Chapter 3

FUNDUSCOPIC FINDINGS

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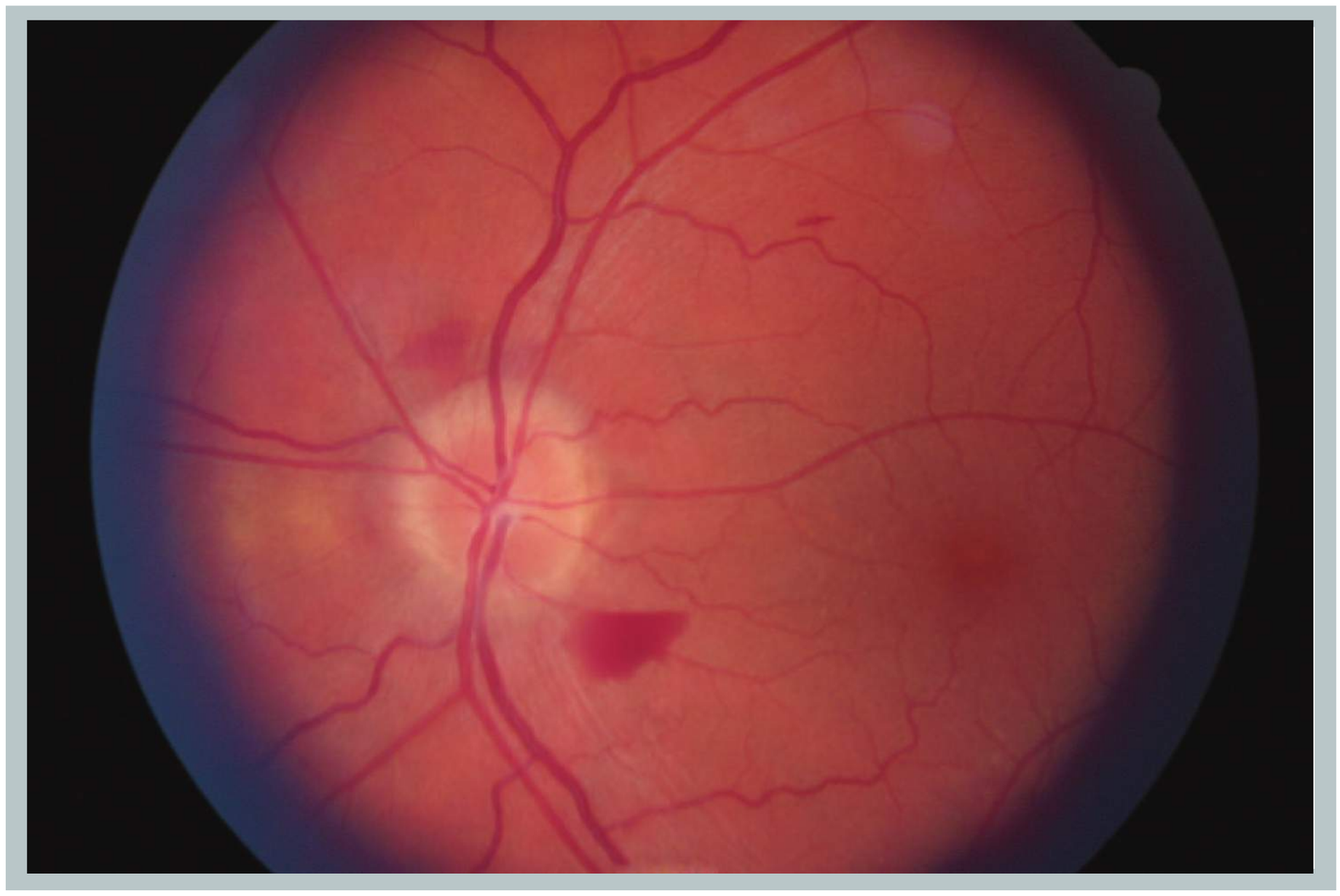

Papilledema. Blurred disc margins indicate papilledema. A small subhyaloid hemorrhage is also seen inferior to the disc at 5-o'clock position. (Photo contributor: Aaron Sobol, MD.)

NORMAL FUNDUS

Clinical Summary

Disk

The disk is pale pink, approximately 1.5 mm in diameter, with sharp, flat margins. The physiologic cup is located within the disk and usually measures less than six-tenths the disk diameter. The cups should be approximately equal in both eyes.

Vessels

The central retinal artery and central retinal vein travel within the optic nerve, branching near the surface into the inferior and superior branches of arterioles and venules, respectively. Normally the walls of the vessels are not visible; the column of blood within the walls is visualized. The venules are seen as branching, dark red lines. The arterioles are seen as bright red branching lines, approximately two-thirds or three-fourths the diameter of the venules.

Macula

This is an area of the retina located temporal to the disk; it is void of visible vessels. The fovea is an area of depression approximately 1.5 mm in diameter (similar to the optic disk) in the center of the macula. The foveola is a tiny pit located in the center of the fovea. These areas correspond to central vision.

Background

The background fundus is red; there is some variation in the color, depending on the amount of individual pigmentation and the visibility of the choroidal vessels beneath the retina.

Pearls

1. Fundal examination should be an integral part of any eye examination.
2. The cup/disk ratio is slightly larger in the African American population.
3. The normal fundus should be void of any hemorrhages, exudates, or tortuous vasculature.

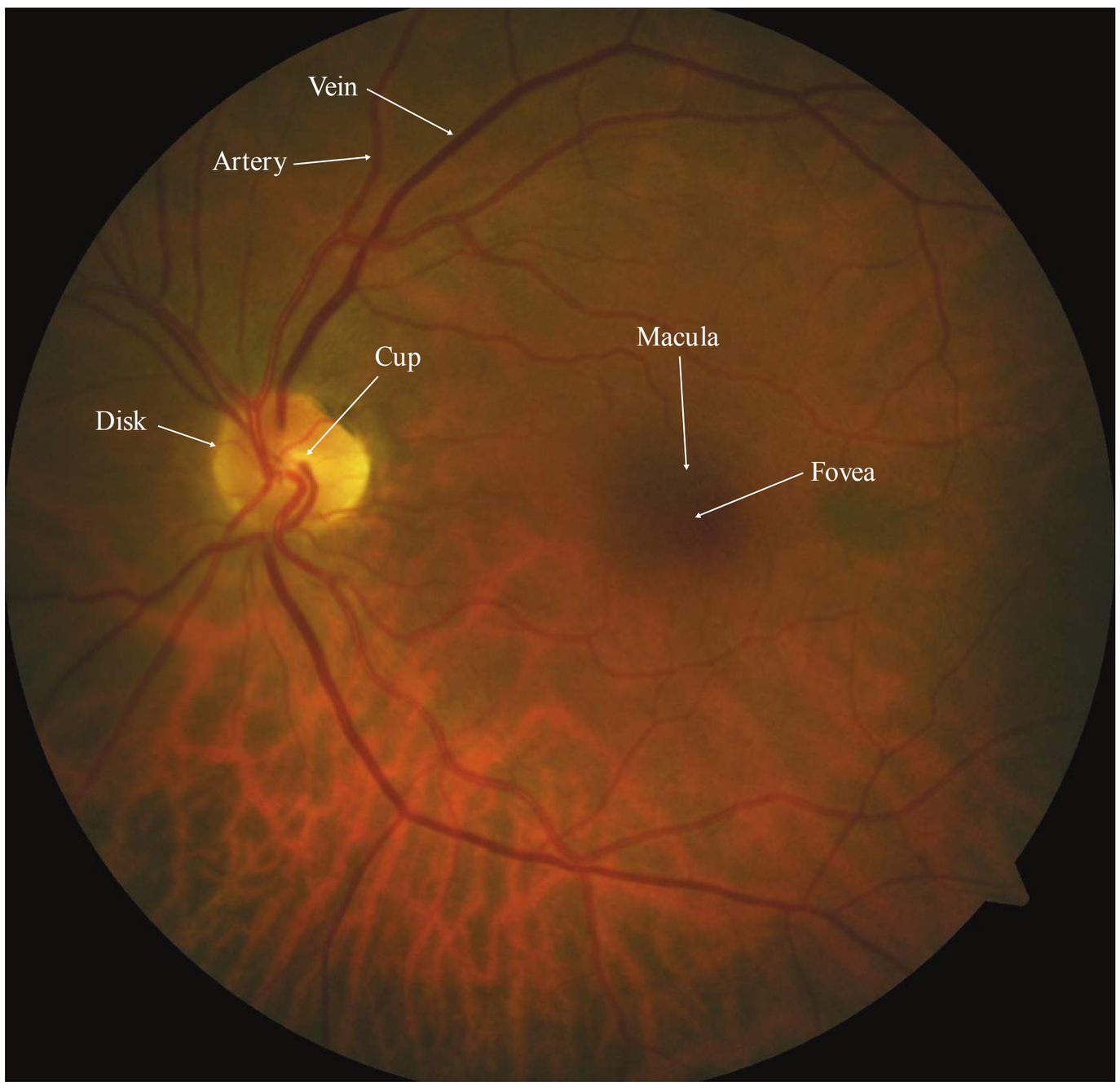

FIGURE 3.1 ■ **Normal Fundus.** The disk has sharp margins and is normal in color, with a small central cup. Arterioles and venules have normal color, sheen, and course. Background is in normal color. The macula is enclosed by arching temporal vessels. The fovea is located by a central pit. (Photo contributor: Jeffrey Goshe, MD.)

AGE-RELATED MACULAR DEGENERATION

Clinical Summary

Age-related macular degeneration increases in incidence with each decade over 50 and is evidenced by accumulation of either hard drusen (small, discrete, round, punctate nodules) or soft drusen (larger, pale yellow or gray, without discrete margins that may be confluent). Most patients with drusen have good vision, although there may be decreased visual acuity and distortion of vision. There may be associated pigmentary changes and atrophy of the retina. Vision may slowly deteriorate if atrophy occurs.

Patients with early or late degenerative changes of the macula are at risk of developing choroidal neovascularization (CNV), which is associated with distortion of vision, blind spots, and decreased visual acuity. Macular appearance may show dirty gray lesions, hemorrhage, retinal elevation, and exudation.

Management and Disposition

Patients with drusen need ophthalmologic evaluation every 6 to 12 months or sooner if visual distortion or decreasing visual acuity develops. If a patient complains of deterioration of visual acuity or image distortion, prompt ophthalmic evaluation is warranted.

Pearls

1. Age-related macular degeneration is the leading cause of blindness in the United States in patients above 65 years of age.
2. Patient may have normal peripheral vision.
3. Untreated CNV can lead to visual loss within a few days.
4. Patients frequently complain of distortion with CNV.

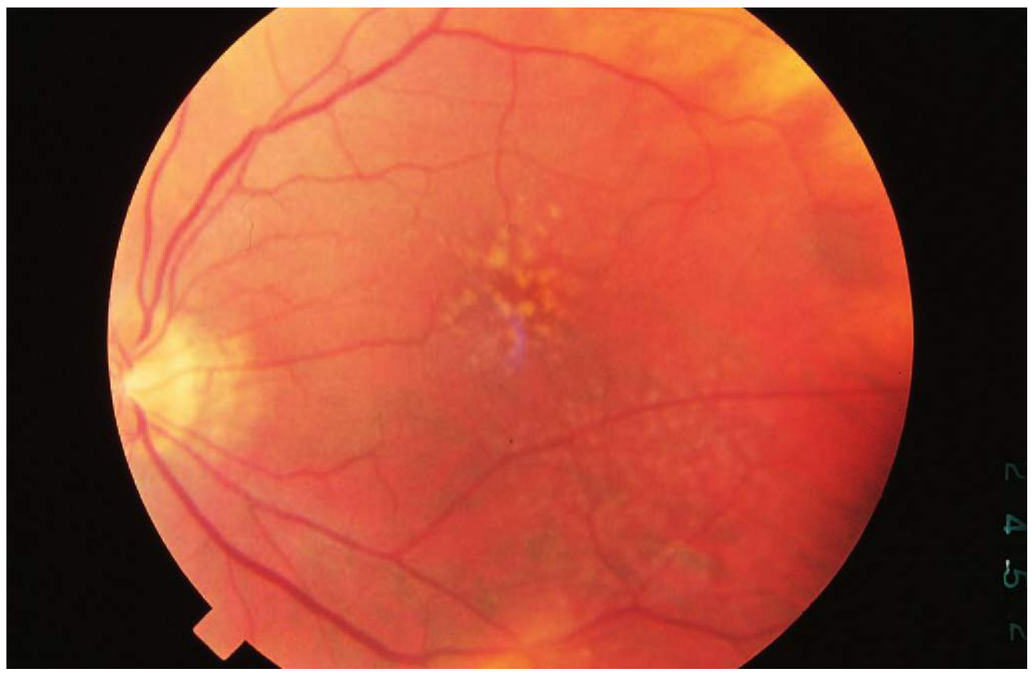

FIGURE 3.3 ■ **Age-Related Macular Degeneration, Drusen.** Drusen are clustered in the center of the macula. (Photo contributor: Richard E. Wyszynski, MD.)

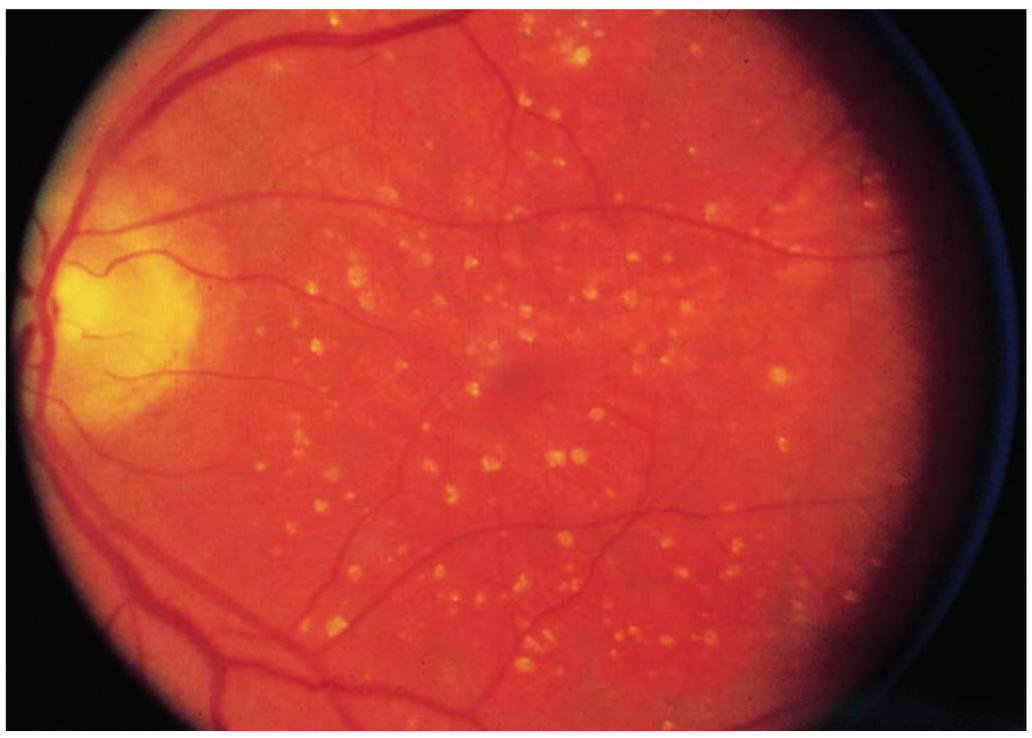

FIGURE 3.2 ■ **Age-Related Macular Degeneration, Drusen**. Typical macular drusen and retinal pigment epithelial (RPE) atrophy (scalloped pigment loss) in age-related macular degeneration.

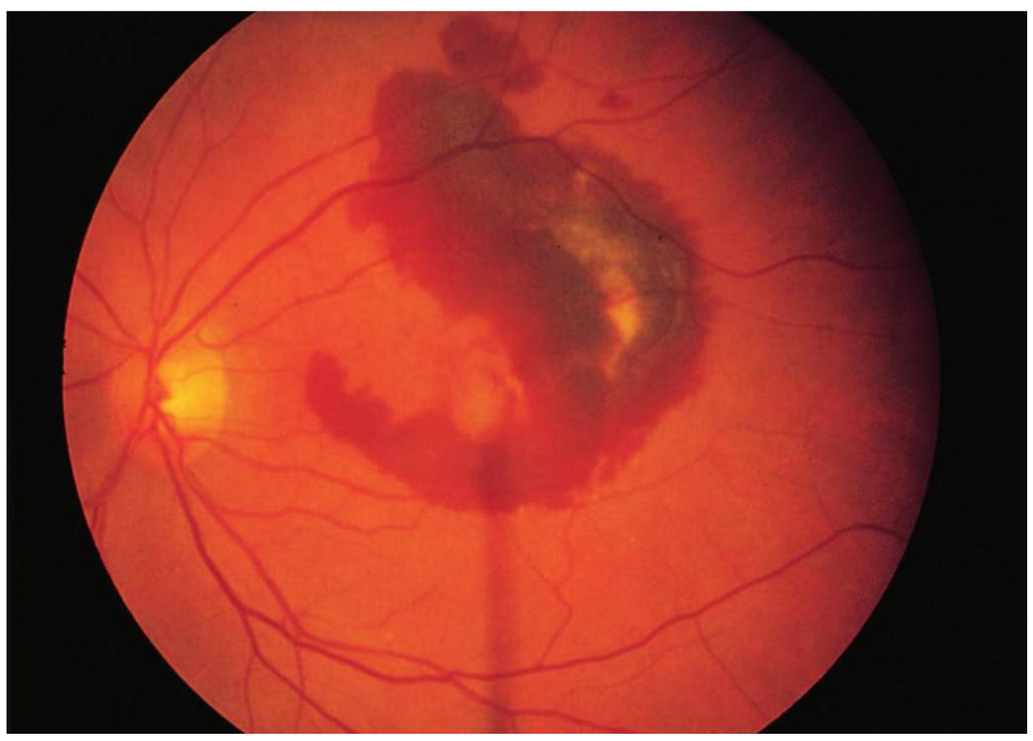

FIGURE 3.4 ■ **Age-Related Macular Degeneration.** Hemorrhage seen beneath the retina in association with subretinal neovascularization. Note the retinal vessels are superficial to the hemorrhage, which lies just beneath the retina. (Photo contributor: Richard E. Wyszynski, MD.)

Clinical Summary

Hard exudates are refractile, yellowish deposits with sharp margins composed of fat-laden macrophages and serum lipids. Occasionally the lipid deposits form a partial or complete ring (called a circinate ring) around the leaking area of pathology. If the lipid leakage is located near the fovea, a spoke or star-type distribution of the hard exudates may be seen.

Cotton wool spots, or soft "exudates," are actually microinfarctions of the retinal nerve fiber layer, and appear white with soft or fuzzy edges.

Inflammatory exudates are secondary to retinal or chorioretinal inflammation.

Hard exudation and cotton wool spots are associated with vascular diseases such as diabetes mellitus, hypertension, and collagen vascular diseases but can be seen with papilledema and other intrinsic ocular conditions. Inflammatory exudates are seen in patients with such diseases as sarcoidosis and toxoplasmosis.

Management and Disposition

Routine referral for ophthalmologic and medical workup is appropriate.

Pearl

1. Hard exudates that are intraretinal may easily be confused with drusen occurring near Bruch membrane, which separates the retina from the choroid.

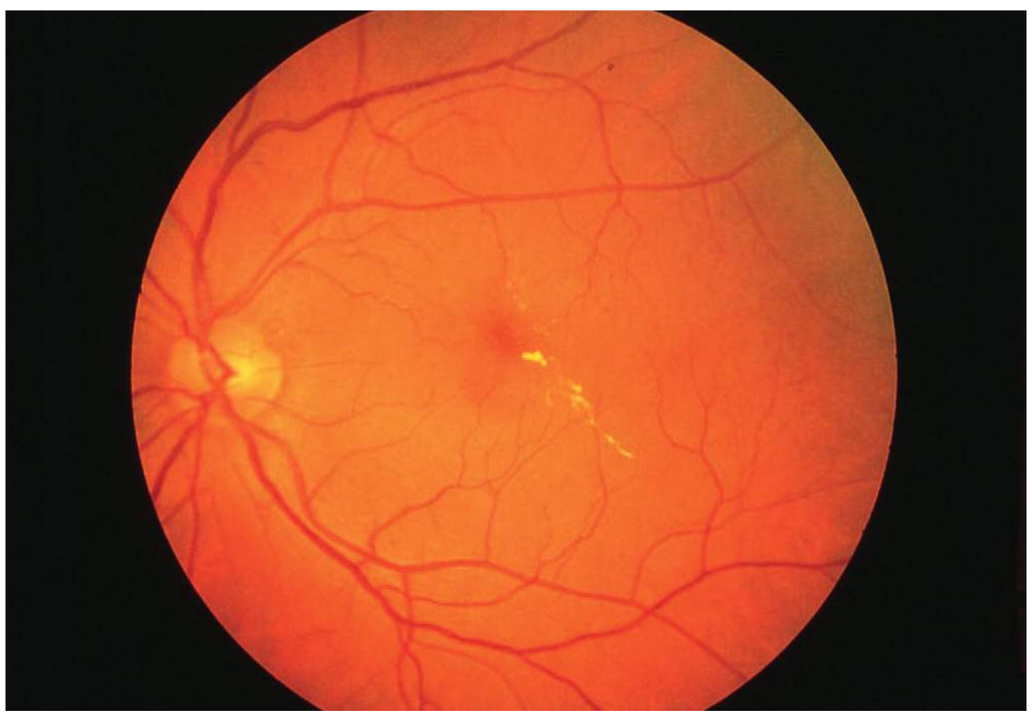

FIGURE 3.5 ■ **Hard Exudates.** Linear collection of yellow lipid deposits with sharp margins in macula. (Photo contributor: Beverly C. Forcier, MD.)

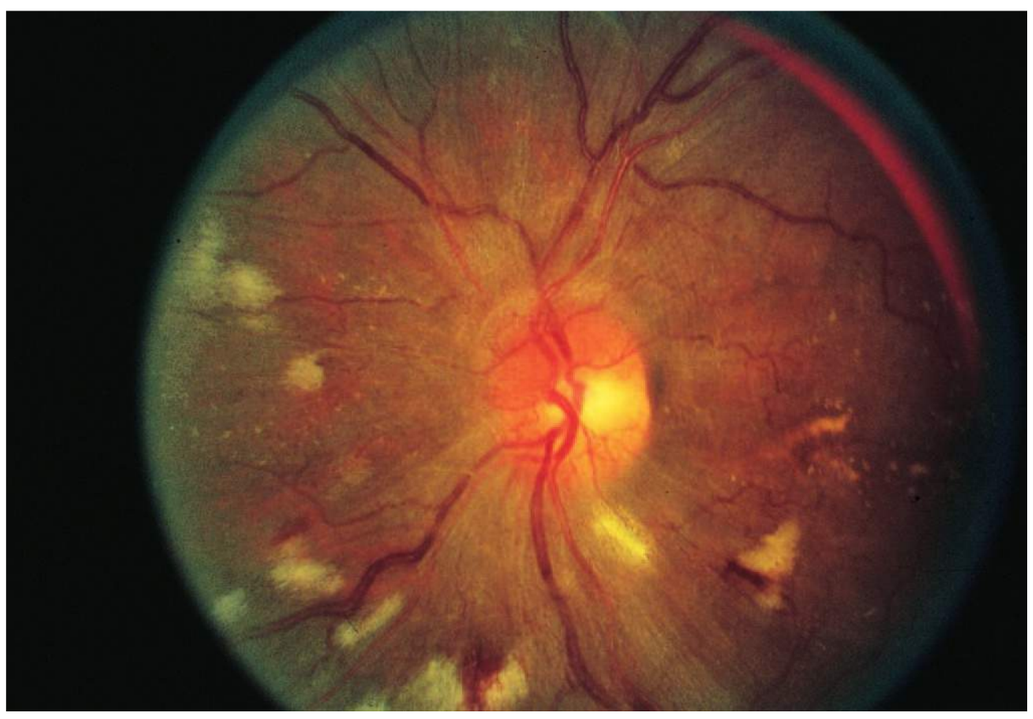

FIGURE 3.6 ■ **Cotton Wool Spots.** White lesions with fuzzy margins, seen here approximately one-fifth to one-fourth disk diameter in size. Orientation of cotton wool spots generally follows the curvilinear arrangement of the nerve fiber layer. Intraretinal hemorrhages and intraretinal vascular abnormalities are also present. (Photo contributor: Richard E. Wyszynski, MD.)

Clinical Summary

Roth spots are retinal hemorrhages with a white or yellow center. They may be seen in patients with a host of diseases such as anemia, leukemia, multiple myeloma, diabetes mellitus, collagen vascular disease, other vascular diseases, intracranial hemorrhage in infants, septic retinitis, and carcinoma. Flame-shaped or splinter hemorrhages or dot-blot hemorrhages may resemble Roth spots.

Management and Disposition

Routine referral for general medical evaluation is appropriate.

Pearls

1. Roth spots are not pathognomonic for any particular disease process and can represent a variety of clinical conditions.
2. These lesions represent red blood cells surrounding inflammatory cells.

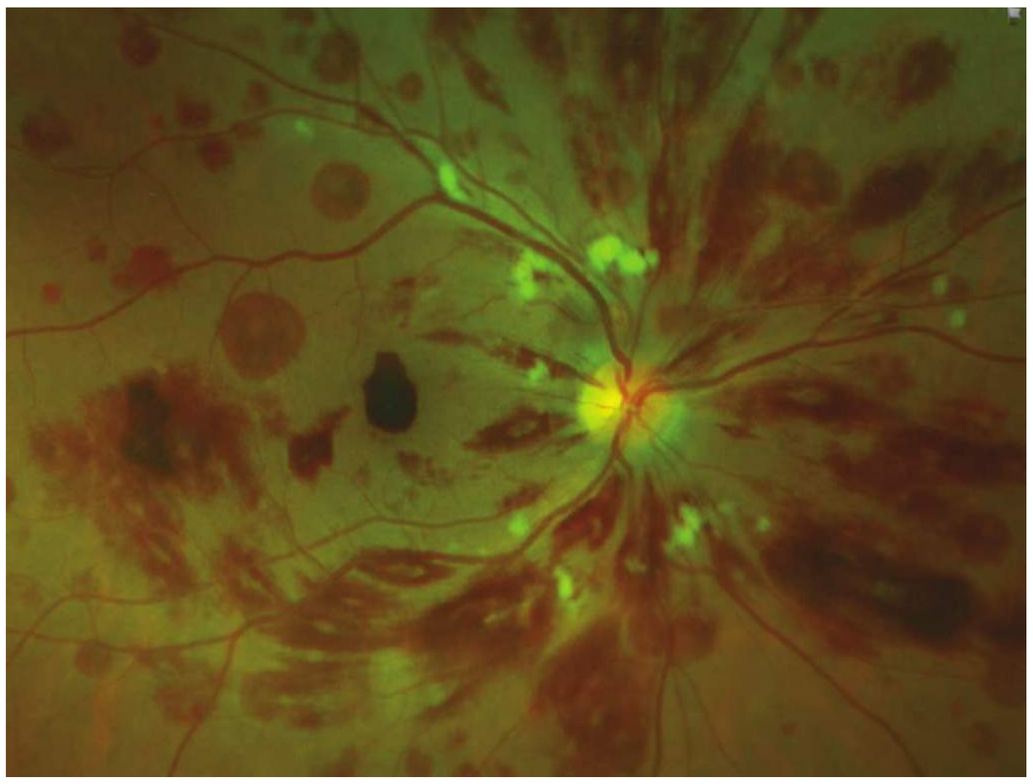

FIGURE 3.7 ■ **Roth Spots.** Multiple retinal hemorrhages with pale centers are seen. (Photo contributor: Tal Rubinstein, MD.)

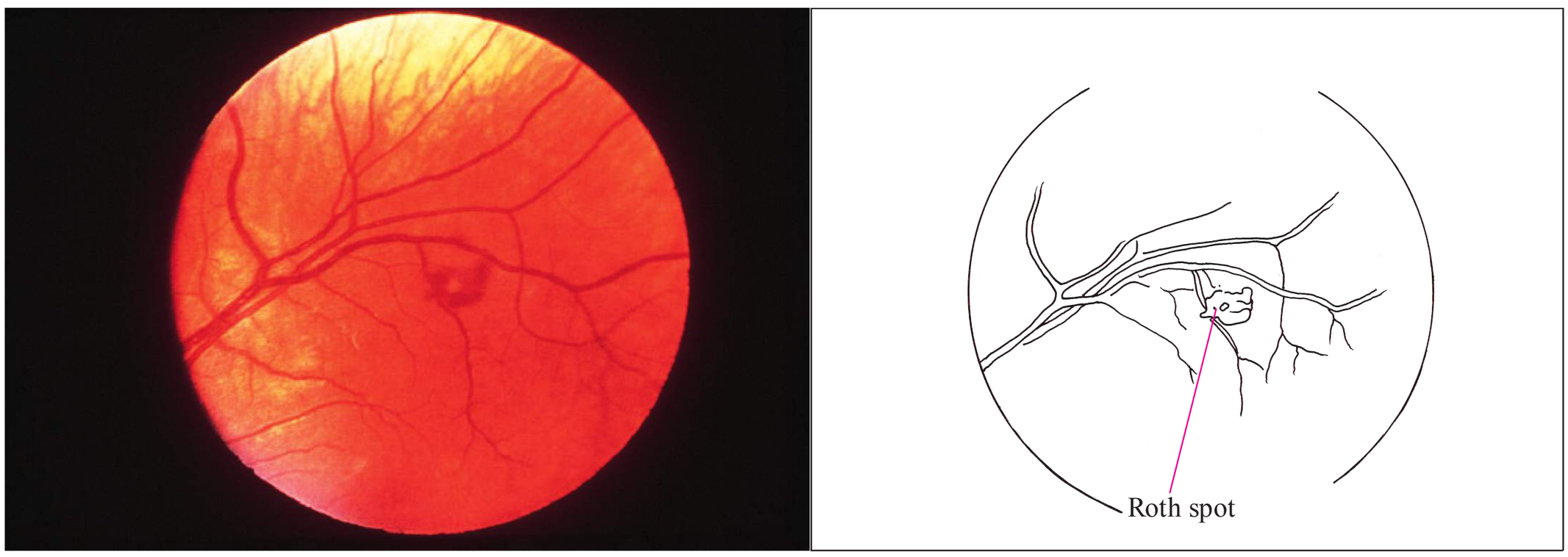

FIGURE 3.8 ■ **Roth Spot.** Retinal hemorrhage with pale center. (Photo contributor: William E. Cappaert, MD.)

Clinical Summary

Plaques, if present, are often found at arteriolar bifurcations. Patients may have signs and symptoms of vascular disease such as carotid bruits or stenosis, aortic stenosis, aneurysms, or atrial fibrillation. Amaurosis fugax, a transient loss of vision often described as a curtain of darkness obscuring vision with sight restoration within a few minutes, may be present in the history.

Cholesterol emboli (Hollenhorst plaques), associated with generalized atherosclerosis, often from carotid atheroma, are bright, highly refractile plaques. Platelet emboli (carotid artery or cardiac thrombus) are white and very difficult to visualize. Calcific emboli (cardiac valvular disease) are irregular and white or dull gray and much less refractile.

Management and Disposition

Referral for routine general medical evaluation is appropriate unless the patient presents with signs or symptoms consistent with showering of emboli, transient ischemic attack, or cerebrovascular accident, in which case admission should be considered.

Pearls

1. Retinal emboli may produce a loss of vision, either transient or permanent in nature.
2. Arteriolar occlusion may occur in either a central or a peripheral branch location.
3. Occurrence of retinal emboli should prompt the clinician to search for an embolic source as the event may be a precursor to impending ischemic stroke.

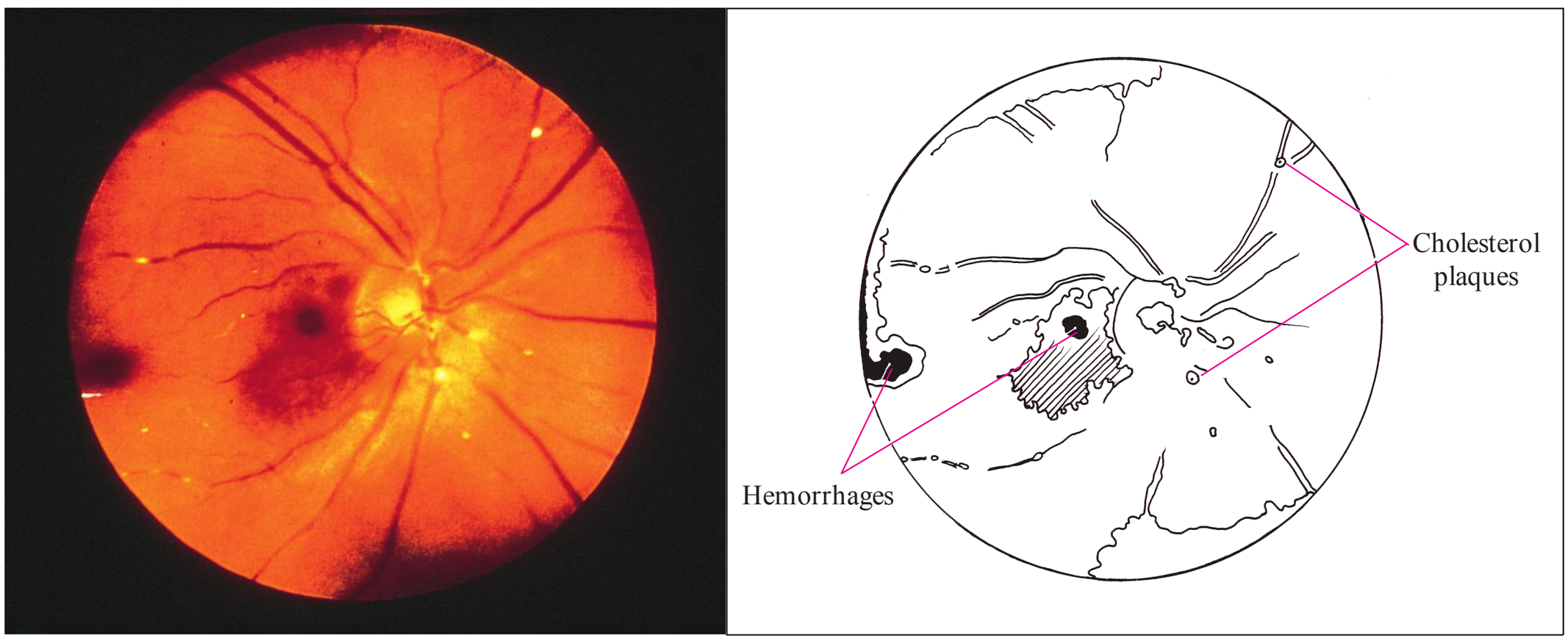

FIGURE 3.9 ■ **Emboli.** Refractile cholesterol plaques usually lodge at vessel bifurcations. (Photo contributor: William E. Cappaert, MD.)

Clinical Summary

In central retinal artery occlusion (CRAO), the typical patient experiences a sudden painless monocular loss of vision, either segmental or complete. Visual acuity may range from finger counting or light perception to complete blindness. Fundal findings may include the following: fundal paleness caused by retinal edema; the fovea does not have the edema and thus appears as a cherry-red spot; narrow and irregular retinal arterioles; and a "boxcar" appearance of the retinal venules.

Management and Disposition

Attempts to restore retinal blood flow may be beneficial if performed in a very narrow time window after the acute event. This may be accomplished by (1) decreasing intraocular pressure (IOP) with topical β-blocker eye drops or intravenous acetazolamide; (2) ocular massage, applied with cyclic pressure on the globe for 10 seconds, followed by release and then repeated. Urgent consultation with an ophthalmologist is indicated to determine if more aggressive acute therapy (paracentesis) is warranted. However, such aggressive treatment rarely alters the poor prognosis. Medical evaluation and treatment of associated findings may be warranted. Tissue plasminogen activator may be considered for lysis of an occluding thrombus.

Pearls

1. Visual decrement may be caused by a "low-flow" state (vs total occlusion). As this cannot be identified on presentation and can present hours later, immediate treatment and consultation is indicated regardless of the time of onset.
2. Sudden, painless monocular vision loss is typical.
3. CRAO may be associated with temporal arteritis. This diagnosis should be strongly considered in all patients presenting with signs and symptoms of CRAO who are older than 55 years.

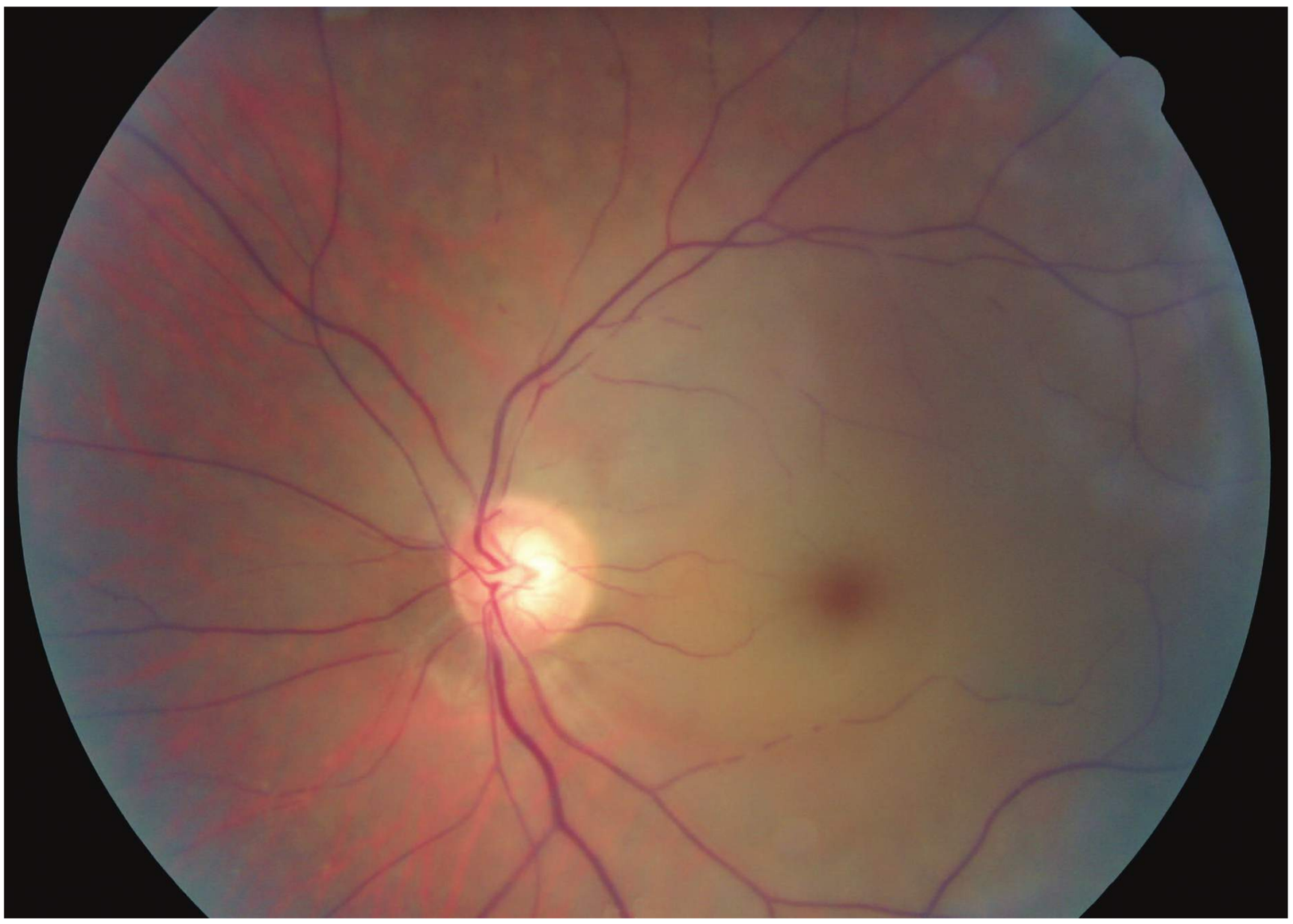

FIGURE 3.10 ■ **Central Retinal Artery Occlusion.** The retinal pallor caused by retinal edema is well demonstrated, contrasting with the "cherry-red spot" of the nonedematous fovea. Note the vascular narrowing and the "boxcar" appearance of the venules. (Photo contributor: Aaron Sobol, MD.)

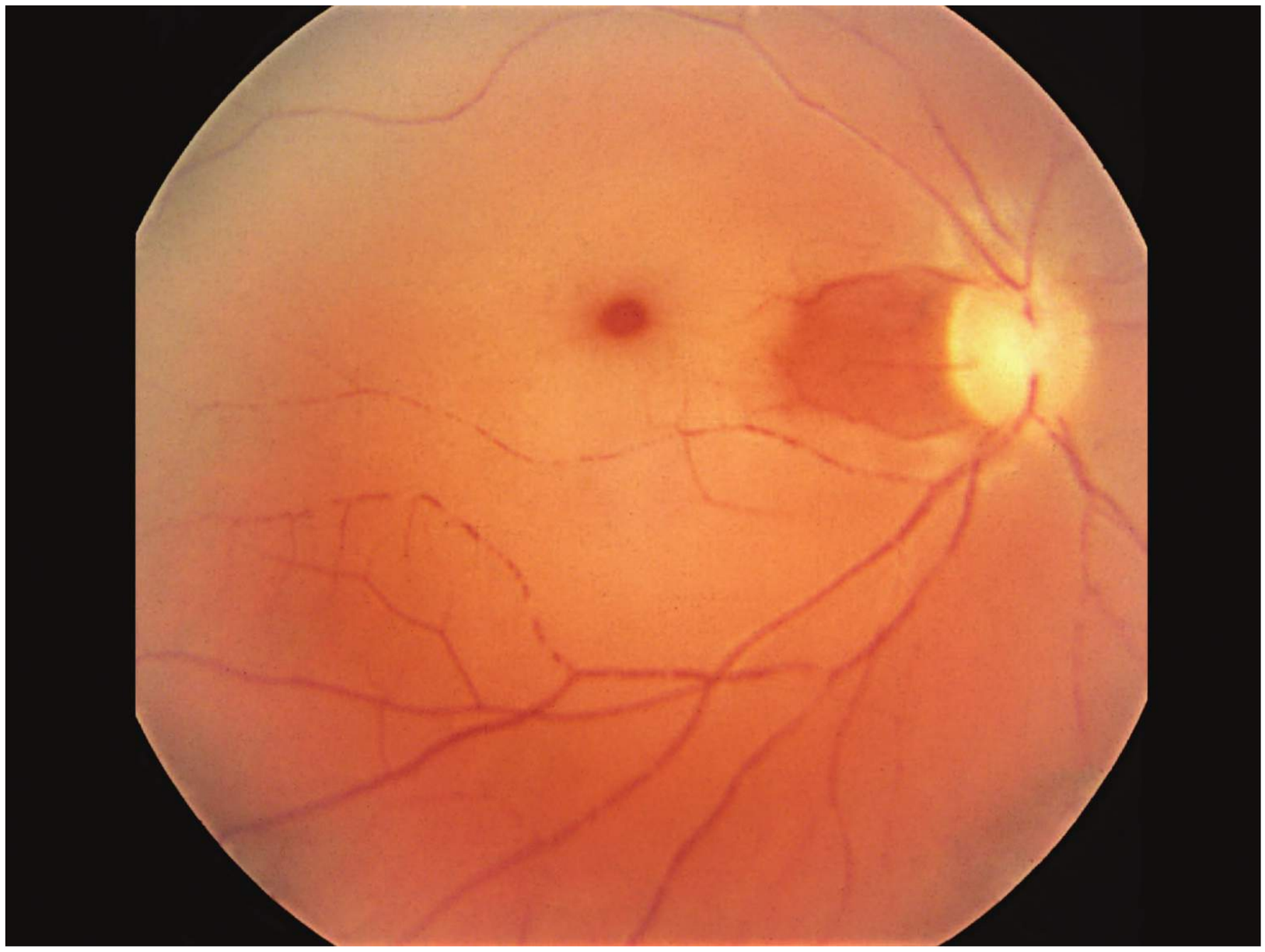

FIGURE 3.11 ■ **CRAO with Cilioretinal Artery Sparing**. "Hyperemia" of the fundus on the temporal side of the disc and sparing of the macular region owing to the presence of a patent cilioretinal artery. (Photo contributor: Thomas R. Hedges III, MD.)

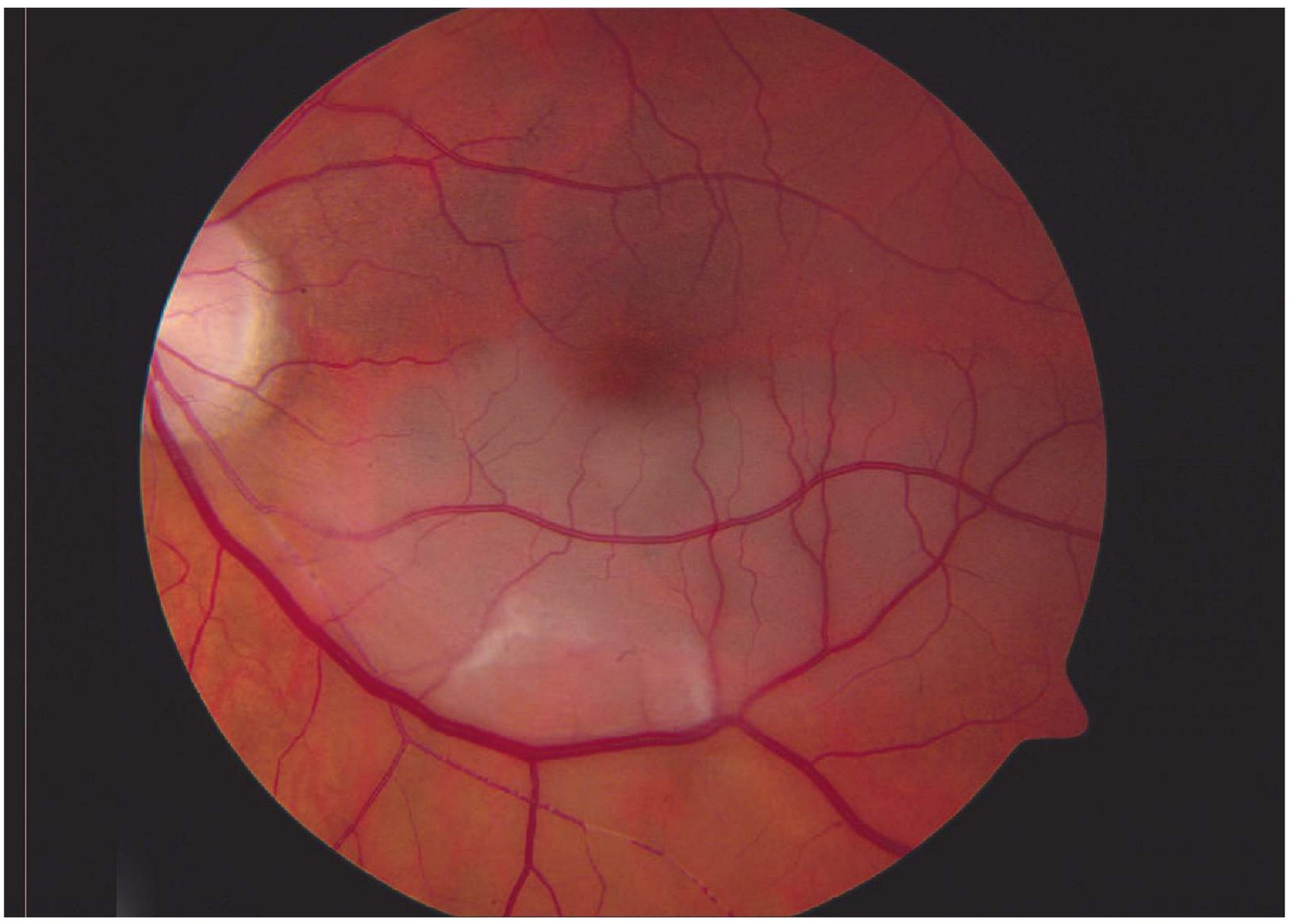

FIGURE 3.12 ■ **Branch Central Retinal Artery Occlusion.** Retinal whitening (owing to ischemia) and absence of blood flow in the infero-termporal branch retinal arteriole. (Photo contributor: Arun D. Singh, MD.)

Clinical Summary

Patients are usually older individuals and complain of sudden, painless visual loss in one eye. The vision loss is usually not as severe as CRAO and may vary from normal to hand motion. Funduscopy in a classic, ischemic central retinal vein occlusion (CRVO) shows a "blood and thunder" fundus: hemorrhages (including flame, dot, or blot, preretinal, and vitreous) and dilation and tortuosity of the venous system. The arterial system often shows narrowing. The disk margin may be blurred. Cotton wool spots and edema may be seen.

Management and Disposition

Treatment is rarely effective in preventing or reversing the damage done by the occlusion and is directed toward systemic evaluation to identify and treat contributing factors, hopefully decreasing the chance of contralateral CRVO. Ophthalmologic evaluation is necessary to confirm the diagnosis, estimate the amount of ischemia, and follow the patient so as to minimize sequelae of possible complications such as neovascularization and neovascular glaucoma.

Pearls

1. Sudden, painless visual loss in one eye should be evaluated promptly to determine its etiology.
2. Look for the classic "blood and thunder" funduscopic findings.
3. Consider the differential diagnosis of acute painful (glaucoma, retrobulbar neuritis) versus painless vision loss (CRAO, anterior ischemic optic neuropathy [AION], retinal detachment, subretinal neovascularization, and vitreous hemorrhage).

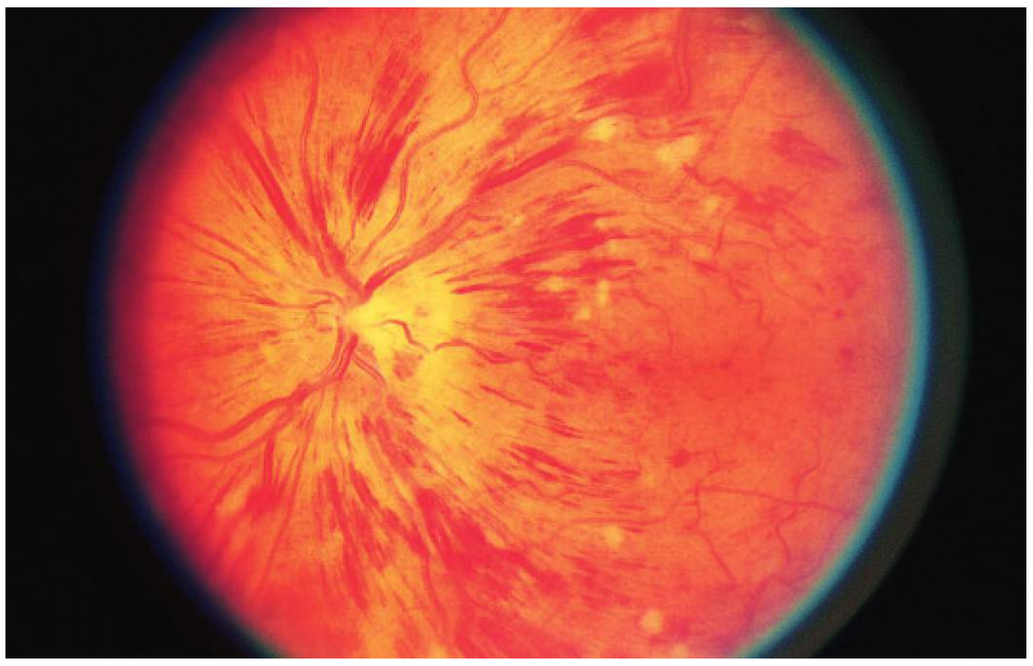

FIGURE 3.13 ■ **Central Retinal Vein Occlusion.** The amount of hemorrhage is the most striking feature in this photograph. Also note the blurred disk margin, the dilation and tortuosity of the venules, and the cotton wool spots. Retinal edema is suggested by blurring of the retinal details. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

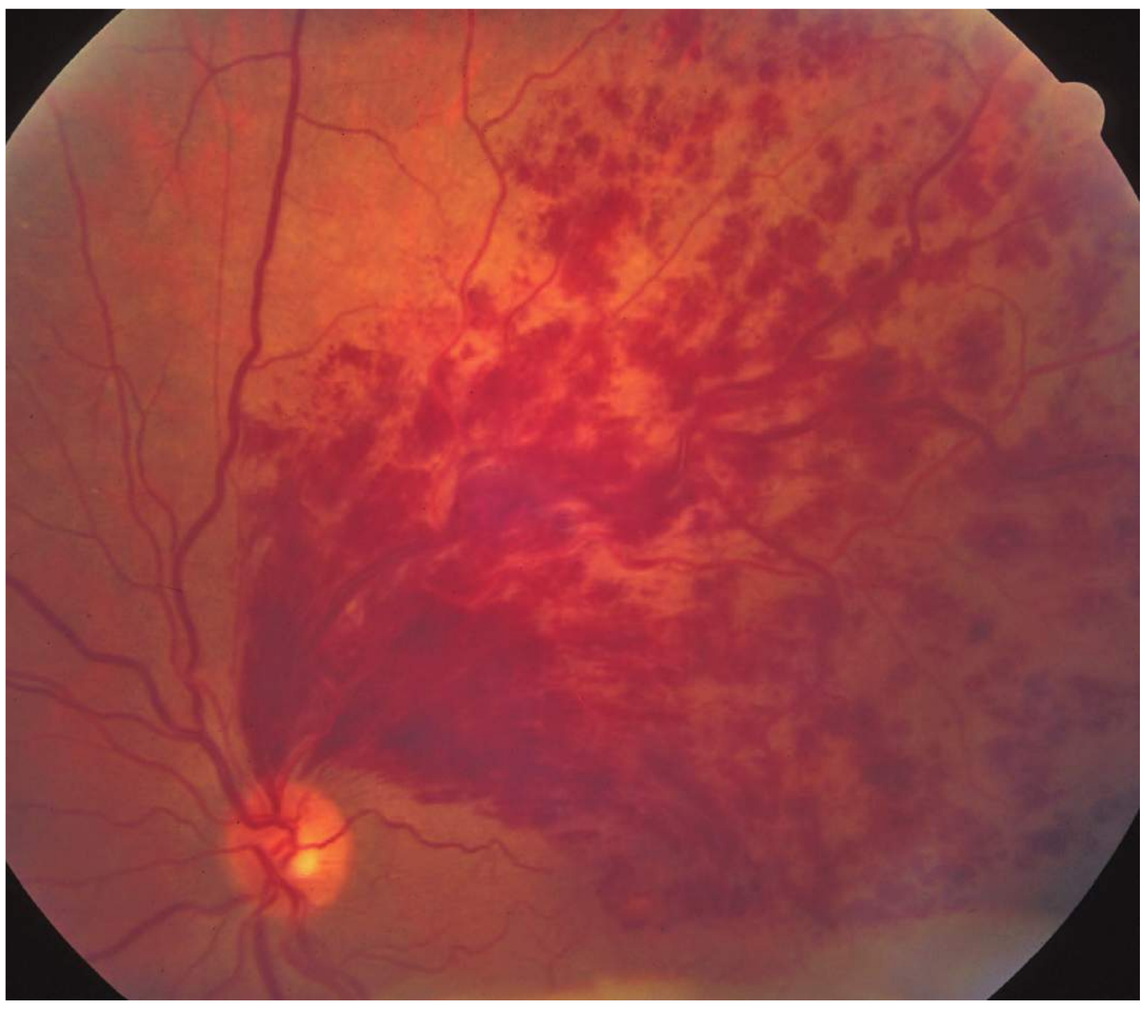

FIGURE 3.14 ■ **Central Retinal Vein Branch Occlusion.** The hemorrhage seen is limited to a sector of the fundus, indicating that a branch occlusion has occurred. There is less edema, and a large portion of the fundus is unaffected. (Photo contributor: Richard E. Wyszynski, MD.)

HYPERTENSIVE RETINOPATHY

Clinical Summary

Fundus changes that may be seen with hypertension include generalized and focal narrowing of arterioles, generalized arteriolar sclerosis (resembling copper or silver wiring), arteriovenous crossing changes, hemorrhages (usually flame-shaped), retinal edema and exudation, cotton wool spots, microaneurysms, and disk edema.

Diabetic retinopathy, many hematologic and vascular diseases, traumas, localized ocular pathology, and papilledema should all be considered.

Management and Disposition

The patient's hypertension should be appropriately treated, and a search for other end-organ damage should be considered. The patient should be referred for appropriate long-term blood pressure management.

Pearls

1. Hypertensive arteriolar findings may be reversible if organic changes have not occurred in the vessel walls.
2. Always consider hypertensive retinopathy in the differential diagnosis of papilledema.

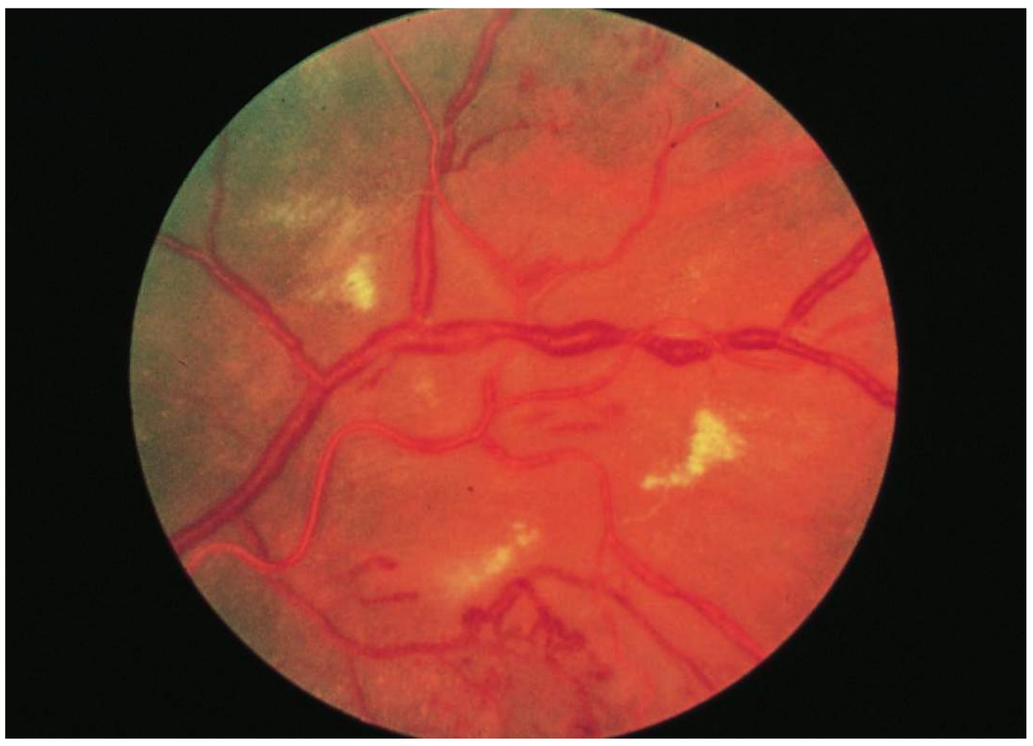

FIGURE 3.15 ■ **Hypertension.** Chronic, severe systemic hypertensive changes are demonstrated by hard exudates, increased vessel light reflexes, and sausage-shaped veins. (Photo contributor: Richard E. Wyszynski, MD.)

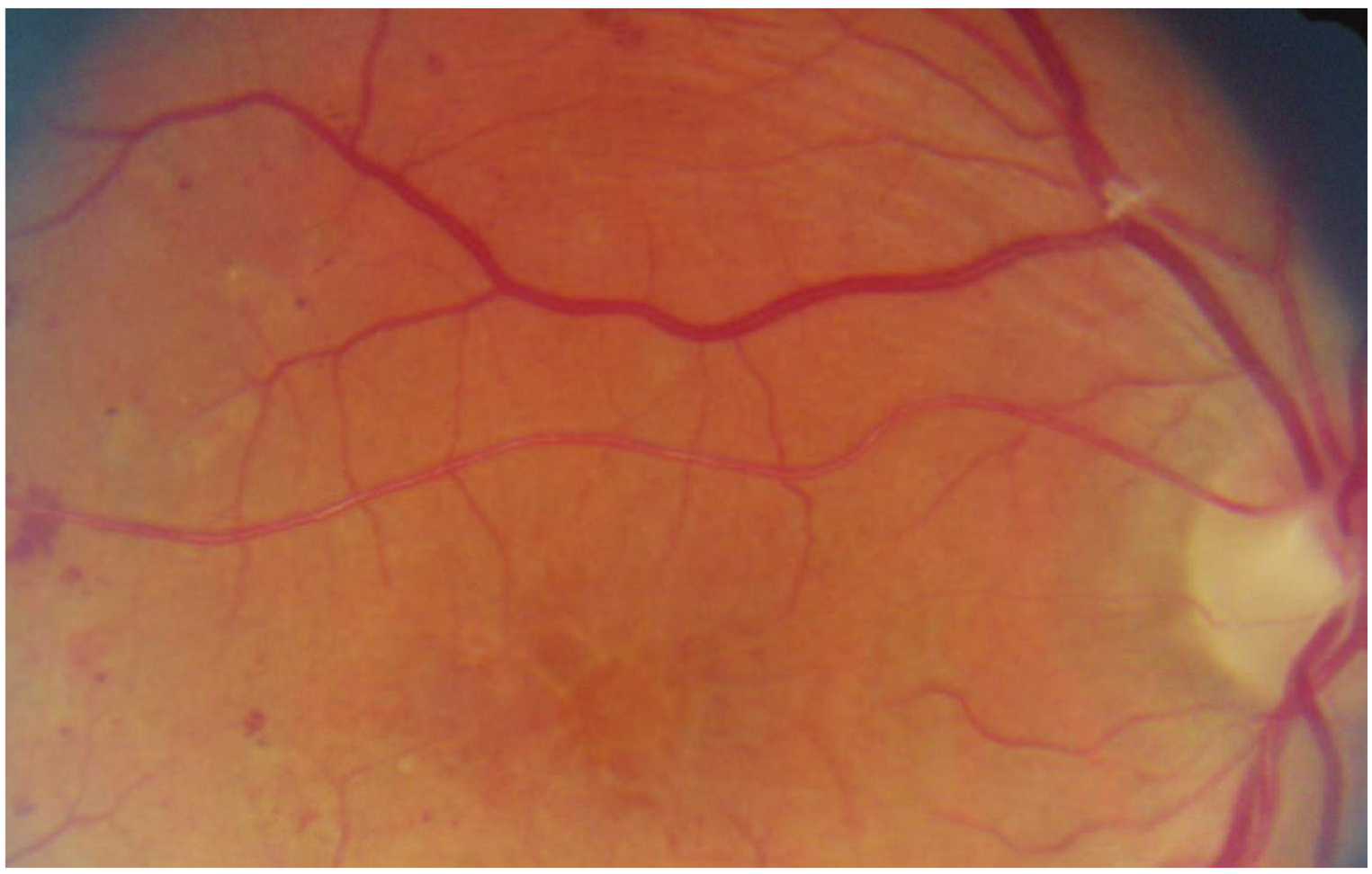

FIGURE 3.16 ■ **Copper and Silver Wiring.** Arteriolar changes seen in hypertensive retinopathy resemble copper (light reflex occupies most of the width) and silver (light reflex occupies the entire width of the arteriole) wiring. (Photo contributor: Aaron Sobol, MD.)

Clinical Summary

The early ocular manifestations of diabetes mellitus are referred to as background diabetic retinopathy (BDR). Fundus findings include flame or splinter hemorrhages (located in the superficial nerve fiber layer) or dot and blot hemorrhages (located deeper in the retina), hard exudates, retinal edema, and microaneurysms. If these signs are located in the macula, the patient's visual acuity may be decreased or at risk of becoming compromised, requiring laser treatment. Preproliferative diabetic retinopathy can show BDR changes plus cotton wool spots, intraretinal microvascular abnormalities, and venous beading. Proliferative diabetic retinopathy is demonstrated by neovascularization at the disk (NVD) or elsewhere (NVE). These require laser therapy owing to risk of severe visual loss from sequelae: vitreous hemorrhage, tractional retinal detachment, and severe glaucoma.

Many vascular and hematologic diseases—such as collagen vascular disease, sickle cell trait, hypertension, hypotension, anemia, leukemia, and inflammatory and infectious states—and ocular conditions can be associated with some or all of the above signs.

Management and Disposition

Routine ophthalmologic referral for laser or surgical treatment is indicated.

Pearls

1. Periodic ophthalmologic evaluations are recommended for all diabetic patients.
2. Microaneurysms typically appear 10 years after the initial onset of diabetes, although they may appear earlier in patients with juvenile diabetes.
3. Control of blood sugar alone does not prevent the development of retinopathy.
4. Blurred vision can also occur from acute increases in serum glucose, causing lens swelling and a refractive shift even in the absence of retinopathy.

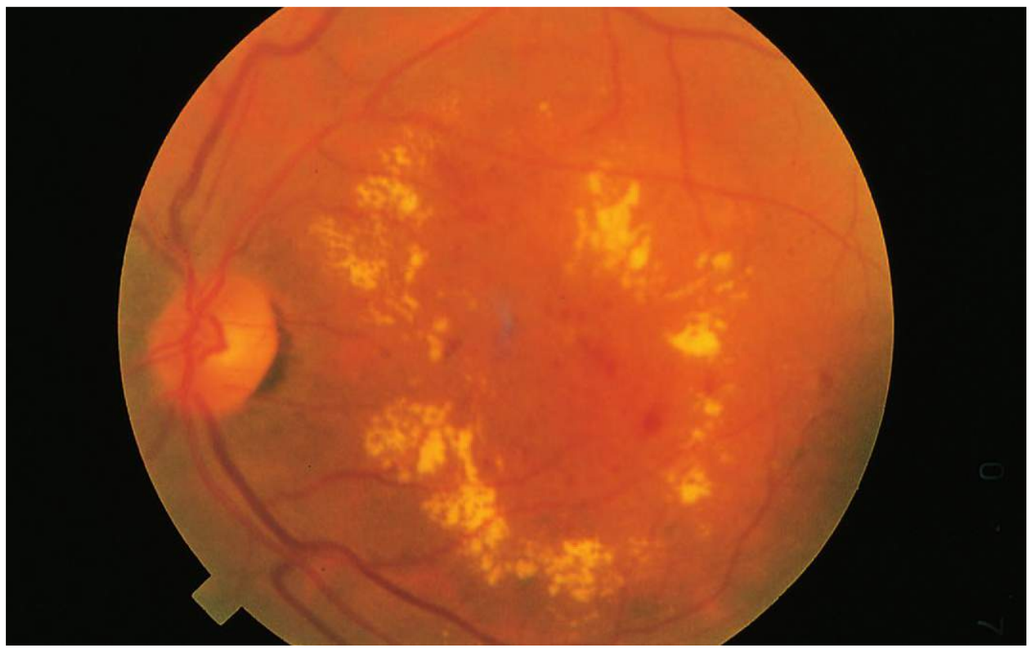

FIGURE 3.17 ■ **Background Diabetic Retinopathy.** An example of diabetic maculopathy with a typical circinate lipid ring. (Photo contributor: Richard E. Wyszynski, MD.)

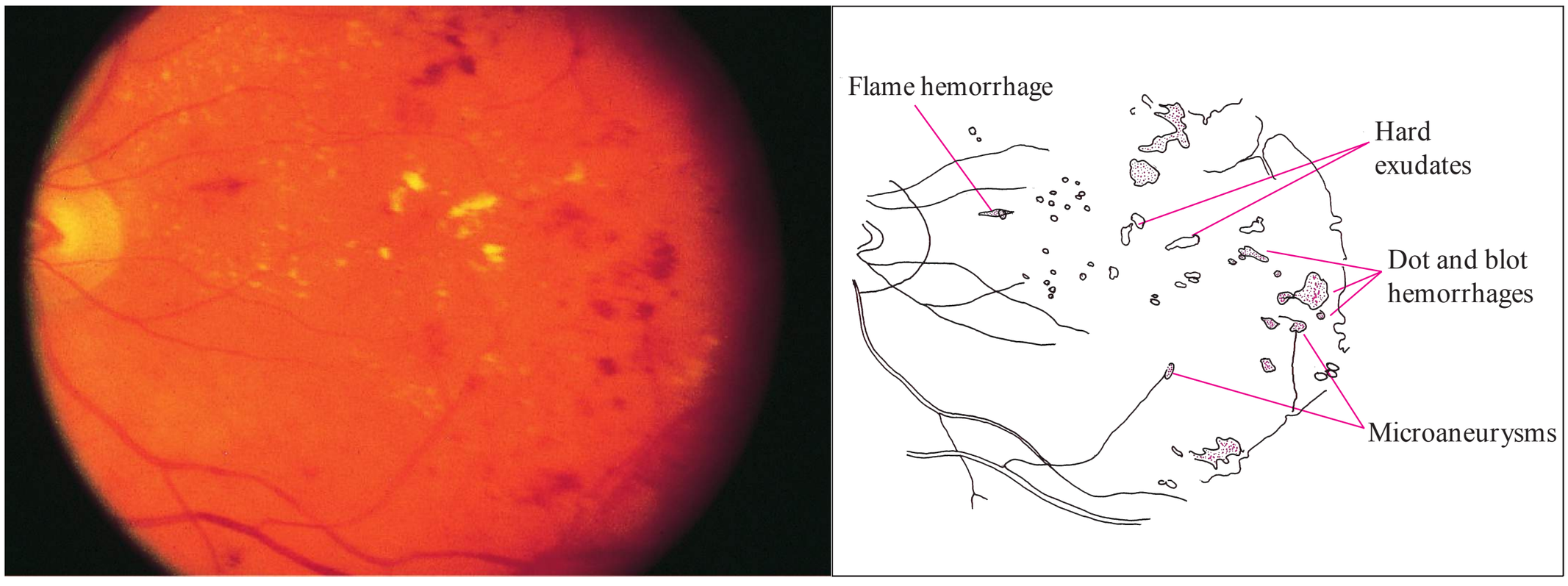

FIGURE 3.18 ■ **Background Diabetic Retinopathy.** Hard exudates, dot hemorrhages, blot hemorrhages, flame hemorrhages, and microaneurysms are present. Because these changes are located within the macula, this is classified as diabetic maculopathy. (Photo contributor: Richard E. Wyszynski, MD.)

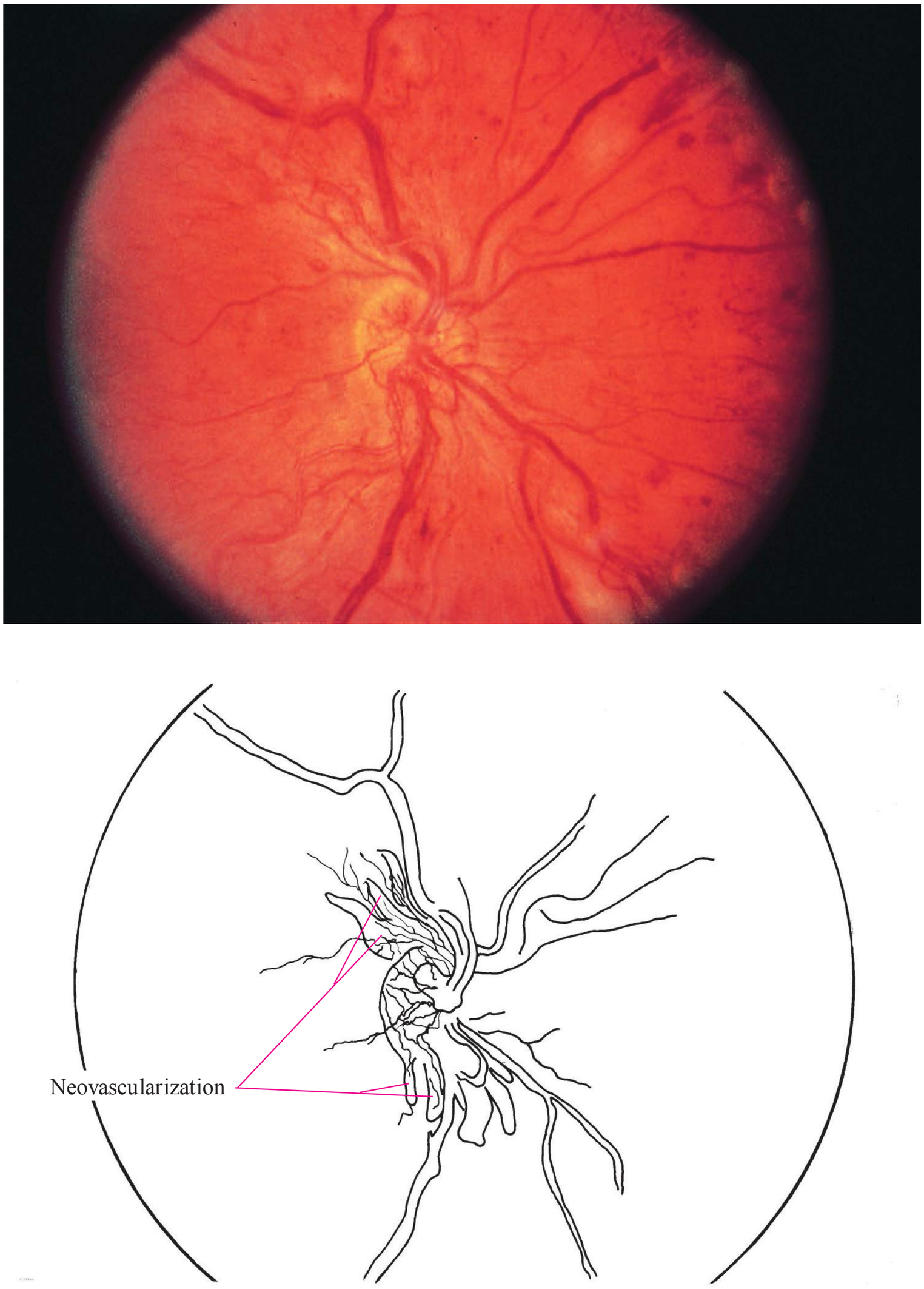

FIGURE 3.19 ■ **Proliferative Diabetic Retinopathy.** In addition to the signs seen in background and preproliferative diabetic retinopathy, neovascularization is seen here coming off the disk. (Photo contributor: Richard E. Wyszynski, MD.)

Clinical Summary

Patients may complain of floaters followed by the sudden loss or deterioration of vision in the affected eye, although bilateral hemorrhage can occur. The red reflex is diminished or absent, and the retina is obscured because of the bleeding. Large sheets or three-dimensional collections of red to red-black blood may be detected.

Multiple underlying etiologies include proliferative diabetic retinopathy, retinal or vitreous detachments, hematologic diseases, trauma (ocular or shaken impact syndrome), subarachnoid hemorrhage (SAH), collagen vascular disease, infections, macular degeneration, and tumors.

Management and Disposition

Refer to an ophthalmologist and an appropriate physician for associated conditions. Ophthalmic observation, photocoagulation, and surgery are all therapeutic options. Bed rest may help to increase visualization of the fundus.

Pearl

1. The patient's vision may improve somewhat after a period of sitting or standing as the blood layers out.

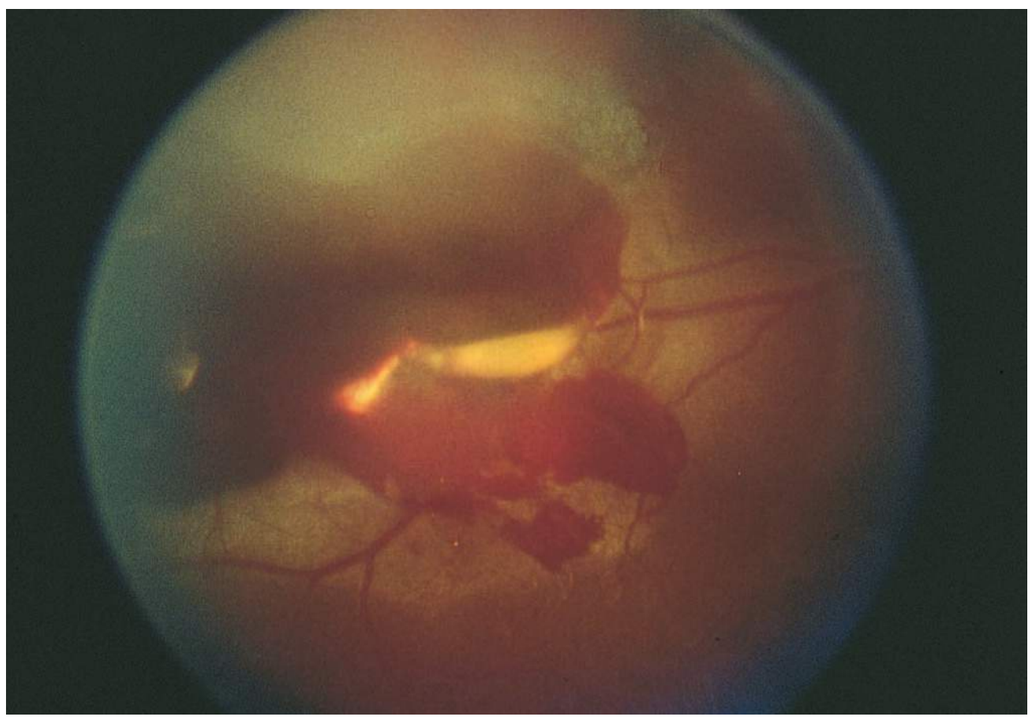

FIGURE 3.20 ■ **Vitreous Hemorrhage.** Large amount of vitreous hemorrhage associated with metallic intraocular foreign body. The large quantity of blood obscures visualization of retinal details. (Photo contributor: Richard E. Wyszynski, MD.)

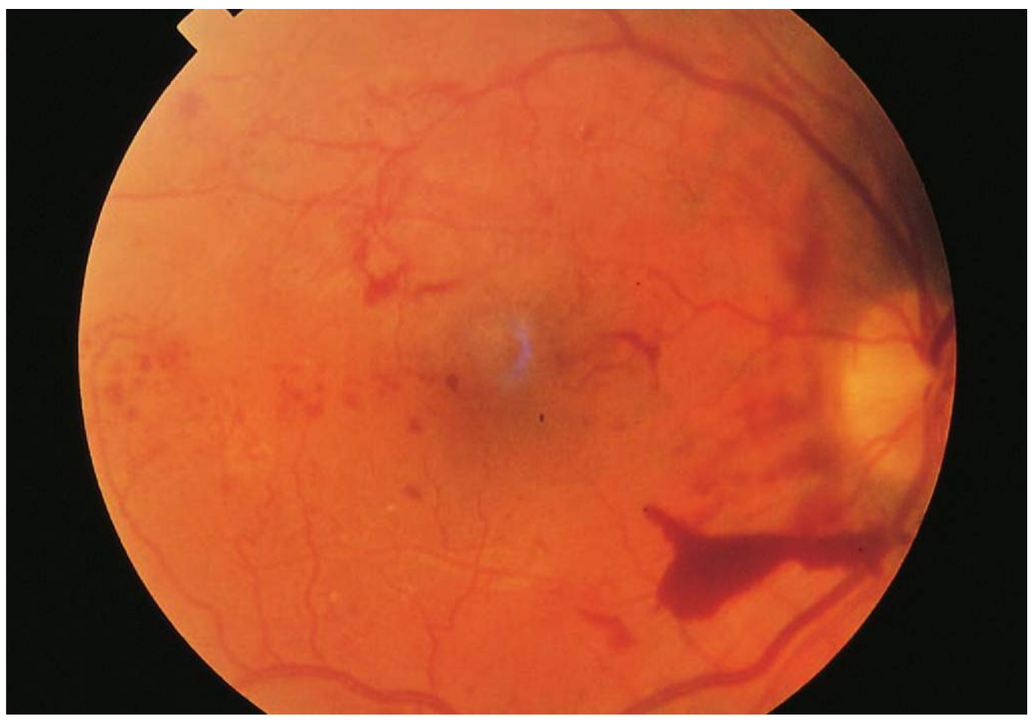

FIGURE 3.21 ■ **Vitreous Hemorrhage.** A smaller amount of vitreous hemorrhage is more easily photographed. Gravitational effect on the vitreous blood creates the appearance of a flat meniscus (keel-shaped blood) in this patient with vitreous hemorrhage associated with proliferative diabetic retinopathy. (Photo contributor: Richard E. Wyszynski, MD.)

RETINAL DETACHMENT

Clinical Summary

Patients often complain of monocular, decreased visual function and may describe a shadow or curtain descending over the eye. Other complaints include cloudy or smoky vision, floaters, or momentary flashes of light. Monocular visual field defects may be noted, and central visual acuity is diminished with macular involvement. Fundal examination may reveal a billowing or tentlike elevation of retina compared with adjacent areas. The elevated retina often appears gray. Retinal holes and tears may be seen, but often the holes, tears, and retinal detachment cannot be seen without indirect ophthalmoscopy.

Retinal detachments caused by retinal tears or holes can be associated with trauma, previous ocular surgery, nearsightedness, family history of retinal detachment, and Marfan disease. Retinal detachments caused by traction on the retina by an intraocular process can be because of systemic influences in the eye, such as diabetes mellitus or sickle cell trait. Occasionally retinal detachments are caused by tumors or exudative processes that elevate the retina. Symptoms of "light flashes" may occur with vitreous changes in the absence of retinal pathology. Patients may note flashes of light occurring only in a darkened environment because of the mechanical stimulation of the retina from the extraocular muscles, usually in a nearsighted individual.

Management and Disposition

Urgent ophthalmologic evaluation and treatment are warranted.

Pearls

1. Often patients have had sensation of flashes of light that occur in a certain area of a visual field in one eye, corresponding to the pathologic pulling on the corresponding retina.
2. Visual loss may be gradual or sudden.

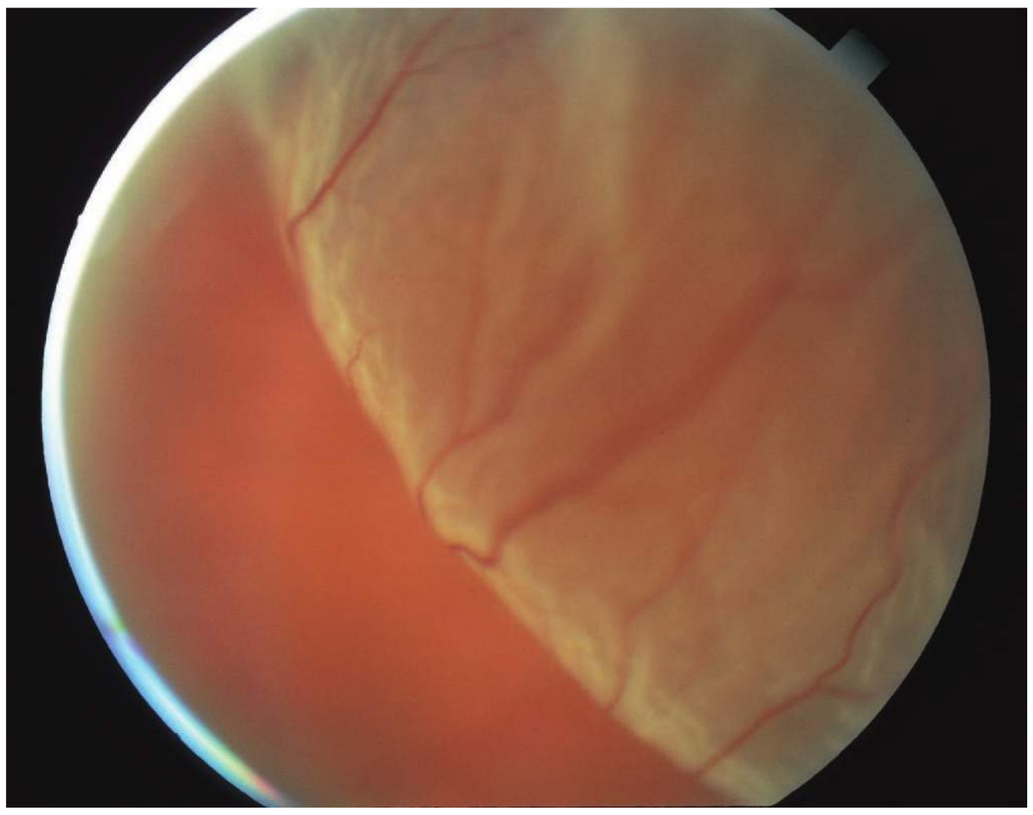

FIGURE 3.22 ■ **Retinal Detachment**. A fold of detached retina is seen drooping well in front of the posterior pole giving the appearance of a "three-dimensional" fundus. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

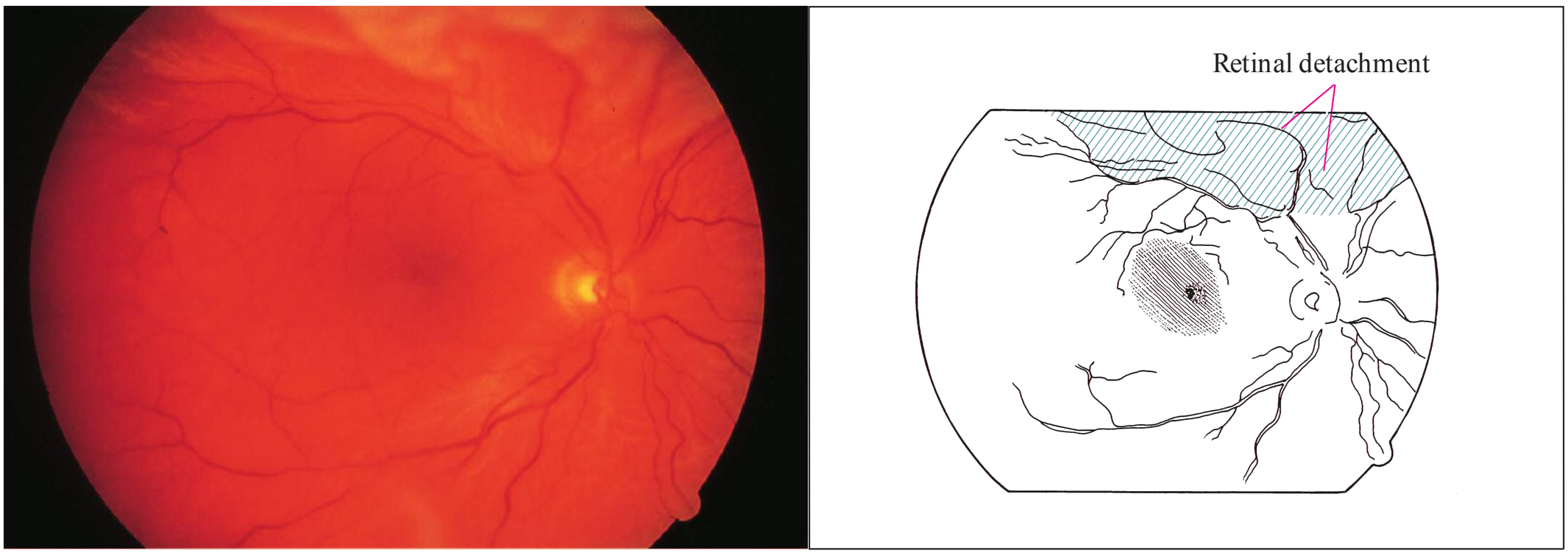

FIGURE 3.23 ■ **Retinal Detachment.** Undulating, out-of-focus, elevated retina is seen with few vessels in focus. (Photo contributor: Richard E. Wyszynski, MD.)

Clinical Summary

Patients may complain of the gradual and usually painless onset of the following visual sensations: floaters, scintillating scotomas (quivering blind spots), decreased peripheral visual field, and metamorphopsia (wavy distortion of vision). Cytomegalovirus (CMV) infiltrates appear as focal, small (but may be larger, confluent) white lesions in the retina that look like cotton wool spots. CMV is a necrotizing virus that is spread hematogenously, so that damage is concentrated in the retina adjacent to the major vessels and the optic disk. Often hemorrhage is involved with significant retinal necrosis (dirty white with a granular appearance), giving the "pizza pie" or "cheese and ketchup" appearance. Optic nerve involvement and retinal detachments can be present.

The differential includes other infections such as toxoplasmosis, other herpesviruses, syphilis, and occasionally other opportunistic infections.

Management and Disposition

Reversal, if possible, of immunosuppression; antiviral agents have been used effectively to threat this condition.

Pearls

1. HIV retinopathy consists of scattered retinal hemorrhages and scattered, multiple cotton wool spots that resolve over time, whereas CMV lesions will typically progress.
2. Although exposure to the CMV virus is widespread, the virus rarely produces a clinically recognized disease in nonimmunosuppressed individuals.

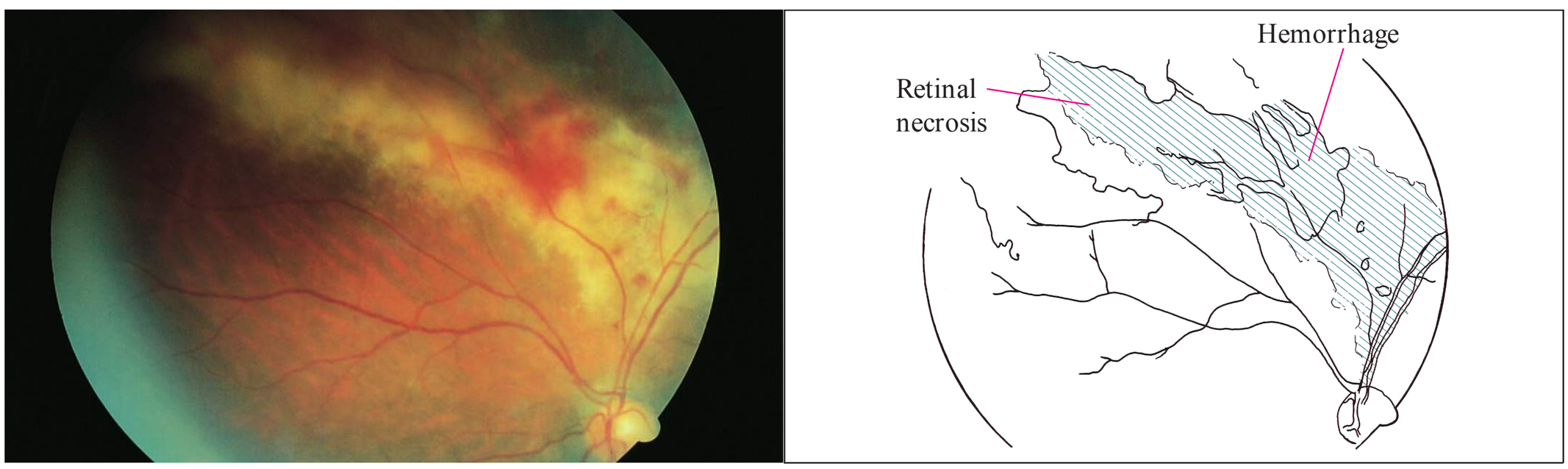

FIGURE 3.24 ■ **CMV Retinitis.** "Pizza pie" or "cheese and ketchup" appearance is demonstrated by hemorrhages and the dirty, white, granular-appearing retinal necrosis adjacent to major vessels. (Photo contributor: Richard E. Wyszynski, MD.)

Clinical Summary

Papilledema involves swelling of the optic nerve head, usually in association with elevated intracranial pressure. The optic disks are hyperemic with blurred disk margins; the venules are dilated and tortuous. The optic cup may be obscured by the swollen disk. There may be flame hemorrhages and infarctions (white, indistinct cotton wool spots) in the nerve fiber layer and edema in the surrounding retina.

Ocular inflammation (eg, papillitis), tumors or trauma, central retinal artery or vein occlusion, optic nerve drusen, and marked hyperopia may present with similar findings.

Management and Disposition

Expeditious ophthalmologic and medical evaluation is warranted.

Pearls

1. The top of a swollen disk and the surrounding unaffected retina will not both be in focus on the same setting on direct ophthalmoscopy.
2. Papilledema is a bilateral process, though it may be slightly asymmetric. A unilateral swollen disk suggests a localized ocular or orbital process.
3. Vision is usually normal acutely, though the patient may complain of transient visual changes. The blind spot is usually enlarged.
4. Diplopia from sixth cranial nerve palsy can be associated with increased intracranial pressure and papilledema.

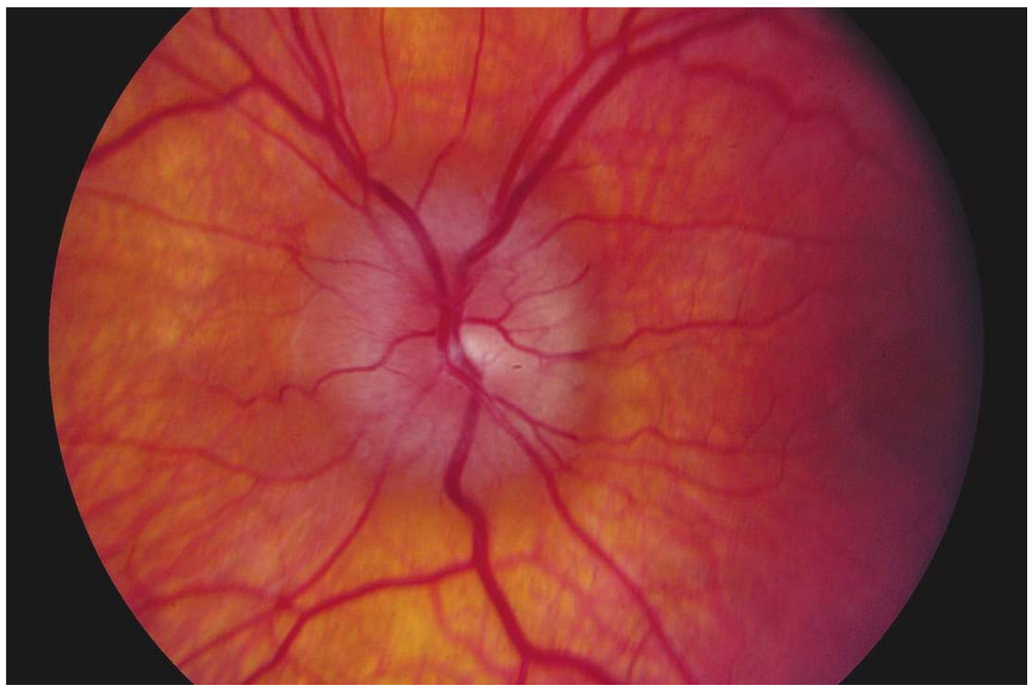

FIGURE 3.25 ■ **Papilledema.** Note blurred disk margins and congested disk. (Photo contributor: Arun D. Singh, MD.)

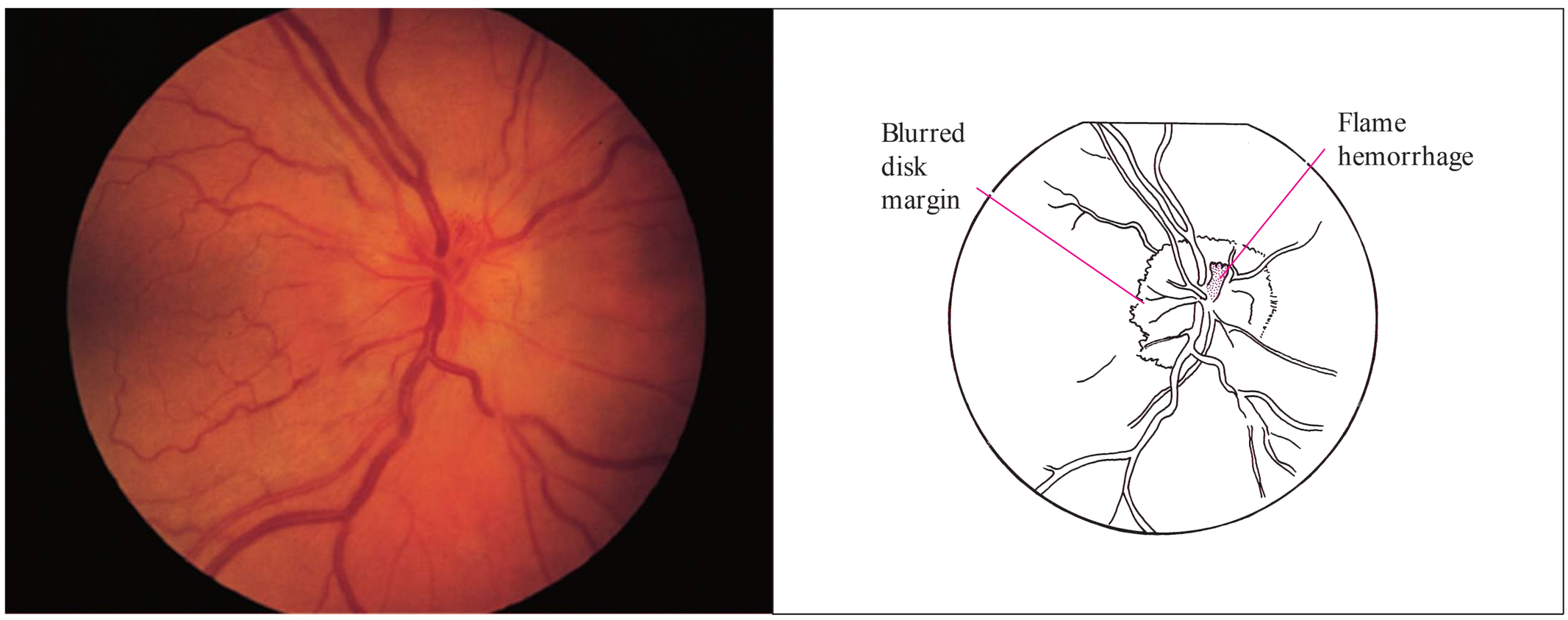

FIGURE 3.26 ■ **Papilledema.** Disk is hyperemic and swollen with loss of sharp margins. The venules are dilated and tortuous. The cup is obscured. A small flame hemorrhage is seen at 12- to 1-o'clock position on the disk margin. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

Clinical Summary

Most cases of optic neuritis are retrobulbar and involve no changes in the fundus, or optic disk, during the acute episode. With time, variable optic disk pallor may develop. Typical retrobulbar optic neuritis presents with sudden or rapidly progressing monocular vision loss in patients younger than 50 years. There is a central visual field defect that may extend to the blind spot. Pain on movement of the globe is common. The pupillary light response is diminished in the affected eye. Over time the vision improves partially or completely; minimal or severe optic atrophy may develop. Papillitis, inflammation of the intraocular portion of the optic nerve, will accompany disk swelling, with a few flame hemorrhages and possible cells in the vitreous.

Optic neuritis must be differentiated from papilledema (bilateral disk swelling, typically with no acute visual loss with the exception of transient visual changes), ischemic neuropathy (pale, swollen disk in an older individual with sudden monocular vision loss), tumors, and metabolic or endocrine disorders. Most cases of optic neuritis are of unknown etiology. Some known causes of optic neuritis include demyelinating disease, infections (including viral, syphilis, tuberculosis, sarcoidosis), or inflammations from contiguous structures (sinuses, meninges, orbit).

Management and Disposition

Treatment is controversial; often none is recommended. Oral steroids may worsen prognosis in certain cases. Intravenous steroids should be considered after consultation with an ophthalmologist.

Pearls

1. Monocular vision loss with pain on palpation of the globe or with eye movement and decreased color vision and color desaturation with loss of contrast are clinical clues to the diagnosis.
2. Sudden or rapidly progressing central vision loss is characteristic.
3. Most cases of acute optic neuritis are retrobulbar. Thus, ophthalmoscopy shows a normal fundus.
4. Suspect temporal arteritis in older patients.

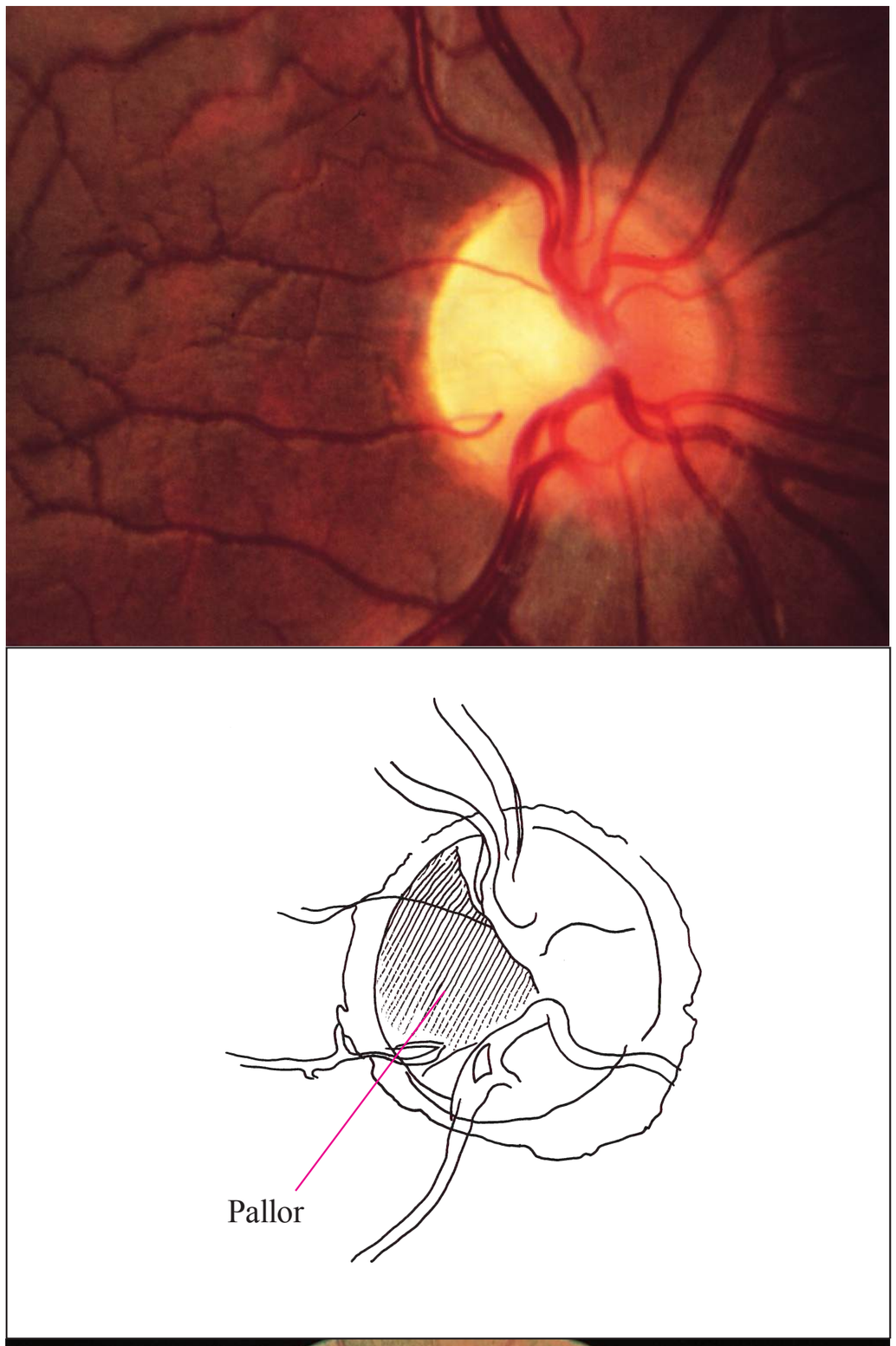

FIGURE 3.27 ■ **Optic Nerve Pallor.** Optic nerve pallor, either segmental (top) or generalized (bottom), is a nonspecific change that may be associated with a previous episode of optic neuritis or other insults to the optic nerve. (Photo contributor: Richard E. Wyszynski, MD.)

ANTERIOR ISCHEMIC OPTIC NEUROPATHY

Clinical Summary

Anterior ischemic optic neuropathy (AION) presents with a sudden loss of visual field (often altitudinal), usually involving fixation, in an older individual. The loss is usually stable after onset, with no improvement, and only occasionally, progressive over several days to weeks. Pale disk swelling is present involving a sector or the full disk, with accompanying flame hemorrhages. The cup to disc ratio is typically small (0.1-0.2) bilaterally.

The common, nonarteritic causes of AION (probably arteriosclerosis) need to be differentiated from arteritic ones, such as giant cell arteritis. If untreated, the latter will involve the other eye in 75% of cases, often in a few days to weeks. These elderly individuals often have weight loss, masseter claudication, weakness, myalgias, elevated sedimentation rate, and painful scalp, temples, or forehead.

Management and Disposition

Routine ophthalmologic and medical evaluation is appropriate.

Pearls

1. Consider AION in an elderly patient with sudden, usually painless, visual field loss.
2. Rule out giant cell arteritis. These patients tend to be older (age > 55 years) and may have associated CRAO or cranial nerve palsies (III, IV, or VI) with diplopia.

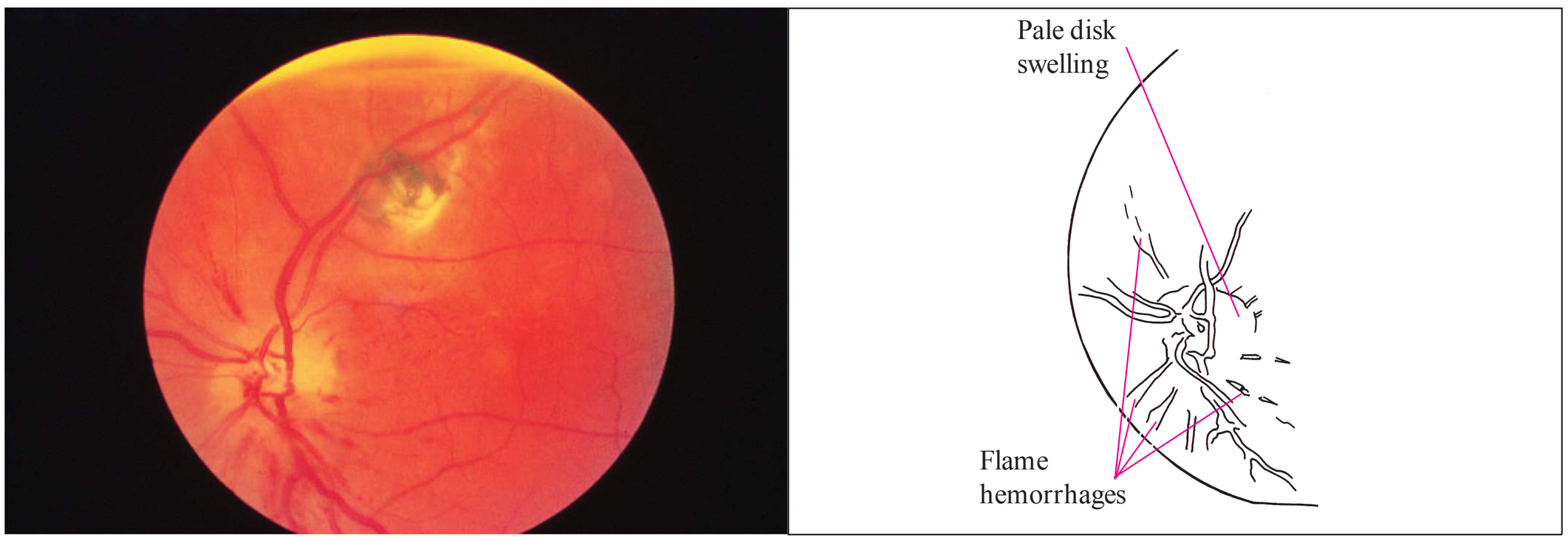

FIGURE 3.28 ■ **Anterior Ischemic Optic Neuropathy.** Pale disk swelling and flame hemorrhages are present. This patient also has an unrelated retinal scar owing to toxoplasmosis. (Photo contributor: William E. Cappaert, MD.)

Clinical Summary

Acute narrow or closed-angle glaucoma (ACG) results from a physical impedance of aqueous humor outflow. Symptoms range from colored halos around lights and blurred vision to severe pain (described as a headache or brow ache) with nausea and vomiting. IOP is markedly elevated. Perilimbal vessels are injected, the pupil is mid-dilated and poorly reactive to light, and the cornea may be hazy and edematous.

Two-thirds of glaucoma patients have open-angle glaucoma. Often they are asymptomatic. They may have a family history of glaucoma. Funduscopy may show asymmetric cupping of the optic nerves. The optic nerve may show notching, local thinning of tissue, or disk hemorrhage. Optic cups enlarge, especially vertically, with progressive damage. Tissue loss is associated with visual field abnormalities. The IOP is often but not always greater than 21 mm Hg.

Management and Disposition

Acute ACG requires emergent ophthalmologic consultation and administration of medications to decrease IOP such as β-blocker drops (timolol), carbonic anhydrase inhibitors (acetazolamide), cholinergic-stimulating drops (pilocarpine), hyperosmotic agents (osmoglyn), and α-adrenergic agonists (apraclonidine). Open-angle glaucoma is treated with long-term ophthalmic evaluation and treatment with medications and laser or surgery.

Pearls

1. Nausea, vomiting, and headache may obscure the diagnosis. Use digital globe palpation routinely in patients with these complaints.
2. Open-angle glaucoma usually causes no symptoms other than gradual loss of vision.
3. Congenital glaucoma is rare. However, because of prognosis if diagnosis is delayed, consider congenital glaucoma in infants and children with tearing, photophobia, enlarged eyes, or cloudy corneas.
4. Asymmetric cupping, enlarged cups, and elevated IOP are hallmarks of open-angle glaucoma.

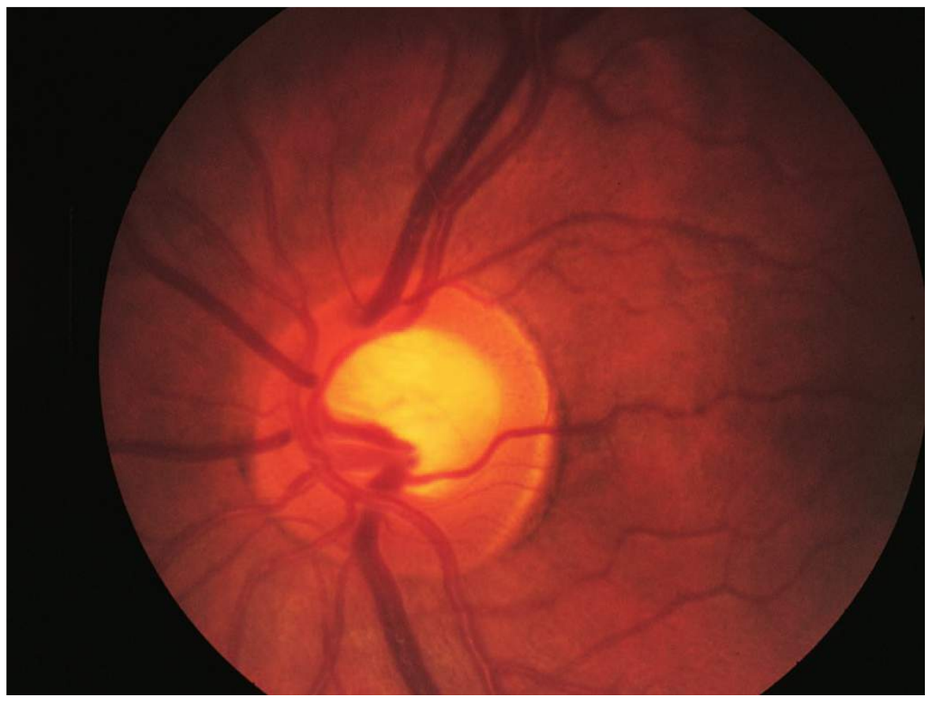

FIGURE 3.30 ■ **Glaucomatous Cupping.** The cup is not central; it is elongated toward the rim superotemporally. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

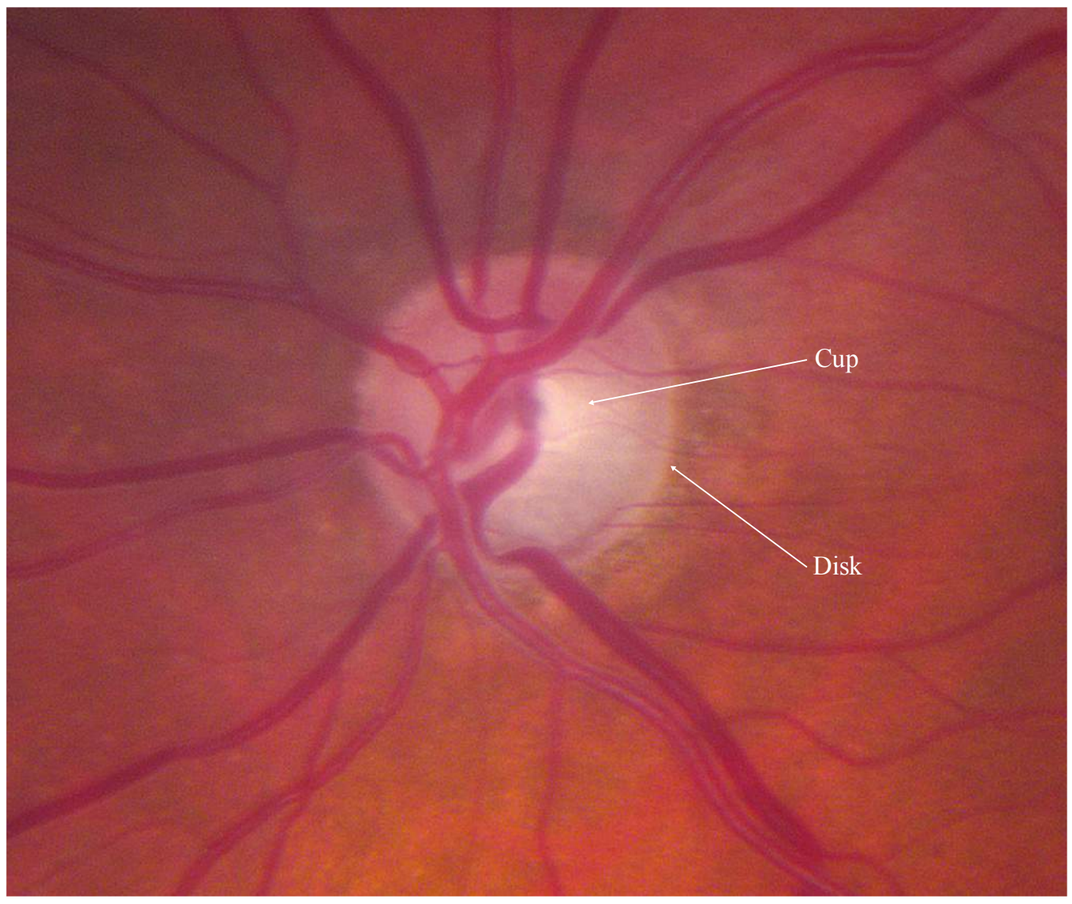

FIGURE 3.29 ■ **Normal Cupping.** The cup is central and is less than one half the diameter of the disc. (Photo contributor: Jeffrey Goshe, MD.)

Clinical Summary

Subhyaloid hemorrhage appears as extravasated blood beneath the retinal layer. These are often described as "boat-shaped" hemorrhages to distinguish them from the "flame-shaped" hemorrhages on the superficial nerve fiber layer of the retina. They may occur as a result of blunt trauma but are perhaps best known as a marker for subarachnoid hemorrhage (SAH). In SAH, the hemorrhages appear as a "puff" of blood emanating from the central disk.

SAH, shaken impact syndrome, hypertensive retinopathy, and retinal hemorrhage should all be considered and aggressively evaluated.

Management and Disposition

No specific treatment is required for subhyaloid hemorrhage. Treatment is dependent on the underlying etiology. Appropriate specialty referral should be made in all cases.

Pearls

1. A funduscopic examination looking for subhyaloid hemorrhage should be included in all patients with severe headache, unresponsive pediatric patients, or those with altered mental status.
2. The appearance of a retinal hemorrhage indicates its anatomic location. A subhyaloid hemorrhage lies over the retinal vessels—thus, they cannot be seen on funduscopic examination. In a subretinal hemorrhage (see Fig. 3.4), the vessels lie superficial to the hemorrhage and thus are easily seen.

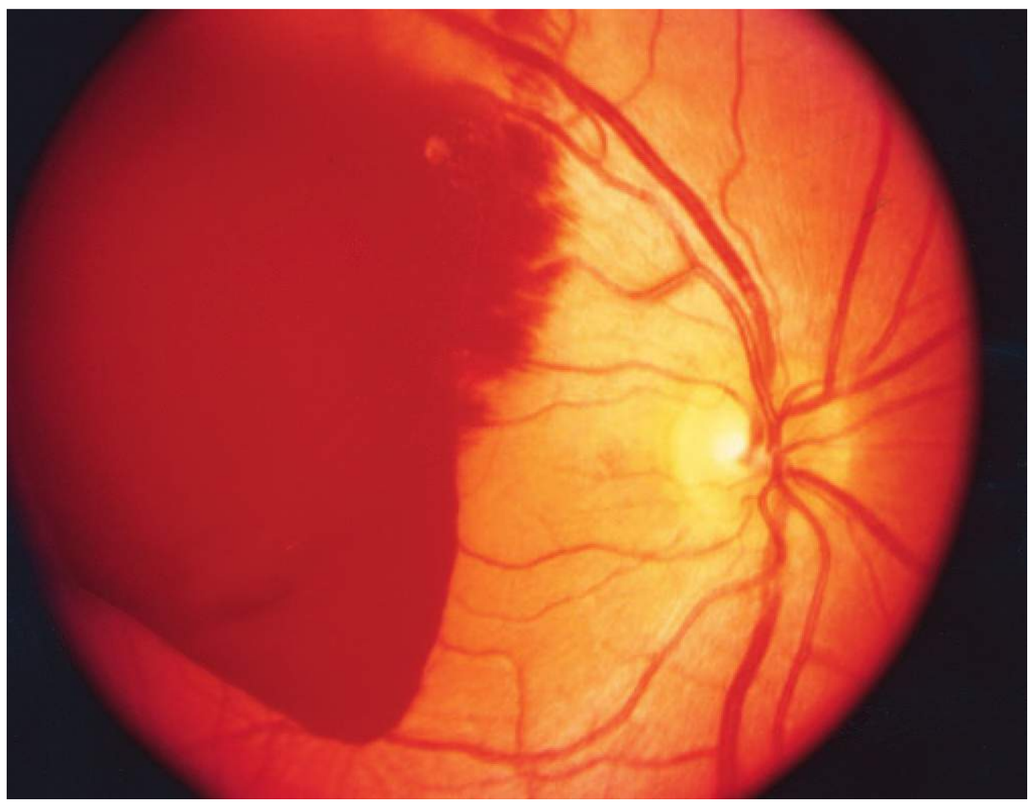

FIGURE 3.31 ■ **Subhyaloid Hemorrhage.** Subhyaloid hemorrhage seen on funduscopic examination in a patient with subarachnoid hemorrhage. (From Edlow JA, Caplan LR. Primary care: avoiding pitfalls in the diagnosis of subarachnoid hemorrhage. N Engl J Med. 2000;342:29-36. Reproduced with permission from John J. Weiter, MD, PhD.)

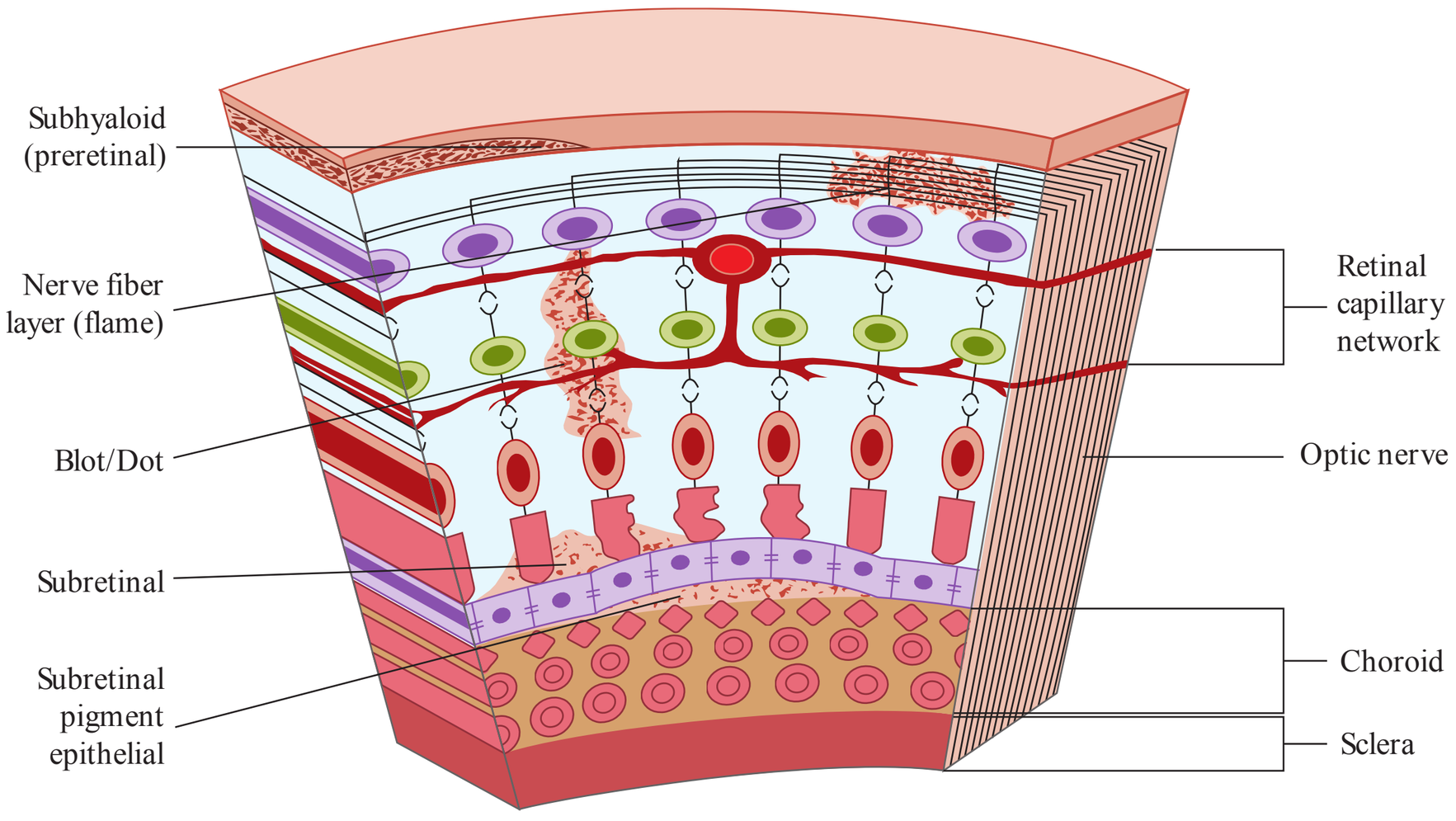

FIGURE 3.32 ■ **Retinal Hemorrhages—Anatomic Location.**