



Figure 29-11 *Upper left*, The normal eye. Note that the surface of the iris is highly textured with crypts and folds. *Upper right*, The normal flow of aqueous humor. Aqueous humor, produced in the posterior chamber, flows through the pupil into the anterior chamber. The major pathway for the egress of aqueous humor is through the trabecular meshwork, into the Schlemm canal. Minor outflow pathways (uveoscleral and iris, not depicted) contribute to a limited extent to aqueous outflow. *Lower left*, Primary angle-closure glaucoma. In anatomically predisposed eyes, transient apposition of the iris at the pupillary margin to the lens blocks the passage of aqueous humor from the posterior chamber to the anterior chamber, bowing the iris forward (iris bombé) and occluding the trabecular meshwork. Pressure builds in the posterior chamber. *Lower right*, A neovascular membrane has grown over the surface of the iris, smoothing the iris folds and crypts. Myofibroblasts within the neovascular membrane cause the membrane to contract and to become apposed to the trabecular meshwork (peripheral anterior synechia). Outflow of aqueous humor is blocked, and the intraocular pressure becomes elevated.

Both open-angle and angle-closure glaucoma can be subclassified into primary and secondary types. In *primary open-angle glaucoma*, the most common form of glaucoma, the angle is open, and few changes are apparent structurally. Mutations in the myocilin (*MYOC*) gene have been associated with a subset of individuals with juvenile and adult primary open-angle glaucoma. Mutations in optineurin (*OPTN*) may also be responsible for a subset of adult patients with open angle glaucoma. The role of these genes in the pathogenesis of glaucoma is not clear.

There are multiple causes of *secondary open-angle glaucoma*. Pseudoexfoliation glaucoma, perhaps the most common form of secondary open angle glaucoma, is associated with the deposition of fibrillar material of varying composition throughout the anterior segment. Pseudoexfoliation glaucoma has been associated with

single nucleotide polymorphisms in the lysyl oxidase like 1 (*LOX1*) gene. In addition to deposition in the anterior chamber, fibrillar material is deposited around blood vessels in connective tissue and in many visceral organs such as liver, kidney and gall bladder.

Particulate material such as high-molecular-weight lens proteins produced by phacolysis, senescent red cells after trauma (*ghost cell glaucoma*), iris epithelial pigment granules (*pigmentary glaucoma*), and necrotic tumors (*melanolytic glaucoma*) can clog the trabecular meshwork in the presence of an open angle. Elevations in the pressure on the surface of the eye (episcleral venous pressure) in the presence of an open angle also contribute to secondary open-angle glaucoma. This type of glaucoma is associated with surface ocular vascular malformations seen in *Sturge-Weber syndrome* or as a consequence of arterIALIZATION of the