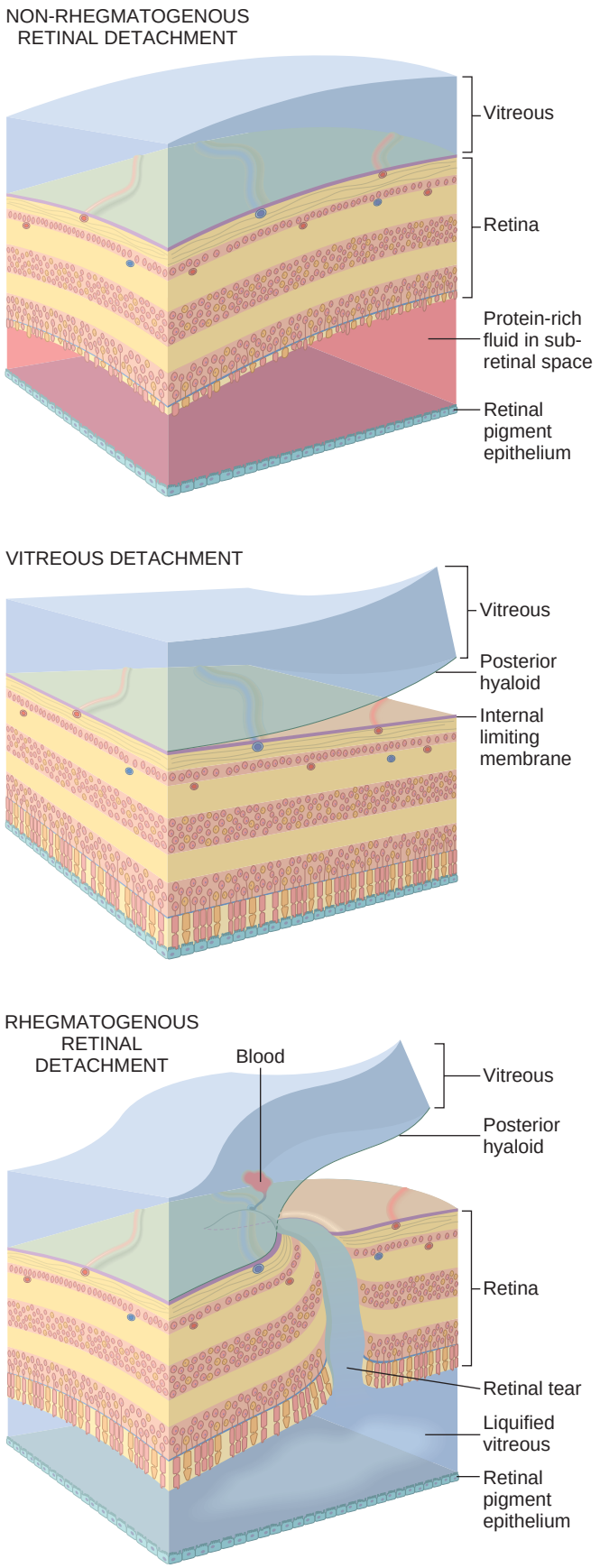

Figure 29-17 Retinal detachment is defined as the separation of the neurosensory retina from the RPE. Retinal detachments are classified broadly into non-rhegmatogenous (without a retinal break) and rhegmatogenous (with a retinal break) types. *Top*, In non-rhegmatogenous retinal detachment the subretinal space is filled with protein-rich exudate. Note that the outer segments of the photoreceptors are missing (see Fig. 29-16 for orientation of layers). This indicates a chronic retinal detachment, a finding that can be seen in both non-rhegmatogenous and rhegmatogenous detachments. *Middle*, Posterior vitreous detachment involves the separation of the posterior hyaloid from the internal limiting membrane of the retina and is a normal occurrence in the aging eye. *Bottom*, If during a posterior vitreous detachment the posterior hyaloid does not separate cleanly from the internal limiting membrane of the retina, the vitreous humor will exert traction on the retina, which will be torn at this point. Liquefied vitreous humor seeps through the retinal defect, and the retina is separated from the RPE. The photoreceptor outer segments are intact, illustrating an acute detachment.

color of the blood column may change from bright red to copper and to silver depending on the degree of vascular wall thickness (Fig. 29-18*A*).** Retinal arterioles and veins share a common adventitial sheath. Therefore, in pronounced retinal arteriolosclerosis the arteriole may compress the vein at points where both vessels cross (Fig. 29-18*B*). Venous stasis distal to arteriolar-venous crossing may precipitate occlusions of the retinal vein branches.

In malignant hypertension vessels in the retina and choroid may be damaged. Damage to choroidal vessels may produce focal choroidal infarcts, seen clinically as *Elschnig spots*. Damage to the choriocapillaris, the internal layer of the choroidal vasculature, may, in turn, damage the overlying RPE and permit the exudate to accumulate in the potential space between the neurosensory retina and the RPE, thereby producing a retinal detachment. Exudate from damaged retinal arterioles typically accumulates in the outer plexiform layer of the retina (Fig. 29-18*A*). The ophthalmoscopic finding of a macular star—a spokelike arrangement of exudate in the macula in malignant hypertension—results from exudate accumulating in the outer plexiform layer of the macula that is oriented obliquely instead of perpendicular to the retinal surface.

Occlusion of retinal arterioles may produce infarcts of the nerve fiber layer of the retina (axons of the retinal ganglion cell layer populate the nerve fiber layer). Axoplasmic transport in the nerve fiber layer is interrupted at the point of axonal damage, and accumulation of mitochondria at the swollen ends of damaged axons creates the histologic illusion of cells *(cytoid bodies)*. Collections of cytoid bodies populate the nerve fiber layer infarct, seen ophthalmoscopically as "cotton-wool spots" (Fig. 29-19). Although nerve fiber layer infarcts are described here in the context of hypertension, they may be detected in a variety of retinal occlusive vasculopathies. For example, retinal nerve fiber layer infarcts may develop in individuals with AIDS due to a retinal vasculopathy that is similar to the brain vasculopathy that may develop in this condition.

Diabetes Mellitus

The eye is profoundly affected by diabetes mellitus. The effects of hyperglycemia on the lens and iris have already been mentioned. Thickening of the basement membrane of the epithelium of the pars plicata of the ciliary body is a reliable histologic marker of diabetes mellitus in the eye (Fig. 29-20) and is reminiscent of similar