Glaucoma refers to a group of optic neuropathies that present with progressive optic nerve head (ONH) damage and characteristic visual field (VF) loss. Elevated intraocular pressure (IOP) is the strongest risk factor for glaucoma, but it need not be present—IOP can be normal, or even relatively low. *We still don’t know the exact mechanism of axonal death in glaucoma*—but die they do.
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In addition to being the strongest risk factor for glaucoma, IOP has another quality that renders it unique: *It is the only glaucoma risk factor that is modifiable in a manner proven to mitigate the risk of glaucoma progression.* Thus, at present IOP reduction is the sole arrow in our glaucoma-treatment quiver. IOP reduction can be accomplished via hypotensive drops, laser surgery, or incisional (aka *filtering*) surgery; which modality is employed depends upon a number of clinical factors including (but far from limited to) glaucoma type and severity.
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In the course of this overview, we will unpack and expand upon the ideas presented above. Let’s start with IOP. We’ll look first at the variables that determine it, then at the physical principle underlying its measurement in the clinic.
The Goldmann equation identifies the variables that determine IOP and indicates how they relate to one another.

\[ \text{IOP} = \frac{\text{Aqueous Formation Rate (} \mu\text{L/min)} }{\text{Outflow Facility (} \mu\text{L/min/mmHg)}} + \text{Episceral Venous Pressure (mmHg)} \]
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Glaucoma Overview

The Goldmann equation

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Note also that the units \( \mu L/min \) cancel out, leaving IOP in mmHg.
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Let’s interpret it anyway, just to be sure we’re on the same page:
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--If the fraction’s numerator (aqueous formation) goes up, its value goes up.
The Goldmann equation

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Let’s interpret it anyway, just to be sure we’re on the same page:

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--If its denominator (aqueous outflow) goes up, its value goes down
Glaucoma Overview

The Goldmann equation

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Note that the Goldmann equation implies three means by which IOP can be lowered:

a) 

b) 

c)
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Glaucoma Overview

The Goldmann equation

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b) increase the fraction’s denominator, ie, increase aqueous outflow; or
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The Goldmann equation

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**The Goldmann equation**

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IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episceral Venous Pressure (mmHg)}
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Let’s take a look at IOP reduction via decreasing aqueous formation.
The Goldmann equation identifies the variables that determine IOP and indicates how they relate to one another. Note first that the mathematics of the equation—\( IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episceral Venous Pressure (mmHg)} \)—are straightforward, and easy to interpret.

Note that the Goldmann equation implies three means by which IOP can be lowered: 

a) reduce the fraction’s numerator, i.e., decrease aqueous formation;

b) increase the fraction’s denominator, i.e., increase aqueous outflow; or

c) decrease that number at the end, i.e., reduce EVP.

Three classes of meds decrease aqueous formation:

--\( \beta \) blockers

--Carbonic anhydrase inhibitors (CAIs)

--\( \alpha \) agonists
The Goldmann equation identifies the variables that determine IOP and indicates how they relate to one another. Note first that the mathematics of the equation—\( IOP = \text{fraction} + \text{number} \)—are straightforward, and easy to interpret.

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Two related laser procedures—cyclophotocoagulation and endocyclophotocoagulation—can reduce aqueous production surgically.
Glaucoma Overview

**The Goldmann Equation**

\[
IOP = \frac{\text{Aqueous Formation Rate (μL/min)}}{\text{Outflow Facility (μL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}
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a) reduce the fraction’s numerator, ie, decrease aqueous formation;

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c) decrease that number at the end, ie, reduce EVP.

**Next let’s consider IOP reduction via increasing aqueous outflow**
The Goldmann equation

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So as implied by the equation, lowering IOP requires that one or more of the following be accomplished:

a) reduce the fraction’s numerator, ie, decrease aqueous formation;
b) increase the fraction’s denominator, ie, increase aqueous outflow;
c) decrease that number at the end, ie, reduce EVP.

There are two types of outflow: through the trabecular meshwork (TM), and via the uveoscleral pathway.
Glaucoma Overview

The Goldmann equation

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IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episceral Venous Pressure (mmHg)}
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---

The TM is located in the angle. Aqueous passes through the TM to enter Schlemm’s canal; from Schlemm’s canal it passes through collector channels to empty into the episcleral venous plexus.

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Glaucoma Overview

**The Goldmann equation**

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TM outflow can be enhanced medically with parasympathomimetics, e.g., pilo. (This med is no longer commonly prescribed.)

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Aqueous Formation Rate, Outflow Facility, Episceral Venous Pressure.
Glaucoma Overview

**The Goldmann equation**

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There are two types of outflow: through the trabecular meshwork (TM), and via the uveoscleral pathway.

TM outflow can be enhanced medically with parasympathomimetics, e.g., pilo. (This med is no longer commonly prescribed.) Surgically, TM outflow can be enhanced several ways. Laser trabeculoplasty is a highly effective office-based procedure.
Glaucoma Overview

The Goldmann equation

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There are two types of outflow: through the trabecular meshwork (TM), and via the uveoscleral pathway.

TM outflow can be enhanced medically with parasympathomimetics, eg, pilo. (This med is no longer commonly prescribed.) Surgically, TM outflow can be enhanced several ways. Laser trabeculoplasty is a highly effective office-based procedure. Additionally, TM outflow can be surgically enhanced at the time of cataract extraction via one of several different so-called minimally-invasive glaucoma surgeries (MIGS). These include disruption or removal of a portion of the TM, creating a permanent breach in it with an implantable conduit, or cannulating and dilating Schlemm’s canal.
Creation of an artificial conduit through it with an implanted bypass stent

Disruption or removal of a portion of the TM

MIGS

Enlargement of Schlemm’s canal via cannulation and dilation
Glaucoma Overview

The Goldmann equation

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**Uveoscleral outflow** occurs when aqueous percolates through the ciliary body and into the suprachoroidal space. From there it passes through the sclera, probably along passageways that accommodate nerves and blood vessels.

There are two types of outflow: through the trabecular meshwork (TM), and via the **uveoscleral pathway**.

- b) increase the fraction's denominator, i.e., **increase aqueous outflow**;
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Glaucoma Overview

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_Uveoscleral outflow_ occurs when aqueous percolates through the ciliary body and into the suprachoroidal space. From there it passes through the sclera, probably along passageways that accommodate nerves and blood vessels. Per the _Glaucoma_ book, it's estimated that 5-15% of total aqueous outflow occurs via the uveoscleral pathway—perhaps even more in younger people.

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Glaucoma Overview

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- a) reduce the fraction’s numerator, ie, decrease aqueous formation;
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Uveoscleral outflow can be enhanced medically with prostaglandin analogues (PGAs), eg, latanaprost, bimatoprost and travaprost. These are among the most commonly prescribed glaucoma drops.
The Goldmann Equation

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Uveoscleral outflow can be enhanced medically with prostaglandin analogues (PGAs), e.g., latanaprost, bimatoprost and travaprost. These are among the most commonly prescribed glaucoma drops. As of this writing, there are no IOP-lowering surgical procedures that work by intentionally increasing uveoscleral outflow. (FDA approval was withdrawn for the one MIGS procedure that attempted to enhance it.)
Glaucoma Overview

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As previously mentioned, the \( \alpha \)-agonists reduce aqueous production. However, one of them (apraclonidine) also lowers EVP to some extent.
Glaucoma Overview

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Finally: There is an important IOP-lowering maneuver that is **not** implied by the Goldmann equation: **Dehydration of the vitreous** with a hyperosmotic agent (eg, mannitol).
Glaucoma Overview

**The Goldmann equation**

\[ \text{IOP} = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episceral Venous Pressure (mmHg)} \]

We’ve discussed suppressing aqueous formation—now let’s talk about where aqueous is made, and review how it circulates.
Glaucoma Overview

The Goldmann equation

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Aqueous is formed in the nonpigmented epithelium of the pars plicata portion of the ciliary body.
Glaucoma Overview

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Aqueous is formed in the **nonpigmented epithelium** of the pars plicata portion of the ciliary body. And yes, as implied by the fact that aqueous is made in a ‘nonpigmented’ epithelium, it is the case that the ciliary body has a pigmented epithelium as well.
Glaucoma Overview

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Let’s take a look at the structures involved in aqueous formation, starting with the ciliary body and its anatomy.
Glaucoma Overview

The ciliary body has two parts: The **pars plana** and the **pars plicata**
Glaucoma Overview

Ciliary body: Another view
Glaucoma Overview

Ciliary body: Another view
An aside: The pars plana of the eye is the structure through which intravitreal injections are performed (it is aka ‘the pincushion of the eye’)
“Aqueous is formed in the nonpigmented epithelium of the pars plicata portion of the ciliary body”

Now let’s look at the CB epithelium. Low power photomicrograph.
“Aqueous is formed in the nonpigmented epithelium of the pars plicata portion of the ciliary body”

Now let’s look at the CB epithelium. **Higher.**
“Aqueous is formed in the nonpigmented epithelium of the pars plicata portion of the ciliary body.”

Now let’s look at the CB epithelium. **High.** Now we can clearly identify both epithelial layers.
This image illustrates how aqueous, created in the pars plicata epithelium, empties into the posterior chamber, flows through the pupil into the AC, and eventually exits via the TM. (Egress via the U/S pathway is not depicted.)
Glaucoma refers to a group of optic neuropathies that present with progressive optic nerve head (ONH) damage and characteristic visual field (VF) loss. Elevated intraocular pressure (IOP) is the strongest risk factor for glaucoma, but it need not be present—IOP can be normal, or even relatively low.

In addition to being the strongest risk factor for glaucoma, IOP has another quality that renders it unique: it is the only glaucoma risk factor that is modifiable in a manner proven to mitigate the risk of glaucoma progression. Thus, at present IOP reduction is the sole arrow in our glaucoma-treatment quiver. IOP reduction can be accomplished via hypotensive drops, laser surgery, or incisional (aka filtering) surgery; which modality is employed depends upon a number of clinical factors including (but far from limited to) glaucoma type and severity. Each modality reduces IOP via one of two mechanisms—either by interfering with the production of aqueous humor, or by facilitating its egress from the intraocular space.

In the course of this overview, we will unpack and expand upon the ideas presented above.
The optic nerves are composed of the axons of retinal ganglion cells. Estimates vary, but it’s safe to say the typical optic nerve contains about 1.2M axons. These axons do not synapse in the region of the optic nerve head; rather, most will synapse in the lateral geniculate nucleus, or LGN. (Most of the others are involved in the pupillary light reflex; they peel off just prior to reaching the LGN, heading instead to the pretectum of the dorsal midbrain.)
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Anatomically speaking, the optic nerve has four portions: The *intraocular*, *intraorbital*, *intracanalicular*, and *intracranial*.

<table>
<thead>
<tr>
<th>Portion</th>
<th>Length (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intraocular</td>
<td>1</td>
</tr>
<tr>
<td>Intraorbital</td>
<td>25-30</td>
</tr>
<tr>
<td>Intracanalicular</td>
<td>4-10</td>
</tr>
<tr>
<td>Intracranial</td>
<td>10</td>
</tr>
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</table>
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Anatomically speaking, the optic nerve has four portions: The *intraocular*, *intraorbital*, *intracanalicular*, and *intracranial*.

Also anatomically speaking, the optic nerve head has four portions as well: The *nerve fiber layer (NFL)*, *pre-laminar*, *laminar*, and *retrolaminar*. Each has its own blood supply.

<table>
<thead>
<tr>
<th>Portion</th>
<th>Blood supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>NFL portion</td>
<td>Central retinal artery (CRA)</td>
</tr>
<tr>
<td>Pre-laminar</td>
<td>Short posterior ciliary arteries</td>
</tr>
<tr>
<td>Laminar</td>
<td>Arterial circle of Zinn &amp; Haller</td>
</tr>
<tr>
<td>Retrolaminar</td>
<td>Centrifugal CRA branches, centripetal pial branches</td>
</tr>
</tbody>
</table>
For reasons that have yet to be fully elucidated, glaucomatous optic neuropathy tends to damage the superior and inferior poles of the ONH preferentially and early, producing thinning at the poles (focal thinning is often referred to as a ‘notch.’)
Because of this tendency, ophthalmologists focus on the vertical cup-disc ratio (VCDR) when assessing a pt’s glaucoma status.

For reasons that have yet to be fully elucidated, glaucomatous optic neuropathy tends to damage the superior and inferior poles of the ONH preferentially and early, producing thinning at the poles (focal thinning is often referred to as a ‘notch.’)
Note that the VCDR can be misleading in this regard, as it can be quite pronounced in some normal eyes (especially those with a large disc).
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Thus, in determining the glaucomatous-ness of an ONH, don’t just rely on the VCDR--make sure you also inspect and critically evaluate the status of the neuroretinal rim.
The nonglaucomatous neuroretinal rim tends to follow what’s known as the *ISNT rule*: In decreasing order, the rim is thickest at its **Inferior**, **Superior**, **Nasal**, and **Temporal** portions. If an ONH’s rim adheres to this rule, it *ISNT* glaucomatous.
The nonglaucomatous neuroretinal rim tends to follow what’s known as the **ISNT rule**: In decreasing order, the rim is thickest at its Inferior, Superior, Nasal, and Temporal portions. If an ONH’s rim adheres to this rule, it **ISNT** glaucomatous.

**Note:** Not all glaucoma docs find the ISNT rule to be helpful—YMMV. Ask!
Now consider the ONH and retina in cross section. Note that the RNFL and ONH are both organized in a specific fashion:
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--The RNFL is stacked *vertically*, with fibers that originate at points distant from the ONH running at the bottom (ie, closer to the RPE); and
Now consider the ONH and retina in cross section. Note that the RNFL and ONH are both organized in a specific fashion:

--The RNFL is stacked *vertically*, with fibers that originate at points distant from the ONH running at the bottom (ie, closer to the RPE); and

--The ONH is stacked *horizontally*, with its peripheral-most fibers being those originating in the far retina, and its innermost fibers originating in the peripapillary region.
Now let’s look at the \textit{topography of the retinal nerve fiber layer}, and how that topography relates to the structure of the ONH.
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Now let’s look at the topography of the retinal nerve fiber layer, and how that topography relates to the structure of the ONH.
First, take note of the horizontal raphé. Fibers do not cross this anatomic boundary—those superior to it join the superior ONH, and those inferior to it, the inferior ONH.
Next, the papillomacular (PM) bundle—the swath of nerve fibers originating in the foveal region. Note how this bundle takes up the lion’s share of the temporal ONH.
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Next, the **papillomacular (PM) bundle**—the swath of nerve fibers originating in the foveal region. Note how this bundle takes up the lion’s share of the temporal ONH.

Finally, note how the PM bundle impacts the structure of the ONH. Because the bundle takes up the temporal ONH, fibers from the temporal perifoveal region and beyond are forced to ‘loop around’ it, and end up joining the ONH near its superior and inferior poles.
Because there are so many fibers at the superior and inferior poles, the normal ONH rim tends to be thicker at these sites. (This accounts for the relative proportions of the rim segments as captured by the ISNT rule described previously.)
Glaucoma Overview

Note also that a *vertical meridian* can be described in the retina as well. Unlike the horizontal raphé (which is physically instantiated in the anatomy of the retina), this vertical meridian is purely functional—it cannot be identified via histological examination of the retina.
Glaucoma Overview

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Glaucoma Overview

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The optic chiasm provides the anatomic nexus for the vertical meridian found in the visual fields.
Glaucoma Overview

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The *optic chiasm* provides the anatomic nexus for the vertical meridian found in the visual fields. Recall it is at the chiasm that the afferent signal is divvied up in terms of right and left hemifields, with fibers associated with the right hemifield projecting to the left at the chiasm, and fibers associated with the left hemifield projecting to the right.
Note also that a *vertical meridian* can be described in the retina as well. Unlike the horizontal raphé (which is physically instantiated in the anatomy of the retina), this vertical meridian is purely functional—it cannot be identified via histological examination of the retina. Instead, it is identified via *visual field testing*. Fixation divides the VF into nasal and temporal fields, with the photoreceptors (PRs) responsible for the temporal VF being nasal to the vertical meridian, and those responsible for the nasal VF located temporal to it.

Finally, note that fixation also divides the VF into superior and inferior VFs. The corresponding portions of the retina related topographically to the horizontal raphé.
Putting it all together: The VF can be divided into four quadrants. Together, retinal topography and ONH structure dictate that each quadrants corresponds with a particular anatomic location on the ONH.
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Glaucoma Overview

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We’ve discussed how visual fields relate to the ONH and retina; now let’s take a closer look at VF themselves.
Here is a representation of the VF for each eye. Which is OD, and which OS?
Here is a representation of the VF for each eye. Which is OD, and which OS? Remember, VFs are not drawn as if the pt is looking at you; they’re drawn as if you are the pt!
Measured in degrees from fixation, this is how far the normal VF extends superiorly, inferiorly, nasally and temporally. (Don’t get too fixated on these specific numbers—different sources will give slightly different values.)
Glaucoma Overview

Again when measured in degrees from fixation…
Glaucoma Overview

Again when measured in degrees from fixation… *The perimetry tests used most often in clinical practice assess only the central 24 degrees of the visual field!*
Glaucoma Overview

The **blind spot** on a VF is about 15 degrees from fixation
For reasons that have yet to be fully elucidated, glaucoma initially ‘prefers’ to damage the superior and inferior poles of the ONH. This leads to thinning at the poles (focal thinning is referred to as a ‘notch.’)
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For reasons that have yet to be fully elucidated, glaucoma initially "prefers" to damage the superior and inferior poles of the ONH. This leads to thinning at the poles (focal thinning is referred to as a 'notch'.) Specifically, glaucoma tends initially to affect fibers that originate on the temporal side of the vertical meridian.

The result of this is that glaucomatous VF defects appear in and extend from the nasal visual field.
Define glaucoma. A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss.

Why isn’t elevated IOP mentioned above? Elevated IOP is a strong risk factor for glaucoma, but it need not be present—IOP can be normal, or even low.

In addition to being the strongest risk factor for glaucoma, IOP has another quality that renders it unique—what is it? It is the only risk factor that is modifiable in a manner proven to influence the risk of glaucoma progression.

It was noted initially that glaucoma presents with “characteristic VF loss.” That’s what we’re getting at here. Let's take a detailed look at the way glaucomatous VF defects appear and progress.
Note: The following set of VFs are from a pt who suffered severe, progressive VF loss in a manner classic for glaucomatous optic neuropathy. I am not personally familiar with this case, and thus cannot provide context regarding the clinical circumstances that resulted in such profound, unchecked VF loss.
The first location at which glaucomatous VF manifests is near the nasal limit of a 24-2 field, sitting on (or ‘hanging’ just below) the horizontal midline. This pattern of loss is called a *nasal step*. 
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This location in the VF...is associated with this location on the retina, meaning that the affected nerve fibers originated there...and entered the ONH peripherally.
If left untreated, the nasal step will gradually enlarge.
If left untreated, the nasal step will gradually enlarge.
As glaucoma damage progresses, further loss of nerve fibers joining at that portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an **arcuate**.
Glaucoma Overview

As glaucoma damage progresses, further loss of nerve fibers at the superior portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an *arcuate*.

Note the area of origin for affected fibers now extends all the way to the ONH itself.
As glaucoma damage progresses, further loss of nerve fibers joining at that portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an arcuate.

Note also that an early inferior nasal step is now present.
Glaucoma Overview

If left unchecked, an arcuate will expand into the surrounding portion of the VF.
Once an arcuate has expanded sufficiently, it becomes an altitudinal defect. The superior visual field is now all but gone.
Once an arcuate has expanded sufficiently, it becomes an altitudinal defect. The superior visual field is now all but gone. The inferior nasal step continues to enlarge.
Glaucoma Overview

The inferior step is now an arc, and appears destined to become altitudinal, resulting in blindness.
Glaucoma is a progressive condition, passing from undetectable early disease...
Glaucoma is a progressive condition, passing from undetectable early disease... to asymptomatic-but-detectable (via RNFL imaging) disease...
Glaucoma is a progressive condition, passing from undetectable early disease… to asymptomatic-but-detectable (via RNFL imaging) disease... to functional (ie, marked by VF loss) disease...
Glaucoma is a progressive condition, passing from undetectable early disease... to asymptomatic-but-detectable (via RNFL imaging) disease... to functional (i.e., marked by VF loss) disease... to severe vision loss and blindness.
Glaucoma is a progressive condition, passing from undetectable early disease... to asymptomatic-but-detectable (via RNFL imaging) disease... to functional (ie, marked by VF loss) disease... to severe vision loss and blindness. This stepwise pattern of progression has been coined the glaucoma continuum.
In this regard, a word on the notion of ‘early’ glaucoma. We previously described the above VF defect as an ‘early’ nasal step.
In this regard, a word on the notion of ‘early’ glaucoma. We previously described the above VF defect as an ‘early’ nasal step. But take note of the point along the glaucoma continuum at which such a VF defect occurs—clearly, it doesn’t qualify as ‘early’ disease with respect to the continuum. Don’t mistake early VF changes for early disease!
Finally, let’s look briefly at how one should think through the new glaucoma case sitting in your exam chair.
The first thought you should have when encountering a pt you suspect has glaucoma is…
Glaucoma Overview

What is the status of the angle?

The first thought you should have when encountering a pt you suspect has glaucoma is…

*What is the status of the angle?*
Glaucoma Overview

Glaucoma

Open-angle

Closed- or narrow-angle

The first thought you should have when encountering a pt you suspect has glaucoma is…

What is the status of the angle?

Note that there is but one way to determine the status of the angle, and that is gonioscopy. Don’t assume your glaucoma pt has open angles—prove it by performing gonio!
Glaucoma Overview

OAG

If you have determined a pt has open-angle glaucoma, your next question is…
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Is it primary open-angle glaucoma (POAG), or secondary OAG?
If you have determined a pt has open-angle glaucoma, your next question is…

Is it primary open-angle glaucoma (POAG), or secondary OAG?

There is only one way to make this determination, and that is by ruling out all the causes of secondary OAG. This is because POAG is a diagnosis of exclusion.
These are the causes of secondary OAG that must be ruled out. *Do not memorize the chart*—learning the 2ndry OAGs is a task for another day. For now, just be aware they exist.
POAG is quite prevalent in the US, affecting about 2% of the over-40 population. It is a leading cause of blindness worldwide, second only to cataracts in this regard.
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The BCSC *Glaucoma* book emphasizes five risk factors for POAG (other than IOP):

1. **Race** is well established in this regard, with individuals of black and Hispanic heritage at a significantly greater risk than whites (their relative risk of going blind is higher as well).

2. 

3. 

4. 

5. 
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1. **Race** is well established in this regard, with individuals of black and Hispanic heritage at a significantly greater risk than whites (their relative risk of going blind is higher as well).
2. **Age** is a strong risk factor for both having POAG, and for POAG progression.
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1. **Race** is well established in this regard, with individuals of black and Hispanic heritage at a significantly greater risk than whites (their relative risk of going blind is higher as well).
2. **Age** is a strong risk factor for both having POAG, and for POAG progression.
3. **Family history** of a first-degree relative is significant.
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3. *Family history* of a first-degree relative is significant.
4. *Myopia* has been determined to be a risk factor by most (but not all) studies looking at the subject.
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3. **Family history** of a first-degree relative is significant.
4. **Myopia** has been determined to be a risk factor by most (but not all) studies looking at the subject.
5. **Thin central cornea** was established as a risk factor in the *Ocular Hypertension Treatment Study* (OHTS).
Angle-Closure Glaucoma

Next we will turn our attention to angle-closure glaucoma
If you have determined a pt has angle-closure glaucoma, your next question is:
If you have determined a pt has angle-closure glaucoma, your next question is: *Is it primary angle closure, or secondary?*
If you have determined a pt has angle-closure glaucoma, your next question is: *Is it primary angle closure, or secondary?*

In secondary ACG, a specific pathological cause of the angle closure can be identified, whereas no such cause is present in primary dz.
Like the 2ndry OAGs, the 2ndry ACGs are plentiful in number and diverse in cause. And as with the 2ndry OAGs, learning their names is a task for another day.
If you have determined a pt has angle-closure glaucoma, your next question is:

*Is it primary angle closure, or secondary?*

In secondary ACG, a specific pathological cause of the angle closure can be identified, whereas no such cause is present in primary dz. There are four subtypes of PACG:
If you have determined a pt has angle-closure glaucoma, your next question is: *Is it primary angle closure, or secondary?*

In *secondary ACG*, a specific pathological cause of the angle closure can be identified, whereas no such cause is present in primary dz. There are *four subtypes of PACG*:

- Acute
- Subacute
- Chronic
- Plateau iris

*These are discussed in detail in set G18. For now, let’s talk more generally about primary ACG*
While not as widespread as its open-angle cousin, PACG is a common cause of glaucoma and blindness worldwide. As with POAG, increasing age is a strong risk factor, with the incidence in individuals >40 considerably higher than that of those younger. Likewise, a family history of PACG is a risk factor, just as it is for POAG.
While not as widespread as its open-angle cousin, PACG is a common cause of glaucoma and blindness worldwide. As with POAG, increasing age is a strong risk factor, with the incidence in individuals >40 considerably higher than that of those younger. Likewise, a family history of PACG is a risk factor, just as it is for POAG. As with POAG, ethnic background is a risk factor, but the alignment of ethnicity with risk is roughly reversed—individuals of Asian ancestry are at highest risk, with those of African and European heritage at relatively low risk. (The latest version of the BCSC Glaucoma volume at the time of this writing doesn’t address the risk in the Latinx community.)
While not as widespread as its open-angle cousin, PACG is a common cause of glaucoma and blindness worldwide. As with POAG, increasing age is a strong risk factor, with the incidence in individuals >40 considerably higher than that of those younger. Likewise, a family history of PACG is a risk factor, just as it is for POAG. As with POAG, ethnic background is a risk factor, but the alignment of ethnicity with risk is roughly reversed—individuals of Asian ancestry are at highest risk, with those of African and European heritage at relatively low risk. (The latest version of the BCSC Glaucoma volume at the time of this writing doesn’t address the risk in the Latinx community.) Note that the ethnic heritage associated with the highest relative risk is that of the Inuit peoples of Alaska and Greenland, with a relative risk estimated to be 40-50 times that incurred by those of African/European heritages.
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