hypofluorescence that correlated with the findings seen on color photography (Figure 2). Her visual acuity loss and fundus findings were attributed to choroidal infarction that occurred immediately postoperatively. A cerebral internal carotid artery angiogram performed 4 days postoperatively, once her proptosis resolved, revealed a surgical clip at the expected location and no evidence of a clip or obstruction at the level of the ophthalmic artery, suggesting choroidal reperfusion. Her visual acuity remained no light perception.

Comment. Papaverine is a potent vasodilator used to prevent cerebral vasospasm in cranial surgery. It has been shown to increase vessel diameter by an average of 30.1%. We believe our patient had a choroidal infarct, which is a previously unrecognized complication of intracranial papaverine irrigation. Because of its potent vasodilatory effects, papaverine infusions have been shown to increase cerebral blood volume and create a rapid increase in intracranial pressure. Similarly, we feel that the exposure of our patient’s orbital vessels to this agent may have led to widespread vasodilatation, exudation, and an increase in orbital pressure, thereby compressing the posterior ciliary arteries. The small keyhole created in the sphenoid bone may have provided a conduit whereby intracranial papaverine gained access to the orbit. The spontaneous orbital decompression and choroidal reperfusion seen on fluorescein angiography a few days postoperatively may be explained by the short half-life of papaverine and suggest that orbital hemorrhage was not the culprit.

Choroidal infarction after clipping of a cerebral aneurysm may occur after inadvertent clipping of or trauma to the ophthalmic artery. The observation in our case, with angiographic evidence of a patent ophthalmic artery, suggests that intracranial instillation of papaverine may lead to a choroidal infarct and irreversible vision loss. Although there may be spontaneous resolution of orbital edema, we should counsel neurosurgeons regarding this devastating ocular complication and have them consider other, less potent vasodilators when an aneurysm is accessed via the orbital wall.

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**Enterococcus casseliflavus Endophthalmitis Associated With a Horse Tail Injury**

*Enterococcus casseliflavus* is a gram-positive bacteria that has been rarely reported in human infection. We describe the first case to our knowledge of *E. casseliflavus* endophthalmitis associated with a human’s injury by a horse’s tail.

**Report of a Case.** A 37-year-old healthy man had pain and decreased vision in his left eye. Two days earlier while working on a horse farm, he was whipped across the face by the tail of a horse. The next day, his eye became painful. He denied any fever or chills. On examination, his visual acuity was 20/20 OD and light perception OS. There was no relative afferent pupilary defect. Extraocular movements were intact. There were skin abrasions on his forehead and cheek. Results from slitlamp and fundus examinations of the right eye were unremarkable. Slitlamp examination of the left eye revealed +2 conjunctival chemosis and +2 corneal edema without an epithelial defect (Figure). The anterior chamber had a fibrinous reaction with a 1-mm hypopyon. Dilated funduscopy examination of the left eye showed dense vitritis. Contact B-scan ultrasonography of the left eye showed vitreous opacities without retinal detachment. The patient underwent a diagnostic vitrectomy with injection of vancomycin hydrochloride (1 mg/0.1 mL) and cefazidime (2 mg/0.1 mL) for presumptive endophthalmitis. Vitreous cultures grew *E. casseliflavus* that was sensitive to ampicillin but resistant to vancomycin. The blood culture results were negative.

The patient’s pain and anterior segment inflammation resolved 1 week after surgery. Three weeks postop-
eratively, his visual acuity was hand motions OS. A white cataract and vitreous debris were present. Eight weeks after his initial visit, he underwent a pars plana lensectomy and vitrectomy. Intraoperatively, after the vitreous debris was removed, there was an advanced rhegmatogenous and tractional retinal detachment with proliferative vitreoretinopathy. The retinal detachment was inoperable owing to the advanced proliferative vitreoretinopathy and necrotic retina. His last visual acuity was light perception OS.

Comment. *E. casseliflavus* is a particular strain of enterococcus commonly found in the gastrointestinal tract of livestock such as cattle and horses. It has rarely been associated with human infection. There have been a few cases involving polymicrobial bacterial bacteremia with biliary tract disease in humans. A PubMed search revealed no reports of *E. casseliflavus* and eye infection. In this case, the etiology of the *E. casseliflavus* endophthalmitis is not clear. The blood culture results were negative and the patient was afebrile, making endogenous endophthalmitis less likely. The horse tail injury may have introduced the pathogen. *E. casseliflavus* is the most common strain found in fresh and dry horse manure, the likely source of infection. However, the mechanism is unclear as there was no entry site for the bacteria (ie, no evidence of a gross perforation). The conjunctiva was chemoic, but on surgical exploration there was no evidence of a gross perforation. The horse tail injury might have caused a self-sealing microperforation through the cornea or the conjunctiva, which may explain how the bacterial strain entered the eye. The patient did have abrasions around his face from the horse tail whip, suggesting that the tail could have hit the eye as well. Animal tail whip injuries have been reported to cause traumatic subconjunctival crystalline lens and intraocular penetration with the animal hair. Ophthalmologists must be aware that tail whip injuries can have serious visual consequences.

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**COMMENTS AND OPINIONS

**Evolution: Theory, Not Fact**

I t is appropriate to commemorate the Darwin anniversary; his life’s work merits recognition regardless of one’s ideology, and an ophthalmology theme makes for interesting copy. However, lost in the platitudes is the fact that evolution is still but a theory, not an experimentally verifiable fact. There is no more than B:III evidence for the theory of evolution (ie, there have been no clinical trials, randomized or not, confirming the theory; rather, respected authorities have concluded its parts and, at best, there are case-control series that have been extrapolated to its conclusion) despite pervasive, frequent, and dogmatic proclamations to the contrary.

A letter to the editor will not settle this contentious matter but, in keeping with the Archives’ high standards, some balance is due. The main tenet of evolution is successive improvements via stepwise, chance occurrences whose long odds are explained away by the supposition of “millions on millions of years.” It is important to distinguish natural selection, an observable fact (why are NBA basketball players so tall, after all?), from the huge extrapolation of (macro)evolution. Darwin explicitly stated that if a step-by-step scheme could not explain a change, then his “theory would absolutely break down.”

The 2000 steps in the Nilsson-Pelger photoreceptor-to-globe model, cited and schematically illustrated by Fishman, represents at least 3 major problems. First, if we presume that only 2 competitive options were available for each of these 2000 steps, there would be only 2-2000 chance of forming this rudimentary shell (never mind the unbelievable luck that each step might occur consecutively and take as little as 1 year). This might sound plausible until we do the math to offer a comparative scale of the odds: this 10−602 chance occurrence is still 10−491 as large as the number of collisions of the estimated 1078 atomic particles in the universe if they each collided as fast as a molecule vibrates (10−15 seconds) for the 1018 seconds that 25 billion years represents; the sun would have produced only 1065 photons if production had been steady for those 25 billion years. Apply that to the supposedly 540 million–year-old trilobite compound eye and these odds defy reasonability. Second, each change had to have been written into the DNA code (presuming that had already evolved), and if 1 lethal mutation occurred, the guardian heir of the DNA of an accumulated step would be lost. Third, how could the nascent eye have interfaced with other modularity products? Consider that the eye shell has been outfitted with complex neuronal and vascular hardware, trimmed with muscles, eyelids, lacrimal glands, and other paraphernalia (again, all captured in the DNA blueprint as dominant genes), and that some of these plug-ins are unique and critical to its function (eg, eye movement nuclei, the visual cortex) or involve other complex functions like melanin centers for day/night cycles and jiggle centers. And where did the chiasm come from?