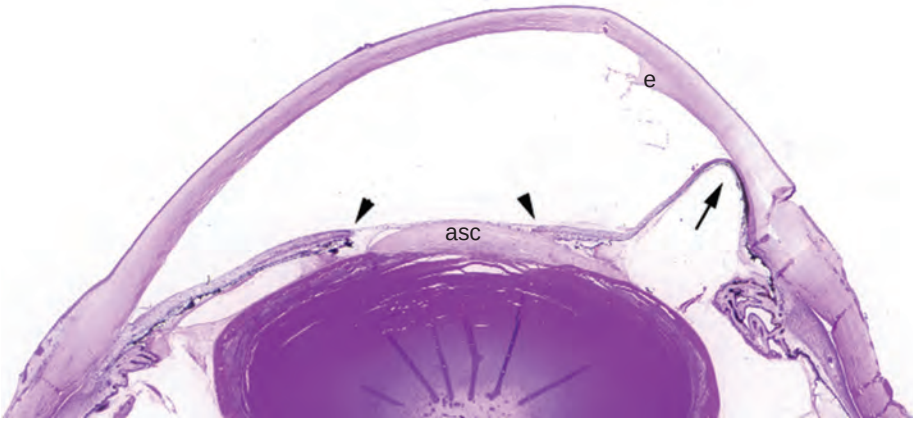

Figure 29-12 Sequelae of anterior segment inflammation. This eye was removed for complications of chronic corneal inflammation (not visible at this magnification). The exudate (e) present in the anterior chamber would have been visualized with a slit lamp as an optical "flare." The iris is adherent focally to the cornea, obstructing the trabecular meshwork (anterior synechia, *arrow*), and to the lens (posterior synechiae, *arrowheads*). An anterior subcapsular cataract (asc) has formed. The radial folds in the lens are artifacts.

episcleral veins following a spontaneous or traumatic carotid-cavernous fistula.

Primary angle-closure glaucoma typically develops in eyes with shallow anterior chambers, often found in individuals with hyperopia. Transient apposition of the pupillary margin of the iris to the anterior surface of the lens may result in obstruction to the flow of aqueous humor through the pupillary aperture *(pupillary block)*. Continued production of aqueous humor by the ciliary body thus elevates pressure in the posterior chamber and may bow the iris periphery forward *(iris bombé)*, apposing it to the trabecular meshwork. These anatomic changes provoke a marked elevation in intraocular pressure (Fig. 29-11). Since the crystalline lens is avascular and the lens epithelium receives its nutrition from the aqueous humor, unremitting elevation in intraocular pressure in primary angle-closure glaucoma can damage the lens epithelium. This leads to minute anterior subcapsular opacities that are visible by slit-lamp examination *(glaukomflecken)*. Although the affected individual might have a normal complement of healthy corneal endothelial cells, sustained elevated intraocular pressure can produce corneal edema and bullous keratopathy.

There are many causes of *secondary angle-closure glaucoma*. Contraction of various types of pathologic membranes that form over the surface of the iris can draw the iris over the trabecular meshwork, occluding aqueous outflow. For example, chronic retinal ischemia is associated with the up-regulation of VEGF and other proangiogenic factors. The appearance of VEGF in the aqueous humor is thought to induce the development of thin, clinically transparent fibrovascular membranes over the surface of the iris. Contraction of myofibroblastic elements in these membranes leads to occlusion of the trabecular meshwork by the iris: *neovascular glaucoma* (Fig. 29-11). Necrotic tumors, especially retinoblastomas, can also induce iris neovascularization and glaucoma. Secondary angle-closure glaucoma may be caused by other mechanisms as well; for example, tumors in the ciliary body can mechanically compress the iris onto the trabecular meshwork, closing off the major pathway of aqueous outflow.

Endophthalmitis and Panophthalmitis

In intraocular inflammation, vessels in the ciliary body and iris become leaky, allowing cells and exudate to accumulate in the anterior chamber. These changes can be visualized with a slit lamp; at times the inflammatory cells may adhere to the corneal endothelium, forming clinically visible *keratic precipitates*. The size and shape of these precipitates can provide clues to the underlying cause of the inflammation. For example, aggregates of macrophages on the endothelium in sarcoid produce characteristic "mutton-fat" keratic precipitates.

Just as pleural exudate in acute bronchopneumonia can lead to adhesions between the visceral and parietal pleura, the presence of exudate in the anterior chamber can facilitate the formation of adhesions between the iris and the trabecular meshwork or cornea *(anterior synechiae)* or between the iris and anterior surface of the lens *(posterior synechiae)*. Anterior synechiae can lead to elevation in intraocular pressure, which may lead to optic nerve damage. Prolonged contact between the iris and the anterior surface of the lens can deprive lens epithelium of contact with aqueous humor and can induce fibrous metaplasia of the lens epithelium: *anterior subcapsular cataract* (Fig. 29-12). The pharmacologic induction of pupillary dilation and cycloplegia in individuals with intraocular inflammation is intended in part to prevent the formation of synechiae and their sequelae.

Although inflammation confined to the anterior segment is technically intraocular inflammation, the term *endophthalmitis* is reserved for inflammation within the vitreous humor. The retina lines the vitreous cavity, and suppurative inflammation in the vitreous humor is poorly tolerated by the retina; a few hours may be sufficient to cause irreversible retinal injury. Endophthalmitis is classified as *exogenous* (originating in the environment and gaining access to the interior of the eye through a wound) or *endogenous* (delivered to the eye hematogenously). The term *panophthalmitis* is applied to inflammation within the eye that involves the retina, choroid, and sclera and extends into the orbit (Fig. 29-13).