

Open-Angle Glaucoma

Slow, painless rise in eye pressure that quietly damages the optic nerve and side vision.

THE STORY IN ONE SENTENCE

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“The drain inside the eye gets clogged over time, so pressure builds and crushes the nerve that sends pictures to the brain—stealing vision from the edges inward without any pain.”

Normal anatomy — the body's job

Your eye makes a clear fluid called aqueous humor every minute to keep the eyeball inflated and nourished. That fluid flows from behind the colored part of your eye (the iris), through the pupil into the front chamber, and then leaves through a spongy drain called the trabecular meshwork, which sits in the angle where the iris meets the cornea (the clear window at the front of your eye). From the drain, fluid flows into a tiny canal and then into the veins outside the eye. When fluid leaves at the same speed it is made, the pressure inside stays steady and safe.

BODY'S JOB

The aqueous humor brings nutrients to parts of the eye that have no blood vessels (like the lens and cornea) and carries away waste. The drain keeps pressure balanced so the eyeball holds its shape and the optic nerve at the back of the eye is not squeezed.

1. Ciliary body (a ring of tissue behind the iris) makes fresh aqueous humor.
2. Fluid flows forward through the pupil into the front chamber of the eye.
3. Fluid reaches the angle (the corner where the iris meets the cornea).
4. Fluid passes through the trabecular meshwork (a tiny spongy mesh drain).
5. Fluid drains into Schlemm's canal (a circular channel around the edge of the cornea).
6. Fluid flows out into small veins and rejoins the bloodstream.
7. Pressure inside the eye stays steady (normally 10–21 mm Hg).

What breaks

The trabecular meshwork—the sponge-like drain at the angle of the eye—slowly gets clogged with microscopic debris and the drain cells stop working as well. Even though the angle itself stays wide open (which is why it is called open-angle glaucoma), fluid cannot leave fast enough. Pressure climbs, and that steady squeezing crushes the delicate nerve fibers in the optic nerve one by one.

CLINICAL TERM

Progressive dysfunction of the trabecular meshwork leading to increased intraocular pressure and optic neuropathy.

How the body compensates

Your eye and brain have almost no way to compensate for rising pressure or dead nerve fibers. The eye cannot grow a new drain or make the old one work faster. The brain fills in missing spots in your side vision by guessing what should be there, so you may not notice blind patches until a large area is gone.

1. {'step': 'The fellow eye covers the deficit', 'why': 'Because you have two eyes with overlapping fields of view, your brain merges the pictures—so a blind spot in one eye is hidden by good vision in the other eye, and you feel like nothing is wrong.'}
2. {'step': 'Central vision stays sharp at first', 'why': 'The nerve fibers that serve the very center of your vision (where you read and recognize faces) are the last to die, so you can still see 20/20 on the letter chart even when large areas of side vision are already gone.'}
3. {'step': 'No compensation for lost nerve fibers', 'why': 'Once a retinal ganglion cell dies, it never grows back and no other cell can take over its job—the vision loss is permanent.'}

Domino effect

1. Trabecular meshwork clogs and outflow resistance rises — Aging, genetics, and debris slowly damage the drain cells, so fluid cannot leave the eye fast enough and pressure climbs above the safe range (often above 21 mm Hg).
2. Intraocular pressure squeezes the optic nerve head — The optic disc (where the nerve exits the back of the eye) is the weakest spot in the wall of the eyeball, so rising pressure bends and crushes the nerve fibers and the tiny blood vessels that feed them.
3. Optic nerve axons lose their blood supply and die — Squeezed capillaries cannot deliver oxygen; the long nerve fibers (axons) that run from the retina to the brain starve and break apart.

4. Retinal ganglion cell bodies die — When an axon is cut or crushed, the whole nerve cell dies—so patches of retina go dark and stop sending signals to the brain.
5. Peripheral visual field shrinks inward (tunnel vision) — The nerve fibers that serve side vision die first (they enter the optic nerve at the edges), so you lose the ability to see things off to the side, above, or below—while straight-ahead vision may still be clear.
6. Optic disc develops cupping — As nerve fibers disappear, the center of the optic disc caves inward like a bowl (increased cup-to-disc ratio), which an eye doctor can see during an exam.
7. Central vision is eventually lost if untreated — In late-stage disease, the pressure and starved blood flow (ischemia) (not enough blood reaching the tissue to feed it) finally destroy the central nerve fibers, and you go blind.
8. Damage is permanent and progressive without treatment — Dead nerve cells never regenerate, and rising pressure continues to kill more fibers every month—so early detection and pressure-lowering treatment (drops, laser, or surgery) are the only way to save the vision that remains.

Symptoms · what the patient feels, and why

- Gradual loss of side vision (peripheral vision): Pressure inside the eye builds up slowly over months or years. This pressure squeezes and damages the optic nerve (the cable that sends pictures from your eye to your brain). The nerve fibers on the outer edges die first, so you lose the ability to see things off to the side while your straight-ahead vision stays clear for a long time.
- Tunnel vision in late stages: As more and more optic nerve fibers die from ongoing pressure, the only vision left is a small circle straight ahead—like looking through a narrow tube. The outer ring of nerve fibers is already gone, so only the center bunch is still working.
- No pain: In open-angle glaucoma, the drain inside the eye clogs very slowly and gently. There is no sudden spike in pressure and no stretching of pain-sensing nerves in the eye, so you feel nothing even as damage builds.
- Difficulty seeing in dim light or at night: When part of the optic nerve is damaged, your brain gets fewer signals about contrast and detail. In low light, your eye needs every nerve fiber to build a clear picture—so any loss makes dim scenes look muddled or hard to read.
- Trouble adjusting when moving from bright to dark spaces: A healthy optic nerve quickly sends updated brightness signals to your brain. When nerve fibers are dying, the communication slows down and becomes patchy, so your brain takes longer to make sense of the new light level.

- Increased cup-to-disc ratio on eye exam: The optic nerve head (where the nerve enters the back of the eye) has a small dip in the center called the cup. Chronic high pressure kills nerve tissue around the cup, making the dip larger and deeper—like a pothole getting bigger. An eye doctor measures this change to track damage.
- Elevated intraocular pressure (often 21 mmHg or higher): Fluid is made inside the eye every day and drains out through a tiny mesh near the edge of the iris. In open-angle glaucoma, the mesh slowly clogs even though the drain opening looks normal. Fluid builds up faster than it leaves, so pressure climbs.
- Normal central vision until very late in the disease: The nerve fibers that carry straight-ahead (central) vision sit in the middle of the optic nerve bundle and are the last to die. Peripheral fibers on the outer ring are more fragile and fail first, so you can read and recognize faces long after your side vision is gone.

Focused exam

- Intraocular pressure (IOP) measured by tonometry: Normal eye pressure is 10-21 mmHg. In open-angle glaucoma, pressure usually stays above 21 mmHg. This high pressure slowly damages the nerve that sends images to your brain (the optic nerve), like water pressing too hard on a garden hose and crushing it over time.
- Optic nerve head examination (cup-to-disc ratio): The optic nerve has a small cup (dip) in the center. In glaucoma, the nerve tissue dies and the cup gets bigger. A cup-to-disc ratio above 0.6, or one eye's cup much bigger than the other (more than 0.2 difference), tells you the nerve is being damaged.
- Visual field testing (perimetry): This test maps what you can see at the edges of your vision. Glaucoma kills nerve fibers from the outside in, so you lose side vision first—often without noticing. The test catches blind spots (scotomas) before you feel anything wrong.
- Gonioscopy (looking at the drainage angle with a special lens): The eye makes fluid all the time, and that fluid drains out through tiny channels in the front angle of the eye. Gonioscopy lets the doctor see if the angle is open or blocked. Open-angle glaucoma means the angle looks open, but the drain still does not work well—like a sink drain that looks clear but water drains slowly anyway.
- Central corneal thickness (pachymetry): A thicker cornea (the clear front window of the eye) makes the pressure reading falsely high; a thinner cornea makes it falsely low. Knowing the thickness helps the doctor understand the true pressure and also tells risk—people with thin corneas (less than 555 microns) have higher risk of glaucoma damage.
- Optical coherence tomography (OCT) of the optic nerve and retinal nerve fiber layer: OCT takes detailed pictures of the nerve fiber layer (the nerve tissue around the optic nerve head). It measures the thickness in microns and compares it to healthy eyes. Thinning proves nerve damage and helps track whether treatment is working.

Diagnostics · every test answers a question

- Intraocular pressure (IOP) measurement by applanation tonometry → Is the pressure inside the eye high enough to damage the optic nerve?
- Automated visual field test (standard automated perimetry, such as Humphrey field analyzer 24-2 or 30-2) → Has the patient lost any side vision or developed blind spots from nerve damage?
- Optic nerve head photography and cup-to-disc ratio assessment → Is the optic nerve cupped or thinned in a way that matches glaucoma damage?
- Optical coherence tomography (OCT) of the retinal nerve fiber layer (RNFL) and ganglion cell complex → How much nerve tissue is left, and is it thinning over time?
- Gonioscopy → Is the drainage angle of the eye open or closed?
- Central corneal thickness (pachymetry) → Is the cornea thick or thin, and does that change how we interpret the eye pressure?

Treatment logic

- Prostaglandin analog eye drops (latanoprost, travoprost, bimatoprost, tafluprost) once nightly → These drops increase fluid drainage out of the eye through a second pathway (the uveoscleral route, which bypasses the clogged main drain). They lower eye pressure by about 25-35%, which is the biggest drop of any single medicine. Lower pressure slows or stops nerve damage.
- Beta-blocker eye drops (timolol, betaxolol, levobunolol) once or twice daily → These drops slow down the eye's production of fluid (aqueous humor), so less fluid builds up. They lower IOP by about 20-25%. Often added when a prostaglandin alone is not enough.
- Alpha-agonist eye drops (brimonidine, apraclonidine) two or three times daily → These drops both reduce fluid production and slightly increase drainage. They lower IOP by about 15-20%. Brimonidine may also protect the optic nerve directly (neuroprotection), though this is still being studied.
- Carbonic anhydrase inhibitor eye drops (dorzolamide, brinzolamide) two or three times daily → These drops reduce fluid production by blocking an enzyme (carbonic anhydrase) in the eye. They lower IOP by about 15-20%. They are often combined with a beta-blocker in one bottle for convenience.
- Selective laser trabeculoplasty (SLT) → A laser gently heats the trabecular meshwork (the drain) to wake it up and make it work better. It lowers IOP by about 20-30% and can replace or reduce the number of eye drops. The effect lasts 1-5 years on average, and the laser can be repeated if pressure goes back up.

- **Trabeculectomy (filtering surgery)** → The surgeon creates a new drain pathway by making a small flap in the sclera (the white of the eye) so fluid can escape into a space under the conjunctiva (the clear skin over the white). This lowers IOP more than drops or laser—often to the low teens or lower—and the effect usually lasts years. It is used when medicines and laser are not enough to stop vision loss.
- **Minimally invasive glaucoma surgery (MIGS, such as iStent, Xen gel stent, or goniotomy)** → These newer surgeries create small drainage pathways or bypass the clogged trabecular meshwork with tiny stents or cuts. They lower IOP modestly (usually to the mid-to-high teens) and have fewer complications than trabeculectomy. They are often done at the same time as cataract surgery.

Medications

Latanoprost (Xalatan) · Prostaglandin analog

Opens extra drainage pipes in the eye so the fluid inside can flow out faster, lowering the pressure.

1. The drug acts like a natural chemical called prostaglandin F2-alpha that tells certain muscle cells to relax.
2. When those muscles relax, the spaces between the cells in the uveoscleral pathway widen.
3. The aqueous humor (the clear fluid made inside the eye every day) drains out through this wider pathway instead of building up.
4. Less fluid sitting in the eye means lower pressure on the optic nerve.

SAFETY WATCH

Eye color may permanently darken (more brown pigment in the iris) over months to years.
· Eyelashes may grow longer and thicker. · Skin around the eyelid may darken slightly. · Eye redness or mild stinging right after the drop is common and usually harmless. · If you wear contact lenses, take them out before the drop and wait 15 minutes before putting them back in (the preservative can stick to soft lenses). · Do not use if you have active inflammation inside the eye (uveitis) or a history of certain viral eye infections, unless your doctor says it is safe.

Timolol (Timoptic) · Beta-blocker (non-selective)

Slows down the pump that makes fluid inside the eye, so less fluid is produced and pressure stays lower.

1. The ciliary body has tiny pumps driven by beta-2 receptors that constantly make new fluid.
2. Timolol blocks those beta-2 receptors like putting a dimmer on a light switch.

3. With the receptors blocked, the pumps slow down and make about 20 to 30 percent less fluid.
4. Less new fluid means less pressure building up inside the eye.

SAFETY WATCH

Can slow your heart rate or lower blood pressure because the drug is absorbed into your bloodstream through the eye. · Avoid if you have asthma, COPD, or certain heart rhythm problems (like heart block or very slow heart rate) — the drug can make breathing harder or heart rate dangerously slow. · May mask the warning signs of low blood sugar if you have diabetes and take insulin. · Can cause fatigue, shortness of breath, or cold hands and feet in some people. · Press gently on the inside corner of your eye (near your nose) for one minute after the drop to keep less drug from draining into your nose and throat, which reduces side effects.

Brimonidine (Alphagan P) · Alpha-2 agonist

Does two things: turns down the pump that makes fluid in the eye and opens an alternate drain so fluid flows out faster.

1. Brimonidine activates alpha-2 receptors on the cells that make aqueous humor, telling them to slow down production.
2. It also relaxes muscle and connective tissue around the uveoscleral pathway so fluid can drain more easily through that route.
3. Together, less fluid coming in and more fluid going out keeps the pressure lower.

SAFETY WATCH

Eye redness and allergic reactions (itching, swelling of the eyelid) are common — about 10 to 15 percent of people develop an allergy after weeks to months of use. · Can cause dry mouth, drowsiness, or fatigue because a small amount gets into your bloodstream. · Do not use in infants or very young children — it can cause severe sleepiness and even slow breathing. · Avoid alcohol and sedatives while using this drop, as drowsiness may be worse. · Press on the inside corner of your eye for one minute after the drop to reduce absorption into your body.

Dorzolamide (Trusopt) or Brinzolamide (Azopt) · Carbonic anhydrase inhibitor (topical)

Blocks an enzyme in the eye that helps make fluid, so the pump that produces aqueous humor runs slower and makes less fluid.

1. The cells in the ciliary body use an enzyme called carbonic anhydrase to move bicarbonate (a salt) and water into the eye, which becomes aqueous humor.
2. Dorzolamide or brinzolamide blocks that enzyme like jamming a gear in a machine.
3. Without the enzyme working well, less bicarbonate and water move into the fluid, so less new fluid is made.
4. Less fluid production means lower pressure in the eye.

SAFETY WATCH

Bitter or sour taste in your mouth right after using the drop is very common (the drug drains into your throat). · Stinging or burning in the eye for a few seconds after the drop. · If you are allergic to sulfa drugs, tell your doctor — these drops are sulfonamides and may cause a reaction (though true cross-allergy is rare). · Do not use if you have severe kidney disease — the drug can build up in your body. · Blurred vision for a few minutes after the drop is normal; wait before driving or doing tasks that need clear sight.

Pilocarpine · Cholinergic agonist (miotic)

Makes the pupil smaller and pulls on the drainage mesh in the eye, opening it wider so fluid drains out faster.

1. Pilocarpine mimics acetylcholine, a natural signal in the eye that tells the iris muscle to tighten.
2. When the iris tightens, the pupil gets smaller.
3. At the same time, the ciliary muscle contracts and pulls on the scleral spur, which stretches open the trabecular meshwork (the main drain).
4. A wider drain lets aqueous humor flow out faster, lowering pressure.

SAFETY WATCH

Your pupil will stay small for several hours, making it harder to see in dim light or at night — be careful driving after dark. · Brow ache or headache is common, especially when you first start the drop. · Objects may look slightly smaller or vision may seem dimmer. · Can rarely trigger a different kind of glaucoma (angle-closure) if your eye anatomy is at risk — your doctor will check this first. · Rarely used as first-line treatment today because newer drugs have fewer side effects, but still helpful in specific cases.

Netarsudil (Rhopressa) · Rho kinase inhibitor

Relaxes the cells in the main drain of the eye so fluid flows through more easily, and also lowers the pressure in the veins that carry fluid away from the eye.

1. Netarsudil blocks an enzyme called Rho kinase that keeps the cells in the trabecular meshwork stiff.
2. When those cells relax, the spaces between them widen and fluid flows through the drain more easily.
3. The drug also reduces pressure in the episcleral veins, which makes it easier for fluid to leave the eye (like opening a downstream valve).
4. Better outflow through the natural drain means lower pressure inside the eye.

SAFETY WATCH

Eye redness is very common (up to 50 percent of people) because the drug dilates tiny blood vessels on the white of the eye — it looks alarming but is usually harmless. · Small broken blood vessels (subconjunctival bleeding (hemorrhage)) on the white of the eye can happen and look like a red patch; they clear up on their own in a week or two. · Tiny deposits on the clear front of the eye (cornea) may form over time but usually do not affect vision. · Use once a day in the evening; it works best on that schedule.

Acetazolamide (Diamox) · Carbonic anhydrase inhibitor (oral)

Blocks the same enzyme as the eye drops (carbonic anhydrase) but works throughout the whole body, slowing down fluid production in the eye.

1. Acetazolamide is a pill that travels through your bloodstream to the ciliary body.
2. It blocks carbonic anhydrase, the enzyme that helps move bicarbonate and water into the aqueous humor.
3. With the enzyme blocked, less fluid is made inside the eye, and pressure drops.
4. The same enzyme is active in your kidneys, so you lose more bicarbonate and water in your urine (which is why you urinate more).

SAFETY WATCH

You will urinate more often and may feel thirsty — drink enough water. · Tingling or numbness in your fingers, toes, or around your mouth is very common (from changes in blood chemistry). · Foods may taste flat or metallic, especially carbonated drinks. · Can cause fatigue, loss of appetite, or nausea. · Lowers potassium levels in your blood — your doctor may check labs and tell you to eat potassium-rich foods (bananas, oranges, potatoes). · Do not use if you are allergic to sulfa drugs or have severe liver or kidney disease. · Usually used short-term (days to weeks) to quickly lower eye pressure in urgent situations, not as everyday treatment.

Red flags

- Sudden, severe eye pain (suggests acute angle-closure glaucoma instead, which is an emergency)
- Rapid vision loss over days or weeks (open-angle glaucoma is slow; fast loss means something else urgent is happening)
- Severe headache with nausea and vomiting along with blurry vision (points to sudden high pressure or another brain/eye emergency)
- Only one eye affected and vision dropping quickly (open-angle is usually both eyes; one-sided fast loss suggests stroke, detachment, or infection)

Nursing priorities

1. Teach the patient how to put in eye drops correctly and build a daily routine so no doses are missed. — The drops are the main treatment to lower eye pressure and protect what vision is left. But studies show about half of patients don't use them correctly or regularly. If drops are used wrong (like touching the dropper to the eye, not waiting between different drops, or missing days), the pressure stays high and the optic nerve keeps getting damaged. Walking through the technique step-by-step and linking it to something they already do every day (like brushing teeth) makes it far more likely to happen.
2. Check if the patient can afford their medications and help connect them to assistance programs if cost is a barrier. — Glaucoma drops can be expensive, and many patients skip doses or stop altogether because they cannot pay for refills. When treatment stops, eye pressure climbs back up and more permanent vision loss happens. Asking directly and non-judgmentally about cost, then helping find manufacturer programs, generic options, or community resources keeps the treatment plan from failing.

3. Assess the patient's current vision and daily safety, especially fall risk and ability to drive or move around safely. — Glaucoma takes away side vision first, which means the patient might not see curbs, steps, or cars coming from the side. This raises the risk of falls, car crashes, and injuries. Understanding what they can and cannot see helps us teach compensation strategies (like turning the head more, using better lighting, removing tripping hazards at home) and refer for low-vision services or occupational therapy when needed.
4. Reinforce the need for regular eye exams every 3 to 6 months or as prescribed by the eye doctor, even if vision feels fine. — Glaucoma damage is silent. The patient will not feel pain or notice vision changes until a large amount of the nerve is destroyed, and that damage cannot be reversed. Only the eye doctor's tests (measuring pressure, looking at the optic nerve, and checking the visual field) can catch worsening early enough to adjust treatment and save the remaining vision.
5. Teach warning signs of angle-closure glaucoma and instruct the patient to go to the emergency room immediately if they happen. — Even with open-angle glaucoma, a sudden blockage of the eye's drainage system can occur (angle-closure attack). This causes severe eye pain, headache, nausea, blurry vision, and seeing halos around lights. The pressure inside the eye spikes so high it can kill the optic nerve within hours. Getting to the ER right away for emergency treatment is the only way to prevent permanent blindness in that eye.

Sources

- American Academy of Ophthalmology Preferred Practice Pattern
- American Glaucoma Society
- National Eye Institute (NEI)
- Cochrane Eyes and Vision

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