Comment. Posterior pituitary ectopia is a neurohypophyseal malformation that is visible on T1-weighted magnetic resonance images (Figure 2). Numerous large, round, circumscribed, whitish areas of retinochoroidal depigmentation were dispersed throughout the retinal periphery. Examination of both parents disclosed normal-sized eyes without evidence of colobomas.

Peripheral blood chromosomes were normal at 450-band resolution. Polymerase chain reaction amplification and sequencing of the coding regions and nearby flanking sequences of the SIX6 (formerly OPTX2) homeobox gene, a developmental regulatory gene that is a candidate gene for oculopituitary disorders,7 showed no mutations or amino acid sequence variants.

In a recent study of children with optic nerve hypoplasia, Phillips et al8 found posterior pituitary ectopia or absence of the pituitary infundibulum in 23 of 26 patients with congenital hypopituitarism vs none of the 41 patients with normal endocrinologic function.

To our knowledge, isolated optic nerve aplasia with hypopituitarism has not been described. However, the association of optic nerve aplasia and hypopituitarism is well recognized in the clinical context of severe microphthalmos and anophthalmos (a condition in which the optic nerves are absent or rudimentary).7,8 Keppen et al9 found hypogonadotropic hypogonadism in 5 of 13 patients with mental retardation and anophthalmos or severe microphthalmos, suggesting a generalized defect in forebrain development. Brodsky and Frindick8 documented a neurohypophyseal malformation closely resembling posterior pituitary ectopia in a male infant with bilateral anophthalmos and low serum cortisol and pituitary gonadotropin levels. Neuroimaging results showed our patient’s central nervous system abnormalities were confined to the anterior visual pathways and pituitary infundibulum. The risk of sudden death from adipsionic crisis in children with corticotropin deficiency mandates early recognition of congenital hypopituitarism in high-risk patients.9 Posterior pituitary ectopia may prove to be a neuroimaging marker for congenital hypopituitarism in children with optic nerve aplasia.

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Visual Improvement in an Adult Amblyopic Eye Following Radiation-Induced Visual Loss in the Contralateral Eye

Amblyopia is a visual deficit defined as decreased best-corrected visual acuity of at least a 2-line difference between the two eyes that is not
due to an organic cause. It is typically associated with strabismus, uncorrected asymmetric refractive error, or ocular disorders that interfere with the development of the fixation reflex. Although the pathophysiological mechanisms in amblyogenesis are not completely understood, a fundamental principle of treatment is that therapy can be effective only when the visual system is sufficiently “plastic” for cortical modification to occur. The sensitive or critical period of visual development and modification is species specific and age defined; in humans it is thought to occur from birth to the arbitrary age of 9 years.

We describe an adult with a childhood history of anisometropic amblyopia who lost central vision in his dominant eye as a result of radiation-induced maculopathy following treatment for a macular choroidal melanoma. The patient subsequently regained excellent vision in the previously amblyopic eye.

**Report of a Case.** In May 1990, a 42-year-old man experienced decreased vision in his right eye. He had a history of anisometropic amblyopia in his left eye since childhood, with a consistently recorded best-corrected Snellen visual acuity of 20/80 OS. Part-time occlusion was instituted in the right eye at age 6 years, with cessation 1 month later because of patch intolerance. This eye was never optically corrected.

At examination his visual acuity was 20/30 OD and 20/80 OS. The left eye was anatomically normal (Figure 1). Fundus examination of the right eye showed an amelanotic, juxtafoveal choroidal mass with a base measuring 6.0 × 4.5 mm and a thickness of 3.8 mm. A secondary serous retinal detachment was noted over the lesion, extending into the foveola and inferior fundus. The tumor was echolucent at ultrasonographic examination and displayed intrinsic vessels on fluorescein angiographic studies, consistent with a choroidal malignant melanoma.

The melanoma was treated with iodine 125 (I 125 ) plaque radiotherapy. The tumor apex received 11500 rad (115 Gy), and the foveola received 24453 rad (244 Gy). Eighteen months later, tumor regression and subretinal fluid resolution were documented. However, radiation-induced maculopathy caused by treatment was found with cystoid macular edema, foveal exudation, retinal hemorrhage, and microvascular ischemia, allowing a visual acuity of only 20/60 OD (Figure 2). Given the amblyopia in the left eye and decreased vision in the right eye, severe visual handi-

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**Table 1. Visual Acuity Recordings of Our Patient at Each Examination**

<table>
<thead>
<tr>
<th>Date, ho/y</th>
<th>Visual Acuity (OD)</th>
<th>Visual Acuity (OS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6/1990*</td>
<td>20/30</td>
<td>20/80†</td>
</tr>
<tr>
<td>12/1990</td>
<td>20/40</td>
<td>20/100</td>
</tr>
<tr>
<td>11/1991‡</td>
<td>20/60</td>
<td>20/80</td>
</tr>
<tr>
<td>11/1992</td>
<td>20/100</td>
<td>20/50</td>
</tr>
<tr>
<td>11/1993</td>
<td>20/220</td>
<td>20/50</td>
</tr>
<tr>
<td>11/1994</td>
<td>20/100</td>
<td>20/50</td>
</tr>
<tr>
<td>11/1995</td>
<td>20/220</td>
<td>20/50</td>
</tr>
<tr>
<td>11/1996</td>
<td>CF</td>
<td>20/30</td>
</tr>
<tr>
<td>11/1997</td>
<td>CF</td>
<td>20/40</td>
</tr>
<tr>
<td>11/1998</td>
<td>CF</td>
<td>20/30</td>
</tr>
<tr>
<td>11/1999</td>
<td>CF</td>
<td>20/25</td>
</tr>
<tr>
<td>4/2002</td>
<td>CF</td>
<td>20/20</td>
</tr>
</tbody>
</table>

*Abbreviation: CF, counting fingers. Diagnoses of choroidal melanoma in the right eye and treatment with iodine 125 plaque radiotherapy.
†Amblyopia.
‡Radiation maculopathy first detected.
cap was anticipated. Surprisingly, visual acuity in the amblyopic eye improved from 20/80 in June 1990 to 20/20 in April 2002 (Table 1).

**Comment.** Amblyopia is generally considered to be a treatable condition when recognized and managed at an early age.2-3 Treatment of amblyopia consists of 2 basic strategies. The first is to optimize the retinal visual image in the amblyopic eye. This is accomplished by providing a clear visual axis and correcting any significant refractive errors. The second strategy is to enhance the neural stimulus to the visual cortex. In most cases, this is accomplished by limiting stimuli to the nonamblyopic eye using occlusion therapy with patching, pharmacologic penalization, or optical defocusing.2,5

Recovery of visual function in adult amblyopic eyes has also been documented following visual loss in the fellow eye4-8 (Table 2). This finding appears to be independent of the age at which the dominant eye loses vision, whether the loss of vision is total or partial, and previous treatment of amblyopia. In a retrospective multicenter study, Vereecken and Brabant6 found an improvement of at least 3 lines in the amblyopic eye in 28.5% of patients with vision loss in the good eye. A favorable prognostic factor for improvement was the existence of foveal fixation associated with anisometropic amblyopia.

Improvement of visual acuity in the amblyopic eye is usually gradual. El Mallah et al7 retrospectively analyzed the records of 465 adult patients with age-related macular degeneration and identified 9 individuals who also had amblyopia in the fellow eye. All but 2 of these 9 patients showed improvement in vision that averaged 1.5 lines at each consecutive 6-month visit. At the final visit, the accumulated gains in the amblyopic eye averaged 3 lines and generally occurred between 1 and 12 months from baseline. In our patient the visual recovery was slow, during nearly 1 decade, possibly related to the slow loss of vision in the dominant eye.

Visual improvement in the amblyopic eye can be sustained even after recovery of visual acuity in the nonamblyopic eye. Wilson8 described 2 adult patients aged 57 and 67 years with documented childhood amblyopia resulting in a visual acuity of 20/200 and 20/100, respectively, in the affected eye. Cataract extraction in the dominant eye of each patient, to a visual acuity of 20/50 and 20/80, respectively, was associated with improvement in visual acuity of the amblyopic eye to 20/30 and 20/25. Most remarkably, the improvement was sustained after the nonamblyopic eye was rehabilitated by cataract extraction to a visual acuity of 20/20.

In summary, we describe an adult patient whose childhood anisometropic amblyopia reversed completely during a period of 12 years when the contralateral eye developed radiation maculopathy secondary to plaque radiotherapy of a macular choroidal melanoma. Acknowledgement of such cases is important when treating and counseling patients with a vision-threatening ocular condition in whom the contralateral eye is amblyopic.

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**Table 2. Case Reports of Patients With Childhood Amblyopia That Improved in Adulthood**

<table>
<thead>
<tr>
<th>Study</th>
<th>Patient(s), Sex/Age, y</th>
<th>Cause of Amblyopia</th>
<th>Initial Visual Acuity in the Amblyopic Eye</th>
<th>Time Course of Improvement</th>
<th>Final Visual Acuity in the Amblyopic Eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rabini5</td>
<td>F/55</td>
<td>High myopia</td>
<td>Subfoveal CNVM</td>
<td>20/160</td>
<td>3 y</td>
</tr>
<tr>
<td>Hamed et al6</td>
<td>M/54</td>
<td>Anisometropia</td>
<td>AION</td>
<td>20/200</td>
<td>6 mo</td>
</tr>
<tr>
<td></td>
<td>M/70</td>
<td>Strabismus</td>
<td>AION</td>
<td>20/80</td>
<td>1 mo</td>
</tr>
<tr>
<td></td>
<td>F/72</td>
<td>Strabismus</td>
<td>AION</td>
<td>20/400</td>
<td>4 mo</td>
</tr>
<tr>
<td></td>
<td>F/57</td>
<td>Accommodative esotropia</td>
<td>Cataract</td>
<td>20/200</td>
<td>4 y</td>
</tr>
<tr>
<td></td>
<td>F/67</td>
<td>Accommodative esotropia</td>
<td>Cataract</td>
<td>20/100</td>
<td>10 y</td>
</tr>
<tr>
<td></td>
<td>Current case</td>
<td>M/42</td>
<td>Anisometropia</td>
<td>Radiation maculopathy following plaque radiotherapy for choroidal melanoma</td>
<td>20/80</td>
</tr>
</tbody>
</table>

Abbreviations: AION, anterior ischemic optic neuropathy; CNVM, choroidal neovascular membrane.

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**References**