is marked by the final establishment of tawny owls’ breeding territories. The fact that the injury was caused by the bird’s claw may be typical for this species because this is their strongest weapon. This is in contrast to most other reports on bird attacks, in which injuries are inflicted by beak hits.

**Report of a Case.** On a late December night, a hunter on a raised hide was attacked by a tawny owl (Strix aluco) immediately after he had imitated the characteristic birdcall of this species. During the summer, he had mimicked this birdcall many times. Never before had he experienced any aggressive behavior by the bird, but instead he was usually answered by the owl’s song. The attack resulted in several superficial skin wounds on his right cheek and nose and radial limbal-scleral perforation with uveal, vitreal, and retinal incarceration. The lesions’ distribution revealed the typical pattern of the bird’s claw (Figure). Because of intraocular hemorrhage and massive inflammatory infiltration, which is typical for perforating injuries by organic material, vision was reduced to light perception. After resection of the incarcerated tissue, the corneal and scleral wounds were sutured, and cryocoagulation was applied to the retinal defects. Ofloxacin eyedrops (four times daily) and dexamethasone eyedrops (one week four times daily, another week twice daily) were given topically. Oral ciprofloxacin (750 mg twice daily for one week) and oral prednisone (100 mg once a day for 4 days, then down-titrated for 10 days until discontinued) were given as systemic treatment. Eight weeks later, vitreous traction caused a retinal break, which was successfully treated by laser coagulation. During a 5-month follow-up, the vitreous cleared progressively and visual acuity increased to 20/25.

**Comment.** In conclusion, this case demonstrates that, depending on species and season, special caution is needed when attracting birds by imitating their call. Furthermore, this example confirms that successful treatment of perforating eye injuries after bird attacks is possible if immediate surgical care as well as consequential treatment of the usually subsequent severe septic inflammation is provided, in our case with high-dose systemic and local antibiotics and steroids.

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In this report, 2 unusual cases of CHR extending 360° in the midperipheral fundus with no other associated vascular anomalies are described. To our knowledge, no other cases of such characteristics have been reported in the literature.

**Report of Cases.**

**Patient 1.** A 21-year-old white man was referred to the Medical Retina Service, Aberdeen Royal Infirmary, Aberdeen, Scotland, in November 2000 with the diagnosis of diffuse retinal hemorrhages in the right eye. He was asymptomatic. His ocular and medical history was unremarkable. He was born at full term and did not receive supplementary oxygen. His mother had experienced no problems during pregnancy. At birth he was noted to have a strawberry birthmark on the back of his neck, which resolved spontaneously. His family medical history included glaucoma in his maternal grandfather and a peripheral retinal tear in his maternal grandmother. There was no history of fits in the family.

On examination, best-corrected visual acuity was measured at 20/20 OU. Intraocular pressures and results of the slitlamp examination were normal in both eyes. Results of the fundus examination of the left eye were normal. In the right eye, dilated retinal veins with clusters of saccular dilations involving 360° of the midperipheral retina were seen (**Figure 1A**). A few retinal hemorrhages adjacent to these vascular anomalies were present, but no hard exudates were seen. The optic nerve and macula disclosed no abnormalities. No cutaneous angiomatous lesions were present. Results of fundus and cutaneous examinations of his mother, father, and sister showed no abnormalities.

Fluorescein angiography (FFA) demonstrated delayed perfusion of the lesion with a typical blood-fluorescein interface occurring within the saccular dilations (**Figure 1B and C**). Anterior to this venous malformation, the retina appeared avascular. There was no leakage of fluorescein present in the late frames of the angiogram.

At the last follow-up, 28 months after our initial examination, his visual acuity remains 20/20 OU, and the lesion remains unchanged.

**Patient 2.** A 25-year-old white woman was found to have a vascular abnormality in her right eye in February 2000 and was referred to the Medical Retina Service, Aberdeen Royal Infirmary, for evaluation. She was asymptomatic. Her ocular and medical history was unremarkable. She had been born at full term and had received no supplementary oxygen therapy. Her mother had experienced no problems during pregnancy.

On examination, her best-corrected visual acuity was 20/20 OU. Anterior segments and intraocular pressures were normal in both eyes. Results of the fundus examination of the left eye were normal. In the right eye, dilated veins involved 360° of the midperipheral retina, with clusters of saccular dilations (**Figure 2A**). There were no hemorrhages or hard exudates associated with this lesion. Results of a general examination disclosed no ab-

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**Figure 1.** A, Composite color fundus photograph of the right eye of a 21-year-old white man with cavernous hemangioma of the retina (CHR) (patient 1). Involvement of 360° of the midperipheral fundus by the CHR is observed. B, Composite fluorescein angiogram of the same patient. C, Magnified view of the nasal retina shows clusters of saccular venous dilations with characteristic erythrocyte-plasma interfaces (white arrows) (original magnification unknown).
normal neurological signs and no cutaneous angiomas. Results of fundus and cutaneous examinations of her father, mother, and brother showed no abnormalities.

Fluorescein angiography showed delayed perfusion of the involved areas. Slow filling of the saccular aneurysms and typical blood-fluorescein interfaces within these saccular dilations were observed. Anterior to the venous malformation, the retina appeared avascular. No fluorescein leakage was seen in the late frames of the angiogram.

At the last follow-up, 3 years after our initial examination, her visual acuity remains at 20/20 OU, and the CHR remains unchanged.

Comment. Cavernous hemangioma of the retina is a rare, benign, often unilateral vascular tumor, usually recognized in young individuals. Female subjects are more commonly affected. Although most patients with CHR are asymptomatic, neurological symptoms and signs or decreased vision can occur. There is an association between CHR and cavernous hemangiomas involving the central nervous system. In some of these cases, an autosomal dominant inheritance has been noted.

Typically, ophthalmoscopy reveals clusters of saccular aneurysms filled with dark venous blood. Superficial retinal hemorrhages are occasionally seen, but hard exudation has not been observed. In some cases, a gray-white preretinal membrane can partly obscure the surface of the tumor. Although the retinal vasculature elsewhere is usually normal, other associated vascular anomalies can occur. In early frames of the FFA, the vascular channels within the CHR remain hypofluorescent. As the angiogram progresses, these vascular channels fill slowly with the dye. In late frames, a blood-fluorescein interface is characteristically observed in the saccular dilations within the tumor. This phenomenon seems to be related to sedimentation of erythrocytes in the inferior aspect of the venous aneurysm, which appears hypofluorescent. The presence of plasma in the superior aspect of this vascular space, which stains with fluorescein, appears hyperfluorescent. There is no extravascular leakage of dye in CHR.

The patients described herein had unusual CHR. The vascular tumors in these cases extended circumferentially throughout the 360° of the midperipheral retina. The retina anterior to the cavernous hemangioma seemed to be avascular. To our knowledge, only 1 similar case, published by Messmer and coworkers in 1983, has been reported in the literature. In that case, however, a large peripheral shunt vessel extended temporally from the superonasal to the inferonasal quadrants. Unlike the cases reported herein, there was a slight reduction in visual acuity in the affected eye (20/50) and some pigment epithelial mottling at the fovea.

The visual prognosis of patients with extensive CHR remains uncertain. Most patients with localized CHR retain good vision, and visual problems related to contraction of the preretinal membrane overlying the tumor or vitreous hemor-
rhage develop in only a few. However, the risk for development of these complications in extensive lesions such as those reported herein may be higher. Similarly, given that the most anterior retina seemed to be avascular, in these cases there may be also an increased risk for neovascularization of the disc, retina, and/or iris.

The differential diagnosis of CHR should include retinal capillary hemangioma; racemose hemangioma; retinal telangiectasis, including Coats disease; and familial exudative vitreoretinopathy.

Unlike CHR, retinal capillary hemangioma appears characteristically as a discrete red or pink nodular mass with a feeder artery and a draining vein. Retinal capillary hemangioma represents a large arteriovenous shunt and, thus, it fills early on FFA. This differs from the saccular venous dilations of CHR, which fill slowly and progressively with fluorescein. Unlike CHR, hard exudates and retinal detachment are frequently observed associated with retinal capillary hemangioma.

Racemose hemangioma is characterized by tortuous dilated retinal vessels, which represent direct arteriovenous communications. Like CHR, racemose hemangioma does not produce exudation or retinal detachment. However, the saccular venous dilations and the characteristic blood-fluorescein interfaces observed on FFA in CHR are not a feature of racemose hemangioma.

Retinal telangiectasis, including Coats disease, is a nonfamilial vascular abnormality characterized by irregular dilation of the retinal capillaries or arteries. Unlike CHR, retinal telangiectasis is associated with intraretinal and subretinal exudation and an abnormal increased permeability on FFA. Exudative retinal detachment and cystoid macular edema may develop in patients with retinal telangiectasis.

Familial exudative vitreoretinopathy is inherited as an autosomal dominant or X-linked recessive trait. Unlike CHR, familial exudative vitreoretinopathy is characterized by the presence of vitreous bands, neovascularization of the disc and peripheral retina, retinal and subretinal exudation, fibrous proliferation with traction and dragging of the major retinal vessels and macula temporally, and retinal detachment.

Given that in a small proportion of patients with CHR the condition is transmitted in an autosomal dominant fashion, it is important to examine other members of the family to rule out the presence of similar retinal lesions, as there may be an increased chance of cavernous hemangiomas being present in the central nervous system in familial cases. Magnetic resonance imaging of the brain may be recommended in these latter cases.

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