Akinetopsia From Nefazodone Toxicity
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PURPOSE: To investigate two cases of selective impairment of motion perception (akinetopsia) induced by
toxicity from the antidepressant nefazodone, a new drug that blocks serotonin reuptake and antagonizes 5-HT2 receptors.

METHODS: Case reports.

RESULTS: A 47-year-old man receiving nefazodone (Serzone; Bristol-Meyers Squibb, New York, N.Y.) (100 mg
twice daily), reported a bizarre derangement of motion perception. Moving objects were followed by a trail of multiple “freeze-frame” images, which dissipated promptly when motion ceased. A 48-year-old woman receiving nefazodone (400 mg daily at bedtime) reported a similar phenomenon, with visual trails following moving objects. In both patients, vision returned to normal after the dosage of nefazodone was reduced or eliminated.

CONCLUSIONS: Nefazodone toxicity can result in akinetopsia, characterized by the inability to perceive motion in a normal, smooth fashion; persistence of multiple, strobelike images; and visual trails behind moving objects. In this rare syndrome, stationary elements are perceived normally, indicating that nefazodone causes selective impairment of pathways involved in motion processing in the visual system. (Am J Ophthalmol 1999;128:530–531. © 1999 by Elsevier Science Inc. All rights reserved.)

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Zeke has coined the term “akinetopsia” to refer to a selective deficit in the ability to perceive motion. Most reports pertaining to this rare clinical phenomenon have concerned a single subject (L.M.), who developed akinetopsia after suffering large, bilateral parietal strokes. Presumably she sustained damage to area MT (V5), an extrastriate visual region thought to be involved in motion perception.1,3 We describe two cases of akinetopsia, without other neurologic deficits, induced by toxicity from nefazodone, an antidepressant drug.

CASE 1: A 47-year-old man reported seeing streams of multiple, frozen images trailing in the wake of moving objects. As soon as motion ceased, the images collapsed into each other. He compared his vision to a scene lit by a flashing strobe, except that stationary elements were perceived normally. In fact, if nothing was in motion and he held perfectly still, his vision was entirely normal. The moment anything moved, however, it left a stream of static copies in its path. For example, while out for an evening stroll, he saw a pack of identical dogs lined up behind his West Highland terrier. Driving was impossible because he was confused by multiple snapshots of cars, streets, and signs. Moving lights were followed by a long comet tail.

The patient was taking nefazodone, 100 mg twice a day, for depression. He also had human immunodeficiency virus (HIV) infection, with a CD4+ T-cell count of 900 per μl. No history of infection or dementia related to acquired immunodeficiency syndrome was present. His visual symptoms began 2 weeks after starting treatment with ritonavir, saquinavir, and nevirapine in November 1997. These HIV protease inhibitors and nefazodone are metabolized by the enzyme cytochrome P450III4A. The patient consulted us in July 1998. Visual acuity, versions, ocular alignment, visual fields, color perception, stereopsis, and fundi were normal. A brain magnetic resonance imaging scan was unremarkable. A plasma nefazodone level, drawn 6 hours after his morning dose of 100 mg, was 4.5 μg per ml. This value was five times greater than expected for a patient taking only 100 mg twice daily and corresponded approximately to the plasma concentration expected in someone taking the maximum recommended dose of nefazodone, 300 mg twice daily. The patient’s symptoms resolved promptly after nefazodone was discontinued.

CASE 2: Our second patient was a 48-year-old woman who began treatment with nefazodone (400 mg every night) in February 1998 for seasonal affective disorder. She was in good health and took no other medications. Several weeks after starting the drug she reported that, on arising at 4 AM to urinate, she saw visual trails behind moving objects. For example, as she moved her arm, passage of the limb would be reduplicated by multiple, fuzzy images, the way a cartoonist might draw motion. Once her arm stopped, no visual distortion was present. This parcellation of motion occurred with any moving object and was most
evident in dim light. Her symptoms decreased during the course of the day. She consulted us in March 1998. Reduction of her nefazodone dose to 275 mg every night eliminated the symptoms.

Nefazodone is a new antidepressant drug that blocks serotonin (5-HT$_2$) receptors and the reuptake of 5-HT. In clinical trials, 7% of patients (vs 1% of patients treated with a placebo) had abnormal vision, including “visual trails.” Four prior case reports have documented “ghost shadows” and “light trails” from nefazodone usage. Our patients also had these phenomena but had a more dramatic disturbance of vision consisting of akinetopsia and polyopia (for moving objects only). Hughes and Lessell have reported identical symptoms, which they described as palinopsia, in patients with side effects from trazodone. Nefazodone and trazodone are closely related drugs, with similar pharmacologic actions. Ghost images trailing behind moving objects have also been reported from lysergic acid diethylamide, another substance that binds 5-HT$_2$ receptors.

These cases raise four points. First, bizarre visual complaints are easy to dismiss in patients under psychiatric care. Our patients’ symptoms were genuine and, in fact, iatrogenic. Second, both nefazodone and HIV protease inhibitors are metabolized by the cytochrome P$_{450}$IIIA4 isozyme, creating the potential for adverse drug interactions. The entry under nefazodone in the Physicians’ Desk Reference does not mention explicitly the need to lower nefazodone dosage when HIV protease inhibitors are added to a patient’s medical regimen. Third, the selective disruption of motion perception by nefazodone implies that neurons in area MT (V5) use 5-HT as a neurotransmitter or that cells in various cortical areas engaged in motion perception share a 5-HT–based pharmacology. Fourth, our cases highlight an overlap between akinetopsia and palinopsia. In both phenomena, the brain fails to update the visual percept normally. We have interpreted our patients’ symptoms as akinetopsia rather than palinopsia because the persistence of images was brief and occurred only for moving objects, which were immediately present in the visual scene. In palinopsia, image retention also occurs for stationary objects and for objects that have left the visual scene entirely. A defect in the ability to perceive object motion does occur in palinopsia but only as a secondary feature, whereas impaired motion perception is (by definition) the cardinal feature of akinetopsia.

REFERENCES

Cerebrospinal Fluid Leakage During Endoscopic Forehead Lifting
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PURPOSE: To report a case of endoscopic brow lift in which cerebrospinal fluid leakage was encountered.

METHOD: A 69-year-old otherwise healthy man underwent endoscopic forehead lifting.

RESULTS: An area of strong adherence was encountered in the area of the left superior paracentral scalp incision. As the adherence was released, clear fluid extruded (cerebrospinal fluid) and a burr hole was discovered. Absorbable gelatin sponge was placed over the dural defect and burr hole, and closure of the endoscopic scalp incisions was accomplished.

CONCLUSION: Caution is suggested in performing this procedure when a patient has any history of a head trauma. (Am J Ophthalmol 1999;128:531–532. © 1999 by Elsevier Science Inc. All rights reserved.)

The concept of endoscopic forehead lift was introduced by Gregory Keller in 1991 (American Academy of Cosmetic Surgery World Congress, Scottsdale, Arizona) and is becoming more widely accepted to correct functional and cosmetic eyebrow and forehead ptosis. The advantage of this procedure is that it is minimally invasive. It also has been refined sufficiently to be considered a reasonable addition to cosmetic and functional forehead lifting. Before the endoscopic forehead lift, eyebrow elevation included coronal or pretracheal forehead elevation, midforehead elevation, direct brow lift, and browpexy from a blepharoplasty incision.

We present an unexpected encounter with a burr hole during an endoscopic forehead lift. A 69-year-old man

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