Inadvertent Instillation of Nonophthalmic Antiseptic Drops Due to Packaging Similarity

Previously we have reported on the misuse of nonophthalmic and ophthalmic drops due to packaging similarity. Other reports document the inadvertent instillation of ophthalmic drops into the eye because of bottle size or packaging similarity, including Hemoccult developer, cyanoacrylate, and sodium hydroxide.

Report of a Case. An 87-year-old woman underwent penetrating keratoplasty, anterior vitrectomy, and lens exchange for pseudophakic corneal edema. Six months postoperatively her medications included prednisolone acetate (Pred Forte, Allexergan Inc, Irvine, Calif), four times a day, and 5% sodium chloride drops (Muro 128, Bausch & Lomb Pharmaceutical Inc, Tampa, Fla), four times a day. A nurse’s aide assisting with eye drops mistakenly placed an antiseptic solution, (<1% benzalkonium chloride, Mycocide NS, Woodward Laboratories Inc, Los Alamitos, Calif) into the postoperative eye resulting in immediate eye pain, redness, and tearing. This antiseptic is typically used as a topical treatment for nailbed fungus. The eye was flushed repeatedly with tap water, and the patient was seen 2 hours later in the eye clinic with a visual acuity of 20/100 OD. Diffuse injection and mucoid drainage were seen. Slitlamp examination revealed a large bulbar conjunctival epithelial staining defect extending into the inferior fornix and on to the upper eyelid margin. The corneal transplant showed diffuse punctate keratopathy with staining defects at the graft host junction inferiorly. The patient was started on a combination of neomycin sulfate, polymixin B sulfate, and gramicidin (Neosporin Ophthalmic Solution, Glaxo Wellcome Inc, Research Triangle Park, NC) drops, four times a day, and told to increase the topical steroid to every 2 hours while awake. She was seen 4 days later with a visual acuity of 20/100 OD, a healed ocular surface, and mild corneal graft edema.

Comment. Previous reports have documented the inadvertent use of nonophthalmic drops because of similarities in bottle shape, size, labeling, or cap. This case shows a similarity in bottle label and color (both labels are blue and white). Suggestions for uniform bottle sizing, cap shape, or cap color for all nonophthalmic preparations have not resulted in voluntary changes in packaging by manufacturers. We emphasize that many patients taking eye medicines are partially sighted, and it is the responsibility of manufacturers and health professionals alike to take the necessary steps to avoid preventable misuse of eye drops. We now make it a point to instruct patients that eye drops should always be stored separately from all other nonophthalmic topical preparations.

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This investigation was supported in part by an unrestricted departmental grant from Research to Prevent Blindness Inc, New York, NY.

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Hemifacial Atrophy and Primary Corneal Endothelial Failure

Hemifacial atrophy (Parry-Romberg syndrome) is characterized by a slowly progressive atrophy of the skin, subcutaneous tissue, and muscle and bone of the face, usually beginning in the first 2 decades of life. This disease progresses through an active phase of decompensation, generally lasting 2 to 10 years, followed by a quiescent phase without continued atrophy. Ocular findings most commonly described include progressive enophthalmos, restrictive strabismus, pupillary disturbances, heterochromatic iridocyclitis, and blepharoconjunctivitis. To our knowledge, this case is the first report of primary corneal en-
dothelial failure and penetrating keratoplasty in association with Parry-Romberg syndrome.

Report of a Case. A 59-year-old woman with a history of left hemifacial atrophy since age 15 years had continuous pain in the left eye that was unresponsive to topical lubricants. She had a history of ocular hypertension treated with a topical β-blocker and a single episode of iritis in the left eye.

Ophthalmic examination revealed soft tissue atrophy of the left side of the patient’s face (Figure 1).

The eye examination grossly revealed enophthalmos and approximately 2-mm lagophthalmos on the left side. Best-corrected visual acuity was 20/20 OD and 20/60 OS. Ocular motility was normal. The left pupil was sluggish in its reactivity and slightly oval, but there was no relative afferent pupillary defect. The corneal sensitivity was decreased on the left side. Biomicroscopy confirmed that the right eye was normal. The left eye was injected with full-thickness edema of the inferior two thirds of the cornea and microcystic epithelial changes (Figure 2).

The endothelium was found to have a beaten-metal appearance. The anterior chamber was quiet, and the lens was clear. Intraocular pressure was 13 mm Hg OS confirmed by pneumotonometry. No intraocular pressure was recorded in the right eye. The cup-disc ratio was 0.2 OD and 0.3 OS. Findings from the remainder of the funduscopic examination were unremarkable. Specular microscopy revealed 2100 cells/mm² OD and 300 cells/mm² OS.

The patient underwent penetrating keratoplasty for persistent pain and worsening vision in July 1987. Histological examination of the corneal specimen revealed scant endothelial cells and epithelial edema. She subsequently underwent cataract extraction with lens implantation (July 1988) and medial canthoplasty (January 1989) for exposure. She did well until 3 years after undergoing transplantation when bullous keratopathy from graft failure without signs of rejection developed. She underwent a second penetrating keratoplasty (December 1990) and, during the last 7 years, her corneal transplant has remained clear with good visual acuity (20/30 OS). She has had no bouts of iritis, and the intraocular pressure has remained in good control while receiving topical therapy.

Comment. The cause of corneal endothelial decompensation in this patient is most likely primary endothelial failure. The patient had normal endothelial cell counts and endothelial morphology in the right eye. Although this patient did experience an episode of acute iritis, no stigmata of chronic iritis were present that would lead one to believe that the endothelium decompensated from this (that has been reported by Grayson and Pieroni as a cause of bullous keratopathy in this syndrome). Ocular hypertension is an unlikely cause for the decompensation in this case.

We hypothesize that the primary corneal endothelial failure and the hemifacial atrophy in this case are linked. The etiology of hemifacial atrophy is uncertain. Since the corneal endothelium, skin, soft tissues, and bones of the face are of neural crest origin, a genetic defect
causing an abnormality in the maintenance of these tissues could explain the primary degeneration observed.

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Chronic Angle-closure Mimicking Rubeotic Glaucoma in an Adult With Retinopathy of Prematurity

Adults with cicatricial retinopathy of prematurity (ROP) may develop chronic angle-closure glaucoma due to steeper corneas, shallower anterior chambers, and larger lens-to-axial length ratios than normal eyes. We report a case of chronic angle-closure glaucoma with prominent iris vessels mimicking neovascular glaucoma in an adult with cicatricial ROP.

Report of a Case. A 38-year-old man, born at 7 months’ gestation, had useful vision only in his right eye but was otherwise healthy. Cicatricial ROP and normal intraocular pressure (IOP) were noted on ophthalmic examination at our clinic 5 years earlier. For 1 year he had intermittent right-sided headaches progressing to become daily, and resolving after sleep, that were accompanied by redness and blurred vision in his right eye. His symptoms became constant and his peripheral vision declined; when he was no longer able to read, he was examined. Visual acuity was 20/400 OD and light perception OS. Slitlamp examination of the right eye revealed moderate conjunctival hyperemia, microcystic corneal edema, a peripherally shallow but centrally deep anterior chamber, and a posterior subcapsular cataract. Large arborizing blood vessels were visible on the iris surface. Goldmann applanation tonometry was 44 mm Hg OD, 17 mm Hg OS. Dilated fundus examination of the right eye revealed temporal dragging of the retina, and no apparent macula. In the left eye no details were visible, because the subluxed, cataractous lens filled the irregular pupil.

One day after treatment with isosorbide, acetazolamide, and topical glaucoma medications, the IOP was 10 mm Hg OD. Gonioscopy revealed complete angle closure without neovascularization, but the iris vessels remained prominent. The clearer fundus view confirmed the absence of retinal hemorrhages, detachment, or neovascularization; the optic nerve was dragged temporally and cupped. Blood glucose concentration and carotid pulses and auscultation were normal. Frequent 1% prednisolone acetate was added in anticipation of laser peripheral iridotomy. Two days later the IOP was 50 mm Hg OD, but the iris vessel caliber had decreased greatly (Figure). Despite neodymium:YAG laser iridotomy, the IOP became refractory to medical treatment, and trabeculectomy with mitomycin was performed. Six months later the IOP was 12 mm Hg OD without medications; no iris vessels were visible.

Comment. Rubeosis irides is rare in the absence of retinal or ocular ischemia; active ROP in neonates could conceivably provide the necessary stimulus. Michael et al reported 2 cases of adult-onset neovascular glaucoma associated with cicatricial ROP. In one patient no fundus examination was described, and the second patient had cicatrical changes without mention of detachment or acute ischemic changes (such as hemorrhages or neovascularization), and may represent a case similar to ours. Our patient’s history and ophthalmic examination were consistent with chronic angle-closure glaucoma. Neither retinal detachment nor signs of acute retinal ischemia were evident. The rubeosis responded rapidly to topical corticosteroid treatment and was presumably the result of inflammatory congestion of preexisting vascular channels, perhaps formed in conjunction with “plus” disease in infancy, rather than a true neovascular response to angiogenic factors. Panretinal photocoagulation or cryotherapy usually is considered as part of treatment for neovascular glaucoma but should be deferred in cases similar to ours.

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Progressive outer retinal necrosis (PORN) is a distinct variant of herpetic necrotizing retinopathy caused by the varicella zoster virus that has been seen almost exclusively in patients with end-stage acquired immunodeficiency syndrome. Progressive outer retinal necrosis is characterized by initial geographic opacification of the deep periphereal retina without prominent vasculitis or vitritis followed by rapid progression to involve the entire retina. The prognosis is dismal; the progression to involve the entire retina or vitritis followed by rapid peripheral retina without prominent vasculitis or vitritis characterized by initial geographic progression is typical of progressive outer retinal necrosis.

Despite the administration of high-dose intravenous acyclovir sodium (12.4 mg/kg 3 times a day) instituted on hospital day 2, the retinal opacity progressed to involve the entire retina during the next 2 days (Figure). By 3 days after admission to the hospital, she had no light perception OU that persisted 2 years later, when marked gliosis of the retina was noted. Magnetic resonance imaging was normal. Results of an extensive laboratory evaluation for new rheumatologic and infectious conditions (including human immunodeficiency virus) were negative with the exception of an elevated protein level of 1250 g/L (125 g/dL) in the cerebrospinal fluid. The lower extremity weakness was diagnosed as an unrelated transverse myelitis. A CD4+ count was not obtained, but the patient was lymphopenic (absolute lymphocyte count, −164 cells per microliter [reference range, 150.0-300.0 × 10⁹/L]).

Comment. Although proof of varicella zoster infection was not obtained, the course and appearance of this disease is characteristic of PORN. The presence of optic nerve swelling preceding the peripheral retinal changes is unusual, but this clinical picture has been reported for varicella in patients with acquired immunodeficiency syndrome. Unfortunately, the diagnosis was not made until hospital day 2, when it was too late to salvage any vision despite antiviral treatment.

To our knowledge, this is the second case of PORN that has been described outside of patients with acquired immunodeficiency syndrome; a 15-year-old bone marrow transplant patient receiving multiple immunosuppressive medications for active graft-vs-host disease was reported with a retinitis consistent with PORN. Our case is a further reminder that PORN must be kept in the differential diagnosis for a rapidly progressive retinitis in all immunosuppressed patients, regardless of etiology.

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This investigation was supported in part by an unrestricted grant from Research to Prevent Blindness Inc, New York, NY; the Wisconsin Lions Foundation, Rosholt; and the National Eye Institute, National Institutes of Health, Bethesda, Md, to the Department of Ophthalmology and Visual Sciences at the University of Wisconsin–Madison.

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Cat-scratch Disease Manifesting as Unifocal Helioid Choroiditis

Hong et al have described 6 young, otherwise healthy patients who each had a type of inflammatory lesion of the choroid that had not been de-
scribed previously in the literature. The lesion consisted of a solitary round, yellow-white focus of choroiditis in the posterior pole associated with overlying subretinal fluid. In none of the cases was an infectious or systemic inflammatory origin identified. The authors named the disorder “unifocal helioid choroiditis” to emphasize the lesion’s resemblance to the sun.

We observed a similar ophthalmoscopic picture in a young man who also had historical and serologic evidence of cat-scratch disease. It would appear that infection with Bartonella (formerly Rochalimaea) henselae, the microbial agent of cat-scratch disease, is responsible for at least some cases of helioid choroiditis.

**Report of a Case.** A 20-year-old man reported progressive loss of vision in his right eye over 6 days and a severe headache that had been present for 1 day. He had been scratched by a cat 1 month earlier, after which he experienced intermittent malaise, mild weakness, and fever.

The patient was otherwise healthy. He did not have a history of venereal disease, tuberculosis exposure, foreign travel, recent vaccinations, rashes, arthralgias, or breathing problems, nor had he ingested raw meat. He had been exposed to both puppies and ticks within the previous year.

On ophthalmologic examination, best-corrected visual acuity was 20/40 OD and 20/20 OS. The results of color vision testing with the Hardy-Rand-Rittler plates were normal, although responses in the right eye were somewhat slower. Visual fields demonstrated moderate central and superior depression on the right side and moderate superior depression on the left side. There was no afferent pupillary defect. The anterior segments were unremarkable. Ophthalmoscopy in the right eye revealed a round, yellow, slightly elevated, 1-disc-diameter subretinal lesion that was situated inside the inferotemporal vascular arcade (**Figure 1**). Overlying subretinal fluid extended upward from the lesion and elevated the fovea. The right optic disc appeared to be normal. In the left eye, the optic disc exhibited trace swelling nasally. Fluorescein angiography revealed progressive hyperfluorescence of the lesion in the right eye and very slow filling of the subretinal fluid cavity (**Figure 2**). The right optic disc appeared to be normal. Late staining of the nasal half of the left optic disc confirmed the presence of mild segmental disc swelling.

The results of the following tests were either normal or negative: fluorescent treponemal antibody, toxoplasm IgM and IgG antibody titers, Lyme titer, and a purified protein derivative skin test. *Toxocara* antibody titration results were borderline at 1:32. Titration results for *B henselae* and *Bartonella quintana*, performed by the Centers for Disease Control and Prevention, Atlanta, Ga, were 1:2048 and 1:8192, respectively (reference range, <1:64).

The patient was not treated. Three weeks later, subjective vision in the right eye had almost returned to normal. Visual acuity was 20/20 OD, the yellow subretinal lesion was smaller, and the subretinal fluid had disappeared.

**Comment.** The patient’s fundus picture, age at onset, and clinical course are consistent with the reported description of unifocal helioid choroiditis. Furthermore, the recent occurrence of a cat scratch, the history of fever and constitutional symptoms, and the serologic evidence of *Bartonella* infection strongly suggest that the patient had cat-scratch disease and that the choroidal lesion represented a focus of infection.

Although helioid choroiditis typically manifests as a unilateral, unifocal process, we note that one of the patients described by Hong et al. had associated signs of inflammation in the anterior segment and vitreous body. Our patient had asymptomatic involvement of the optic disc in the left eye. Bilateral intraocular inflammation has been documented in a number of reports of cat-scratch disease.

Visual loss associated with cat-scratch disease is often related to neuroretinitis. In neuroretinitis, inflammation within the optic nerve results in optic disc swelling, local vascular incompetence, and eventual accumulation of fluid and exudate in the macula. In addition, an indistinct mass situated eccentrically on the optic disc is sometimes observed. It is possible that neuroretinitis and helioid choroiditis share a common pathophysiology, even though the primary site of inflammation differs in the 2 conditions.

Unifocal helioid choroiditis should be added to the list of ocular manifestations of cat-scratch disease. Clinicians who encounter patients with this condition should determine if the patient has been exposed to cats and whether constitutional symptoms are present. They also should consider performing serologic tests for *Bartonella* infection.

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**Figure 1.** A solitary round, yellow-white choroidal lesion is situated in the inferotemporal macula of the right eye. Contiguous subretinal fluid elevates the entire fovea.

**Figure 2.** A late-phase fluorescein angiogram shows marked hyperfluorescence of the lesion and more subtle hyperfluorescence of the adjacent subfoveal fluid cavity.
Scleral Buckle Infection With Ciprofloxacin-Resistant Pseudomonas aeruginosa

Fluoroquinolones have gained widespread use for treating ocular infections.1,2 We report a case of scleral buckle infection with Pseudomonas aeruginosa resistant to ciprofloxacin.

Report of a Case. A 35-year-old man was seen by his ophthalmologist in Ecuador with a 1-day history of irritation, redness, and chemosis of the left eye. Eighteen months earlier, he had undergone successful repair of rhegmatogenous retinal detachment in the left eye. A diagnosis of scleral buckle infection was made and therapy was begun with topical norfloxacin and 1% prednisone acetate every 2 hours along with ciprofloxacin, 500 mg twice a day by mouth. No improvement was noted after 1 week and the patient was seen at our institution. Visual acuity was 20/20 OD and 20/80 OS. Left eye showed severe erythema and chemosis of the bulbar conjunctiva. Fundus examination revealed attached retina with good buckle support.

The patient underwent removal of the infected buckle and the scleral bed was irrigated with gentamicin solution. Postoperatively, the patient was initially treated with ofloxacin every 2 hours and ciprofloxacin capsules, 750 mg twice a day by mouth. On postoperative day 2, the patient started experiencing increasing pain and discharge and was noted to have conjunctival pseudomembranes along with inferior symblepharon. Forty-eight-hour culture results from the buckle grew P aeruginosa resistant to ofloxacin and ciprofloxacin, resistance being defined as minimum inhibitory concentration of 4 µg/mL3 or higher. The sensitivities were tested using the automated Vitek test system (bioMerieux Vitek Inc, St Louis, Mo) and confirmed using the conventional disc diffusion method and the “E” test (AB Biodesk, Solna, Sweden). The organism was sensitive to aminoglycosides, ceftazidime, imipenem, mezlocillin, pipericillin, and ticarcillin. Therapy was switched to 1.4% fortified tobramycin (14 mg/mL) every hour. Within 48 hours, the pain subsided. At 2 weeks, visual acuity was 20/25 OS with mild conjunctival erythema. Tobramycin was switched to 0.3% solution, 4 times a day, and the patient returned to Ecuador.

Comment. Animal studies and clinical trials have shown that ciprofloxacin is an effective agent for most ocular infections.1,2 There are reports of ciprofloxacin-resistant systemic isolates including Staphylococcus aureus, coagulase-negative Staphylococcus species, and P aeruginosa.3 The possible mechanisms for acquired resistance include a decrease in the susceptibility of DNA gyrase to the drug-mediated inhibition and a decrease in the amount of drug accumulation within the bacteria.4

In the ophthalmic literature, there are multiple reports of ciprofloxacin-resistant ocular isolates including coagulase-negative Staphylococcus, S aureus, Streptococcus viridans, Corynebacterium pseudodiphtheriticum, Xanthomonas maltophilia, and Mycobacterium chelonae.5,6 To our knowledge, this is the first reported case of an ocular infection from ciprofloxacin-resistant P aeruginosa. There are published cases of Pseudomonas species bacterial keratitis unresponsive to ciprofloxacin therapy, but all those isolates showed in vitro susceptibility to the antibiotic.3 In vitro resistance to a given antibiotic does not mean that the ocular infection will not respond to the antibiotic. The in vitro susceptibilities are based on minimum inhibitory concentration values for serum concentration of antibiotics and do not always equate with in vivo susceptibilities for the treatment of ocular infections. Much higher concentrations of antibiotic may be obtained and host factors also come into play in this setting. However, in our case, clinical failure of treatment correlated with the in vitro results.

In general, mild scleral buckle infection is treated with topical broad-spectrum antibiotics (aminoglycosides or fluoroquinolones).5 In resistant or severe cases, removal of the infected buckle with topical and periocular broad-spectrum antibiotics is usually recommended. The antibiotic is later adjusted according to culture sensitivities from the buckle. In some cases, mere removal of the infected buckle may result in resolution of persistent buckle infection even when the same preoperative antibiotic is used postoperatively. This was the rationale for the postoperative use of fluoroquinolones in our case.

Knauff et al showed a statistically significant increase in ciprofloxacin-resistant systemic isolates from 1988 to 1993. All these systemic isolates are also common ocular pathogens. If the present trend continues, widespread ciprofloxacin resistance among the common ocular isolates may occur in the near future.

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Histologic evidence of giant cell arteritis (GCA) may rarely persist despite long-term treatment with doses of corticosteroids adequate to improve symptoms and normalize the erythrocyte sedimentation rate (ESR).1-3 We describe a patient who had positive findings for GCA on temporal artery biopsy after treatment with prednisone at doses of 30 to 60 mg daily for 6 months. A late biopsy for GCA may be informative, even in the patient treated for months with corticosteroids.

Report of a Case. A 71-year-old woman experienced severe headaches, joint pain, and tenderness of her temporal areas. She had sudden loss of vision in both eyes that was followed by gradual improvement over a few days. The ESR was elevated at 87 mm/h; a presumptive diagnosis of GCA was made, and she was begun on a regimen of prednisone, 60 mg/d, with dramatic improvement in her symptoms. The ESR was 8 and 4 mm/h 1 and 2 months later, respectively, and the prednisone dosage was tapered to 30 mg/d. Approximately 4 months after the onset of symptoms, while on a regimen of 30 mg/d of prednisone and having an ESR of 2 mm/h, she had an uneventful cataract extraction with placement of an intraocular lens implant in the left eye. Her preoperative visual acuities were 20/70 OD and 20/60 OS. Postoperatively, her visual acuity was 20/50 OU. One week after surgery she complained of scalp and temporal area tenderness. Although the ESR was only 5 mm/h, the prednisone dosage was increased to 40 mg/d. Her symptoms abated and the prednisone dosage was tapered to 35 then 30 mg/d over 2-week intervals.

Six months after the initial onset of symptoms, her best-corrected visual acuity was 20/60 OD and 20/25 OS. There was red desaturation in her left eye. There were nuclear sclerotic cataract changes sufficient to account for the decreased visual acuity in her right eye. Her pupils were equal, with a small left relative afferent pupillary defect. The right optic disc was normal and the left showed mild temporal pallor. Kinetic perimetry revealed an inferior altitudinal scotoma on the left and a normal field on the right.

Because of her recurrence of symptoms while taking the 30-mg/d regimen of prednisone when she had a normal ESR, the initial diagnosis of GCA was questioned. Findings from a left temporal artery biopsy revealed a muscular artery with thickened intima. The internal elastic lamina was fragmented and there was granulomatous reaction with multinucleated giant cells centered around the inner elastic lamina. The muscular wall contained a lymphocytic infiltrate. (Figure 1 and Figure 2).

Comment. Histologic evidence of GCA may persist despite treatment with corticosteroids. To and colleagues1 described an 80-year-old woman with GCA who, after 4 weeks of prednisone therapy at 60 mg daily, underwent temporal artery biopsy, the results of which...
demonstrated active disease. Similarly, a patient treated with chronic low-dose prednisone for polymyalgia rheumatica with recurrent symptoms had positive biopsy findings after 1 month of treatment.² In a series of 535 patients, Achkar et al³ reported positive biopsy findings after 11 months of corticosteroid treatment, but the treatment regimen was not described in detail. In another series, McDonnell and colleagues⁴ distinguished between the histologic features of active and healed arteritis. Both are characterized by marked intimal thickening, disruption or loss of the internal elastic lamina, and thickening and scarring of the media and adventitia. Active arteritis shows an inflammatory infiltration with lymphocytes, macrophages, and multinucleated giant cells, whereas healed arteritis shows only occasional foci of lymphocytes. Temporal artery biopsy specimens with evidence of active arteritis were taken after a mean of 7 days of treatment, whereas those with healed arteritis were taken after a mean of 82 days of therapy.⁴ The longest corticosteroid-to-biopsy interval among patients in this series with active arteritis was 45 days.

To our knowledge, our patient has one of the longest reported intervals of positive biopsy findings after corticosteroid treatment for GCA, and the corticosteroid dosage was substantial. Although early biopsy is always recommended, arteritis may still be histologically documented after several months of a corticosteroid regimen adequate to improve symptoms and normalize the ESR. Positive biopsy findings will help facilitate future decisions on corticosteroid therapy, particularly if complications from such therapy are expected. Recognition of healed vs active arteritis is also vital in cases where the patient has been on a regimen of corticosteroids without the benefit of biopsy. The distinction between active and healed arteritis probably does not carry as much diagnostic as prognostic value, since evidence of active disease despite corticosteroids probably indicates that a longer or more aggressive therapeutic approach is needed.

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Supported in part by a departmental grant (Ophthalmology) from Research to Prevent Blindness, Inc, New York, NY, and by CORE grant P30-EY6360 from the National Institutes of Health, Bethesda, Md.

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