Chapter 2

- Intraocular Pressure and Aqueous Humor Dynamics

An understanding of *aqueous humor dynamics* is essential for the evaluation and management of glaucoma. As noted in Figure 1-1, aqueous humor is produced in the posterior chamber and flows throug the pupil into the anterior chamber. Aqueous humor exits the eye by passing through the *trabecular meshwork* and into *Schlemm's canal* before draining into the venous system through a plexus of collector channels, as well as through the uveoscleral pathway, which is proposed to exit through the root of the iri and the ciliary muscle, into the suprachoroidal spaces and through the sclera. The *Goldmann equation* summarizes the relationship between many of these factors and the intraocular pressure (IOP) in the undisturbed eye:

$$P_0 = (F/C) + P_v$$

where P_0 is the IOP in millimeters of mercury (mm Hg), F is the rate of aqueous formation in microliters per minute (μL/min), C is the facility of outflow in microliters per minute per millimeter of mercury (μL/min/mm Hg), and P_v is the episcleral venous pressure in millimeters of mercury. Resistance to outflov *(R)* is the inverse of facility *(C).*

Table 2-1 illustrates the impact of reduced outflow facility (C value) of aqueous humor through the trabecular meshwork both in an open angle and in various amounts of angle closure.

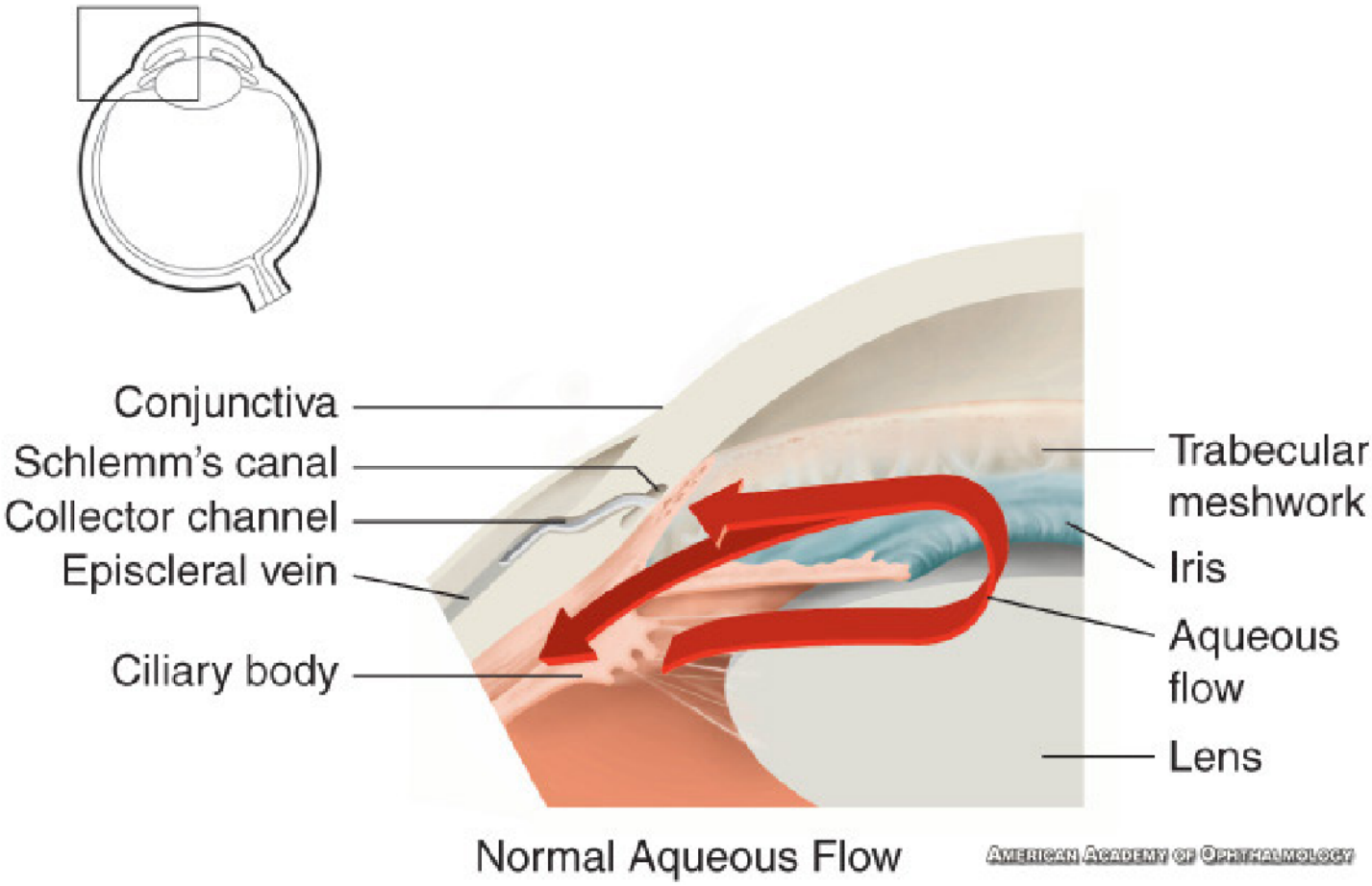

Figure 1-1

Diagrammatic cross section of the anterior segment of the normal eye, showing the site of aqueous production (ciliary body), sites of conventional aqueous outflow (trabecular meshwork–Schlemm's canal system and episcleral venous plexus), and the uveoscleral outflow pathway. Small white arrow shows normal path of outflow and indicates that resistance in this illustration is relative, not total.
(Illustration by Cyndie C. H. Wooley.)

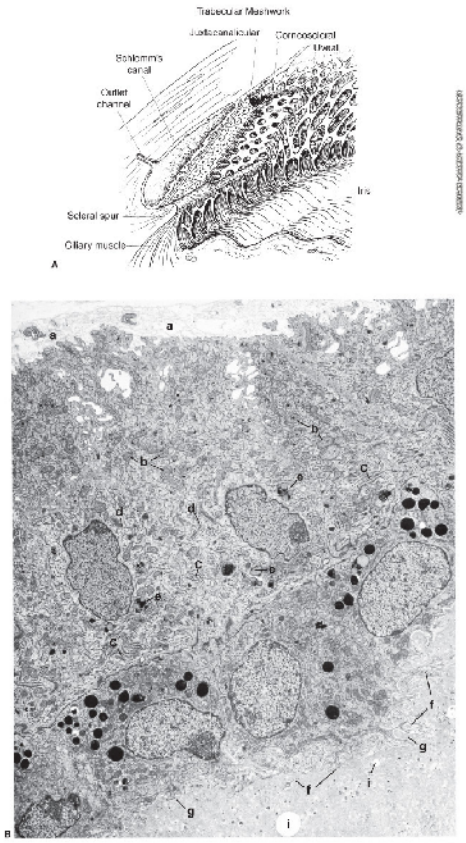

Figure 2-2

A, Three layers of trabecular meshwork (shown in cutaway views): uveal, corneoscleral, and juxtacanalicular. **B,** Pars plicata of the ciliary body showing the 2 epithelial layers in the eye of an older person. The unpigmented epithelial cells measure approximately 20 µm high by 12 µm wide. The cuboidal pigmented epithelial cells are approximately 10 µm high. The thickened internal limiting membrane *(a)* is laminated and vesicular; such thickened membranes are a characteristic of older eyes. The cytoplasm of the unpigmented epithelium is characterized by its numerous mitochondria*(b)* and the cisternae of the rough-surfaced endoplasmic reticulum*(c).* A poorly developed Golgi apparatus*(d)* and several lysosomes and residual bodies *(e)* are shown. The pigmented epithelium contains many melanin granules, measuring about 1 µm in diameter and located mainly in the apical portion. The basal surface is rather irregular, having many fingerlike processes *(f).* The basement membrane of the pigmented epithelium*(g)* and a smooth granular material containing vesicles *(i)* and coarse granular particles are seen at the bottom of the figure. The appearance of the basement membrane is typical of older eyes and can be discerned with the light microscope (×5700).

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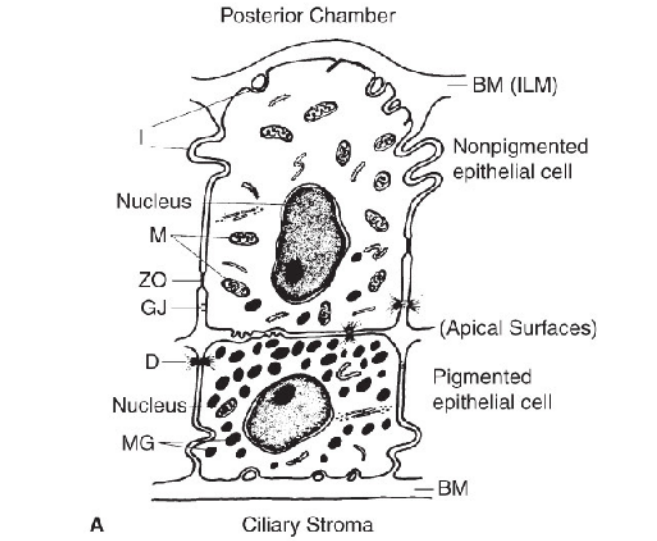

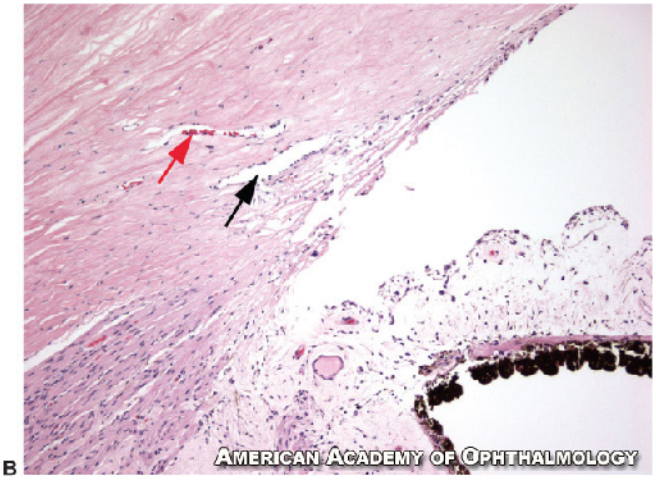

Figure 2-1

A, The 2 layers of the ciliary epithelium showing apical surfaces in apposition to each other. Basement membrane *(BM)* lines the double layer and constitutes the internal limiting membrane *(ILM)* on the inner surface. The nonpigmented epithelium is characterized by large numbers of mitochondria *(M),* zonula occludens *(ZO),* and lateral and surface interdigitations *(I).* The pigmented epithelium contains numerous melanin granules *(MG).* Additional intercellular junctions include desmosomes *(D)* and gap junctions *(GJ).* **B,** Light micrograph of the anterior chamber angle demonstrates Schlemm's canal *(black arrow)* to the trabecular meshwork in the sclera. One of the external collector vessels can be seen adjacent to Schlemm's canal *(red arrow).*

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Episcleral Venous Pressure

Episcleral venous pressure is relatively stable, except with alterations in body position and with certain diseases of the orbit, the head, and the neck that obstruct venous return to the heart or shunt blood from th arterial to the venous system. The usual range of values is 8–10 mm Hg. The pressure in the episcleral veins can be measured with specialized equipment. In acute conditions, according to the Goldmann equation, IOP rises approximately 1 mm Hg for every 1 mm Hg increase in episcleral venous pressure. The relationship is more complex and less well understood, however, in chronic conditions. Chronic elevations of episcleral venous pressure may be accompanied by changes in IOP that are of greater or l magnitude than predicted by the Goldmann equation. In addition, these changes may not vary directly with the episcleral venous pressure. Abnormal elevated episcleral venous pressure can cause the collapse of Schlemm's canal and an increase in aqueous humor outflow resistance. Episcleral venous pressure is often increased in syndromes with facial hemangiomas (eg, Sturge-Weber) and in thyroid-associated orbitopathy and is partially responsible for the elevated IOP seen in thyroid eye disease.

Aqueous Humor Formation

Aqueous humor formation is a biological process that is subject to circadian rhythms. Aqueous humor is formed by the *ciliary processes,* each of which is composed of a double layer of epithelium over a core of stroma and a rich supply of fenestrated capillaries (Fig 2-1). Each of the 80 or so processes contains a large number of capillaries, which are supplied mainly by branches of the major arterial circle of the iris. The apical surfaces of both the outer pigmented and the inner nonpigmented layers of epithelium face each other and are joined by tight junctions, which are an important component of the blood–aqueous barrier. The inner nonpigmented epithelial cells, which protrude into the posterior chamber, contain numerous mitochondria and microvilli; these cells are thought to be the actual site of aqueous production. The ciliary processes provide a large surface area for secretion.

Aqueous humor formation and secretion into the posterior chamber result from

- active secretion, which takes place in the double-layered ciliary epithelium
- ultrafiltration
- simple diffusion

*Active secretion,*or *transport,*consumes energy to move substances against an electrochemical gradient and is independent of pressure. The identity of the precise ion or ions transported is not known, but sodium, chloride, and bicarbonate are involved. Active secretion accounts for the majority of aqueous production and involves, at least in part, activity of the enzyme carbonic anhydrase II. *Ultrafiltration*refers to a pressure-dependent movement along a pressure gradient. In the ciliary processes, the hydrostatic pressure difference between capillary pressure and IOP favors fluid movement into the eye, whereas the oncotic gradient between the two resists fluid movement. The relationship between secretion and ultrafiltration is not known. *Diffusion*is the passive movement of ions across a membrane related to charge and concentration

Rate of Aqueous Formation

The most common method used to measure the rate of aqueous formation is *fluorophotometry.* Fluorescein is administered systemically or topically, and the subsequent decline in its anterior chamber concentration is measured optically and used to calculate aqueous flow. As previously noted, the normal flow is approximately 2.0–2.5 μL/min, and the aqueous volume is turned over at a rate of 1% per minute.

Aqueous formation varies diurnally and drops during sleep. It also decreases with age, as does outflow facility. The rate of aqueous formation is affected by a variety of factors, including

- integrity of the blood–aqueous barrier
- blood flow to the ciliary body
- neurohumoral regulation of vascular tissue and the ciliary epithelium

Aqueous humor production may decrease following trauma or intraocular inflammation and following the administration of certain drugs, such as general anesthetics and some systemic hypotensive agents. Carotid occlusive disease may also decrease aqueous humor production.

Suppression of Aqueous Formation

The mechanisms of action of the various classes of drugs that suppress aqueous formation—the *carbonic anhydrase inhibitors*, *β-adrenergic antagonists (beta-blockers)*, and *α_2-agonists*—are not precisely understood. The role of the enzyme carbonic anhydrase has been debated vigorously. Evidence suggests that the bicarbonate ion is actively secreted in human eyes; thus, the function of the enzyme may be to provide this ion. Carbonic anhydrase may also provide bicarbonate or hydrogen ions for an intracellular buffering system.

Current evidence indicates that β_2-receptors are the most prevalent adrenergic receptors in the ciliary epithelium. The significance of this finding is unclear, but β-adrenergic antagonists may affect active transport by causing a decrease either in the efficiency of the Na^+/K^+ pump or in the number of pump sites. For a detailed discussion and illustration of the sodium pump and pump–leak mechanism, see BCSC Section 2, *Fundamentals and Principles of Ophthalmology.*

Distribution in the Population and Relation to Glaucoma

Pooled data from large western epidemiologic studies indicate that the mean IOP is approximately 16 mm Hg, with a standard deviation of 3 mm Hg. However, IOP has a non-Gaussian distribution with a skew toward higher pressures, especially in individuals older than age 40 (Fig 2-3). The value 22 mm Hg (greater than 2 standard deviations above the mean) has been used in the past both to separate normal and abnormal pressures and to define which patients required ocular hypotensive therapy. This division was based on the erroneous clinical assumptions that glaucomatous damage is caused exclusively by pressures that are higher than normal and that normal pressures do not cause damage. An example of th shortcomings created by these assumptions is that screening for glaucoma based solely on IOP >21 mm Hg misses up to half of the people with glaucoma and optic nerve damage in the screened population.

General agreement has been reached that, for the population as a whole, there is no clear IOP level belov which IOP can be considered “normal” or safe and above which IOP can be considered “elevated” or unsafe: some eyes undergo damage at IOPs of 18 mm Hg or less, whereas others tolerate IOPs in the 30 However, elevation of IOP is still seen as a very important risk factor for the development of glaucomatou optic nerve damage. Although other risk factors affect an individual’s susceptibility to glaucomatous damage, IOP is the only one that can be effectively altered at this time.

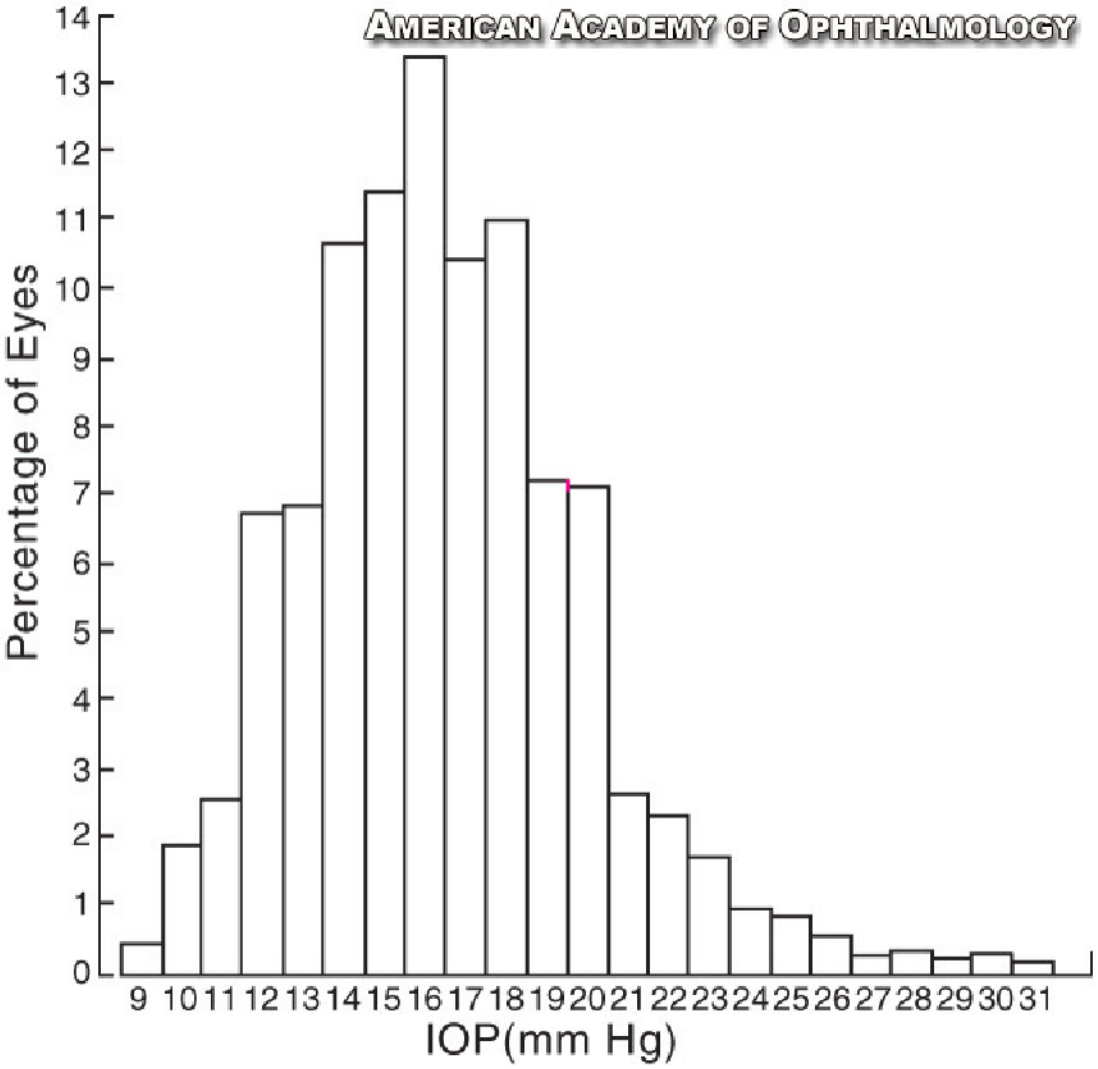

Figure 2-3

Frequency distribution of intraocular pressure: 5220 eyes in the Framingham Eye Study. *(Reproduced from Colton T, Ederer F. The distribution of intraocular pressures in the general population. Surv Ophthalmol. 1980;25:123–129.)*

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Aqueous Humor Outflow

Aqueous humor outflow occurs by 2 major mechanisms: pressure-dependent outflow and pressure-independent outflow. The facility of outflow (*C* in the Goldmann equation; see the beginning of the chapter) varies widely in normal eyes. The mean value reported ranges from 0.22 to 0.30 μL/min/mm Hg. Outflow facility decreases with age and is affected by surgery, trauma, medications, and endocrine factors. Patient with glaucoma and elevated IOP have decreased outflow facility.

Trabecular Outflow

Traditional thought contended that most of the aqueous humor exits the eye by way of the trabecular meshwork–Schlemm's canal–venous system. However, recent evidence questions the exact ratio of trabecular to uveoscleral outflow. As with outflow facility, this ratio is affected by age and by ocular health. The meshwork is classically divided into 3 parts (Fig 2-2). The uveal part is adjacent to the anterior cham and is arranged in bands that extend from the iris root and the ciliary body to the peripheral cornea. The corneoscleral meshwork consists of sheets of trabeculum that extend from the scleral spur to the lateral w of the scleral sulcus. The juxtacanalicular meshwork, which is thought to be the major site of outflow resistance, is adjacent to, and actually forms the inner wall of, Schlemm's canal. Aqueous moves both across and between the endothelial cells lining the inner wall of Schlemm's canal.

The trabecular meshwork is composed of multiple layers, each of which consists of a collagenous connective tissue core covered by a continuous endothelial layer covering. It is the site of pressure-dependent outflow. The trabecular meshwork functions as a 1-way valve that permits aqueous to leave the eye by bulk flow but limits flow in the other direction, independent of energy. Its cells are phagocytic, a function they may exhibit in the presence of inflammation and after laser treatment.

In most older eyes, trabecular cells contain a large number of pigment granules within their cytoplasm that give the entire meshwork a brown or muddy appearance. In addition, the number of trabecular cells decreases with age, and the basement membrane beneath them thickens. There are relatively few trabecular cells—approximately 200,000–300,000 cells per eye. An interesting effect of all types of laser trabeculoplasty is to induce trabecular cell division and cause a change in the production of cytokines and other structurally important elements of the extracellular matrix. The extracellular matrix material is found through the dense portions of the trabecular meshwork.

Schlemm's canal is completely lined with an endothelial layer that does not rest on a continuous basemen membrane. The canal is a single channel, with an average diameter of approximately 370 μm, and is transversed by tubules. The inner wall of Schlemm's canal contains giant vacuoles that have direct communication with the intertrabecular spaces. The outer wall is actually a single layer of endothelial cells that do not contain pores. A complex system of vessels connects Schlemm's canal to the episcleral vein which subsequently drain into the anterior ciliary and superior ophthalmic veins. These, in turn, ultimately drain into the cavernous sinus.

When IOP is low, the trabecular meshwork may collapse, or blood may reflux into Schlemm's canal and l visible on gonioscopy.

Uveoscleral Outflow

In the normal eye, any nontrabecular outflow is termed *uveoscleral outflow*. Uveoscleral outflow is also termed *pressure-independent outflow.* A variety of mechanisms are likely involved, predominantly aqueou passage from the anterior chamber into the ciliary muscle and then into the supraciliary and suprachoroid spaces. The fluid then exits the eye through the intact sclera or along the nerves and the vessels that penetrate it. As noted, uveoscleral outflow is largely pressure-independent and is believed to be influence by age. There is evidence that humans, like nonhuman primates, have significant outflow via the uveoscleral pathway. Uveoscleral outflow has been estimated to account for 5%–15% of total aqueous outflow, but recent studies indicate it may be a higher percentage of total outflow, especially in normal eye of young people. It is increased by cycloplegia, adrenergic agents, prostaglandin analogs, and certain complications of surgery (eg, cyclodialysis) and is decreased by miotics.

Tonography

Tonography is a method used to measure the facility of aqueous outflow. The clinician can take the measurement by using a Schiøtz tonometer of known weight. The tonometer is placed on the cornea, acutely elevating the IOP. The rate at which the pressure declines with time is related to the ease with whi the aqueous leaves the eye. The decline in IOP over time can be used to determine outflow facility in µL/min/mm Hg through a series of mathematical calculations.

Unfortunately, tonography depends on a number of assumptions (eg, the elastic properties of the eye, stability of aqueous formation, and constancy of ocular blood volume) and is subject to many sources of error, such as calibration problems, patient fixation, and eyelid squeezing. These problems reduce the accuracy and reproducibility of tonography for an individual patient. In general, tonography is best used as a research tool for the investigation of pharmacokinetics and is rarely used clinically.

Factors Influencing Intraocular Pressure

IOP varies with a number of factors, including the following (Table 2-2):

- time of day
- heartbeat
- respiration
- exercise
- fluid intake
- systemic medications
- topical medications

Alcohol consumption results in a transient decrease in IOP. In most studies, caffeine has not shown an appreciable effect on IOP. Cannabis decreases IOP but has not been proven clinically useful because of its short duration of action and poor side effect profile. IOP is higher when an individual is recumbent rath than upright, predominantly because of an increase in the episcleral venous pressure. Some people hav an exaggerated rise in IOP when they lie down, and this tendency may be important in the pathogenesis of some forms of glaucoma. IOP usually increases with age and is genetically influenced: higher pressures are more common in relatives of patients with POAG than in the general population.

Diurnal Variation

In normal individuals, IOP varies 2–6 mm Hg over a 24-hour period, as aqueous humor production and outflow change. Higher IOP is associated with greater fluctuation, and a diurnal fluctuation of greater than 10 mm Hg is suggestive of glaucoma. The time at which peak IOPs occur in any individual is quite variable; however, many people reach their peak daytime pressures in the morning hours. Such fluctuatio can be detected through measurement of ocular pressure at multiple times around the clock. Recent evidence suggests that with around-the-clock IOP measurement performed in individuals in habitual bod positions (standing or sitting during the daytime and lying down at night), many individuals, those with glaucoma and those without, will show peak pressures in the early morning hours while they are still in be Measurement of IOP during nonoffice hours may be useful for determining why optic nerve damage occu despite apparently adequately controlled pressure. However, the impact of IOP fluctuations on the optic nerve remains unknown. The relationship between blood pressure and IOP may be important in optic nerv damage: systemic hypotension, especially during sleep, has been suggested as a possible cause of decreased optic nerve perfusion resulting in damage.

Clinical Measurement of Intraocular Pressure

Measurement of IOP in a clinical setting requires a force that indents or flattens the eye. *Applanation tonometry* is the method used most widely. It is based on the Imbert-Fick principle, which states that the pressure inside an ideal dry, thin-walled sphere equals the force necessary to flatten its surface divided by the area of the flattening:

$$P = F/A$$

where P = pressure, F = force, and A = area. In applanation tonometry, the cornea is flattened, and IOP is determined by measuring the applanating force and the area flattened (Fig 2-4).

The *Goldmann applanation tonometer* measures the force necessary to flatten an area of the cornea of 3.06 mm diameter. At this diameter, the resistance of the cornea to flattening is counterbalanced by the capillary attraction of the tear film meniscus for the tonometer head. Furthermore, the IOP (in mm Hg) equals the flattening force (in grams) multiplied by 10. A split-image prism allows the examiner to determine the flattened area with great accuracy. Fluorescein in the tear film is used to outline the area of flattening. The semicircles move with the ocular pulse, and the endpoint is reached when the inner edges the semicircles touch each other at the midpoint of their excursion (Fig 2-5).

Applanation measurements are safe, easy to perform, and relatively accurate in most clinical situations. Of the currently available devices, the Goldmann applanation tonometer is the most valid and reliable. Because applanation does not displace much fluid (approximately 0.5 µL) or substantially increase the pressure in the eye, this method is relatively unaffected by ocular rigidity. Table 2-3 lists possible sources of error in tonometry.

An excessive amount of fluorescein results in wide mires and an inaccurately high reading, whereas an inadequate amount of fluorescein leads to artificially low readings.

Marked corneal astigmatism causes an elliptical fluorescein pattern. To obtain an accurate reading, the clinician should rotate the prism so the red mark on the prism holder is set at the least curved meridian of the cornea (along the negative axis). Alternatively, 2 pressure readings taken 90° apart can be averaged.

Applanation tonometry measurements are also affected by the central corneal thickness (CCT). Recently the importance of CCT and its effect on the accuracy of IOP measurement has become better understood The Goldmann tonometer is most accurate, with a CCT of 520 µm; however, population studies have show

Increased CCT may give an artificially high IOP measurement; decreased CCT, an artificially low readin IOP measured after photorefractive keratectomy (PRK) and laser in situ keratomileusis (LASIK) may be reduced because of changes in the corneal thickness induced by these and other refractive procedures. As a rough guide, using an overview of published studies, it can be estimated that for every 10-µm difference in CCT from the population mean (approximately 542 µm), there is a 0.5 mm Hg difference between actua IOP and the IOP measured with a Goldmann tonometer. However, because the relationship of measured IOP and CCT is not linear, it is important to remember that such correction factors as this are only estimates at best. In addition, the biomechanical properties of an individual cornea may vary, resulting in changes of the relative stiffness or rigidity of the cornea and altering the measurement. The Goldmann tonometer, Perkins tonometer, pneumatonometer, noncontact tonometer, and Tono-Pen are all affected b CCT. Currently, there is no validated correction factor for the effect of CCT on applanation tonometers;

The Ocular Hypertension Treatment Study (OHTS) found that a thinner central cornea was a strong

The *Perkins tonometer* is a counterbalanced applanation tonometer that is portable and can be used with the patient either upright or supine. It is similar to the Goldmann tonometer in using a split-image device and fluorescein staining of the tears.

Table 2-1 Theoretical Examples of Difference in the Degree to Which the Intraocular Pressure (*P*) Is Calculated to Be Affected by Changes in Flow (*F*) and Facility of Outflow (*C*) in Different Types of Eyes, Assuming Constant Episcleral Venous Pressure (P_e [or P_v])

	(mm Hg) P_e	+	(μL/min) *F*	÷	(μL/min/mm Hg) *C)*	=	(mm Hg) *P*
Normal =	9		1.5		0.22		15
	9		1 to 2		0.22		13 to 17
	9		1.5		0.30		14
Glaucoma =	9		1.5		0.05		39
	9		1 to 2		0.05		29 to 49
	9		1.5		0.10		24
Good normal =	9		1.5		0.30		14
½ angle closed =	9		1.5		0.15		19
¾ angle closed =	9		1.5		0.075		29
Poor normal =	9		1.5		0.15		19
½ angle closed =	9		1.5		0.075		29
¾ angle closed =	9		1.5		0.0375		49

Modified with permission from Epstein DL, Allingham RR, Schuman JS. *Chandler and Grant's Glaucoma*. 4th ed. Baltimore: Williams & Wilkins; 1997:21.

Table 2-1

Theoretical Examples of Difference in the Degree to Which the Intraocular Pressure *(P)* Is Calculated to Be Affected by Changes in Flow *(F)* and Facility of Outflow *(C)* in Different Types of Eyes, Assuming Constant Episcleral Venous Pressure (P_e [or P_v])

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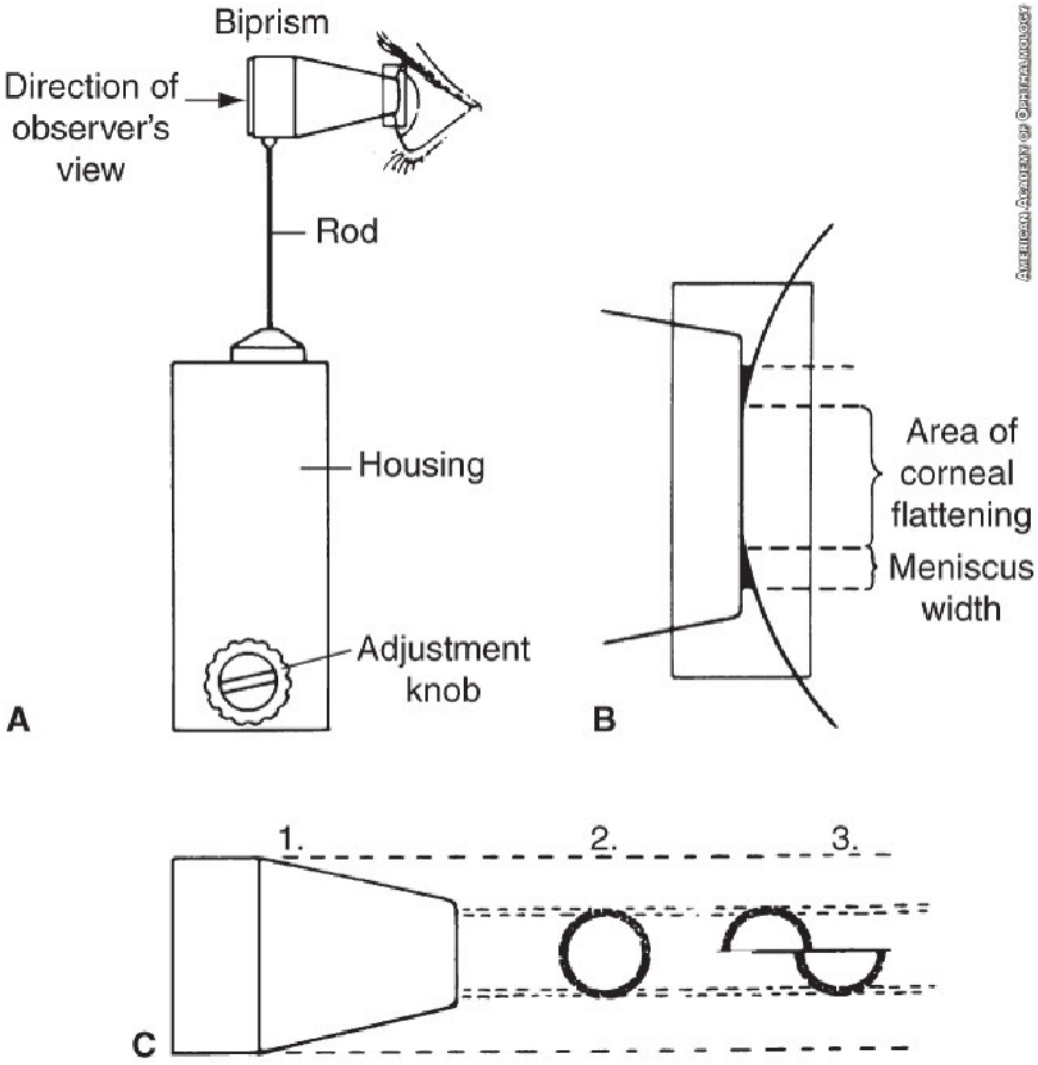

Figure 2-4

Goldmann-type applanation tonometry**A,** Basic features of tonometer, shown in contact with patient's cornea.**B,** Enlargement shows tear film meniscus created by contact of biprism and cornea.**C,** View through biprism*(1)* reveals circular meniscus *(2),* which is converted into semicircle *(3)* by prisms.

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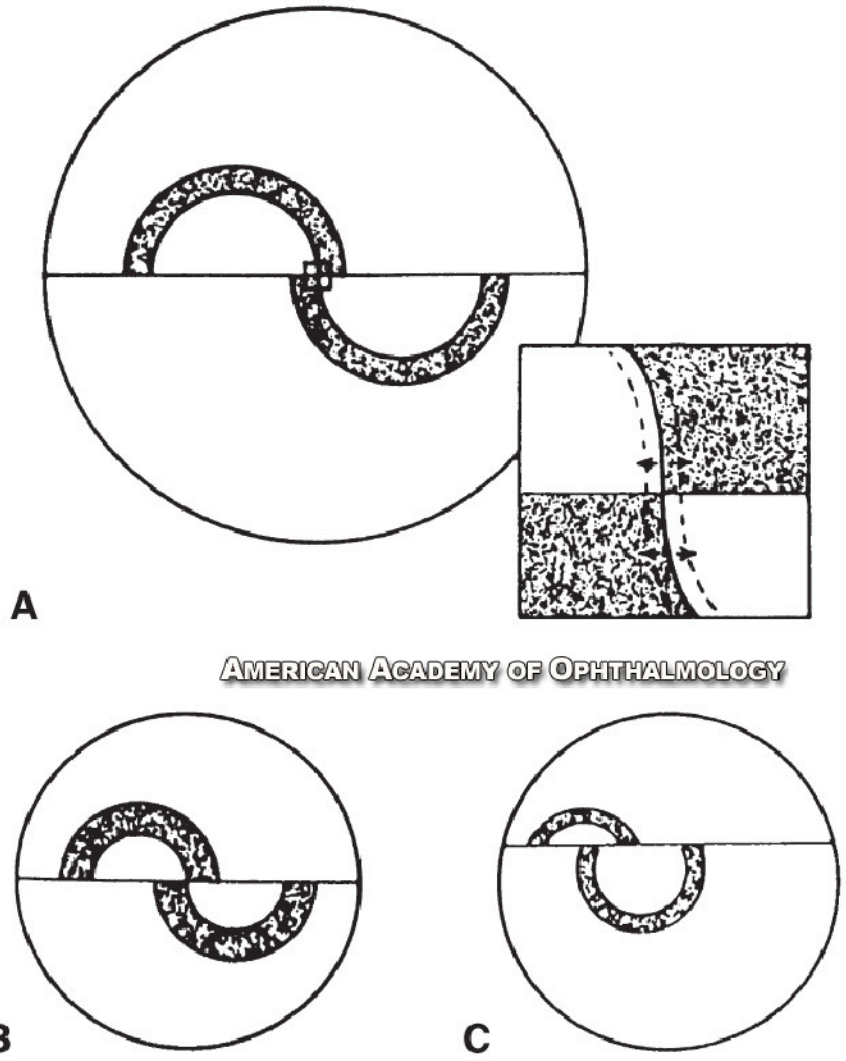

Figure 2-5

Semicircles of Goldmann-type applanation tonometer**A,** Proper width and position. Enlargement depicts excursions of semicircles caused by ocular pulsations. **B,** Semicircles are too wide. **C,** Improper vertical and horizontal alignment.
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Table 2-2 Factors That Affect Intraocular Pressure

Factors that may increase intraocular pressure
- Elevated episcleral venous pressure
  - Valsalva maneuver
  - Breath holding
  - Playing a wind instrument
  - Wearing a tight collar or tight necktie
  - Bending over or being in a supine position
  - Elevated central venous pressure
  - Orbital venous outflow obstruction
  - Intubation
- Pressure on the eye
  - Blepharospasm
  - Squeezing and crying, especially in young children
- Elevated body temperature: associated with increased aqueous humor production
- Hormonal influences
  - Hypothyroidism
  - Thyroid ophthalmitis
- Drugs unrelated to therapy
  - Lysergic acid diethylamide (LSD)
  - Topiramate (Topamax)
  - Corticosteroids
  - Anticholinergics: may precipitate angle closure

Factors that may decrease intraocular pressure
- Aerobic exercise
- Anesthetic drugs
  - Ketamine
  - Depolarizing muscle relaxants such as succinylcholine
- Metabolic or respiratory acidosis: decreases aqueous humor production
- Hormonal influences
  - Pregnancy
- Drugs unrelated to therapy
  - Alcohol consumption
  - Heroin
  - Marijuana (cannabis)

Table 2-2

Factors That Affect Intraocular Pressure

Table 2-3 Possible Sources of Error in Tonometry

Squeezing of the eyelids
Breath holding or Valsalva maneuver
Pressure on the globe
Extraocular muscle force applied to a restricted globe
Tight collar or tight necktie
Obesity or straining to reach slit lamp
An inaccurately calibrated tonometer
Excessive or inadequate amount of fluorescein
High corneal astigmatism
Corneal thickness greater or less than normal
Corneal biomechanical properties (eg, rigidity)
Corneal scarring or band keratopathy
Corneal irregularity
Technician errors

Table 2-3

Possible Sources of Error in Tonometry

Infection Control in Clinical Tonometry

Many infectious agents, including the viruses responsible for acquired immunodeficiency syndrome (AIDS hepatitis, and epidemic keratoconjunctivitis, can be recovered from tears. Tonometers must be cleaned after each use so that transfer of such agents can be prevented:

- The prism head of both Goldmann-type tonometers and the Perkins tonometer should be cleaned immediately after use. The prisms should either be soaked in a 1:10 sodium hypochlorite solution (household bleach), in 3% hydrogen peroxide, or in 70% isopropyl alcohol for 5 minutes, or be thoroughly wiped with an alcohol sponge. If a soaking solution is used, the prism should be rinsed a dried before reuse. If alcohol is employed, it should be allowed to evaporate, or the prism head shou be dried before reuse, to prevent damage to the epithelium.

- The front surface of the air-puff tonometer should be wiped with alcohol between uses because the instrument may be contaminated by tears from the patient.

- Portable electronic applanation devices employ a disposable cover, which should be replaced immediately after each use.

- The Schiøtz tonometer requires disassembly to clean both the plunger and the footplate. Unless the plunger is clean (as opposed to sterile), the measurements may be falsely elevated because of increased friction between the plunger and the footplate. A pipe cleaner can be used to clean the in of the footplate, removing tears and any tear film debris. The same solutions used for cleaning prism heads may then be employed to sterilize the instrument.

For other tonometers, consult the manufacturer’s recommendations.

Methods other than Goldmann-type applanation tonometry

The recognition that the accuracy of applanation tonometry is dependent on many uncontrollable factors has led to a renewed interest in the development of novel tonometric methodologies. In particular, new tonometers aim to lessen the potential inaccuracy secondary to differences in corneal thickness and rigidi One such technology is the *dynamic contour tonometer (DCT),* a nonapplanation contact tonometer that may be more independent of corneal biomechanical properties and thickness than are older tonometers.

Noncontact (air-puff) tonometers measure IOP without touching the eye, by measuring the time necessar for a given force of air to flatten a given area of the cornea. Readings obtained with these instruments var widely, and IOP is often overestimated with these instruments. The instruments are often used in large-scale glaucoma-screening programs or by nonmedical health care providers.

The group of *portable electronic applanation* devices (eg, Tono-Pen) that applanate a very small area of t cornea are particularly useful in the presence of corneal scars or edema. The *pneumatic tonometer,* or *pneumatonometer,* has a pressure-sensing device that consists of a gas-filled chamber covered by a Silastic diaphragm. The gas in the chamber escapes through an exhaust vent. As the diaphragm touches the cornea, the gas vent decreases in size and the pressure in the chamber rises. Because this instrume too, applanates only a small area of the cornea, it is especially useful in the presence of corneal scars or edema.

Schiøtz tonometry determines IOP by measuring the indentation of the cornea produced by a known weig The indentation is read on a linear scale on the instrument and is converted to millimeters of mercury by a calibration table. Because of a number of practical and theoretical problems, however, Schiøtz tonometry i now rarely used.

It is possible to estimate IOP by *digital pressure* on the globe. This test may be used with uncooperative patients, but it may be inaccurate even in very experienced hands. In general, tactile tensions are only useful for detecting large differences between 2 eyes.