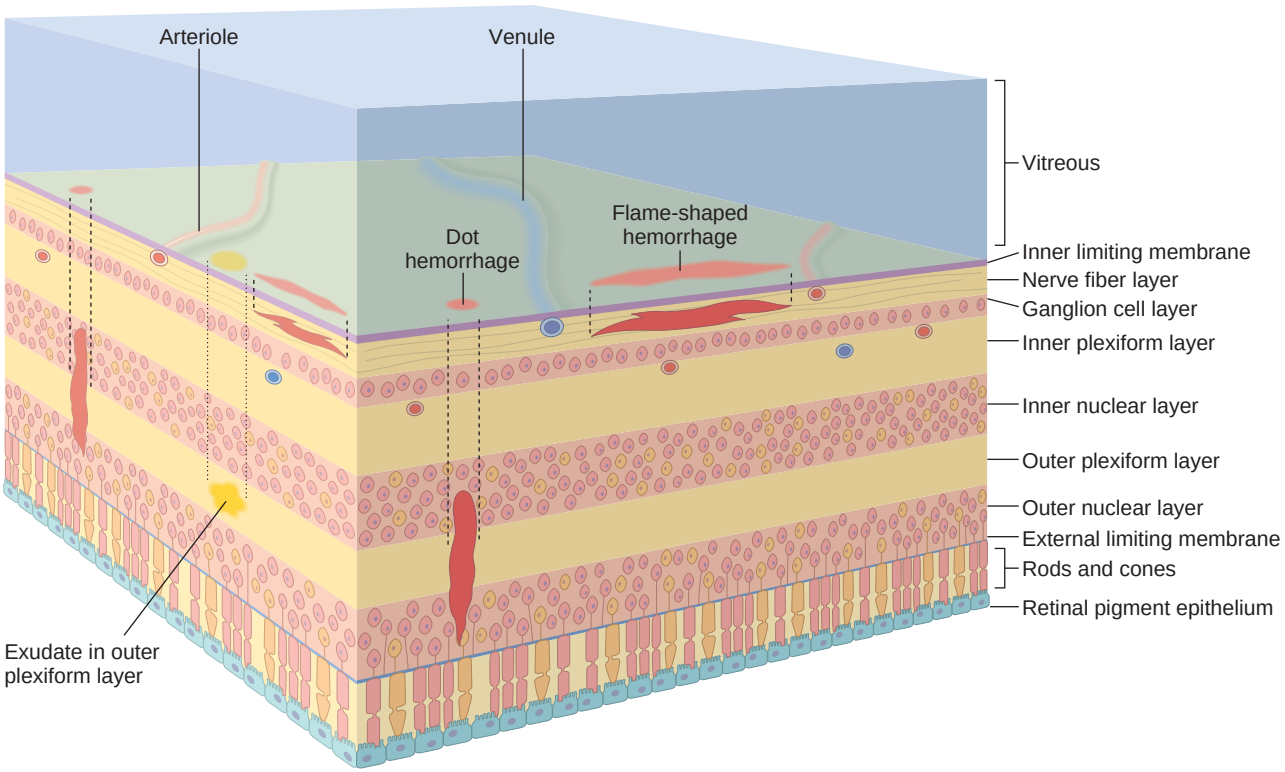

Figure 29-16 Clinicopathologic correlations of retinal hemorrhages and exudates. The location of the hemorrhage within the retina determines its appearance by ophthalmoscopy. The retinal nerve fiber layer is oriented parallel to the internal limiting membrane, and hemorrhages of this layer appear to be flame-shaped ophthalmoscopically. The deeper retinal layers are oriented perpendicular to the internal limiting membrane and hemorrhages in this location appear as cross-sections of a cylinder or "dot" hemorrhages. Exudates that originate from leaky retinal vessels accumulate in the outer plexiform layer.

has an important role in the maintenance of the outer segments of the photoreceptors. Disturbances in the RPE-photoreceptor interface are implicated in hereditary retinal degenerations such as *retinitis pigmentosa*.

The adult vitreous humor is avascular. Incomplete regression of fetal vasculature running through the vitreous humor can produce significant pathology as a retrolental mass *(persistent hyperplastic primary vitreous)*. The vitreous humor can be opacified by hemorrhage from trauma or retinal neovascularization. With age the vitreous humor may liquefy and collapse, creating the visual sensation of "floaters." Also, with aging, the posterior face of the vitreous humor—the posterior hyaloid—may separate from the neurosensory retina *(posterior vitreous detachment)*. The relationship between the posterior hyaloid and the neurosensory retina has a key role in the pathogenesis of retinal neovascularization and in some forms of retinal detachment.

Retinal Detachment

Retinal detachment (separation of the neurosensory retina from the RPE) is broadly classified by etiology based on the presence or absence of a break in the retina. *Rhegmatogenous retinal detachment* is associated with a full-thickness retinal defect. Retinal tears may develop after the vitreous collapses structurally, and the posterior hyaloid exerts traction on points of abnormally strong adhesion to the retinal internal limiting membrane. Liquefied vitreous humor then seeps through the tear and gains access to the potential space between the neurosensory retina and the RPE (Fig. 29-17). Re-attachment of the retina to the RPE generally requires relief of vitreous traction through indenting of the sclera by surgical procedures. This can be accomplished by the application of strips of silicon to the surface of the eye (scleral buckling) and possibly by removal of vitreous material (vitrectomy). Rhegmatogenous retinal detachment may be complicated by *proliferative vitreoretinopathy*, the formation of epiretinal or subretinal membranes by retinal glial cells (Müller cells) or RPE cells.

Non-rhegmatogenous retinal detachment (retinal detachment without retinal break) may complicate retinal vascular disorders associated with significant exudation and any condition that damages the RPE and permits fluid to leak from the choroidal circulation under the retina. Retinal detachments associated with choroidal tumors and malignant hypertension are examples of nonrhegmatogenous retinal detachment.

Retinal Vascular Disease

Hypertension

Normally, the thin walls of retinal arterioles permit a direct visualization of the circulating blood by ophthalmoscopy. In retinal arteriolosclerosis the thickened arteriolar wall changes the ophthalmic perception of circulating blood: vessels may appear narrowed, and the