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Pupilloplasty in a great horned owl with pupillary occlusion and cataracts

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A juvenile great horned owl (Bubo virginianus) of undetermined gender was found by a roadside in a California desert and taken to the University of California-Davis Raptor Center for rehabilitation. No other history was available. Although the bird appeared to be able to see and had no visible evidence of having been traumatized, the Raptor Center staff noticed an abnormal left eye and requested an ophthalmic evaluation.

Examination revealed anisocoria (Fig 1). The right pupil responded normally to light. There was no apparent pupil in the left eye (Fig 2), although when the eye was illuminated the iris sphincter muscle contracted so that the axial, pigmented region of iris formed a small, broad-based rostral projection of iridial tissue (Fig 3). A normal menace reflex was elicited in the right eye, but not in the left. Palpebral reflexes were normal in both eyes. The eyelids, conjunctiva, nictitating membrane, cornea, anterior chamber, and iridocorneal angle of each eye were examined with diffuse light and biomicroscopy and found to be normal. The anterior part of the lens capsule of the right eye had a small, local, axially located cataract. The right fundus was determined to be normal by indirect ophthalmoscopy. The left lens and fundus were not visible because of the pupillary occlusion.

Ultrasonography, using a real-time mechanical sector scanner and a 10MHz transducer, was performed on the left eye to compare the nonvisible posterior segment of that eye with a similar study of the right eye. Abnormalities were not found.

The only apparent abnormality of the left eye was a continuous sheet of iridial tissue covering the lens. Because the left eye was potentially a visual eye, pupilloplasty, the surgical creation of a pupil, was recommended.

The bird was anesthetized and placed in dorsal recumbency, and the left eye was draped in a standard manner for intraocular surgery. An eyelid speculum was used to retract the eyelids. The cornea was kept moist throughout the procedure by topical application of balanced salt solution. Using a No. 65 Beaver blade, a 3-mm incision was made into the anterior chamber at the lateral region of the limbus. A Cilco handpiece, connected to a Peyman Vitrector, was introduced through the incision into the anterior chamber until the aspiration and cutting port were positioned over the central, pigmented region of the iridial sheet. Anterior chamber depth was maintained by continuous irrigation through the handpiece with Ringer's solution containing 1:10,000 epinephrine and 1 U/ml of heparin. The axial portion of the iris was aspirated into the cutting port of the handpiece, which then was tilted to gently lift the iris from the anterior surface of the lens. The central portion of iris was resected by guillotine cutting, creating a pupil that dilated immediately. The handpiece was removed and the limbal incision was closed, using 3-0 Barraquer eyelid speculum, Storz Instrument Co, St Louis, Mo.

BSS, Alcon Laboratories, Fort Worth, Tex.
Beaver Inc, Waltham, Mass.
Cilco, Pomona, Calif.
Cilco I/A Vitrectomy, Peyman Unit V9000, Pomona, Calif.
#Tavenol Laboratories, Deerfield, Ill.
interrupted sutures of 7-0 polyglactin 910. A local cataract of the anterior, axial region of the lens was apparent once a pupil had been created. There was mild swelling and superficial hemorrhage of the lateral region of iris attributable to contact with the handpiece. The bird recovered from anesthesia. To minimize stress to the owl, postoperative medications were not used.

The bird was observed daily from a distance for signs of infection, uveitis, or keratitis. Five days after surgery, the bird was captured and restrained for examination. The right eye was unchanged. The left eye had a normal pupillary light response, and a pupil that was roughly circular. The cataract was more easily examined than at the time of surgery. It was small, circular, and located axially in the anterior region of the lens capsule and cortex. It was connected to a nuclear opacity by a thin, opaque

band (Fig 4). There were several small accumulations of black pigment on the anterior capsule of the lens immediately overlying the cataract. Evidence of anterior uveitis was not detected. The corneal incision had vascularized and appeared to be healing as expected. Indirect ophthalmoscopy of the fundus did not reveal abnormalities.

After an additional 2 weeks of daily observation at a distance, the bird was captured and restrained for close examination. There had been progression of the corneal blood vessels into and across the limbal incision and resorption of the sutures. The cataract and pupil remained unchanged. Five weeks later, the only visible change was scarring of the corneal incision site. A temporary tarsorrhaphy of the right eye was done, using local (lidocaine) and topical (proparacaine) anesthetic agents to evaluate vision of the left eye. With the tarsorrhaphy in place, the bird was able to fly, land on a perch, and track objects and people normally. The tarsorrhaphy was removed at the end of the day. Three months after surgery, the bird was unchanged and had been successful in hunting within a large enclosure. It was released back into a free-living state.

The musculature of the iris of the great horned owl has been demonstrated to be complex, consisting of smooth muscle, striated muscle, and myoepithelium. The circumferentially oriented striated muscle is considered to be primarily responsible for the rapidity of the pupillary constriction in response to light. Because the iris of the bird of this report moved before and after surgery when illuminated, it must have had a functional iris sphincter, ocular motor nerve, and optic nerve, and a retina that was capable of reacting to light.

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Vicryl, Ethicon, Johnson & Johnson Co, Somerville, NJ.

1Lidocaine HCl, Abbott Laboratories, North Chicago, Ill.
2Ophtalmic, Allergan Pharmaceuticals, Irvine, Calif.
The pupillary abnormality and the bilateral cataracts may have been caused by a developmental abnormality or a traumatic event, or both. A penetrating injury into the anterior axial region of the lens might cause a cataract, miosis, and enough inflammation to result in pupillary occlusion and scotomas; however, this seemed an unlikely cause of ocular anomaly of this bird. First, there was no evidence of a corneal scar, damage to the lens via a penetrating injury through the sclera was unlikely because of the axial location and anterior-posterior orientation of the cataract. The possibility of a penetrating injury through the cornea and into the lens, sustained early in life with subsequent healing and growth of the cornea, was a remote possibility that could not be discounted. This type of injury might result in a corneal scar that was so small that it was not detected. Second, the iris color was similar to that in the right eye and was considered normal. Because a severe degree of anterior uveitis probably would have been required to cause pupillary scoliosis or occlusion, a change in iris color resulting from iris degeneration or rearrangement as a result of the inflammation might also have been expected.

There are few reports of developmental ocular anomalies of raptors. Developmental lesions may be underestimated, however, because birds with severe ocular malformation would be expected to have compromised survival skills, and most would probably die without being detected by human beings. Although various developmental lesions in raptors have been reported, reports describing a lesion similar to that of this bird could not be found. The appearance of the left iris of this bird could have been caused by extreme microcoria, so that the pupil was not detected clinically. In human beings, however, microcoria is associated with an inability of the pupil to dilate because of an absence or underdevelopment of the dilator muscle, and this bird's pupil dilated once a pupillary aperture was created.

Another explanation for the appearance of the left iris could be an even rarer condition, acoreia, the absence of a pupil. Pupillary scoliosis could develop by lack of atrophy of the mesodermal tissue that forms the pupillary membrane (the lamina irido-pupillaris) or by hyperplasia of the anterior layer of the iris stroma in the area that should have developed into the pupil. In extreme cases, the accessory iridal tissue may extend across and cover the entire pupillary aperture and insert onto the anterior capsule of the lens. Most commonly, however, persistent pupillary membranes, which have been described extensively in human beings, dogs, and other species, develop as strands of mesodermal tissue rather than the dense sheet of tissue that occluded the pupil of the bird of this report. Although persistent pupillary membranes have not been reported in birds, it seems reasonable to assume they would be similar to those seen in other species.

A possible explanation for the type of cataract seen in the left eye would be abnormal separation of the surface ectoderm from the lens vesicle. If the lens stalk persisted for longer than normal, or detached in an abnormal manner, it could potentially give rise to a nuclear cataract with abnormal lens epithelium causing the adjacent lens cortex to become cataractous. If there were such a cleavage defect between the surface ectoderm and the lens vesicle, there might also have been incomplete atrophy of the sheet of mesodermal tissue that invades between the cornea and lens to form the pupillary membrane. However, persistence of the lens stalk with resultant interference of neural crest migration also results in corneal edema and vascularization caused by defects in Descemet membrane and endothelium; this eye had no evidence of corneal edema or vascularization.

It was surprising that, although there was no pupil visible, there was no evidence of iris bombé. Iris bombé may not have developed because the aqueous humor may have been able to percolate through microscopic openings in the central sheet of iridal tissue or directly through the iris stroma. Eyes with pupillary scoliosis may not have iris bombé if aqueous production has been depressed sufficiently by severe iridocyclitis; the owl of this report, however, had no evidence of iridocyclitis.

Because the critical period of susceptibility to visual deprivation in precocious birds peaks within the first day of hatching, we were uncertain whether this owl had developed functional visual cortical neurons. Because the bird was able to fly and land normally when the right eye was closed, it was presumed that the retina of the left eye and the visual cortex were functional to some degree. Whether the bird had binocular vision could not be determined. Owls that have been deprived of binocular vision early in life have been demonstrated electrophysiologically to lack binocularly-activated neurons within the visual Wulst, although functional input to the Wulst from the visually deprived eye develops. Some monocularly-deprived owls have also had marked strabismus, which was not apparent in this owl. The importance of binocular vision to the survival of birds is uncertain, however, because monocular birds are known to be able to survive in a free-living state. Because the owl of this report was successful in locating and capturing live prey within a large enclosure, the question of binocular vision or cortical changes seemed unimportant.


In the article, “Treatment by digital amputation of subungual squamous cell carcinoma in dogs: 21 cases (1987–1988)” (JAVMA, Sept 1, 1992, pp 759–761), figures 1 and 2 were transposed. The JAVMA regrets the error.