INTRODUCTION

Glaucoma is a group of optic neuropathies that share a slowly progressive degeneration of the retinal ganglion cells and their axons, resulting in a distinct appearance of the optic disc and a concurrent pattern of vision loss (1). Glaucoma is the second leading cause of blindness in the world with estimates that it affects 66.8 million individuals worldwide (2); at least 6.7 million of these affected people suffer from complete blindness (2). The vision loss associated with this disease is irreversible, yet the biological basis of glaucoma and the factors contributing to its progression have not been completely elucidated (1). Intraocular pressure (IOP) is the only proven treatable risk factor in glaucoma (1). As such, glaucoma is theoretically defined as a progressive optic neuropathy as a result of elevation of IOP above the physiological level of individuals (3). The upper limit of “normal” IOP, based on a large number of subjects, is internationally accepted as being approx 21 mmHg as a standard in the clinical diagnosis of glaucoma (3).

There are numerous medical and surgical strategies used in the management of glaucoma. The goal of this chapter is to briefly review the physiology of aqueous humor production and drainage, retinal, and optic nerve anatomy pertinent to glaucoma, and the pathophysiology of glaucoma. Further, the main emphasis of this chapter will be on glaucoma drainage devices (GDDs). In particular, historical GDD designs will be reviewed, and the remainder of the chapter will address current designs and surgical strategies used with GDDs for the management of glaucoma, the complications following GDD implantation, and the challenges encountered in developing and using these devices in the eye.
IOP is regulated by a balance between the secretion and drainage of aqueous humor from the eye (Fig. 1). Aqueous humor is secreted posterior to the iris by the nonpigmented ciliary epithelium of the ciliary body and this fluid then flows anteriorly through the pupil to the anterior chamber. Aqueous humor exits the eye into the venous circulation through the trabecular meshwork (conventional outflow pathway) and independently through the uveoscleral pathway (unconventional outflow pathway) (Fig. 1).

**Ocular Anatomy and Physiology**

**Aqueous Humor Dynamics**

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**Retinal Ganglion Cells and Optic Nerve**

Axons from the retinal ganglion cells consist of the innermost layer of the retina, the nerve fiber layer (Fig. 2B). These axons converge on the optic disc and form the optic nerve, which contains a central depression called the cup. Most optic nerves have a visible physiological cup, which is surrounded by a neuroretinal rim (Fig. 2A). The human optic nerve contains approx 1 million nerve fibers (Fig. 2C), which exit the eye after passing through the lamina cribrosa, a series of perforated connective tissue sheets, and synapse in the lateral geniculate nucleus of the brain (1). Trophic factors are transported both retrogradely from the axonal terminals of the retinal ganglion cells to their cell bodies in the inner retina as well as anterogradely from the retinal ganglion cell

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**Fig. 1.** Diagram illustrating aqueous humor dynamics. Arrows indicate the direction of aqueous humor flow from the ciliary body (located in the posterior chamber) through the pupil, into the anterior chamber, and exiting the eye through the trabecular meshwork (conventional outflow pathway) or uvea (uveoscleral/unconventional outflow pathway) (Reprinted with permission from Elsevier [1]).