INTRODUCTION

Aqueous production

Aqueous humour is produced from plasma by the ciliary epithelium of the ciliary body pars plicata, using a combination of active and passive secretion. A high-protein filtrate passes out of fenestrated capillaries (ultrafiltration) into the stroma of the ciliary processes, from which active transport of solutes occurs across the dual-layered ciliary epithelium. The osmotic gradient thereby established facilitates the passive flow of water into the posterior chamber. Secretion is subject to the influence of the sympathetic nervous system, with opposing actions mediated by beta-2 receptors (increased secretion) and alpha-2 receptors (decreased secretion). Enzymatic action is also critical – carbonic anhydrase is among those playing a key role.

Aqueous outflow

Anatomy

- **The trabecular meshwork** (trabeculum) is a sieve-like structure (Fig. 10.1) at the angle of the anterior chamber (AC) through which 90% of aqueous humour leaves the eye. It has three components (Fig. 10.2).
  - The uveal meshwork is the innermost portion, consisting of cord-like endothelial cell-covered strands arising from the iris and ciliary body stroma. The intertrabecular spaces are relatively large and offer little resistance to the passage of aqueous.
  - The corneoscleral meshwork lies external to the uveal meshwork to form the thickest portion of the trabeculum. It is composed of layers of connective tissue strands with overlying endothelial-like cells. The intertrabecular spaces are smaller than those of the uveal meshwork, conferring greater resistance to flow.
  - The juxtacanalicular (cribriform) meshwork is the outer part of the trabeculum, and links the corneoscleral meshwork with the endothelium of the inner wall of the canal of Schlemm. It consists of cells embedded in a dense extracellular matrix with narrow intercellular spaces, and offers the major proportion of normal resistance to aqueous outflow.
- The **Schlemm canal** is a circumferential channel within the perilimbal sclera. The inner wall is lined by irregular spindle-shaped endothelial cells containing infoldings (giant vacuoles) that are thought to convey aqueous via the formation of transcellular pores. The outer wall is lined by smooth flat cells and contains the openings of collector channels, which leave the canal at oblique angles and connect directly or indirectly with episcleral veins. Septa commonly divide the lumen into 2–4 channels.

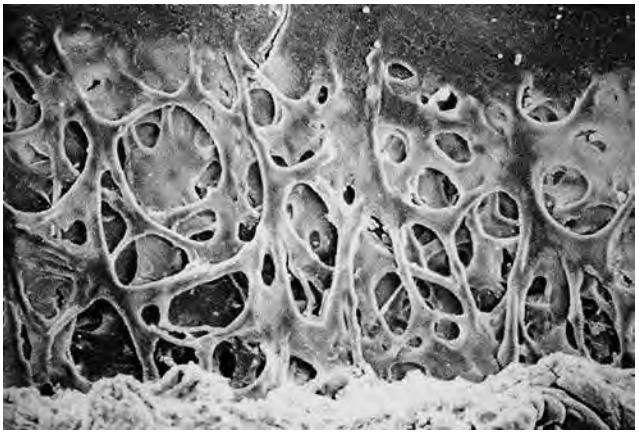

Fig. 10.1 Scanning electron micrograph of the trabecular meshwork

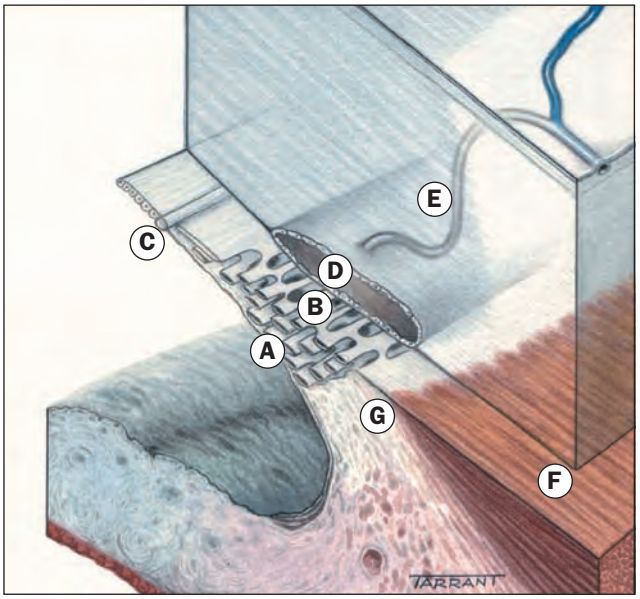

Fig. 10.2 Anatomy of outflow channels: A, Uveal meshwork; B, corneoscleral meshwork; C, Schwalbe line; D, Schlemm canal; E, connector channels; F, longitudinal muscle of the ciliary body; G, scleral spur

Physiology

Aqueous flows from the posterior chamber via the pupil into the AC, from where it exits the eye via three routes (Fig. 10.3).

- **Trabecular** outflow (90%): aqueous flows through the trabeculum into the Schlemm canal and then the episcleral veins. This is a bulk flow pressure-sensitive route so that increasing IOP will increase outflow.
- **Uveoscleral** drainage (10%): aqueous passes across the face of the ciliary body into the suprachoroidal space, and is drained by the venous circulation in the ciliary body, choroid and sclera.
- **Iris**: some aqueous also drains via the iris.

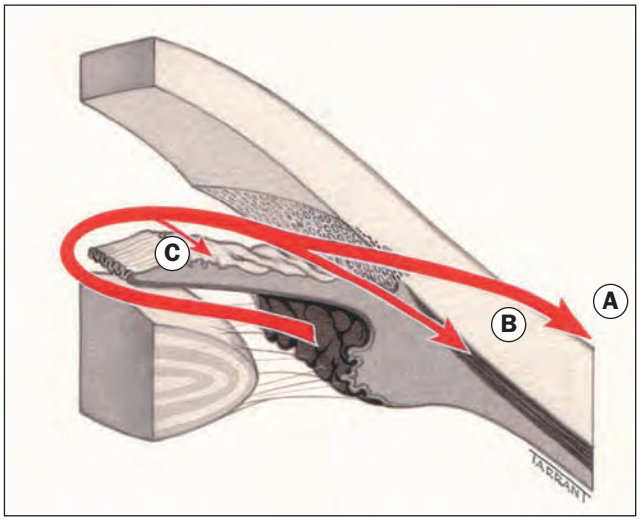

Fig. 10.3 Routes of aqueous outflow: A, trabecular; B, uveoscleral; C, iris

Intraocular pressure

Intraocular pressure (IOP) is determined by the balance between the rate of aqueous production and its outflow, the latter in turn related to factors that include the resistance encountered in the trabeculum and the level of episcleral venous pressure.

Concept of normal intraocular pressure

The average IOP in the general population is around 16 mmHg on applanation tonometry, and a range of about 11–21 mmHg – two standard deviations either side of the average – has conventionally been accepted as normal, at least for a Caucasian population. However, some patients develop glaucomatous damage with IOP less than 21 mm Hg whilst others remain unscathed with IOP well above this level. Whilst reduction of IOP is a key modifiable element in essentially all types of glaucoma, additional incompletely understood factors are critical in determining whether a particular individual or eye develops glaucomatous damage. These include features influencing the IOP reading, such as corneal rigidity, and probably factors affecting the susceptibility of the optic nerve to damage, such as the integrity of its blood supply and structural vulnerability to mechanical stress at the optic nerve head.

Fluctuation

Normal IOP varies with time of day (diurnal variation), heartbeat, blood pressure and respiration. The diurnal pattern varies, with a tendency to be higher in the morning and lower in the afternoon and evening. This is at least partially due to a diurnal pattern in aqueous production, which is lower at night. Glaucomatous eyes exhibit greater than normal fluctuation, the extent of which is directly proportional to the likelihood of progressive visual field damage, and a single reading may therefore be misleading. It is good practice always to note the time of day in conjunction with a recorded IOP.

Overview of glaucoma

Definition

It is difficult to define glaucoma precisely, partly because the term encompasses a diverse group of disorders. All forms of the disease have in common a characteristic potentially progressive optic neuropathy that is associated with visual field loss as damage progresses, and in which IOP is a key modifiable factor.

Classification

Glaucoma may be congenital (developmental) or acquired. Open-angle and angle-closure types are distinguished based on the mechanism by which aqueous outflow is impaired with respect to the AC angle configuration. Distinction is also made between primary and secondary glaucoma; in the latter a recognizable ocular or non-ocular disorder contributes to elevation of IOP.

Epidemiology

Glaucoma affects 2–3% of people over the age of 40 years; 50% may be undiagnosed. Primary open-angle glaucoma (POAG) is the most common form in white, Hispanic/Latino and black individuals; the prevalence is especially high in the latter. On a worldwide basis, primary angle closure (PAC) constitutes up to half of cases, and has a particularly high prevalence in individuals of Asian descent, although with improved assessment such as the routine performance of gonioscopy in a darkened rather than a bright environment, PAC is known to be more prevalent in Caucasian individuals than previously realized.

TONOMETRY

Goldmann tonometry

Principles

Goldmann applanation tonometry (GAT) is based on the Imbert–Fick principle, which states that for a dry thin-walled sphere, the pressure (P) inside the sphere equals the force (F) necessary to flatten its surface divided by the area (A) of flattening (i.e. $P = F/A$). Theoretically, average corneal rigidity (taken as 520 µm for GAT) and the capillary attraction of the tear meniscus cancel each other out when the flattened area has the 3.06 mm diameter contact surface of the Goldmann prism, which is applied to the cornea using the Goldmann tonometer with a measurable amount of force from which the IOP is deduced (Fig. 10.4). The tonometer prism should be disinfected between patients and replaced regularly in accordance with the manufacturer's instructions. Disposable tonometer prisms and caps have been introduced to address concerns of infection from reusable prisms.