Pregnant women should exercise special caution to avoid exposure. Uterine contractions and decreased fetal blood pressure may occur after accidental systemic exposure.

Persons with known hypersensitivity to any of the ingredients should avoid contact with Zenalpha.

Caution should be exercised when handling sedated animals. Handling or any other sudden stimuli, including noise, may cause a defense reaction in an animal that appears to be heavily sedated.

Note to physician: Zenalpha contains medetomidine, an alpha₂-adrenoceptor agonist, in combination with vatinoxan, a peripherally selective alpha₁-adrenoceptor antagonist. Symptoms after absorption or accidental self-injection may include dose-dependent sedation, respiratory depression, bradycardia, tachycardia, and hypotension.

Animal Safety Warnings

Zenalpha should not be administered in the presence of pre-existing hypotension, hypoxia or bradycardia. Due to the pronounced cardiovascular effects of alpha₂-adrenoceptor agonists, only clinically healthy dogs (American Society of Anesthesiologists [ASA] classes I and II) should be administered Zenalpha. Dogs should be monitored frequently for cardiovascular function and body temperature during sedation.

Zenalpha is not intended for use in cats. The use of Zenalpha in cats has been associated with hypotension.

PRECAUTIONS: Dogs should be monitored frequently during sedation for changes in heart rate, blood pressure, respiratory rate and body temperature. Tachycardia may occur in some dogs after recovery from sedation.

In the event of hypoxia or apnea, supplemental oxygen should be administered.

Following administration of Zenalpha, a decrease in body temperature may occur and an external heat source may be needed to maintain body temperature. Hypothermia may persist longer than sedation and analgesia.

The analgesic effect of Zenalpha will not last longer than the sedative effects. Additional analgesic(s) should be administered as needed.

Nervous, excited or agitated dogs with high levels of endogenous catecholamines may exhibit a reduced pharmacological response to Zenalpha (ineffectiveness). The onset of sedative/analgesic effects could be slowed, or the depth and duration of effects could be diminished or nonexistent. Therefore, allow the dog to rest quietly for 10 to 15 minutes after injection.

With the alpha₂-adrenoceptor agonist drug class, including Zenalpha, the potential for isolated cases of hypersensitivity, including paradoxical response (excitation) exists.

Repeat dosing with Zenalpha has not been evaluated.

Zenalpha has only been evaluated in fasted dogs; therefore, the effects on fed dogs (for example occurrence of vomiting) have not been characterized.

The concurrent use of anticholinergic medications and Zenalpha has not been evaluated.

Zenalpha may decrease serum glucose in healthy dogs and this effect may persist longer than sedation.

The safe use of Zenalpha has not been evaluated in dogs with hepatic or renal impairment, dogs younger than 4.5 months old, or dogs that are pregnant, lactating, or intended for breeding.

ADVERSE REACTIONS: The most common adverse reactions observed in the field study were decreased body temperature (not requiring external heat support), reduced respiratory rate, diarrhea, muscle tremor, signs of colitis, hypothermia (requiring external heat support).

To report suspected adverse events, for technical assistance or to obtain a copy of the SDS, contact Dechra Veterinary Products at (866) 933-2472. For additional information about adverse drug experience reporting for animal drugs, contact FDA at 1-888-FDA-VETS or online at <http://www.fda.gov/reportanimal>.

MANUFACTURED FOR:

Dechra Veterinary Products
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Overland Park, KS 66211 USA

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Author



Caryn E. Plummer

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.