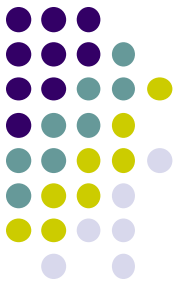
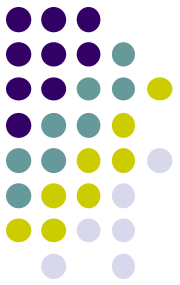


Glaucoma Overview



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.*

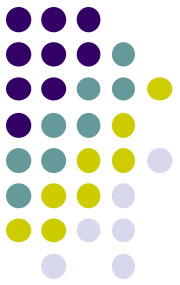
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In addition to being the strongest risk factor for glaucoma, IOP has another quality that renders it unique: *It is the only glaucoma risk factor that is modifiable in a manner proven to mitigate the risk of glaucoma progression.* Thus, at present 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.

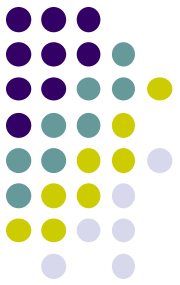
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Glaucoma Overview

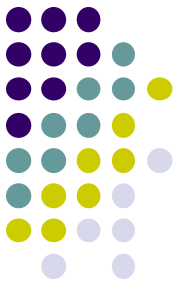


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In the course of this overview, we will unpack and expand upon the ideas presented above. Let's start with IOP. We'll look first at the variables that determine it, then at the physical principle underlying its measurement in the clinic.

Glaucoma Overview

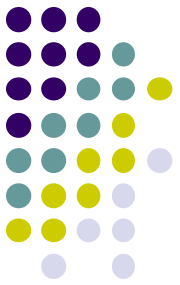


The Goldmann equation

$$IOP = \frac{\text{Aqueous Formation Rate } (\mu\text{L/min})}{\text{Outflow Facility } (\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}$$

The *Goldmann equation* identifies the variables that determine IOP and indicates how they relate to one another.

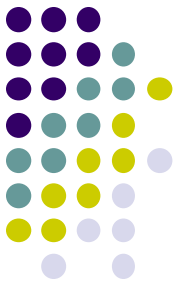
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Glaucoma Overview

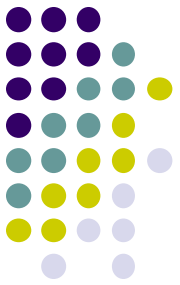
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Note also that the units $\mu\text{L}/\text{min}$ cancel out, leaving IOP in mmHg.

Glaucoma Overview



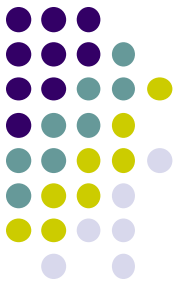
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Glaucoma Overview



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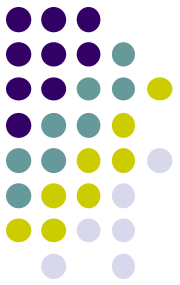
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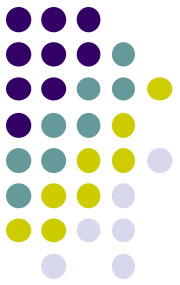
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Glaucoma Overview



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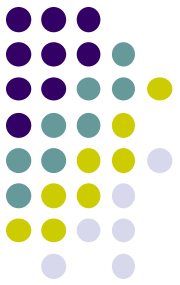
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Glaucoma Overview



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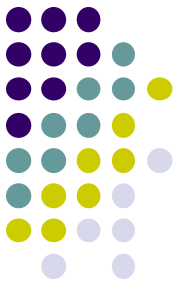
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Glaucoma Overview



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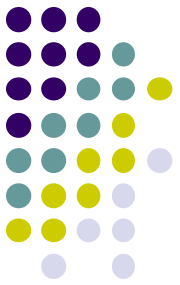
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Glaucoma Overview



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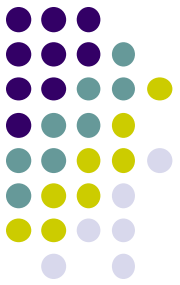
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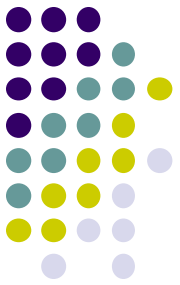
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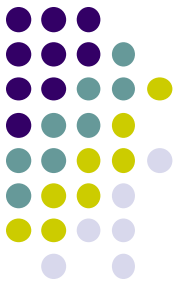
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Let's take a look at IOP reduction via decreasing aqueous formation

Glaucoma Overview



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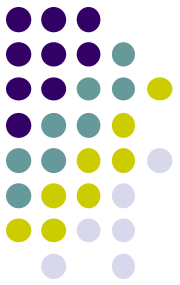
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Three classes of meds decrease aqueous formation:

- β blockers
- Carbonic anhydrase inhibitors (CAIs)
- α agonists

Glaucoma Overview



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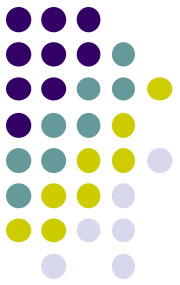
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Two related laser procedures—*cyclophotocoagulation* and *endocyclophotocoagulation*—can reduce aqueous production surgically

Glaucoma Overview



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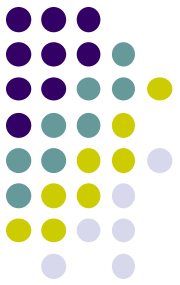
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*Next let's consider IOP reduction via **increasing aqueous outflow***

Glaucoma Overview



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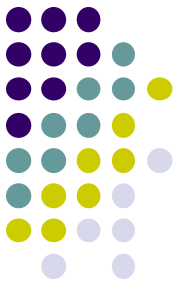
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There are two types of outflow: through the *trabecular meshwork* (TM), and via the *uveoscleral pathway*.

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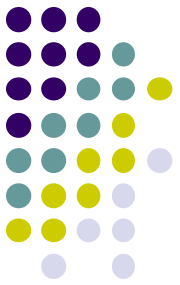
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The TM is located in the angle. Aqueous passes through the TM to enter Schlemm's canal; from Schlemm's canal it passes through collector channels to empty into the episcleral venous plexus.

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Glaucoma Overview



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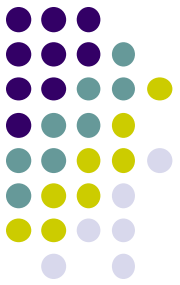
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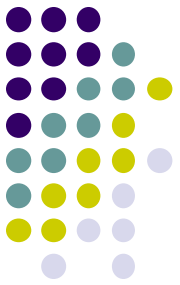
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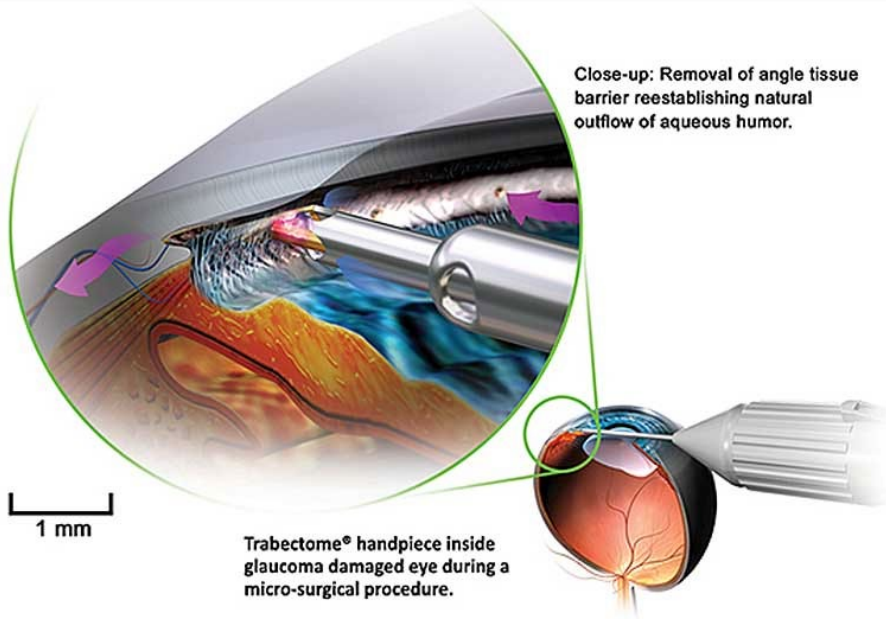
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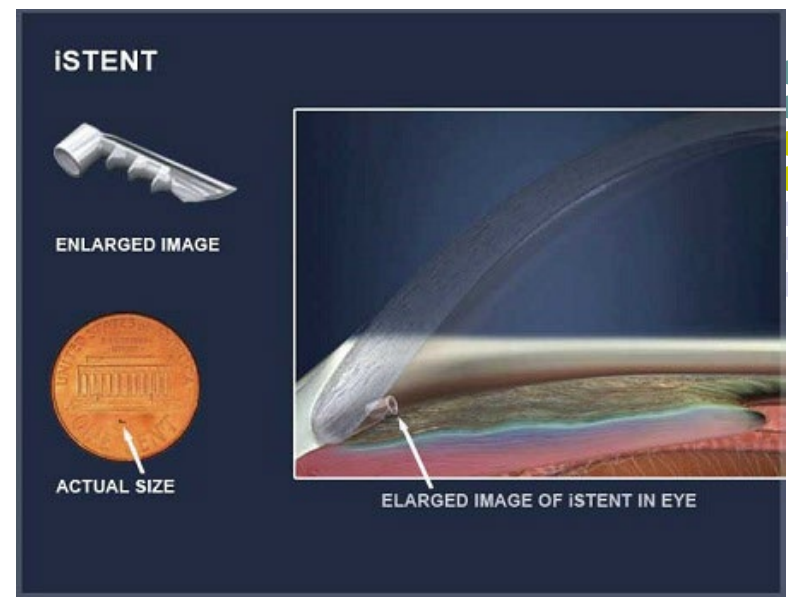
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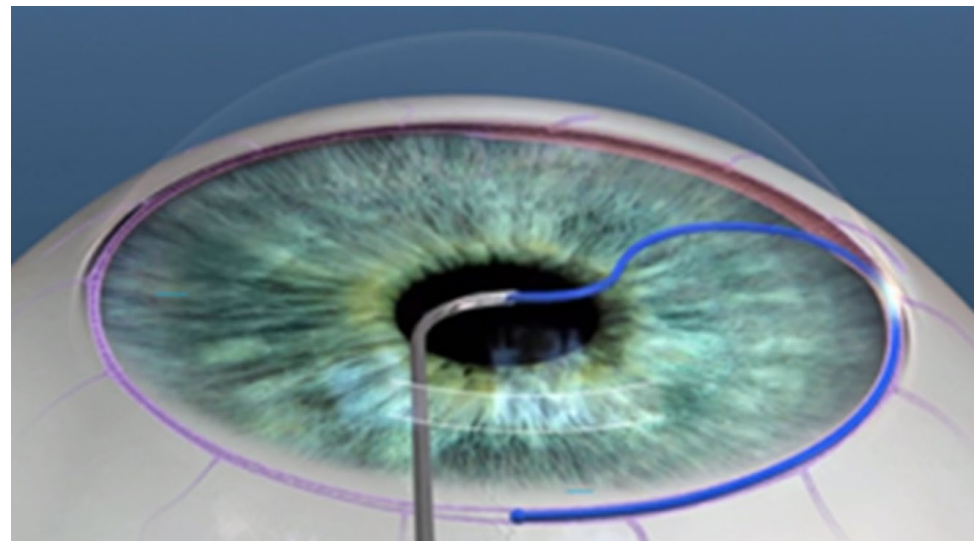


Disruption or removal
of a portion of the TM

MIGS

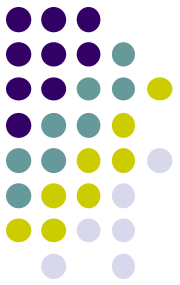


Creation of an artificial conduit through
it with an implanted bypass stent



Enlargement of Schlemm's canal
via cannulation and dilation

Glaucoma Overview



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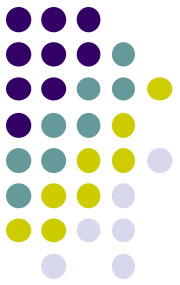
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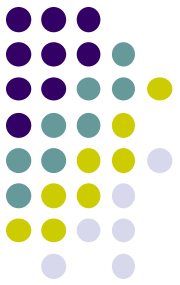
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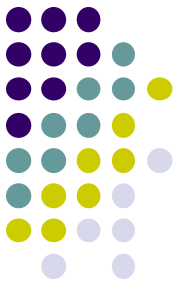
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Uveoscleral outflow can be enhanced medically with prostaglandin analogues (PGAs), eg, *latanaprost*, *bimatoprost* and *travaprost*. These are among the most commonly prescribed glaucoma drops.

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The Goldmann equation

$$IOP = \frac{\text{Aqueous Formation Rate } (\mu\text{L/min})}{\text{Outflow Facility } (\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}$$

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.

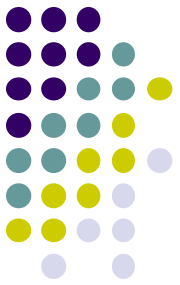
indicates
equation—
interpret.

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

- b) increase the fraction's denominator, ie, **increase aqueous outflow**;
- c) decrease that number at the end, ie, reduce EVP.

Uveoscleral outflow can be enhanced medically with prostaglandin analogues (PGAs), eg, *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



The Goldmann equation

$$IOP = \frac{\text{Aqueous Formation Rate } (\mu\text{L/min})}{\text{Outflow Facility } (\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}$$

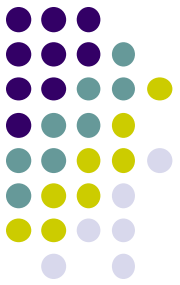
The *Goldmann equation* identifies the variables that determine IOP and indicates how they relate to one another. Note first that the mathematics of the equation—*IOP* equals a *fraction*, plus a *number*—are straightforward, and easy to interpret.

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

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

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

Glaucoma Overview



The Goldmann equation

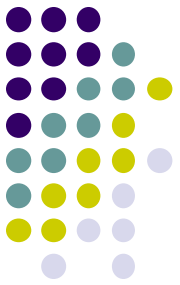
$$IOP = \frac{\text{Aqueous Formation Rate } (\mu\text{L/min})}{\text{Outflow Facility } (\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}$$

The *Goldmann equation* identifies the variables that determine IOP and indicates how the IOP is determined. 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).

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

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

Glaucoma Overview

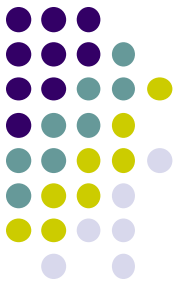


The Goldmann equation

$$IOP = \frac{\text{Aqueous Formation Rate } (\mu\text{L}/\text{min})}{\text{Outflow Facility } (\mu\text{L}/\text{min}/\text{mmHg})} + \text{Episcleral Venous Pressure (mmHg)}$$

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

Glaucoma Overview

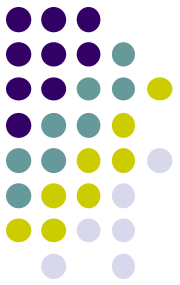


The Goldmann equation

$$IOP = \frac{\text{Aqueous Formation Rate } (\mu\text{L}/\text{min})}{\text{Outflow Facility } (\mu\text{L}/\text{min}/\text{mmHg})} + \text{Episcleral Venous Pressure (mmHg)}$$

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

Glaucoma Overview

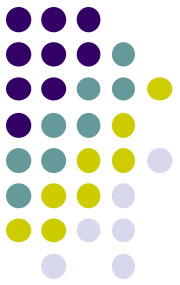


The Goldmann equation

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



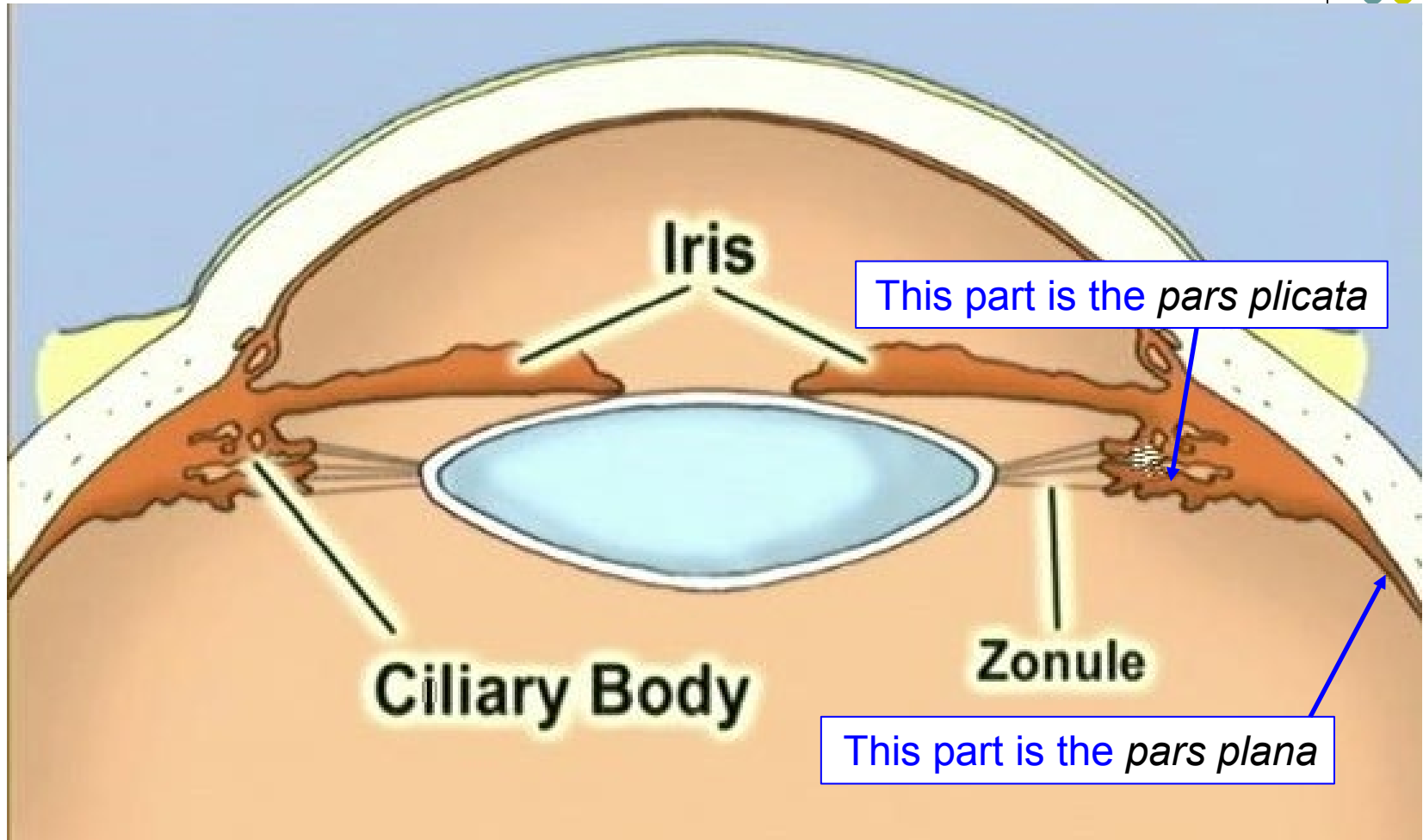
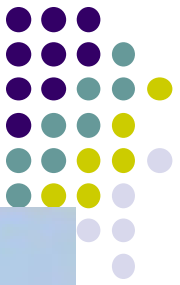
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Aqueous is formed in the **nonpigmented epithelium** of the pars plicata portion of the ciliary body. And yes, as implied by the fact that aqueous is made in a 'nonpigmented' epithelium, it is the case that the ciliary body has a pigmented epithelium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

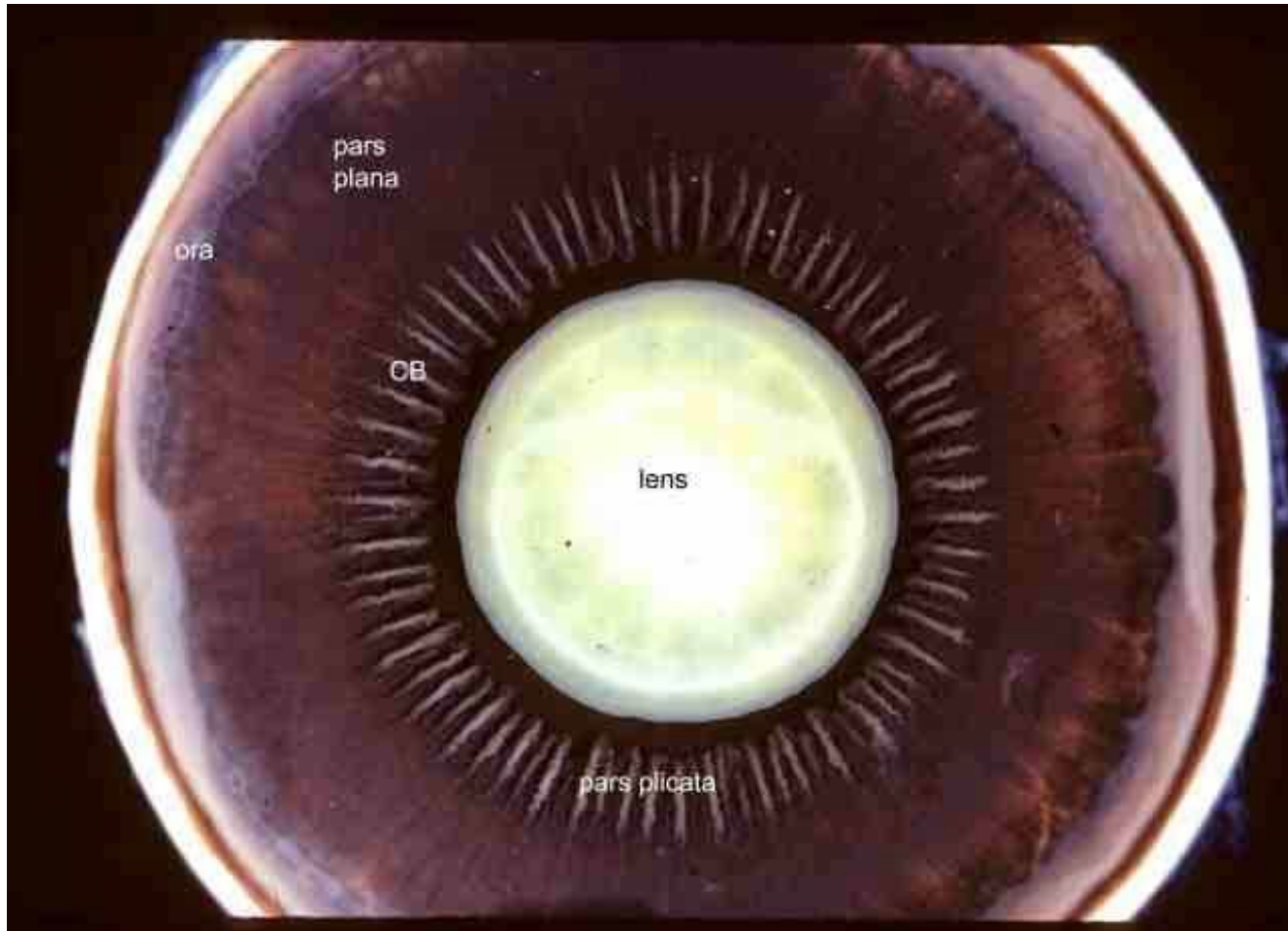
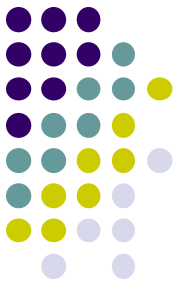
Let's take a look at the structures involved in aqueous formation, starting with the ciliary body and its anatomy.

Glaucoma Overview



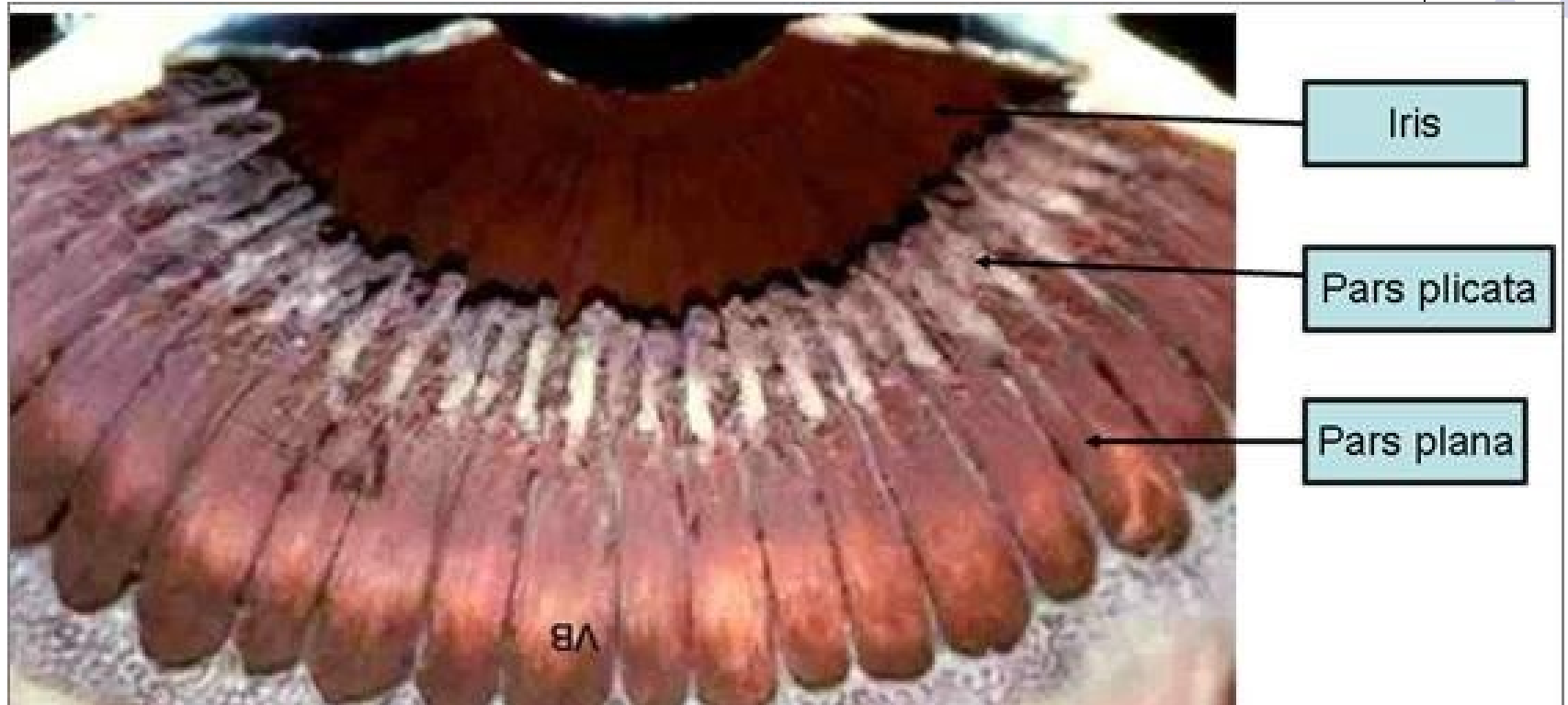
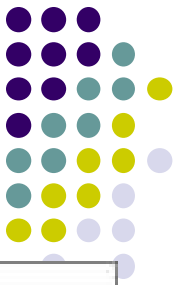
The ciliary body has two parts: The *pars plana* and the *pars plicata*

Glaucoma Overview



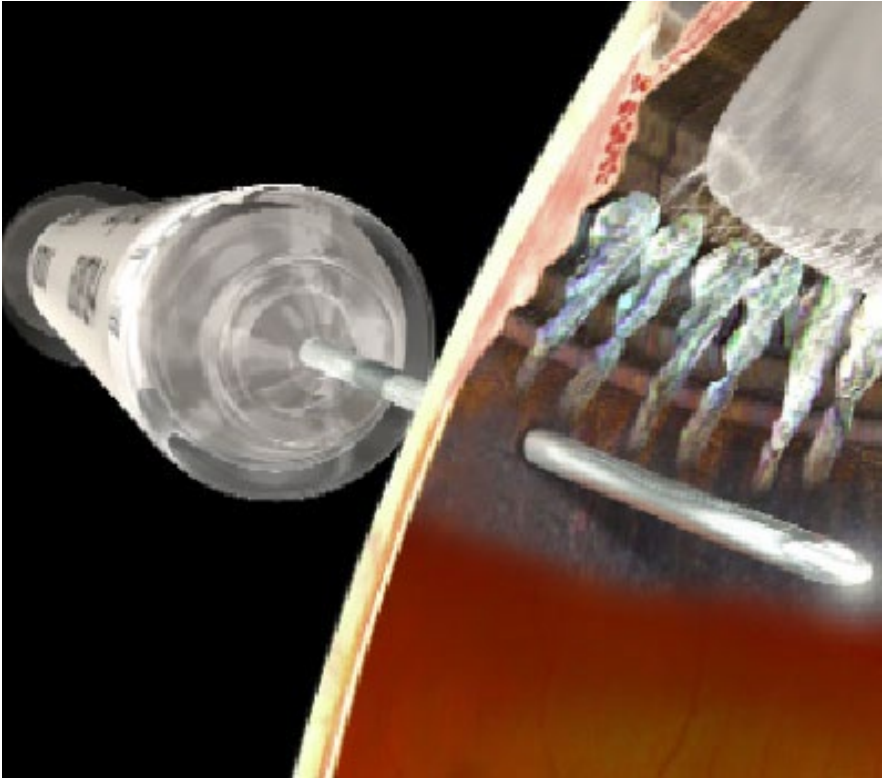
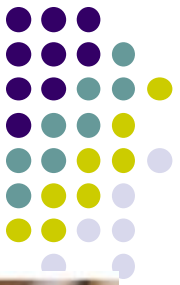
Ciliary body: Another view

Glaucoma Overview



Ciliary body: Another view

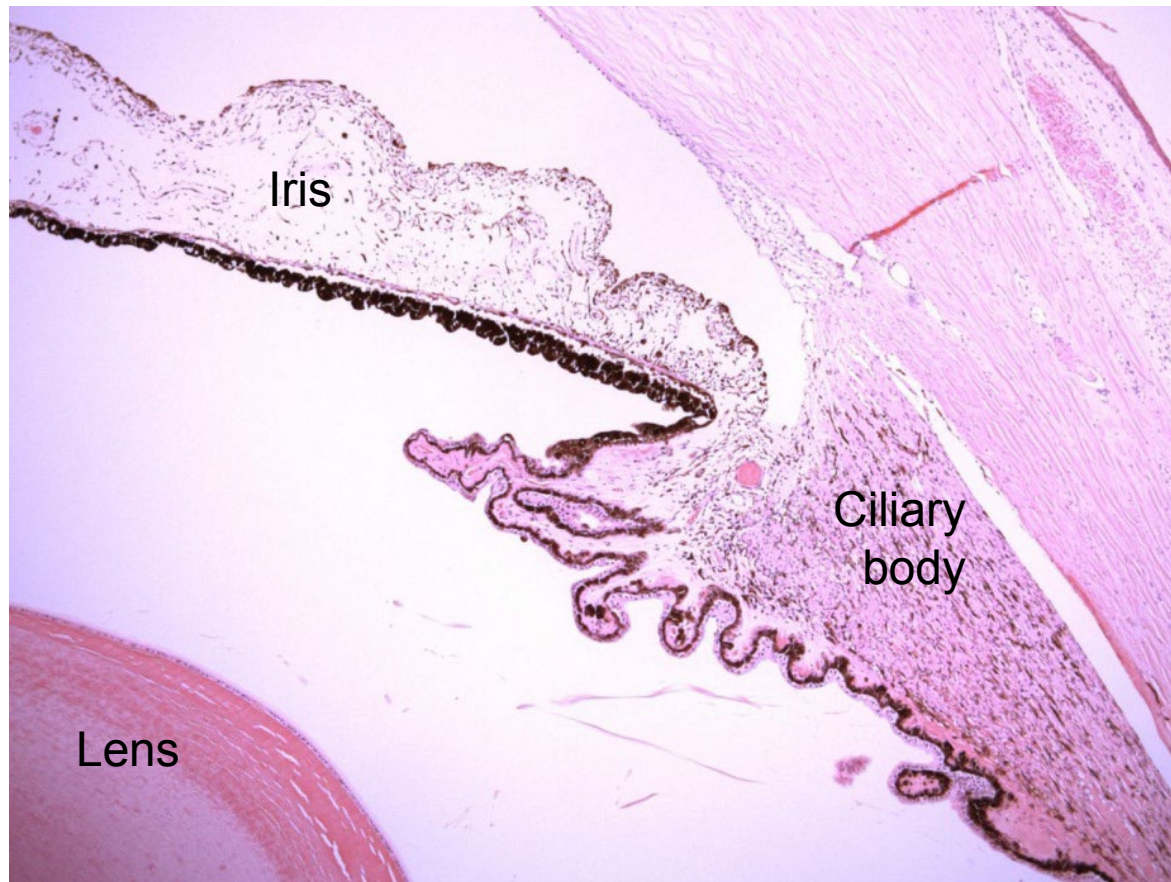
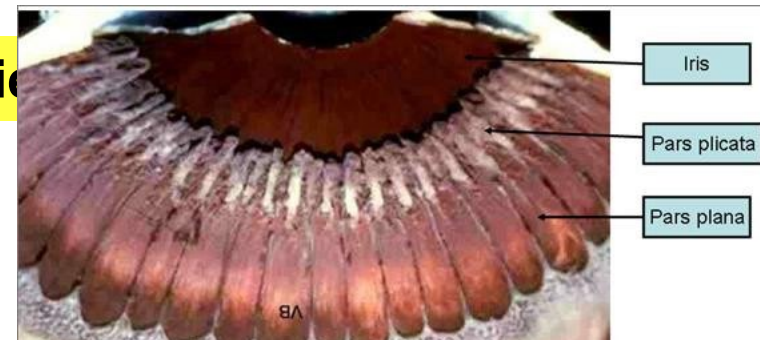
Glaucoma Overview



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')

Glaucoma Overview

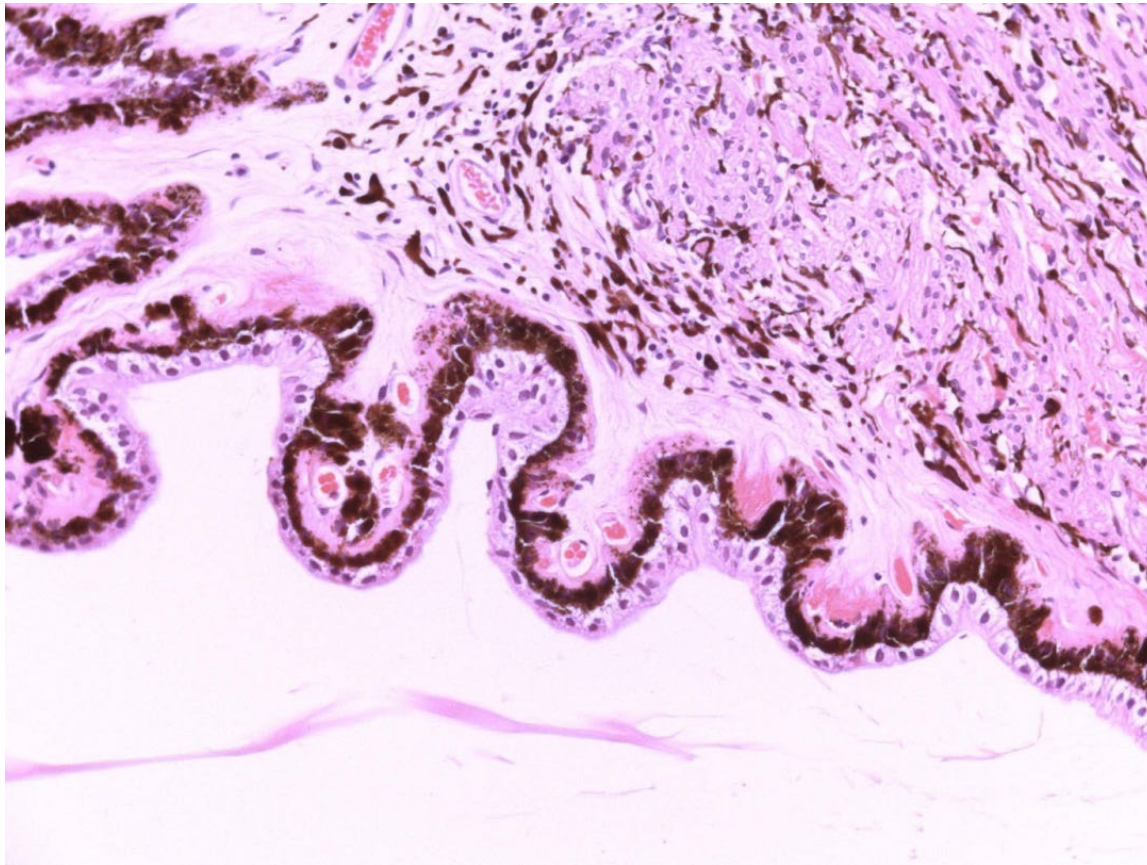
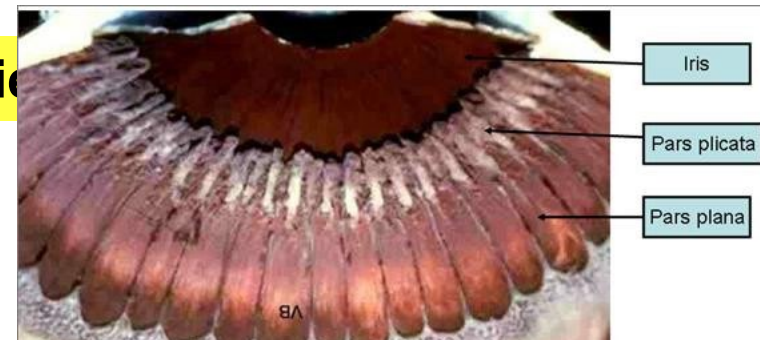
“Aqueous is formed in the nonpigmented epithelium of the pars plicata portion of the ciliary body”



Now let's look at the CB epithelium. **Low power** photomicrograph.

Glaucoma Overview

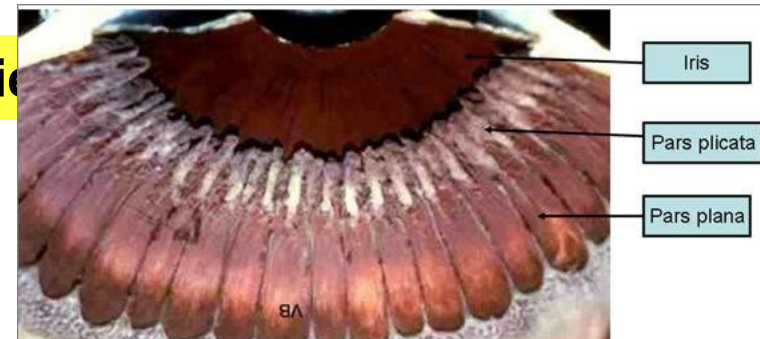
“Aqueous is formed in the nonpigmented epithelium of the pars plicata portion of the ciliary body”



Now let's look at the CB epithelium. **Higher.**

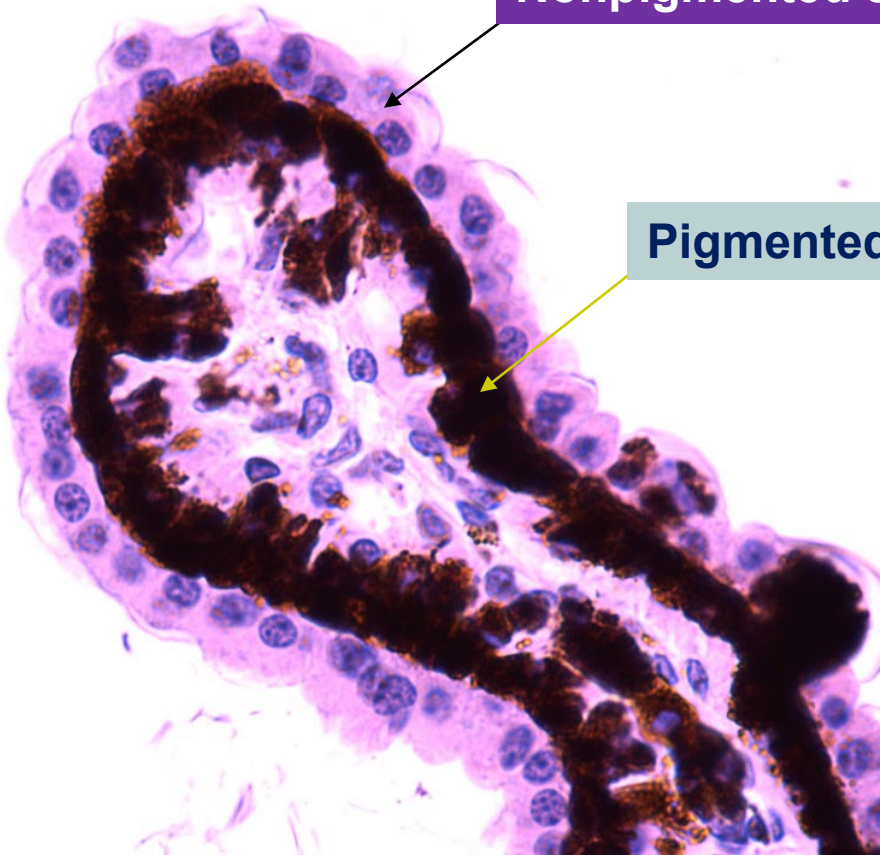
Glaucoma Overview

“Aqueous is formed in the nonpigmented epithelium of the pars plicata portion of the ciliary body”



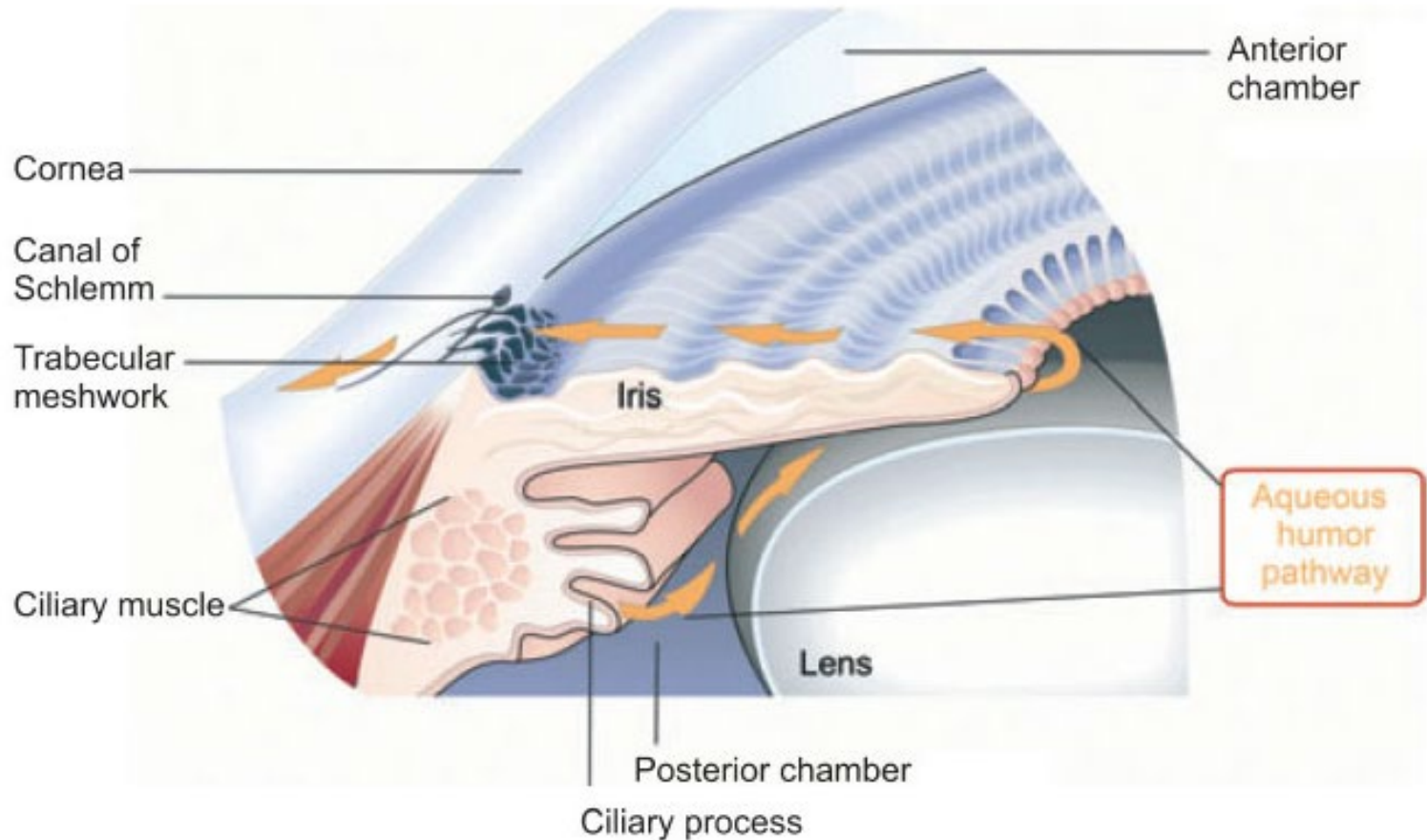
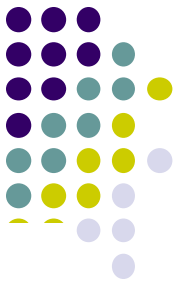
Nonpigmented epithelium

Pigmented epithelium



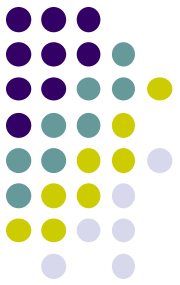
Now let's look at the CB epithelium. **High**. Now we can clearly identify both epithelial layers.

Glaucoma Overview



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 Overview



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.

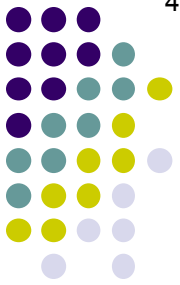
We mentioned that glaucoma presents with “progressive ONH damage.” Let’s drill down on the structure of the ONH, and how the retina relates to it.

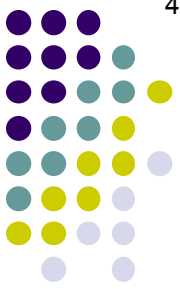
In addition to being a leading cause of blindness, glaucoma is a disease that renders it unique: it is the only one for which there is no proven way to mitigate the risk of blindness. While there are many ways to treat glaucoma—our glaucoma-treatment modalities include medication, laser surgery, or incisional surgery—each 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.

Glaucoma Overview

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.)



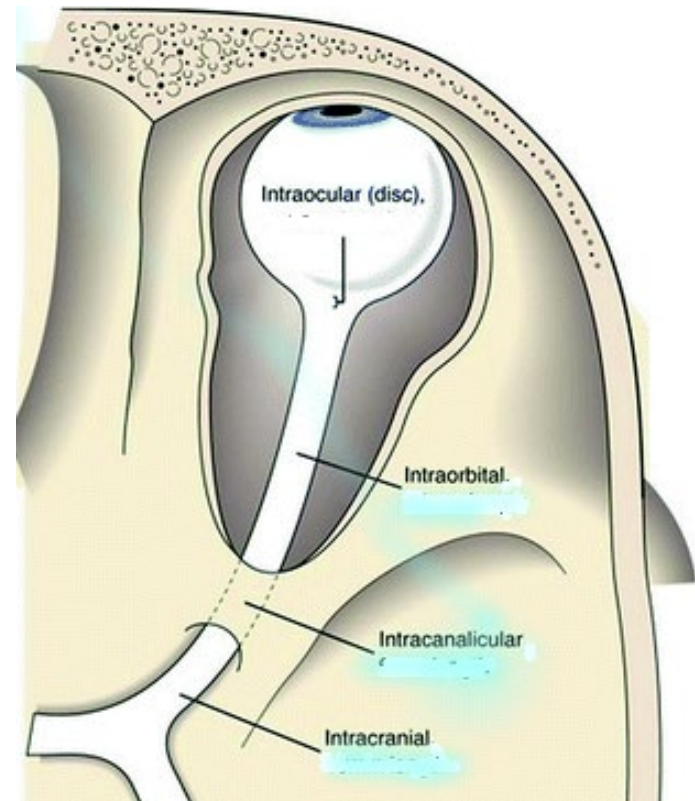


Glaucoma Overview

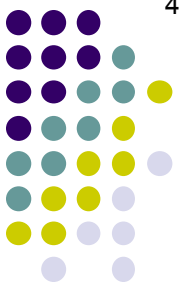
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Anatomically speaking, the optic nerve has four portions: The *intraocular*, *intraorbital*, *intraocular*, and *intracranial*.

Portion	Length (mm)
Intraocular	1
Intraorbital	25-30
Intracanalicular	4-10
Intracranial	10



Glaucoma Overview

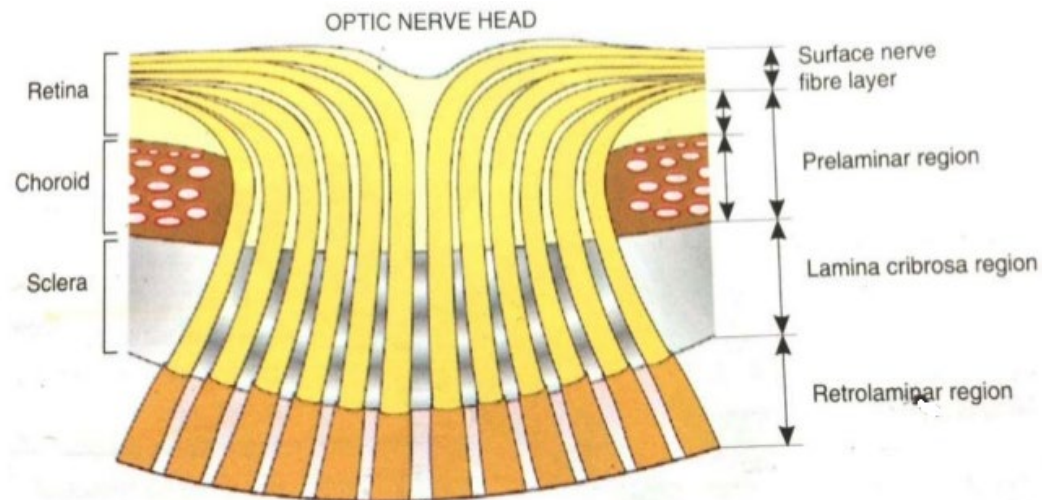


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Anatomically speaking, the optic nerve has four portions: The *intraocular*, *intraorbital*, *intraocular*, and *intracranial*.

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.

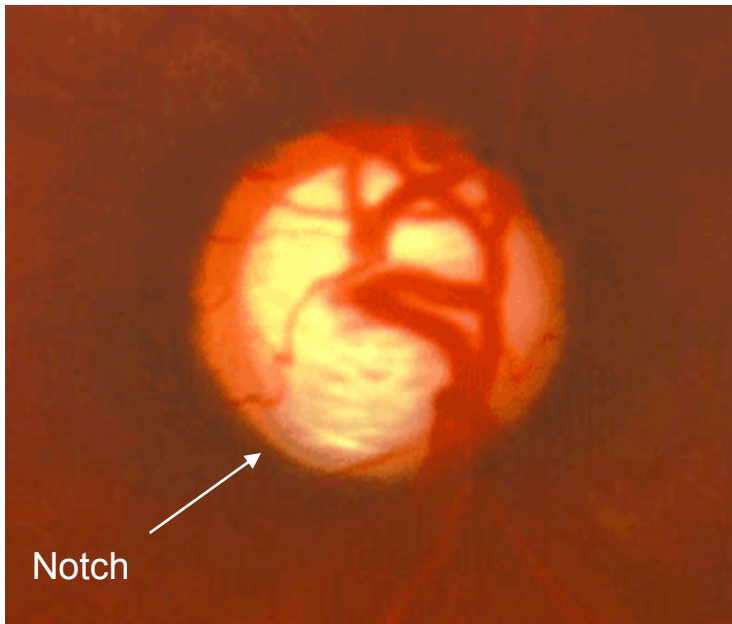
Portion	Blood supply
NFL portion	Central retinal artery (CRA)
Pre-laminar	Short posterior ciliary arteries
Laminar	Arterial circle of Zinn & Haller
Retrolaminar	Centrifugal CRA branches, centripetal pial branches



Glaucoma Overview



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.')



Notch

Glaucomatous ONH



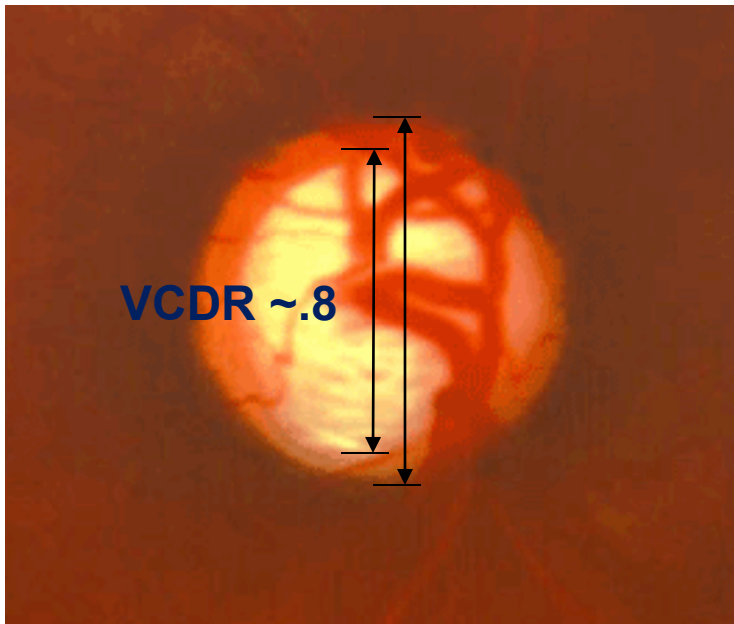
Normal ONH

Glaucoma Overview

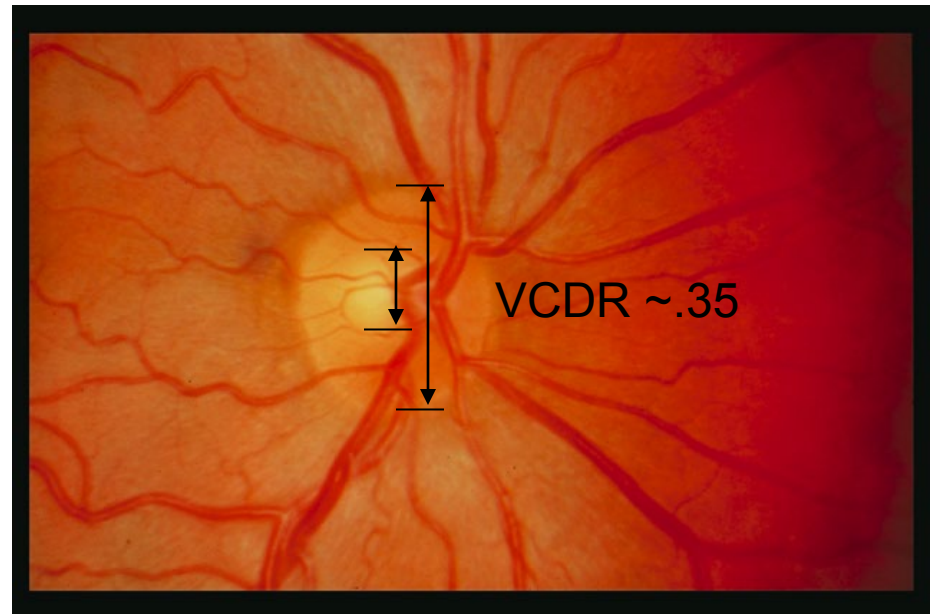


Because of this tendency, ophthalmologists focus on the *vertical cup-disc ratio* (VCDR) when assessing a pt's glaucoma status

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.')



Glaucomatous ONH

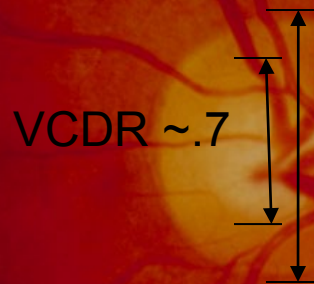


Normal ONH

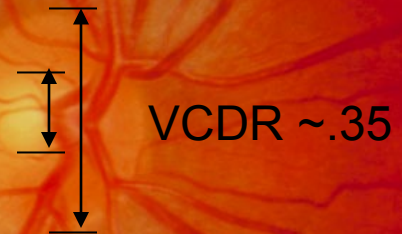
Glaucoma Overview



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).



Normal ONH



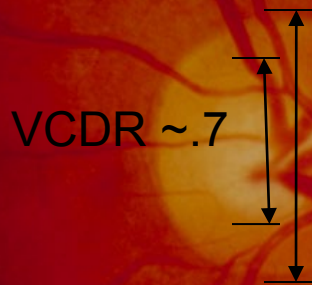
Normal ONH

Glaucoma Overview

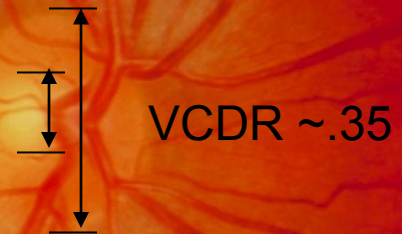


Note that the VCDR can be misleading in this regard, as it can be quite pronounced in some normal eyes (especially those with a large disc).

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*.

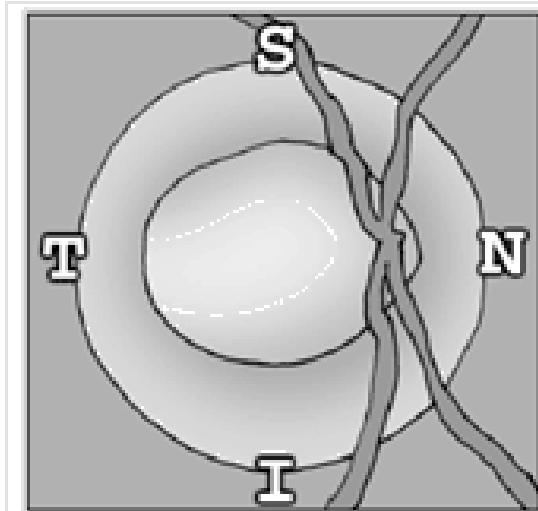
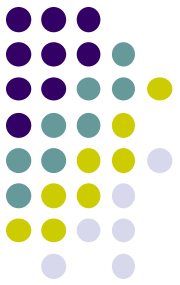


Normal ONH

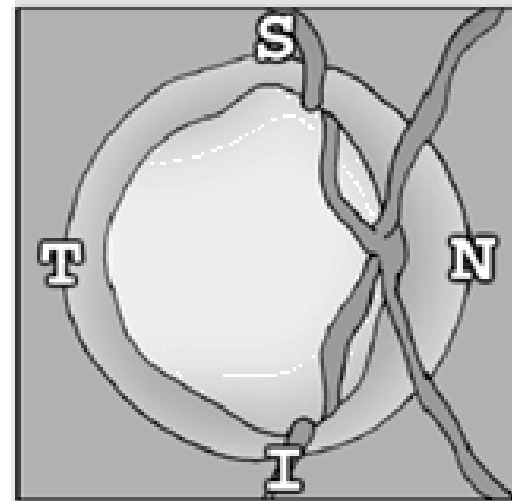


Normal ONH

Glaucoma Overview

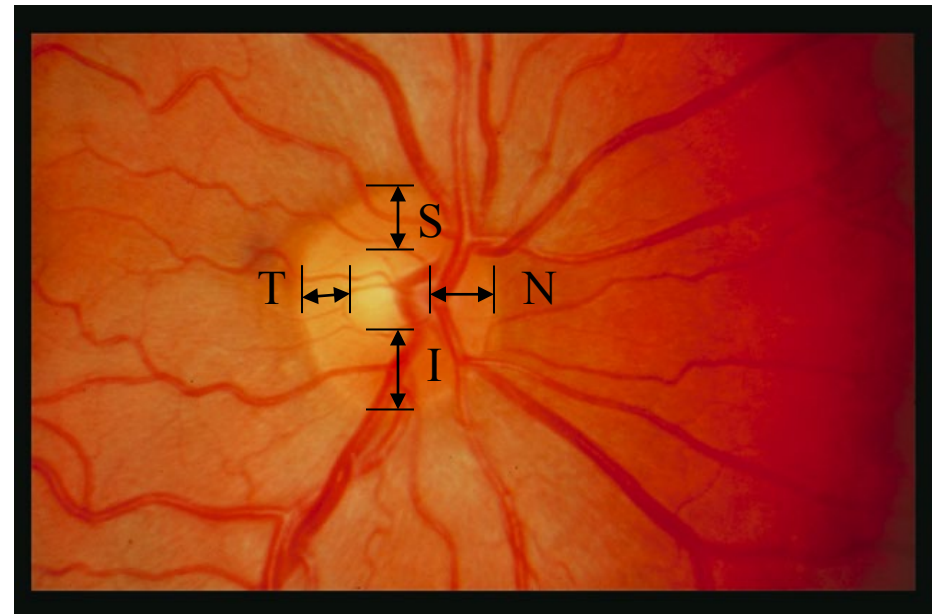


normal



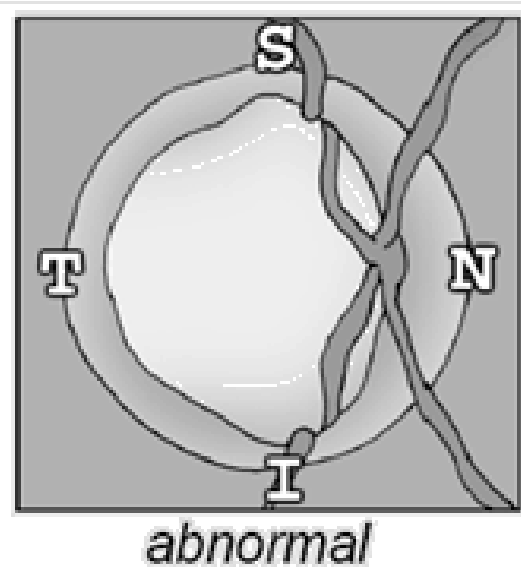
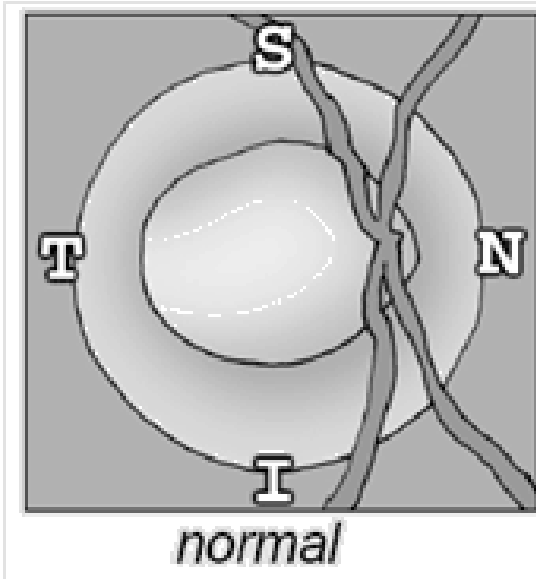
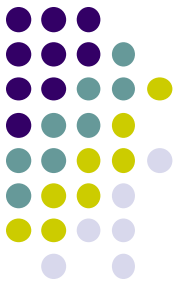
abnormal

The nonglaucomatous neuroretinal rim tends to follow what's known as the **ISNT rule**: In decreasing order, the rim is thickest at its **I**nferior, **S**uperior, **N**asal, and **T**emporal portions. If an ONH's rim adheres to this rule, it **ISNT** glaucomatous.



Normal ONH

Glaucoma Overview

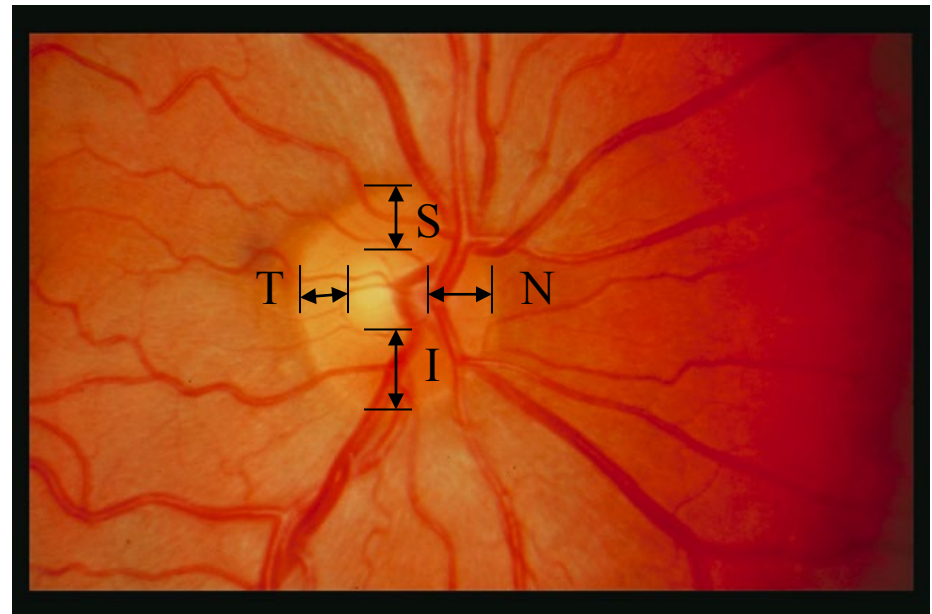


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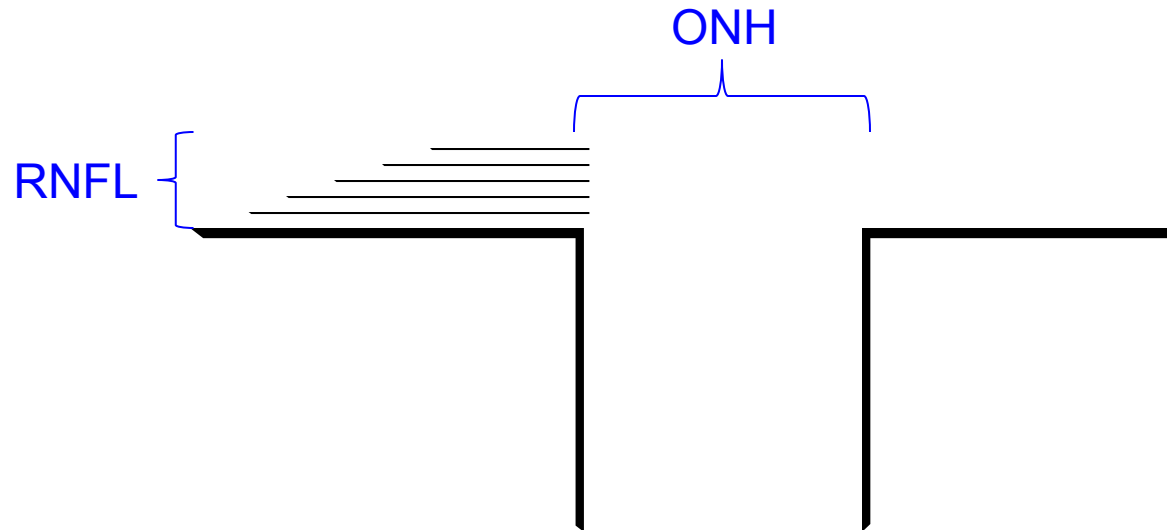
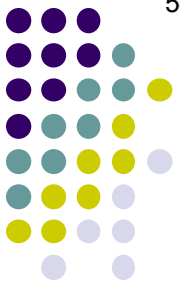
portions. If an ONH's rim adheres to this rule, it **ISNT** glaucomatous.

Note: Not all glaucoma docs find the ISNT rule to be helpful—YMMV. Ask!



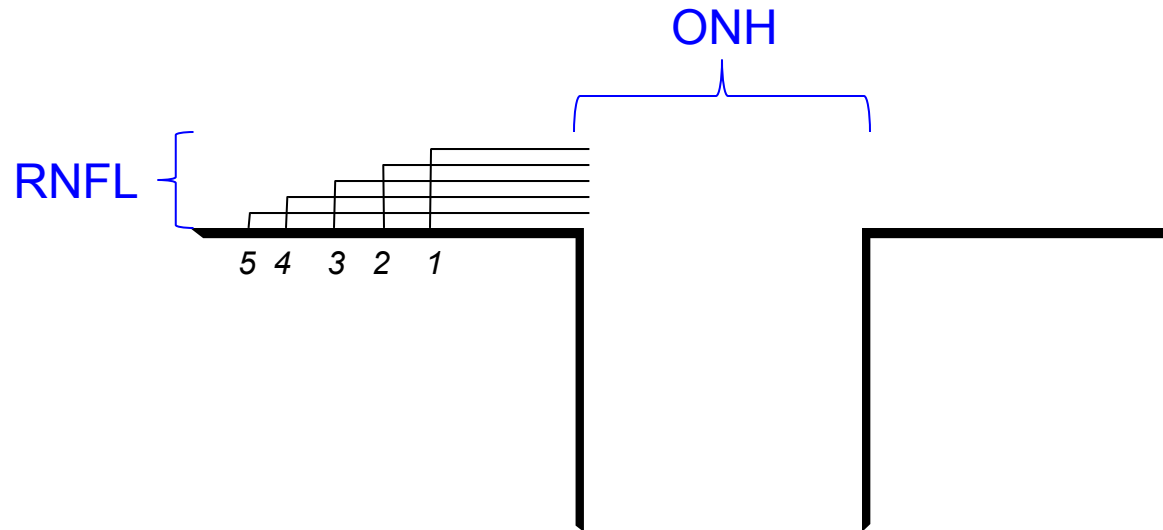
Normal ONH

Glaucoma Overview



Now consider the ONH and retina in cross section. Note that the RNFL and ONH are both organized in a specific fashion:

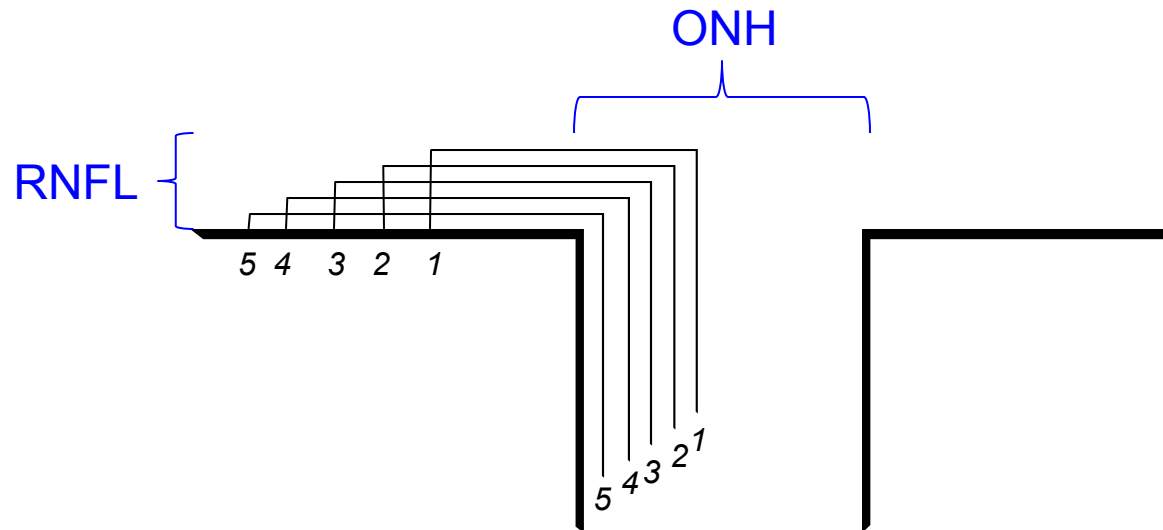
Glaucoma Overview



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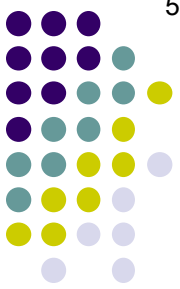
--The RNFL is stacked *vertically*, with fibers that originate at points distant from the ONH running at the bottom (ie, closer to the RPE); and

Glaucoma Overview

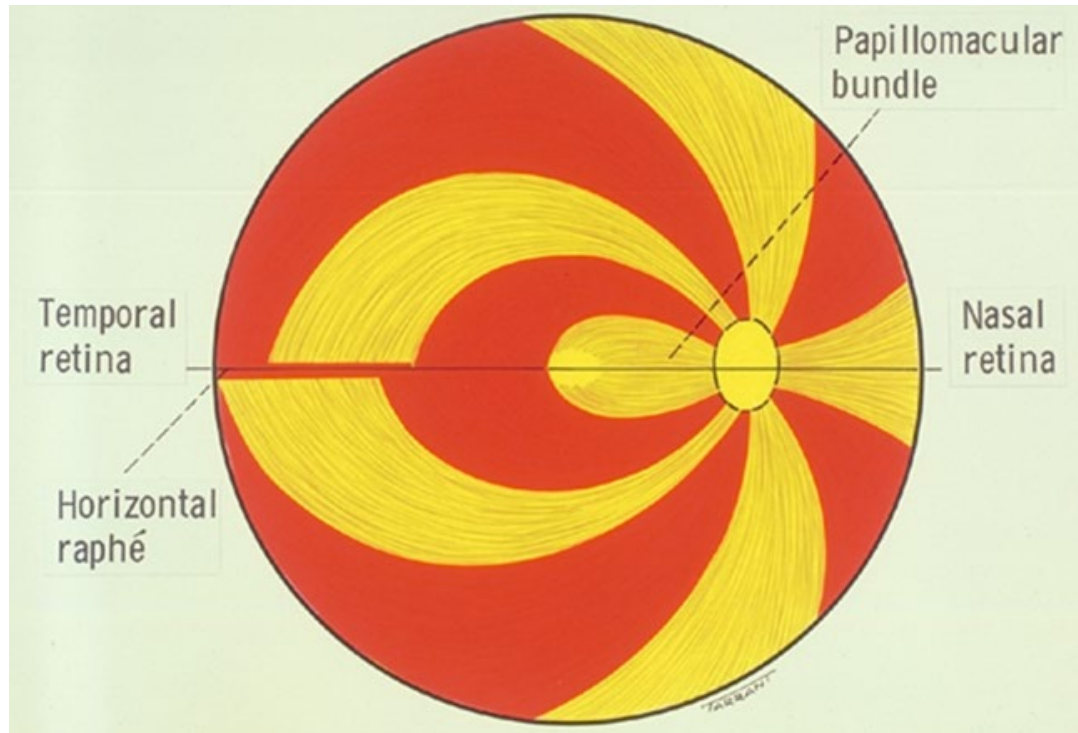
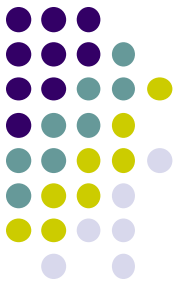


Now consider the ONH and retina in cross section. Note that the RNFL and ONH are both organized in a specific fashion:

- 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.

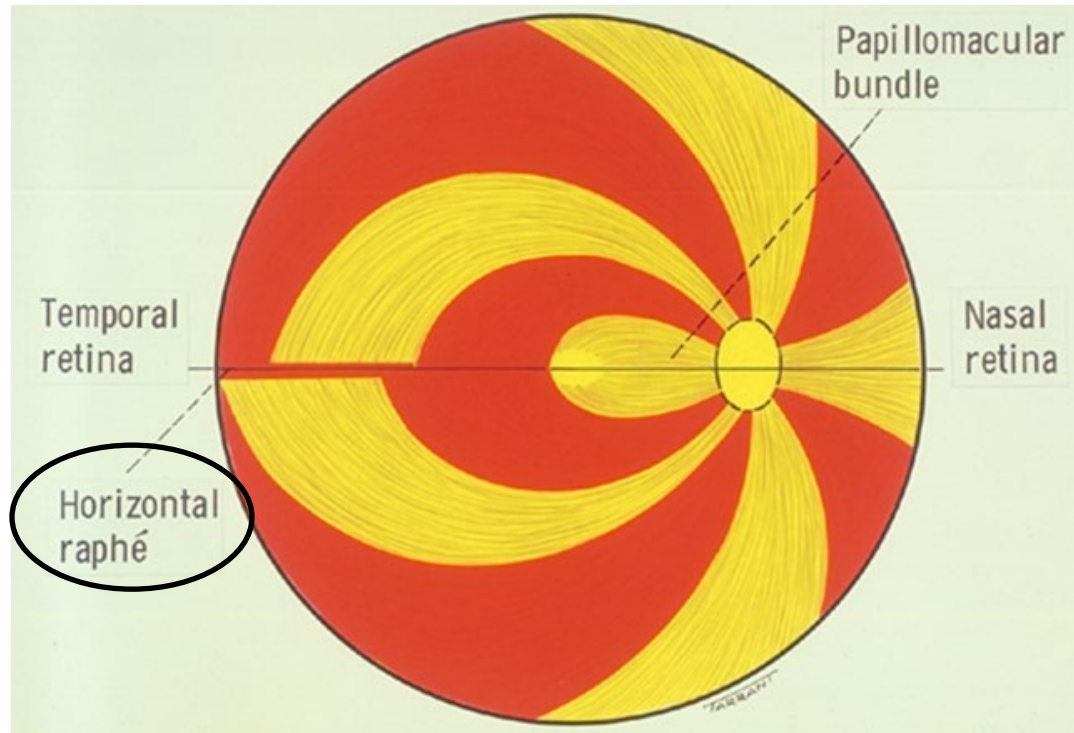
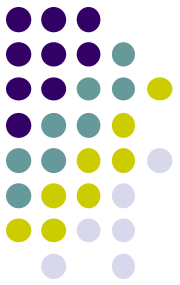


Glaucoma Overview



Now let's look at the *topography of the retinal nerve fiber layer*, and how that topography relates to the structure of the ONH.

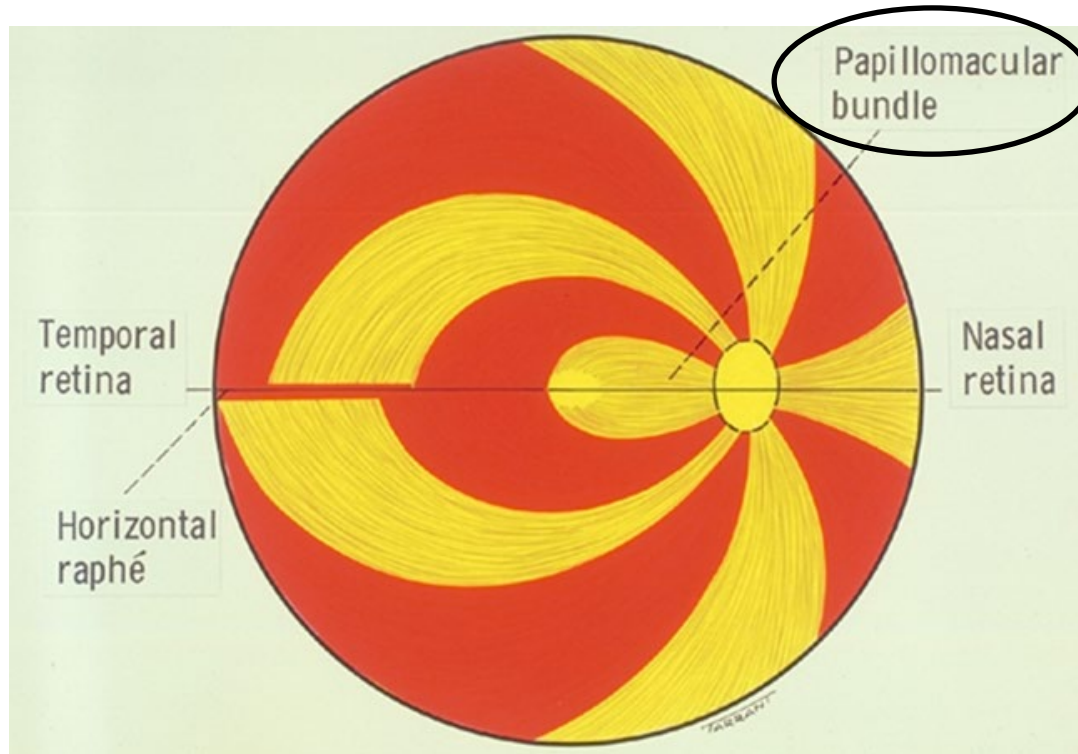
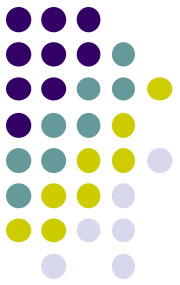
Glaucoma Overview



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.

Glaucoma Overview

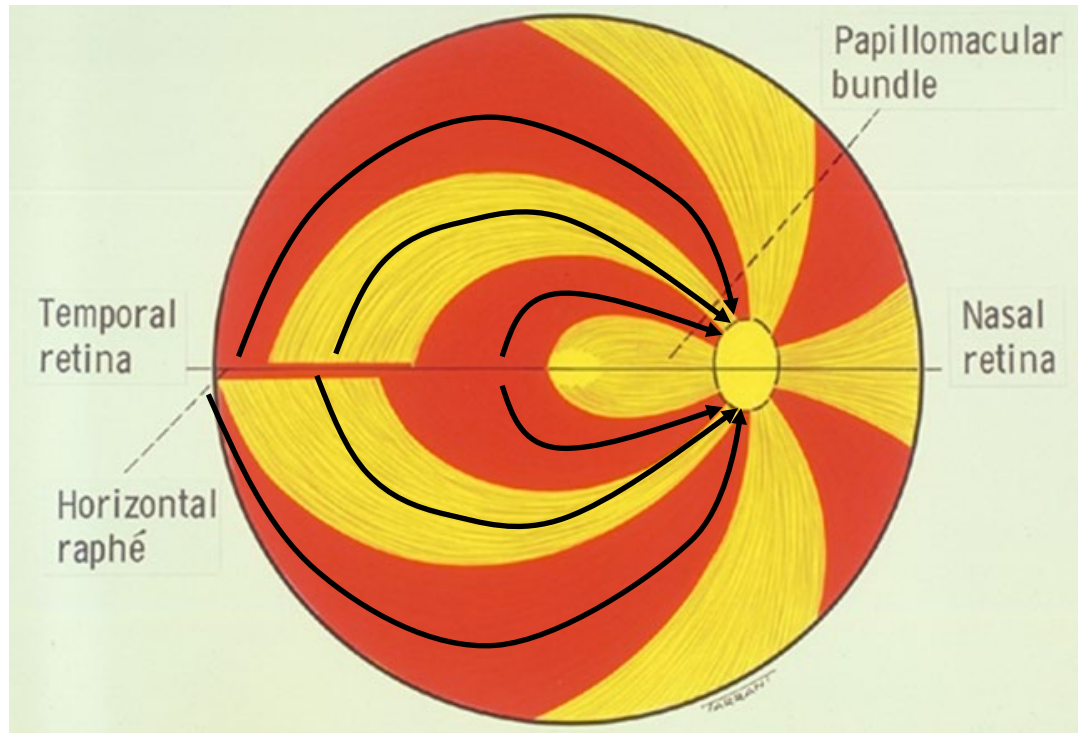
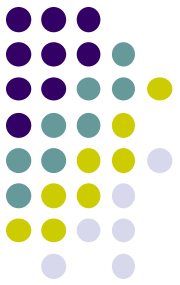


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First, take note of the *horizontal raphé*. Fibers do not cross this anatomic boundary—those superior to it join the superior ONH, and those inferior to it, the inferior ONH.

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.

Glaucoma Overview



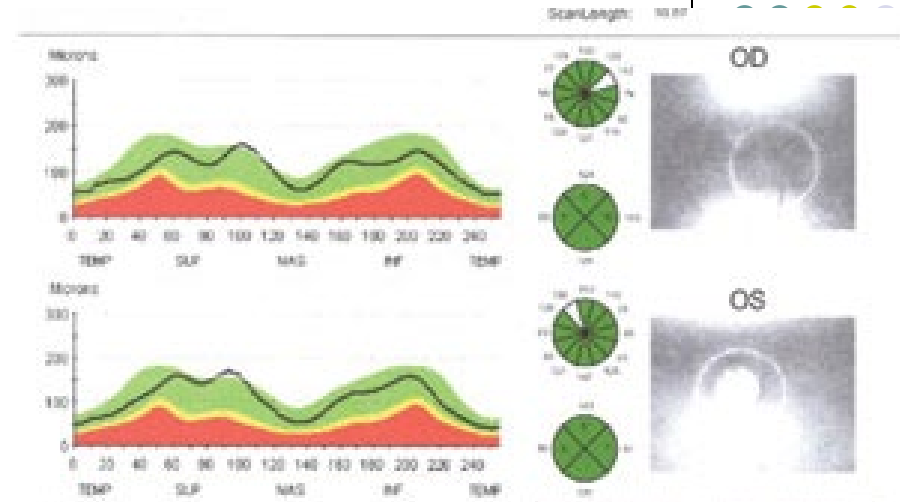
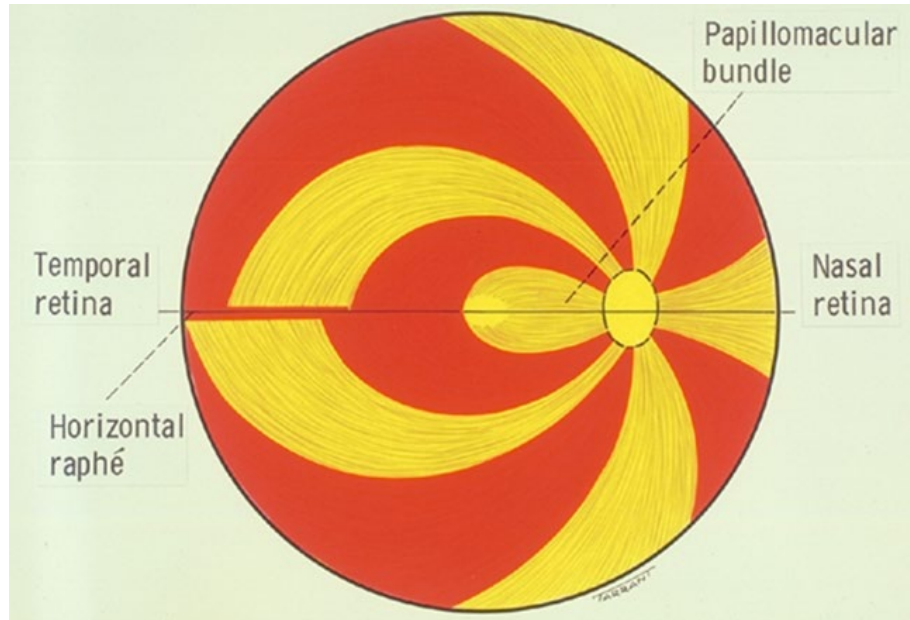
Now let's look at the *topography of the retinal nerve fiber layer*, and how that topography relates to the structure of the ONH.

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.

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.

Glaucoma Overview

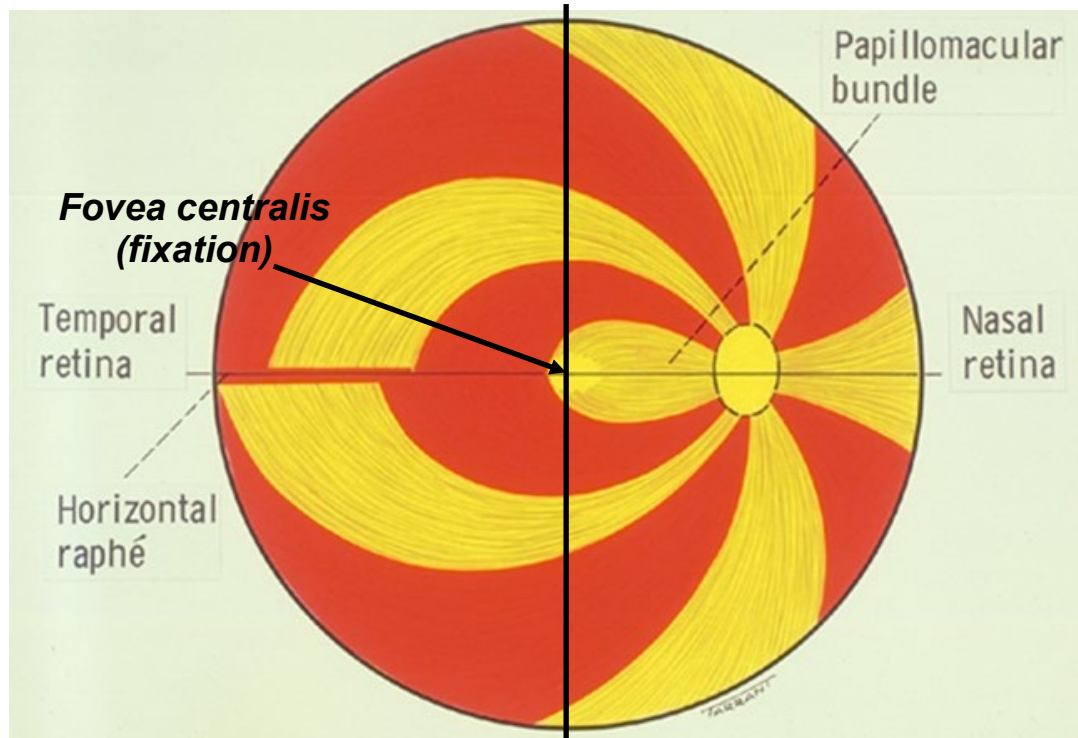
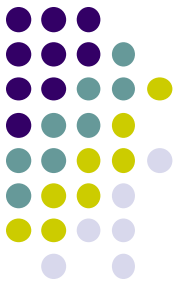


Because there are so many fibers at the superior and inferior poles, the normal ONH rim tends to be thicker at these sites. (This accounts for the relative proportions of the rim segments as captured by the *ISNT rule* described previously.)



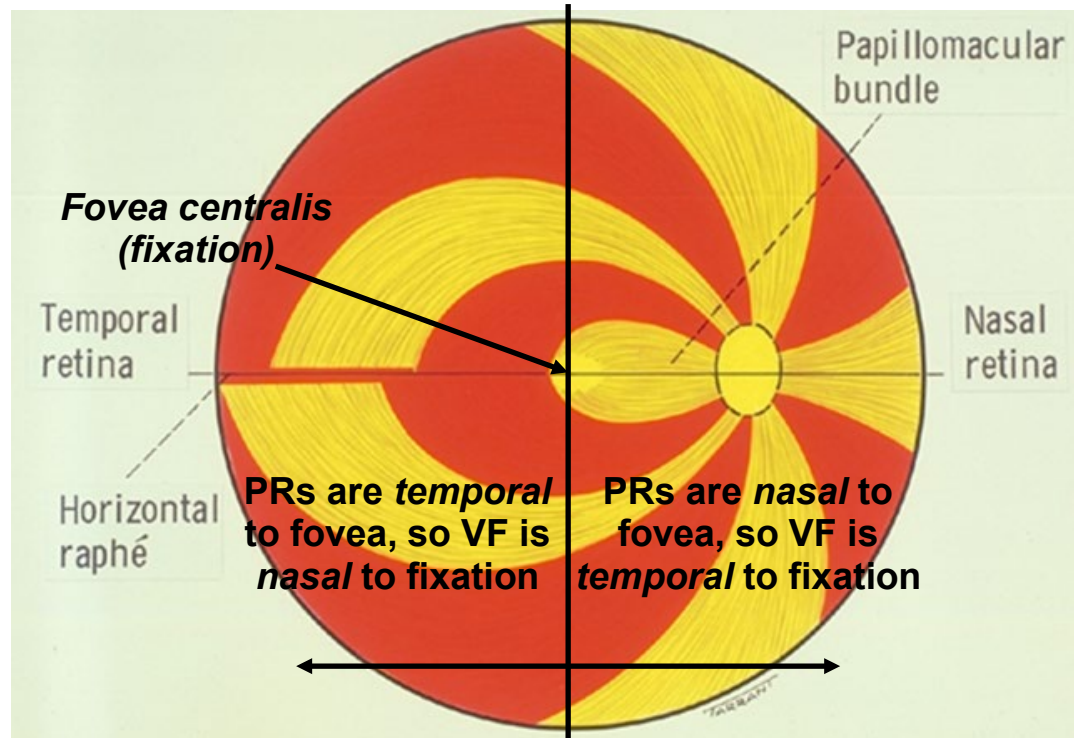
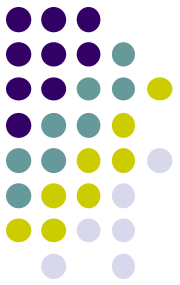
Normal ONH

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.

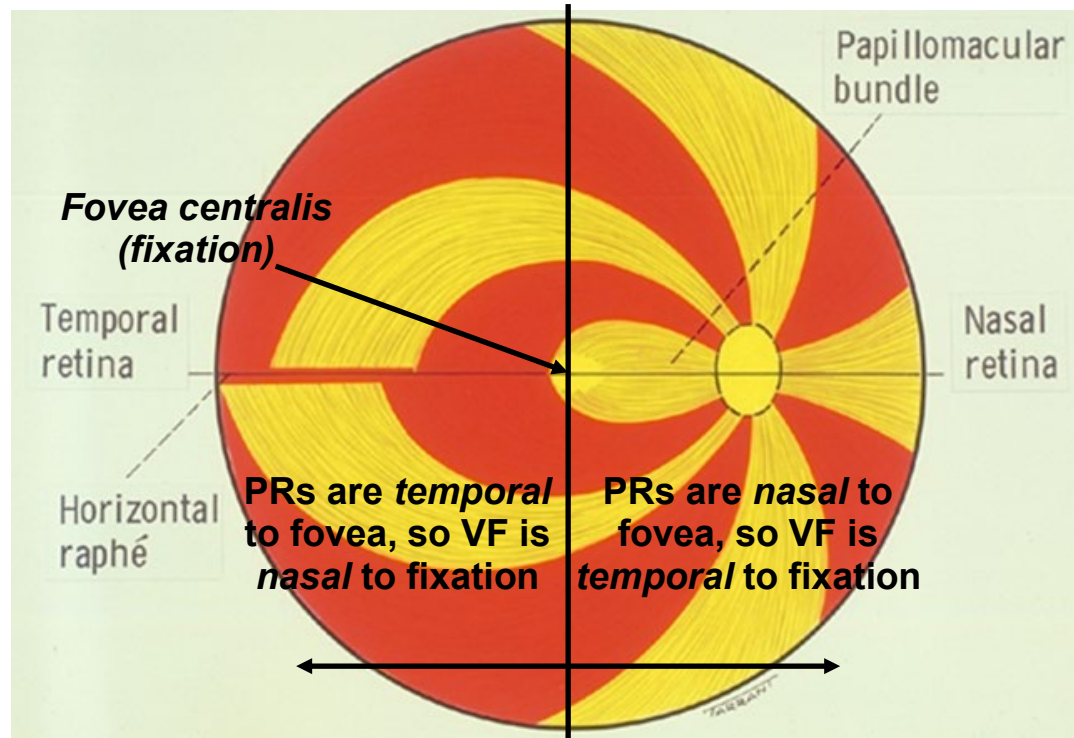
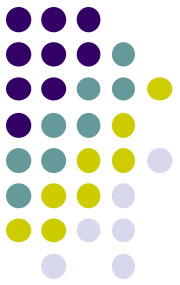
Glaucoma Overview



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.

Glaucoma Overview

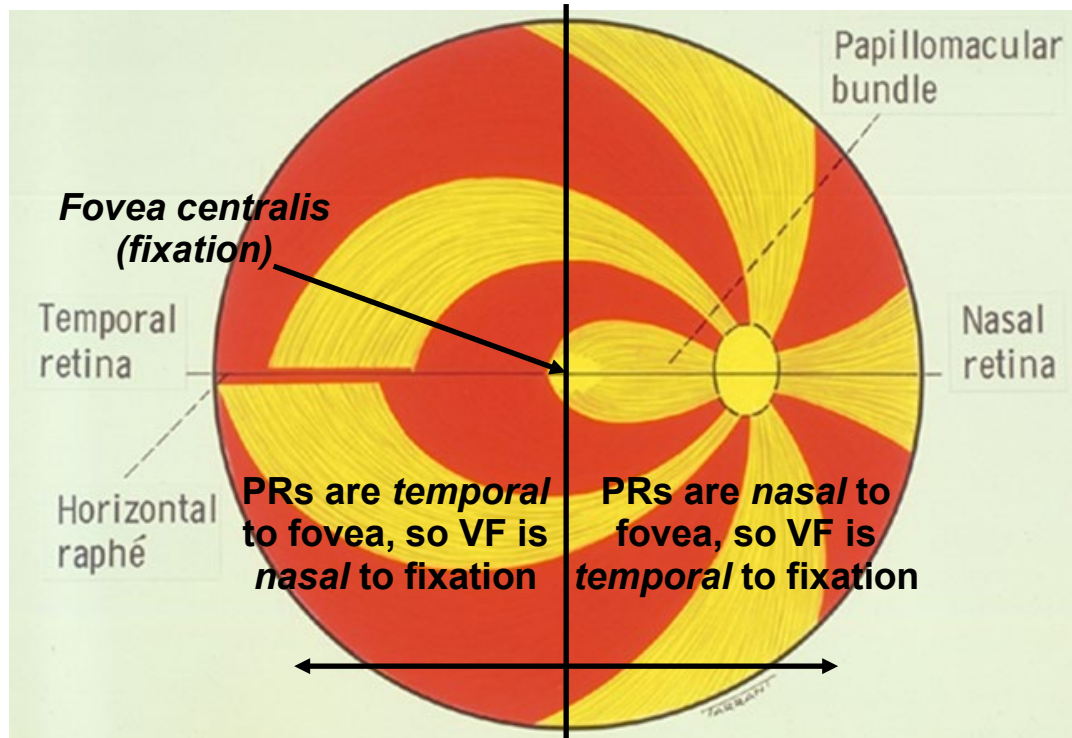
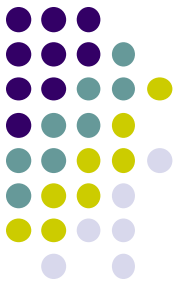


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photo The optic chiasm provides the anatomic nexus for the vertical meridian found in the visual fields.
respo

Glaucoma Overview



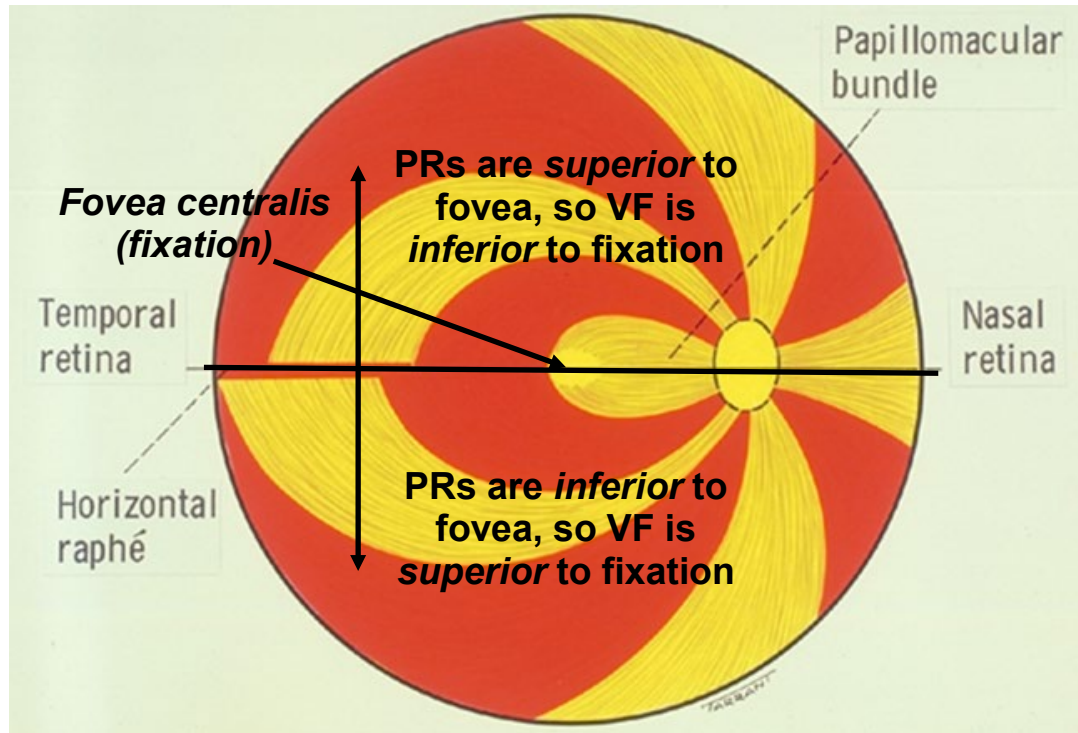
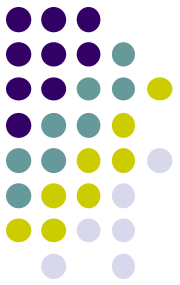
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photo **The optic chiasm provides the anatomic nexus for the vertical meridian found in the visual fields.**

respo 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.

Glaucoma Overview

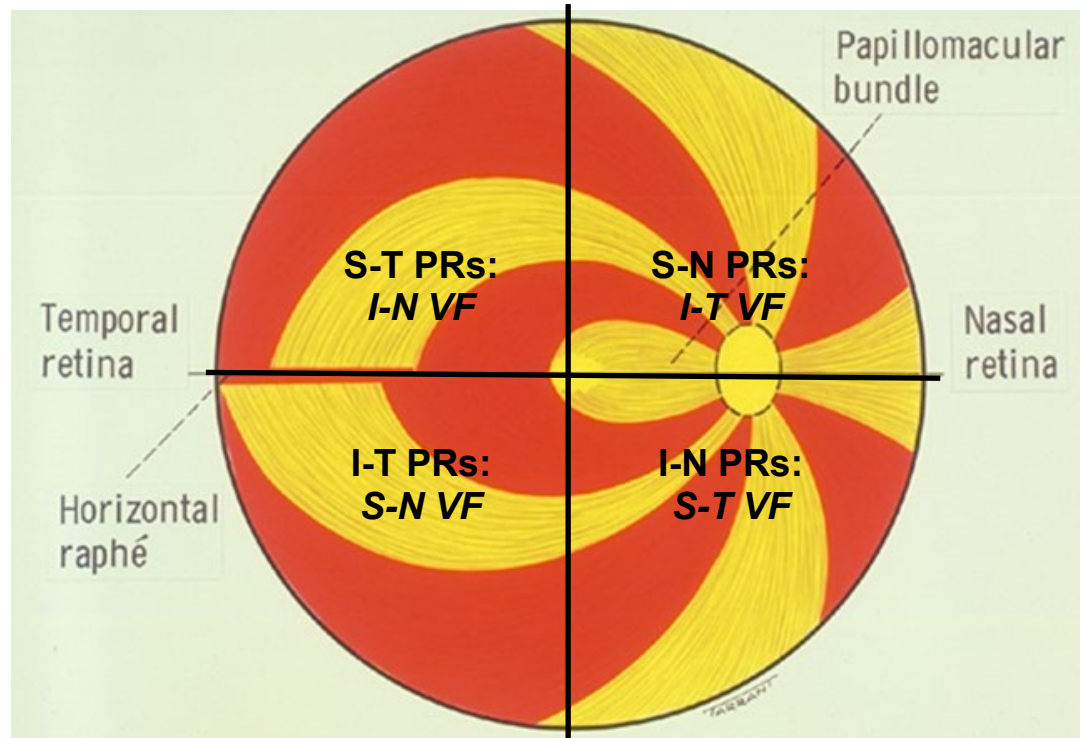
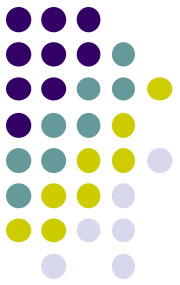


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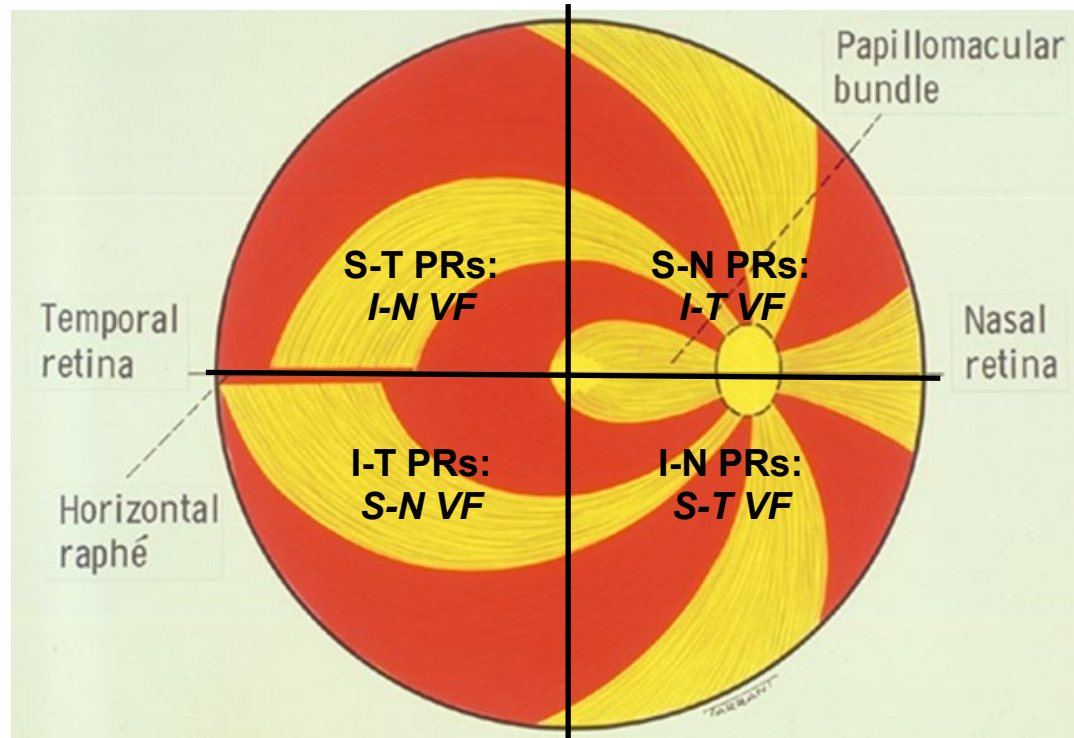
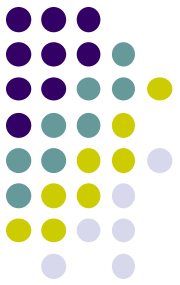
Finally, note that fixation also divides the VF into superior and inferior VFs. The corresponding portions of the retina related topographically to the horizontal raphé.

Glaucoma Overview



Putting it all together: The VF can be divided into four quadrants. Together, retinal topography and ONH structure dictate that each quadrant corresponds with a particular anatomic location on the ONH.

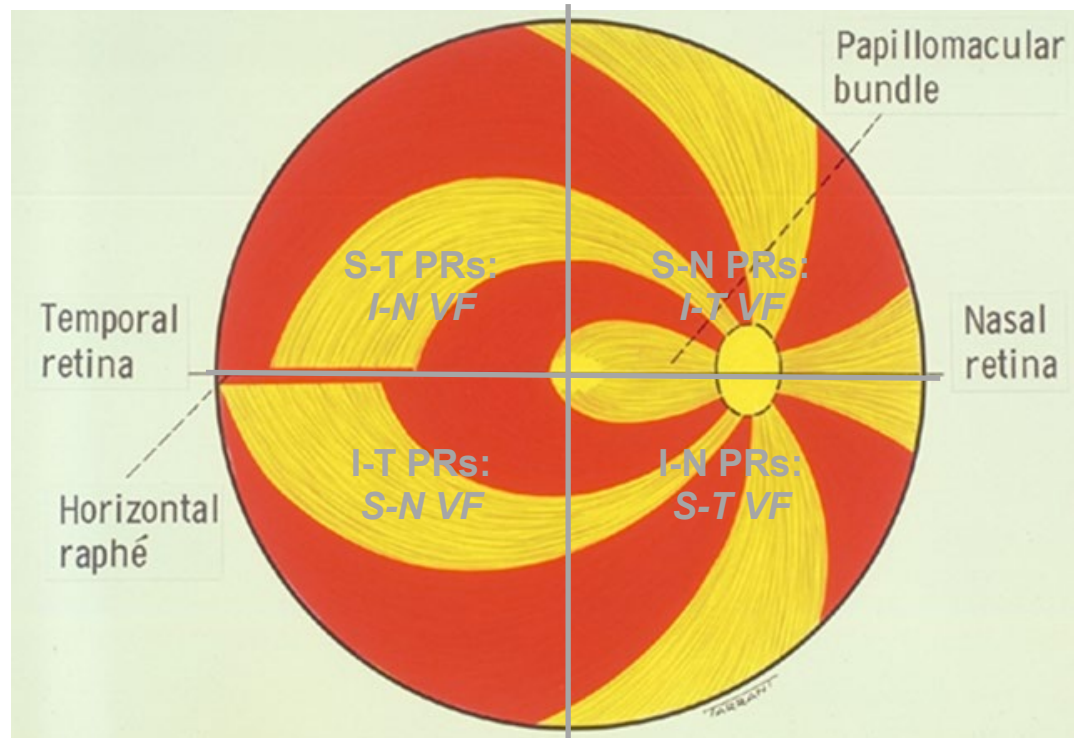
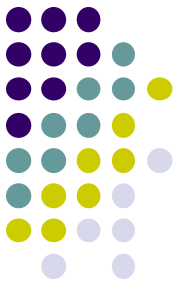
Glaucoma Overview



Putting it all together: The VF can be divided into four quadrants. Together, retinal topography and ONH structure dictate that each quadrant corresponds with a particular anatomic location on the ONH.

This relationship is important to understand as it allows the clinician to determine whether VF changes correlate with structural changes in the ONH as detected via DFE and/or imaging technology.

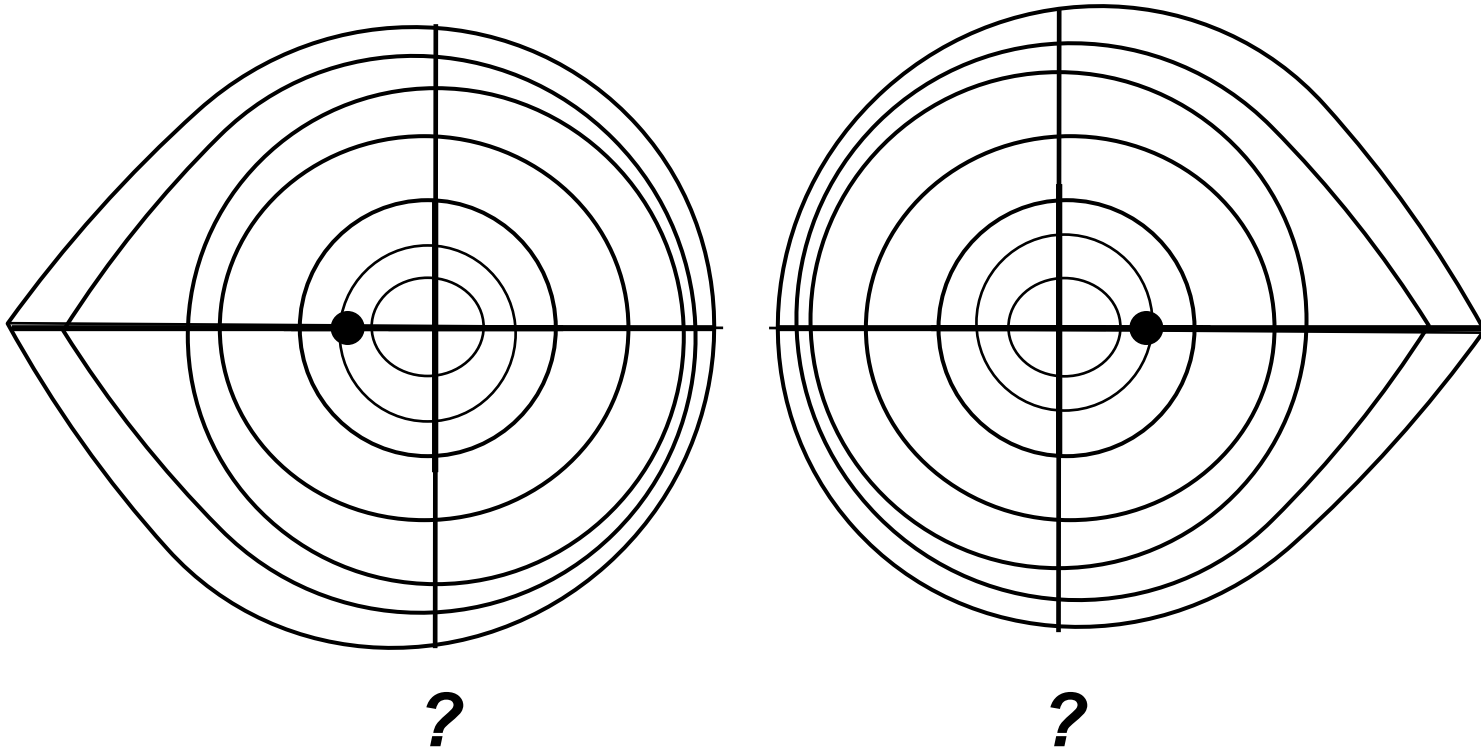
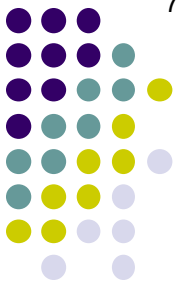
Glaucoma Overview



Putting it all together: The VF can be divided into four quadrants. Together, retinal topography and ONH structure determine the VF. This relationship is crucial for understanding how structural changes in the ONH as detected via DFE and/or imaging technology correlate with structural changes in the VF.

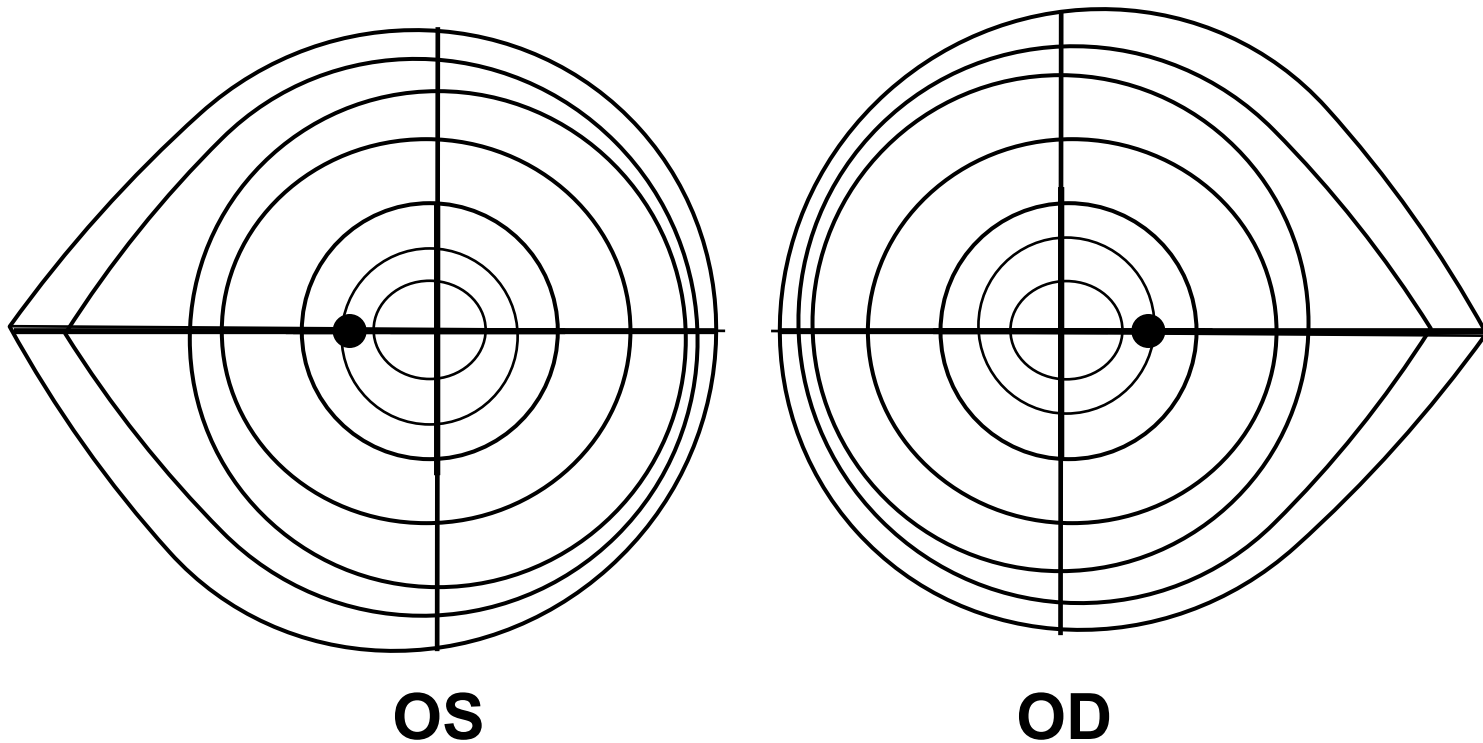
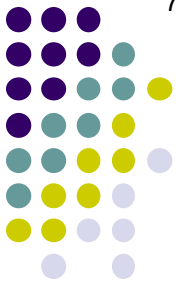
We've discussed how visual fields relate to the ONH and retina; now let's take a closer look at VF themselves

Glaucoma Overview



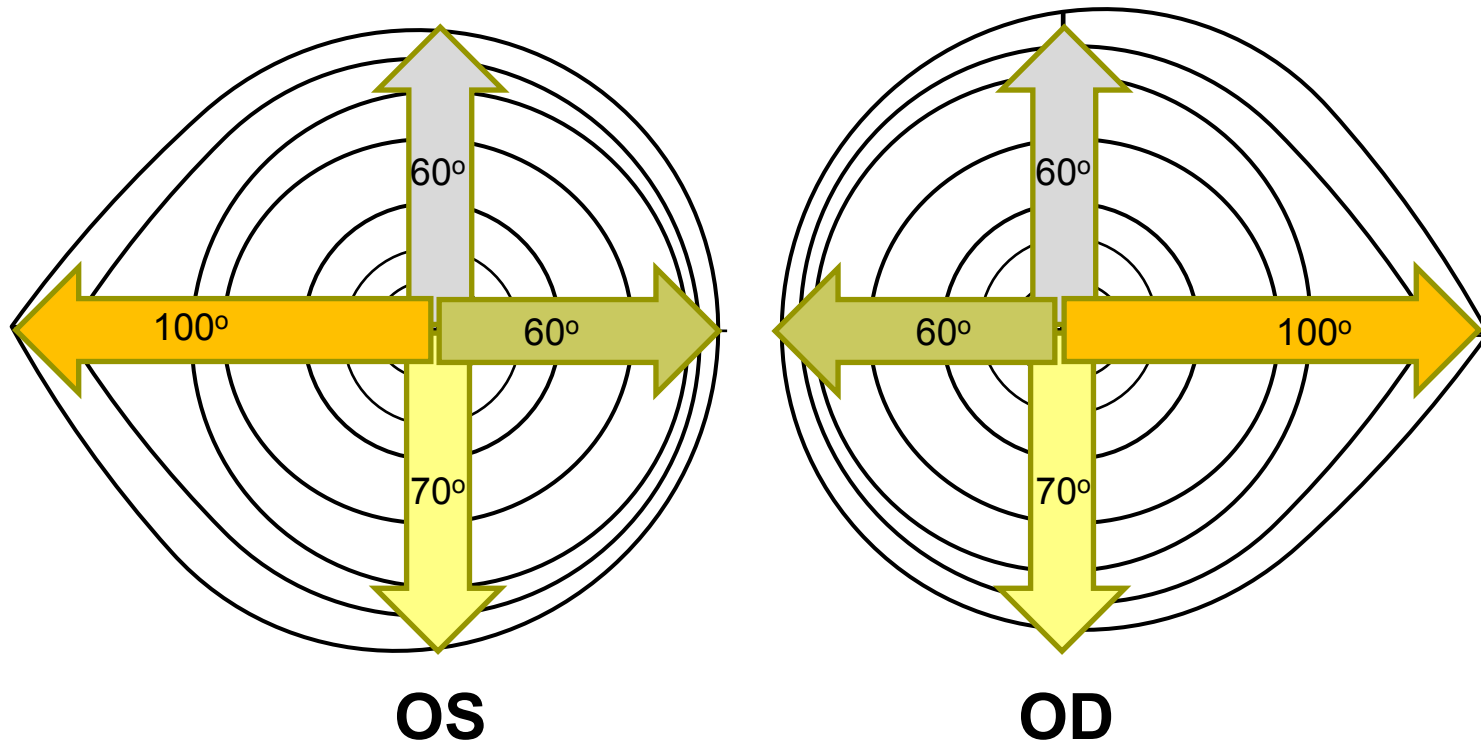
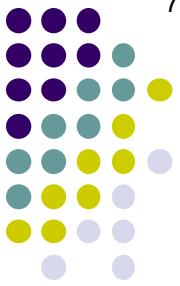
Here is a representation of the VF for each eye. Which is OD, and which OS?

Glaucoma Overview



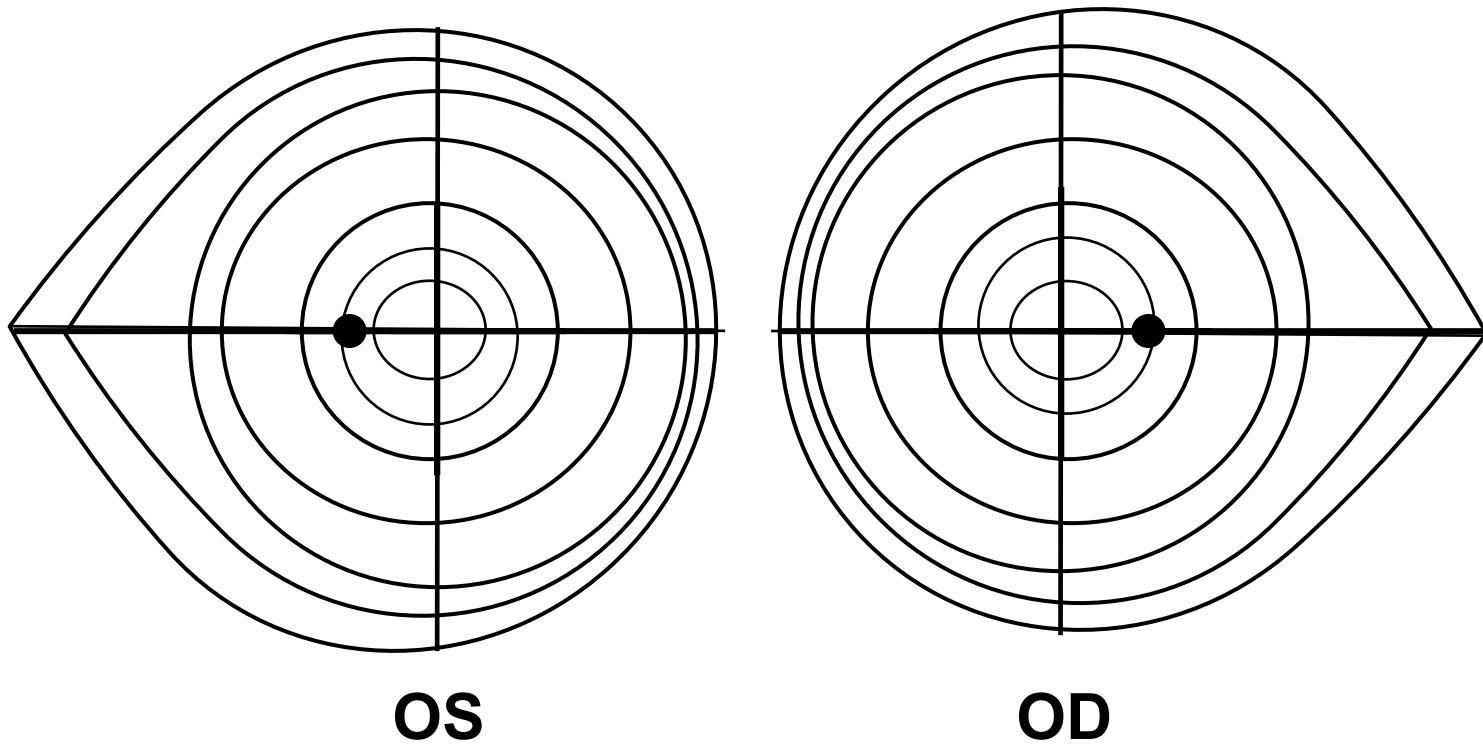
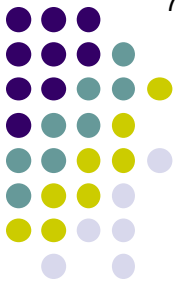
*Here is a representation of the VF for each eye. Which is OD, and which OS? Remember, VFs are **not** drawn as if the pt is looking at you; they're drawn as if **you** are the pt!*

Glaucoma Overview

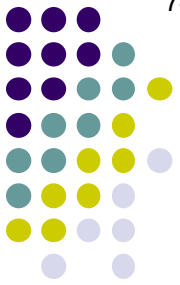


*Measured in degrees from fixation, this is how far the normal VF extends **superiorly, inferiorly, nasally** and **temporally**. (Don't get too fixated on these specific numbers—different sources will give slightly different values.)*

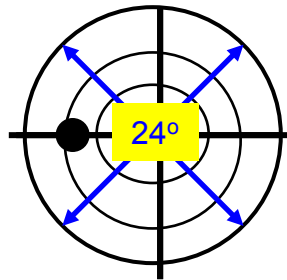
Glaucoma Overview



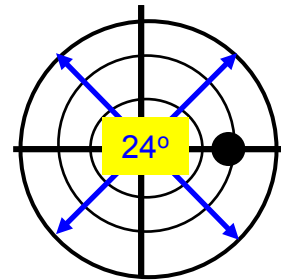
Again when measured in degrees from fixation...



Glaucoma Overview



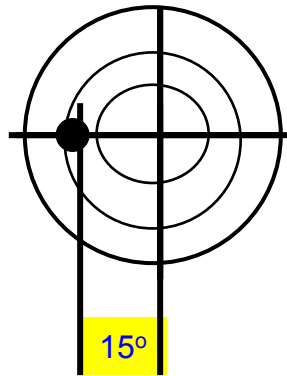
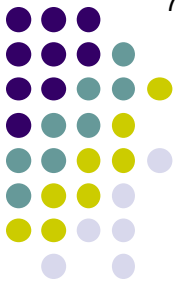
OS



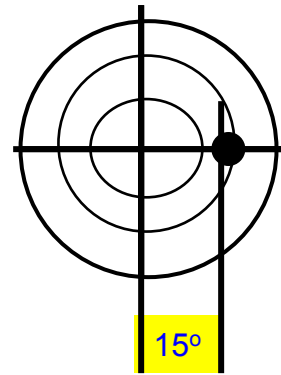
OD

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



OS

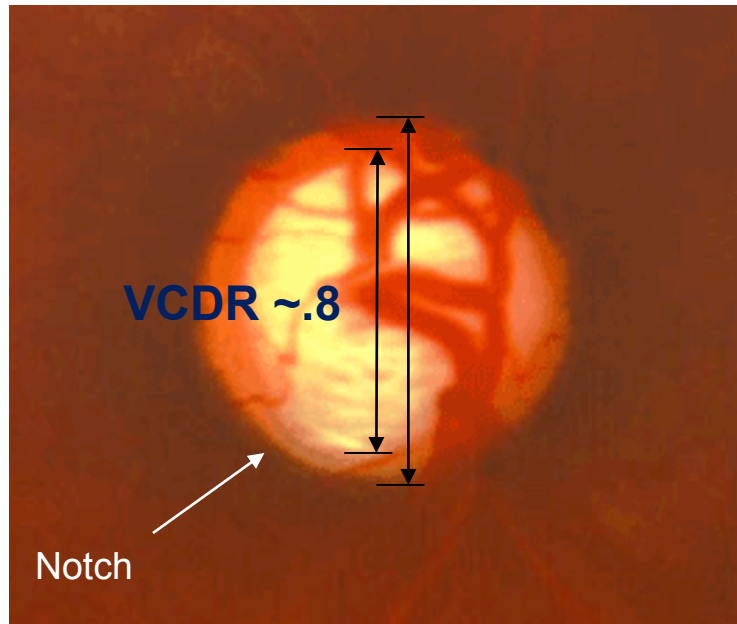


OD

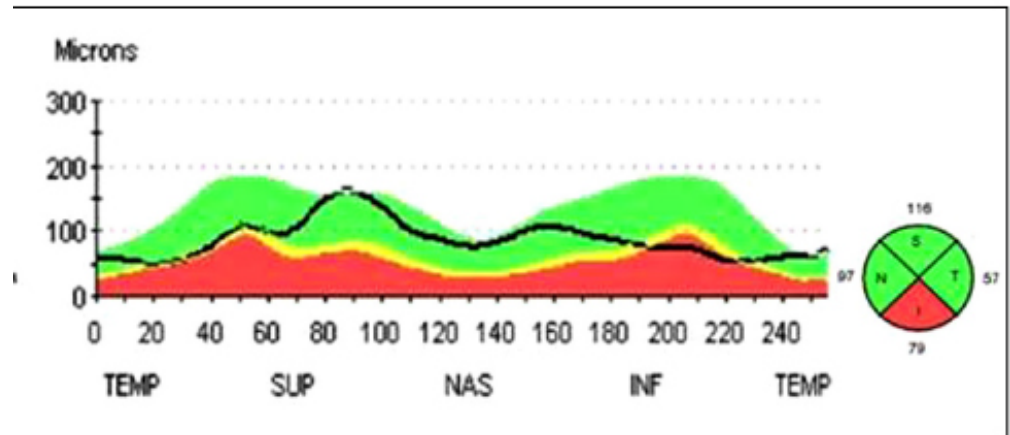
The **blind spot** on a VF is about 15 degrees from fixation

Glaucoma Overview

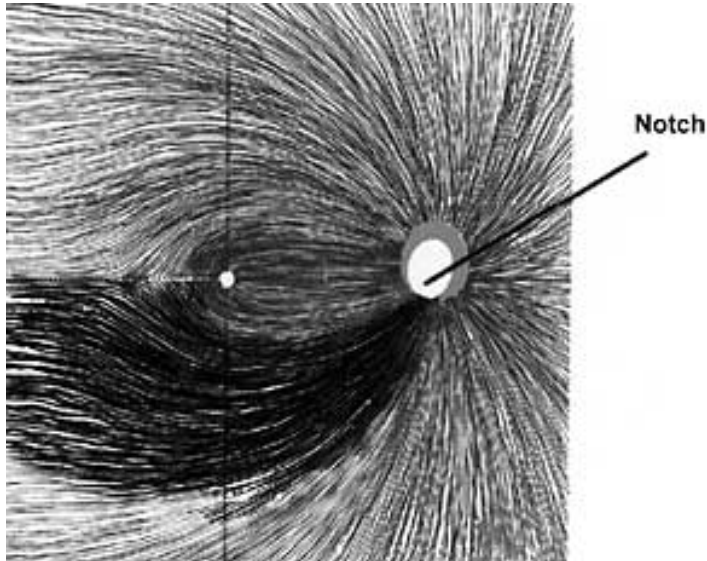
For reasons that have yet to be fully elucidated, glaucoma initially 'prefers' to damage the superior and inferior poles of the ONH. This leads to thinning at the poles (focal thinning is referred to as a 'notch.')



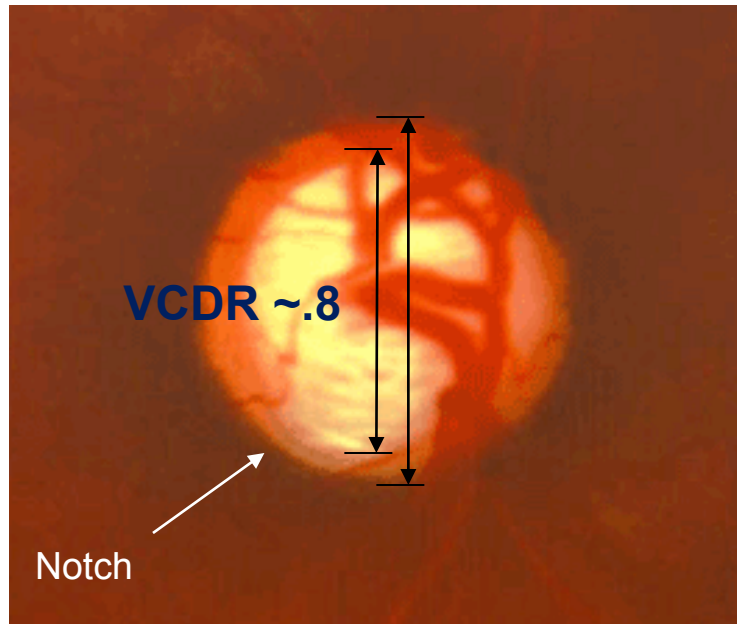
Glaucomatous ONH



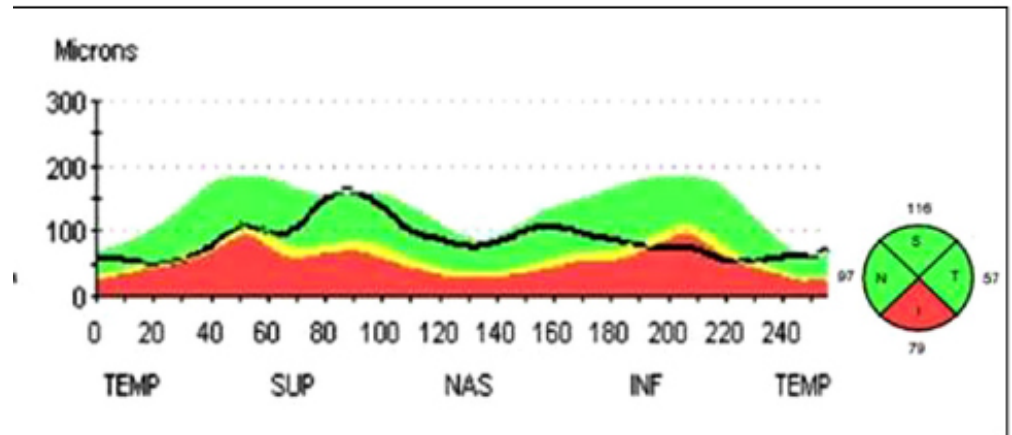
Glaucoma Overview



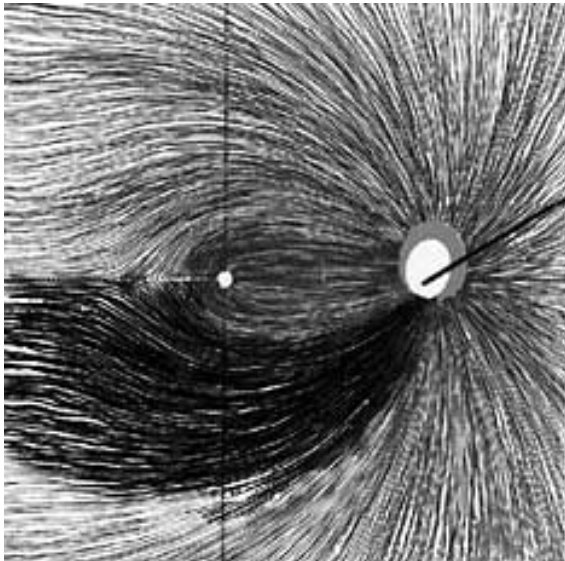
For reasons that have yet to be fully elucidated, glaucoma initially 'prefers' to damage the superior and inferior poles of the ONH. This leads to thinning at the poles (focal thinning is referred to as a 'notch.') Specifically, glaucoma tends initially to affect fibers that originate on the temporal side of the vertical meridian.



Glaucomatous ONH



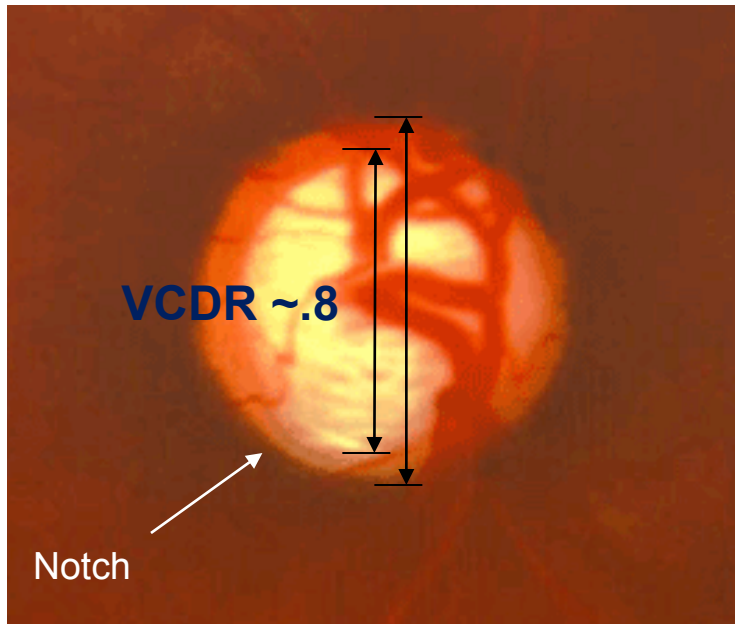
Glaucoma Overview



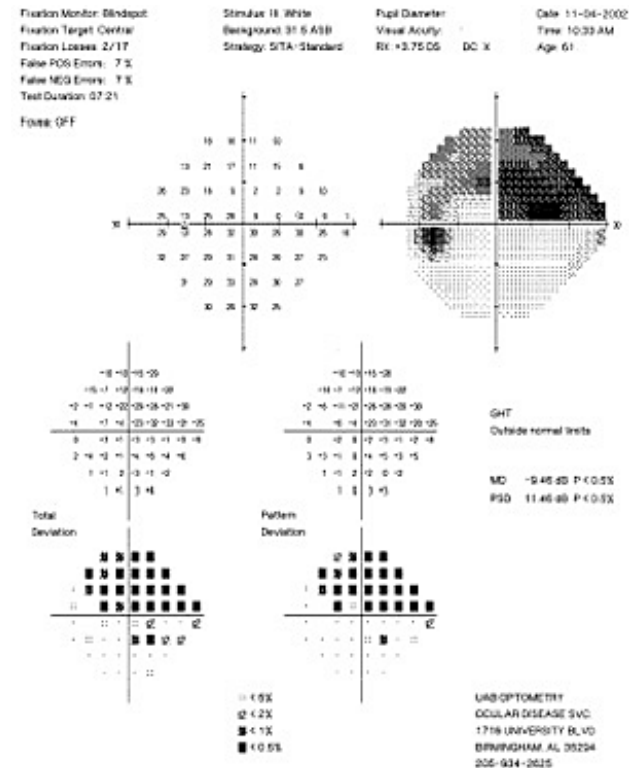
Notch

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.

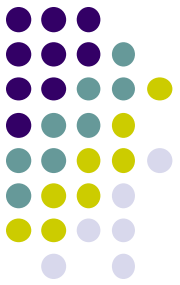
The result of this is that glaucomatous VF defects appear in and extend from the *nasal* visual field.



Glaucomatous ONH



Glaucoma Overview



★ 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.

Glaucoma Overview

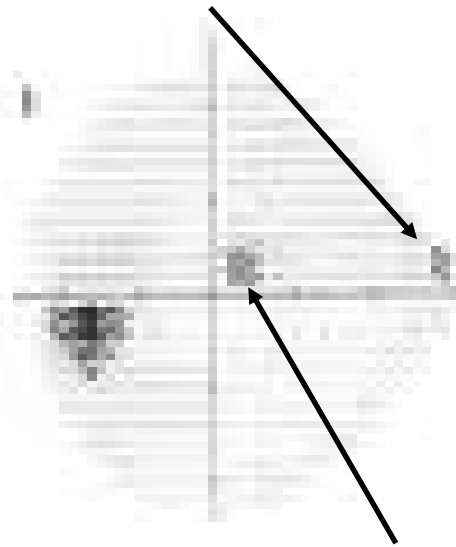


Note: The following set of VFs are from a pt who suffered severe, progressive VF loss in a manner classic for glaucomatous optic neuropathy. I am not personally familiar with this case, and thus cannot provide context regarding the clinical circumstances that resulted in such profound, unchecked VF loss.

Glaucoma Overview



'Early superior nasal step'



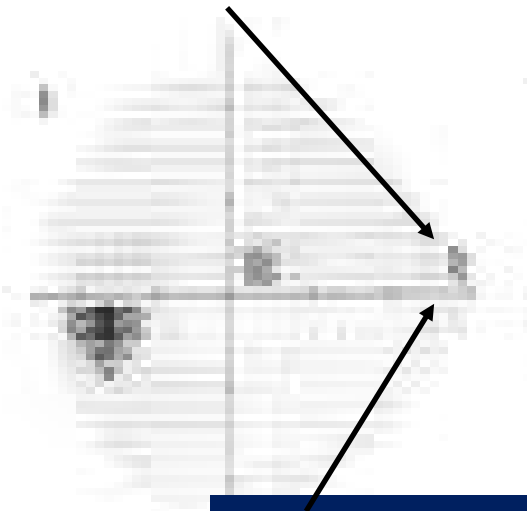
(not real VF loss—going to go away)

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*.

Glaucoma Overview



'Early superior nasal step'



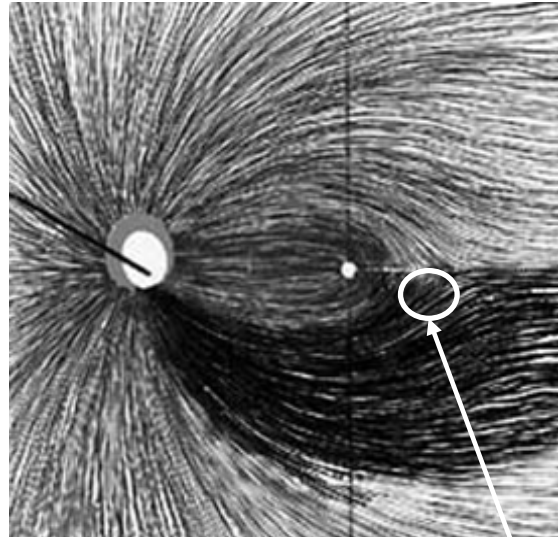
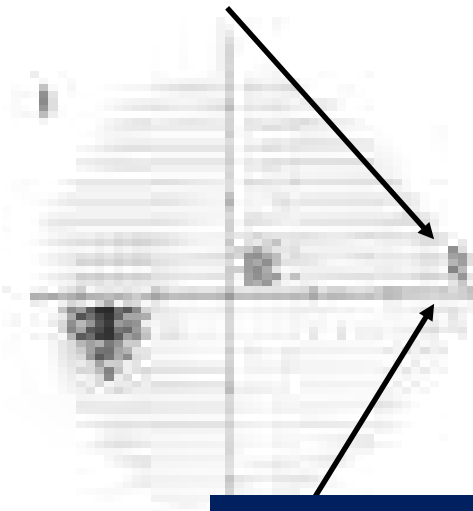
This location in the VF...

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Glaucoma Overview



'Early superior nasal step'



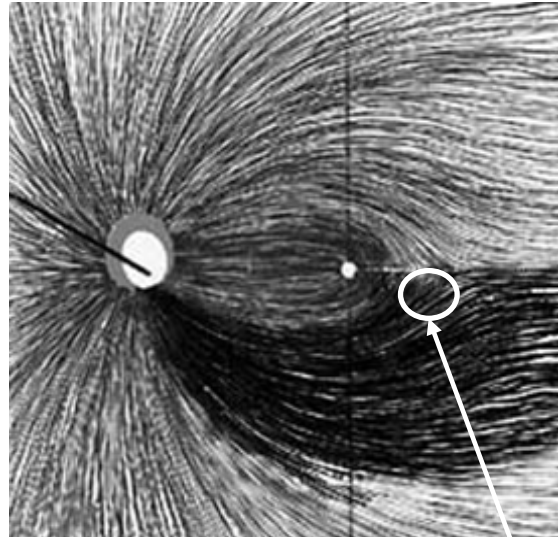
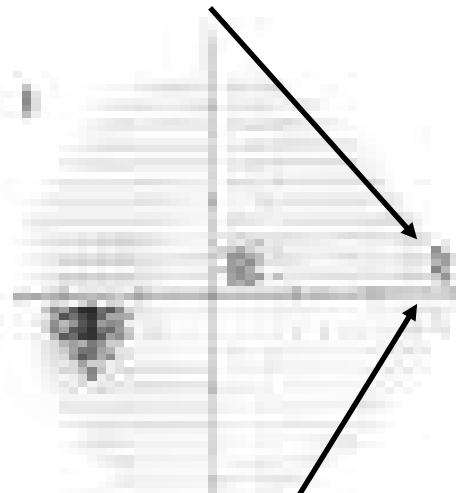
This location in the VF...is associated with **this** location on the retina, meaning that the affected nerve fibers originated there...

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Glaucoma Overview

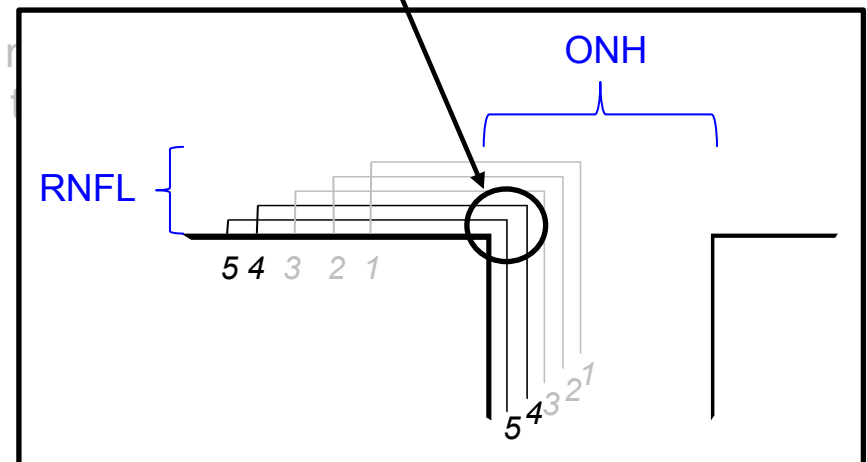


'Early superior nasal step'



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

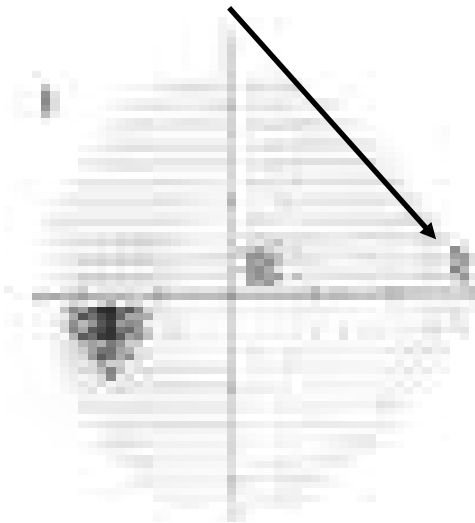
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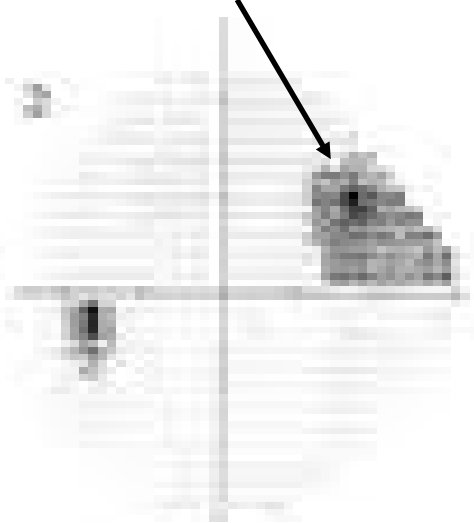
Glaucoma Overview



'Early superior nasal step'



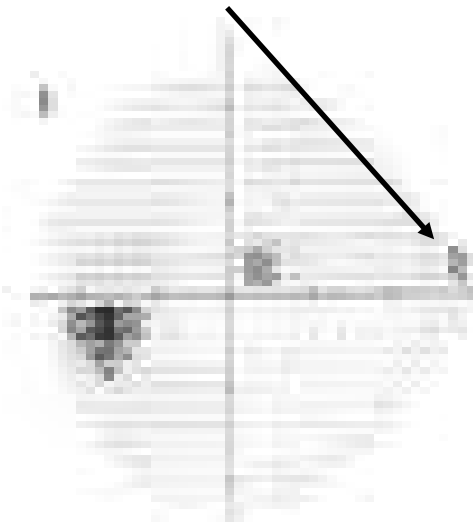
'Superior nasal step'



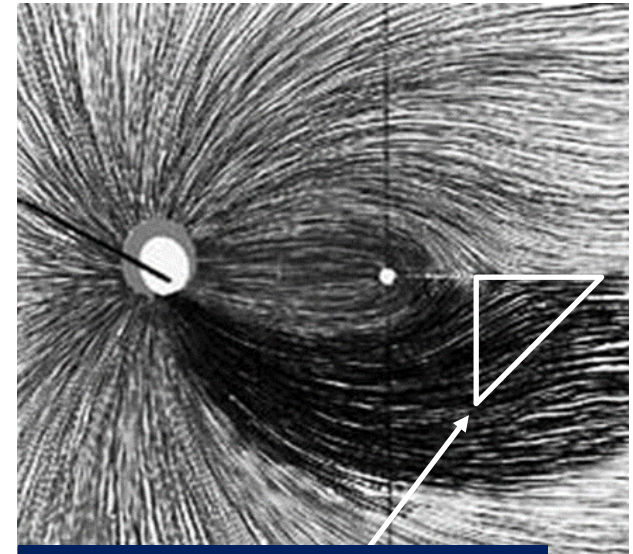
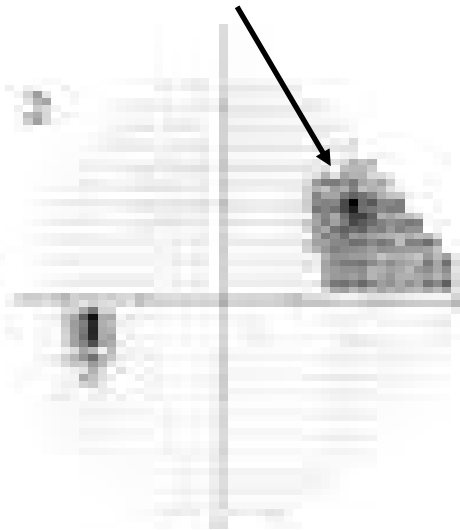
If left untreated, the nasal step will gradually enlarge.

Glaucoma Overview

'Early superior nasal step'



'Superior nasal step'



Note the area of origin for affected fibers has grown

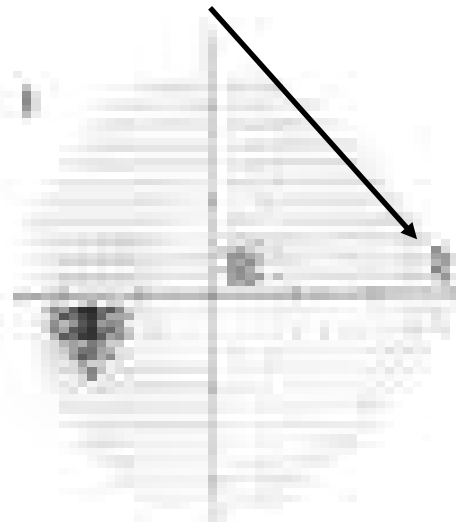


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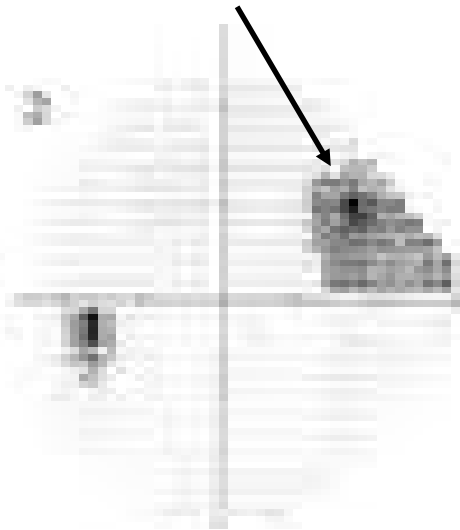
Glaucoma Overview



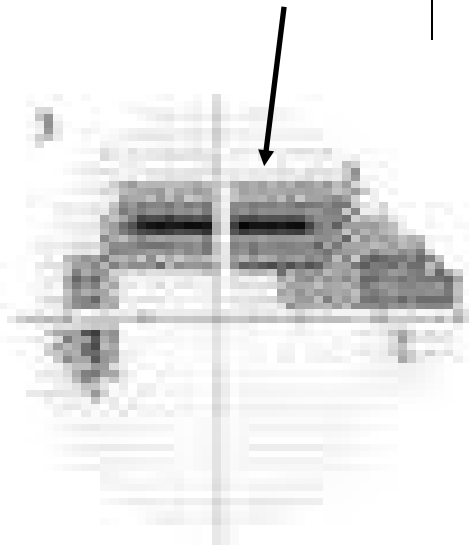
'Early superior nasal step'



'Superior nasal step'



'Superior arcuate'

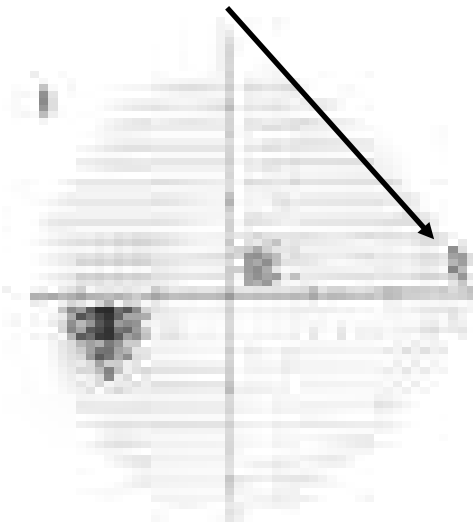


As glaucoma damage progresses, further loss of nerve fibers joining at that portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an *arcuate*.

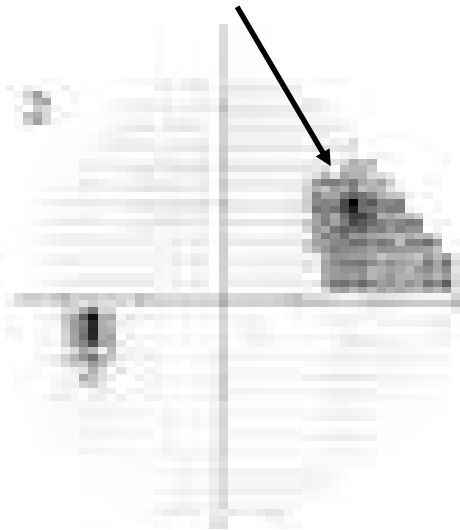
Glaucoma Overview



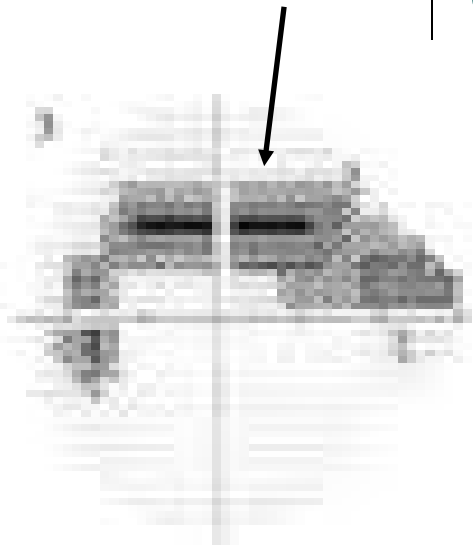
'Early superior nasal step'



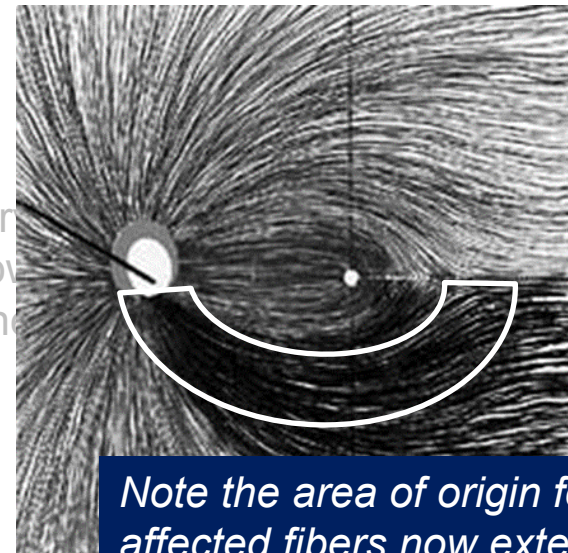
'Superior nasal step'



'Superior arcuate'



As glaucoma damage progresses, further loss of nerve fibers from 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 defect is termed an *arcuate*.

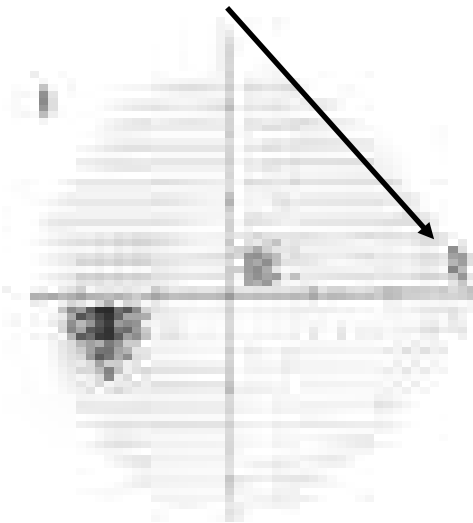


Note the area of origin for affected fibers now extends all the way to the ONH itself

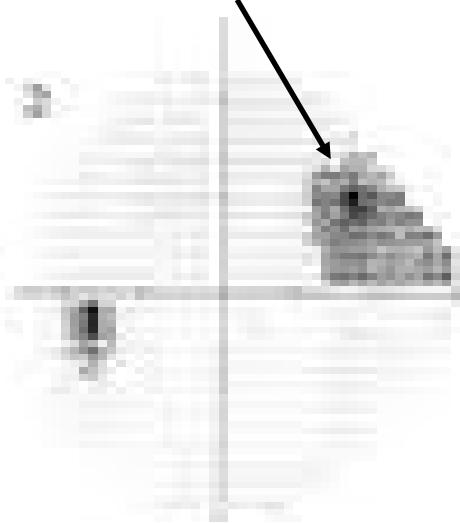
Glaucoma Overview



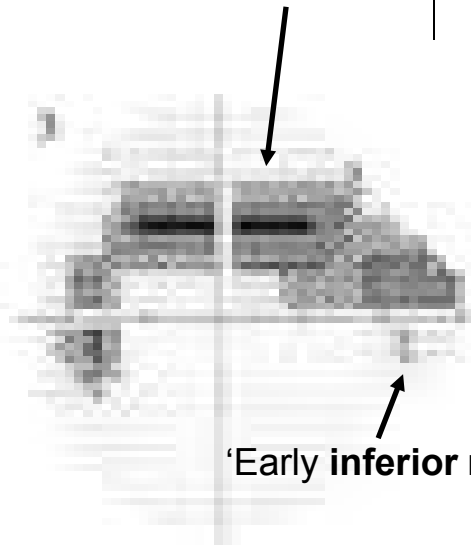
'Early superior nasal step'



'Superior nasal step'



'Superior arcuate'



'Early **inferior** nasal step'

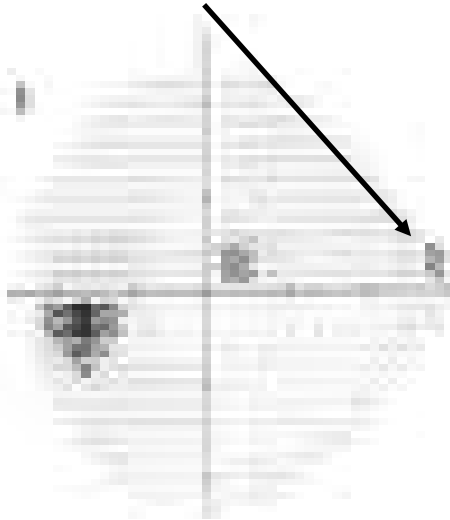
As glaucoma damage progresses, further loss of nerve fibers joining at that portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an *arcuate*.

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

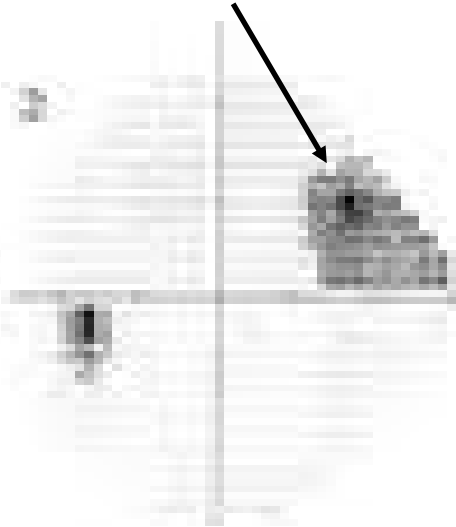
Glaucoma Overview



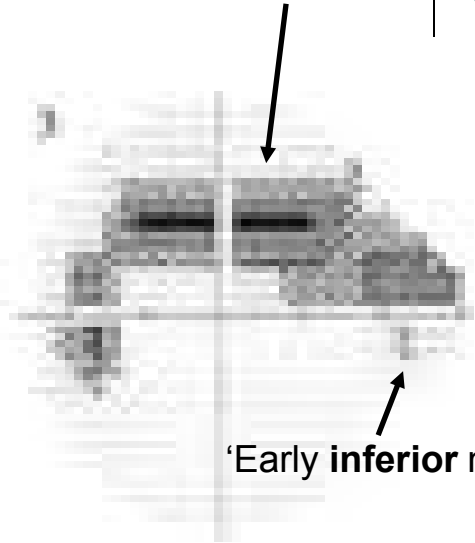
'Early superior nasal step'



'Superior nasal step'

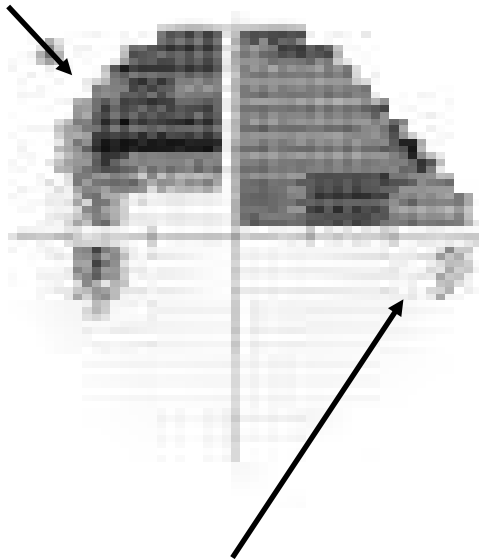


'Superior arcuate'



'Early **inferior** nasal step'

'Advanced arcuate'



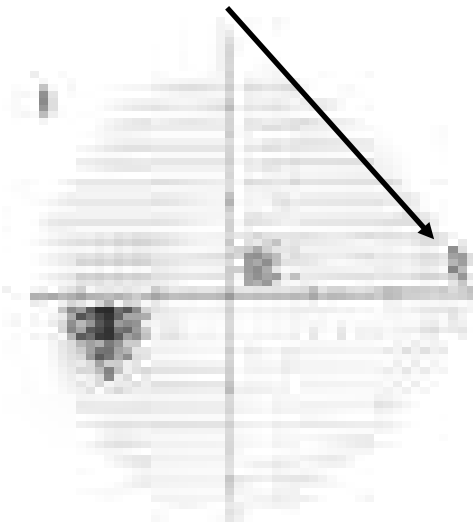
'Early inferior nasal step'

If left unchecked, an arcuate will expand into the surrounding portion of the VF.

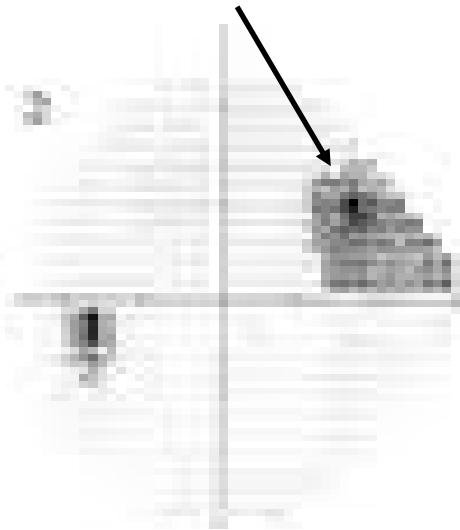
Glaucoma Overview



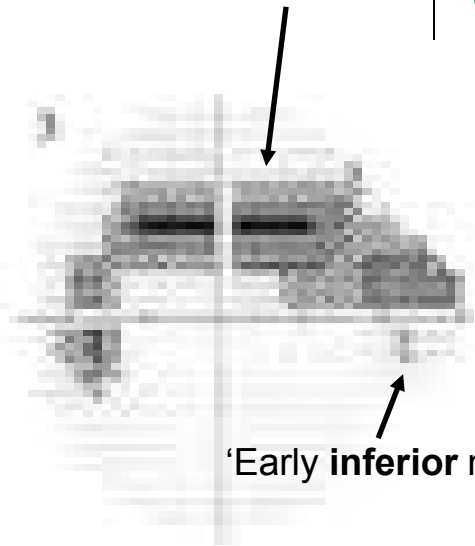
'Early superior nasal step'



'Superior nasal step'

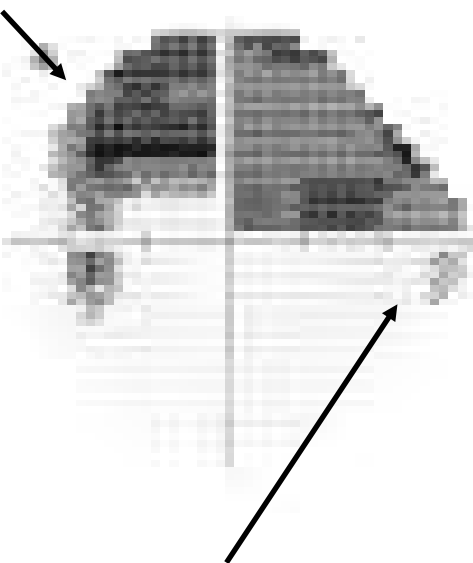


'Superior arcuate'

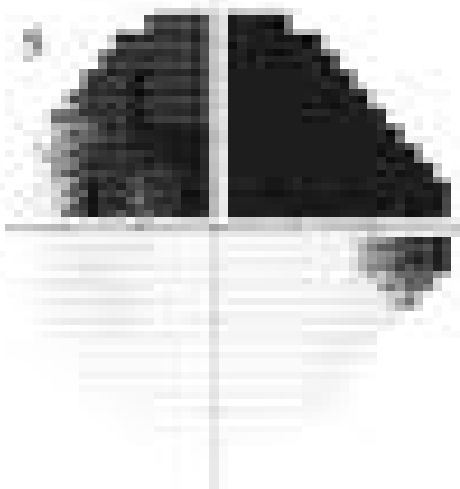


'Early **inferior** nasal step'

'Advanced arcuate'



'Altitudinal defect'



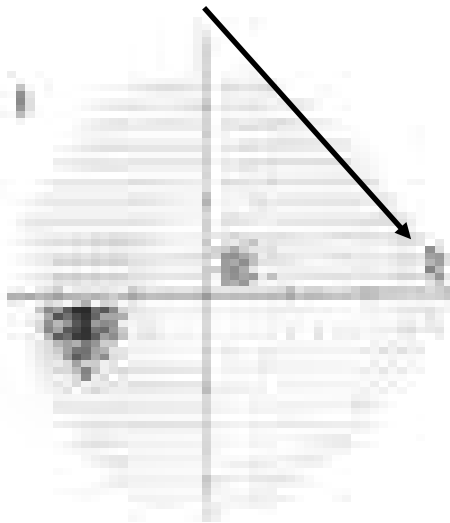
'Early inferior nasal step'

Once an arcuate has expanded sufficiently, it becomes an *altitudinal defect*. The superior visual field is now all but gone.

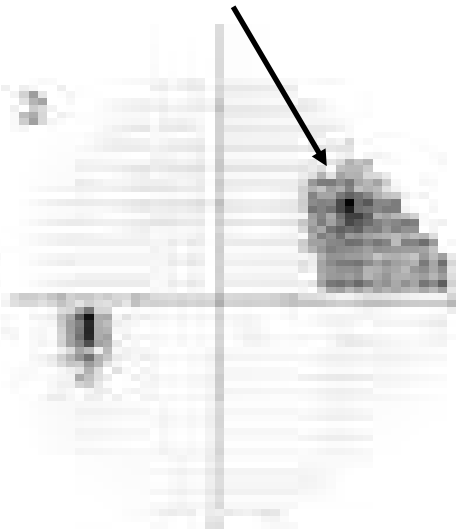
Glaucoma Overview



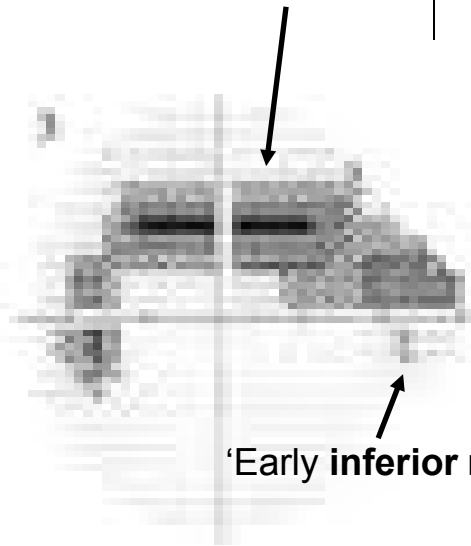
'Early superior nasal step'



'Superior nasal step'

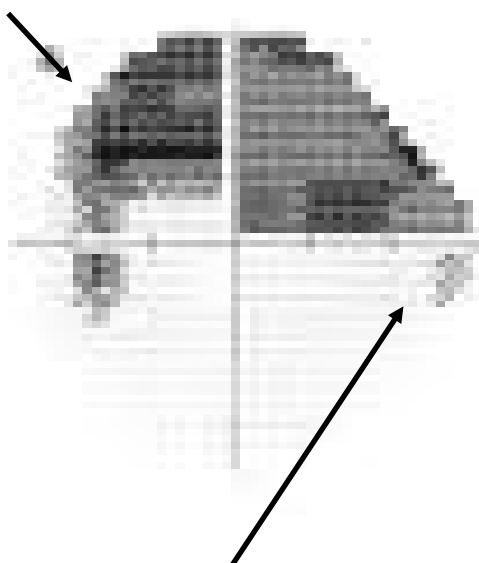


'Superior arcuate'

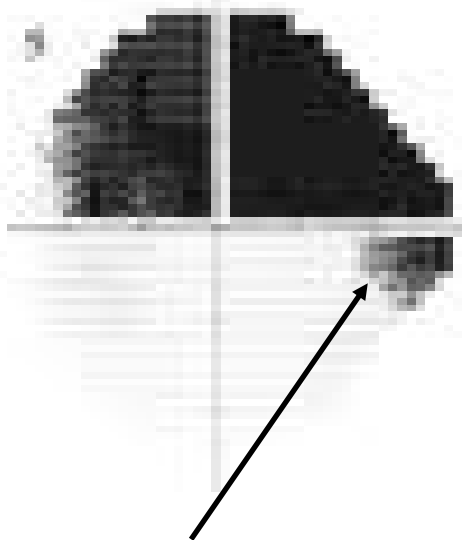


'Early **inferior** nasal step'

'Advanced arcuate'



'Altitudinal defect'



'Early inferior nasal step'

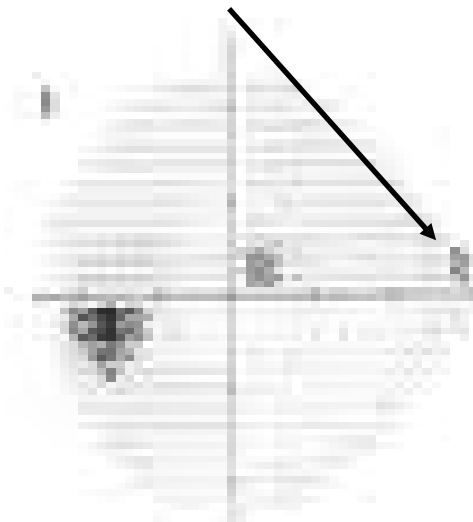
'Inferior nasal step'

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.

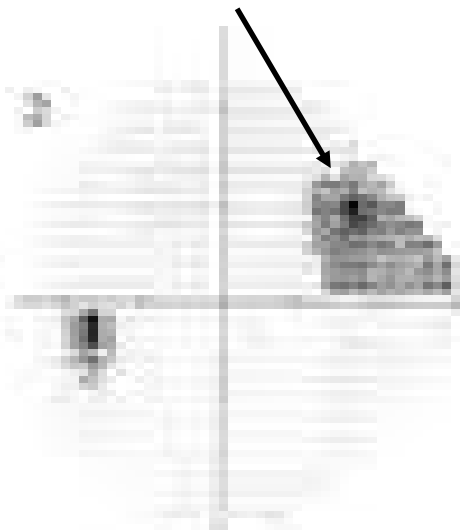
Glaucoma Overview



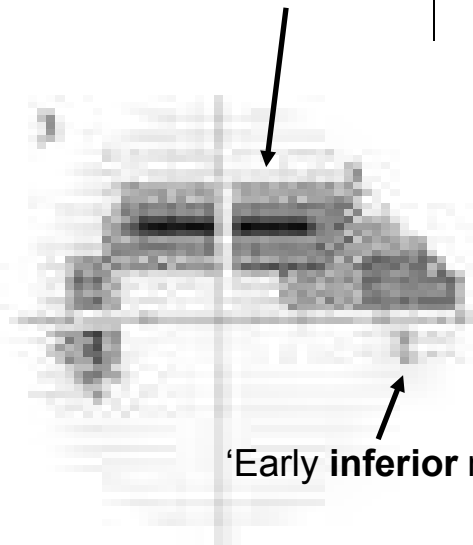
'Early superior nasal step'



'Superior nasal step'

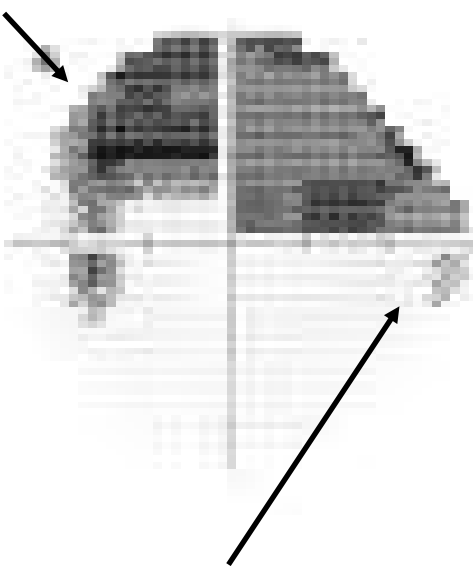


'Superior arcuate'



'Early inferior nasal step'

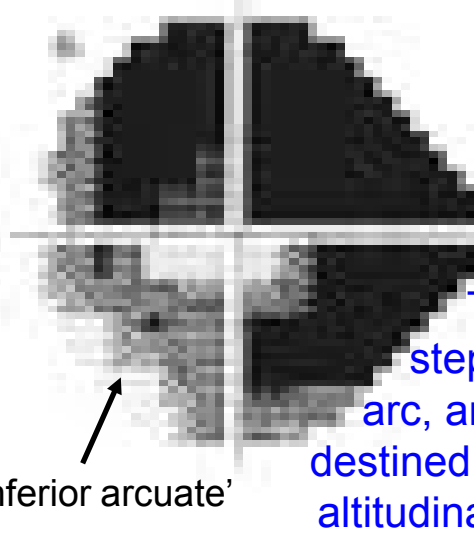
'Advanced arcuate'



'Altitudinal defect'



'Altitudinal defect'



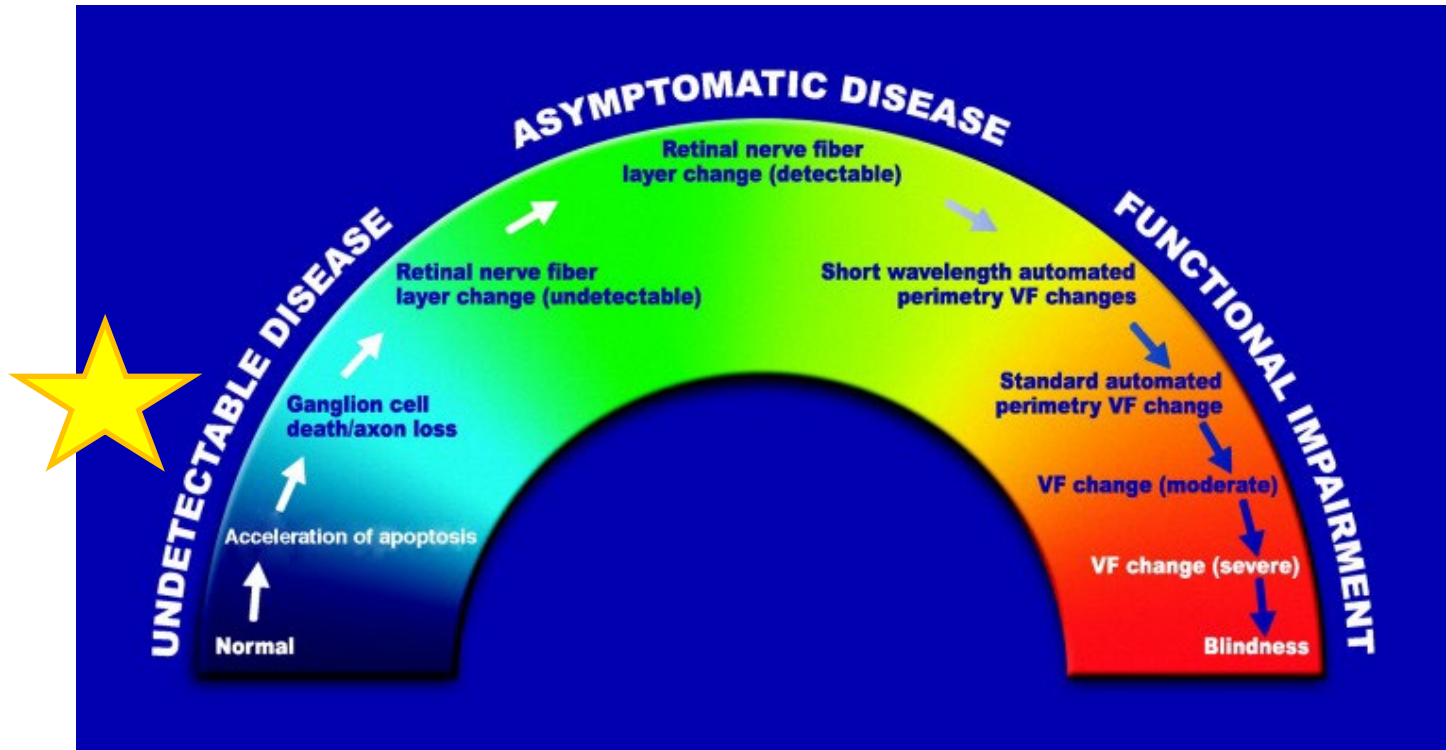
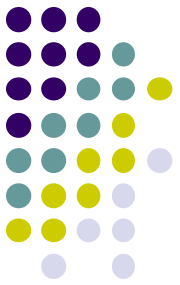
'Early inferior nasal step'

'Inferior nasal step'

'Inferior arcuate'

The inferior step is now an arc, and appears destined to become altitudinal, resulting in blindness.

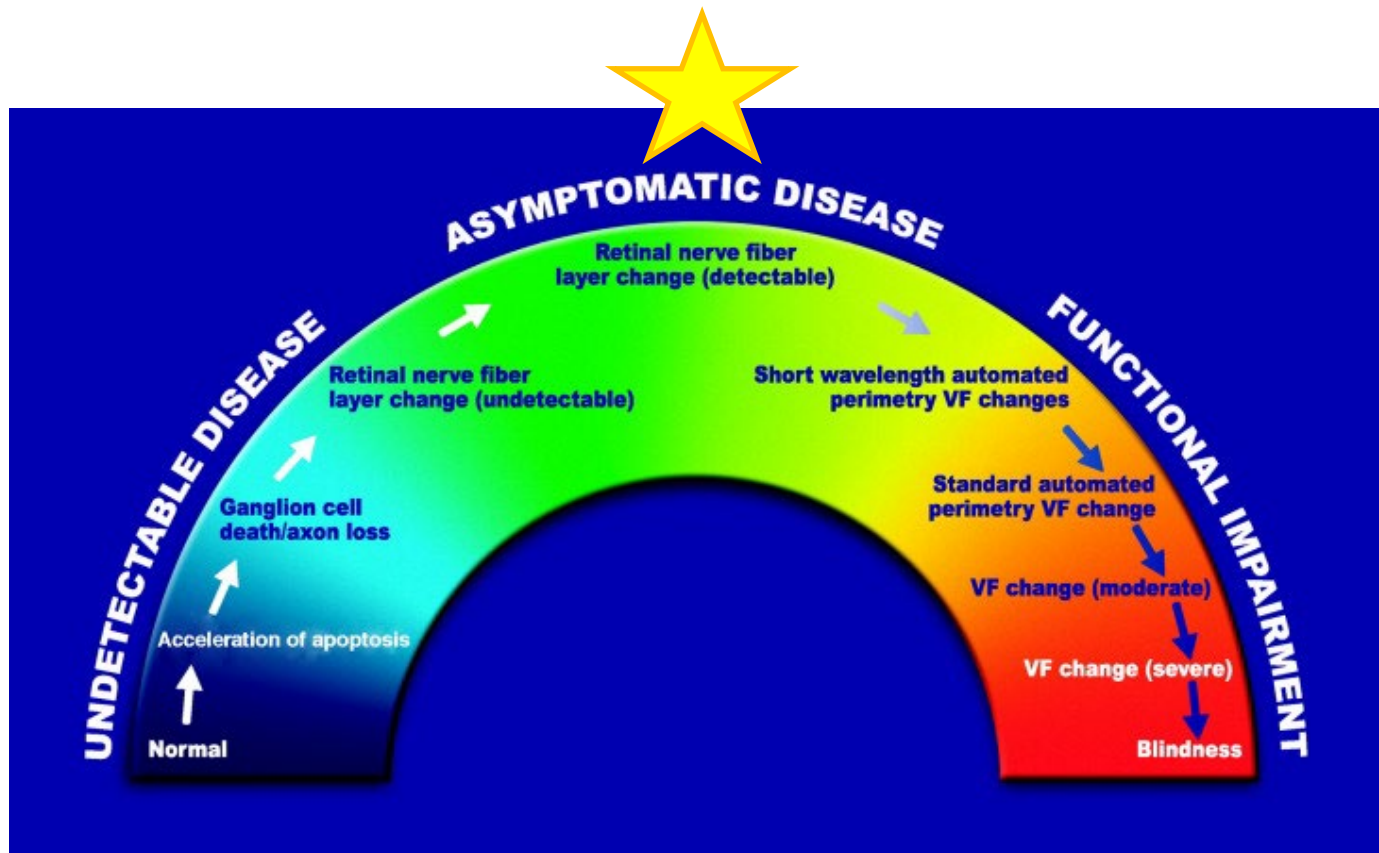
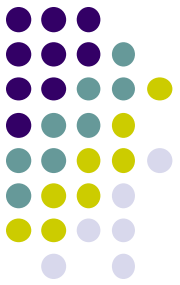
Glaucoma Overview



The 'Glaucoma Continuum'

Glaucoma is a progressive condition, passing from undetectable early disease...

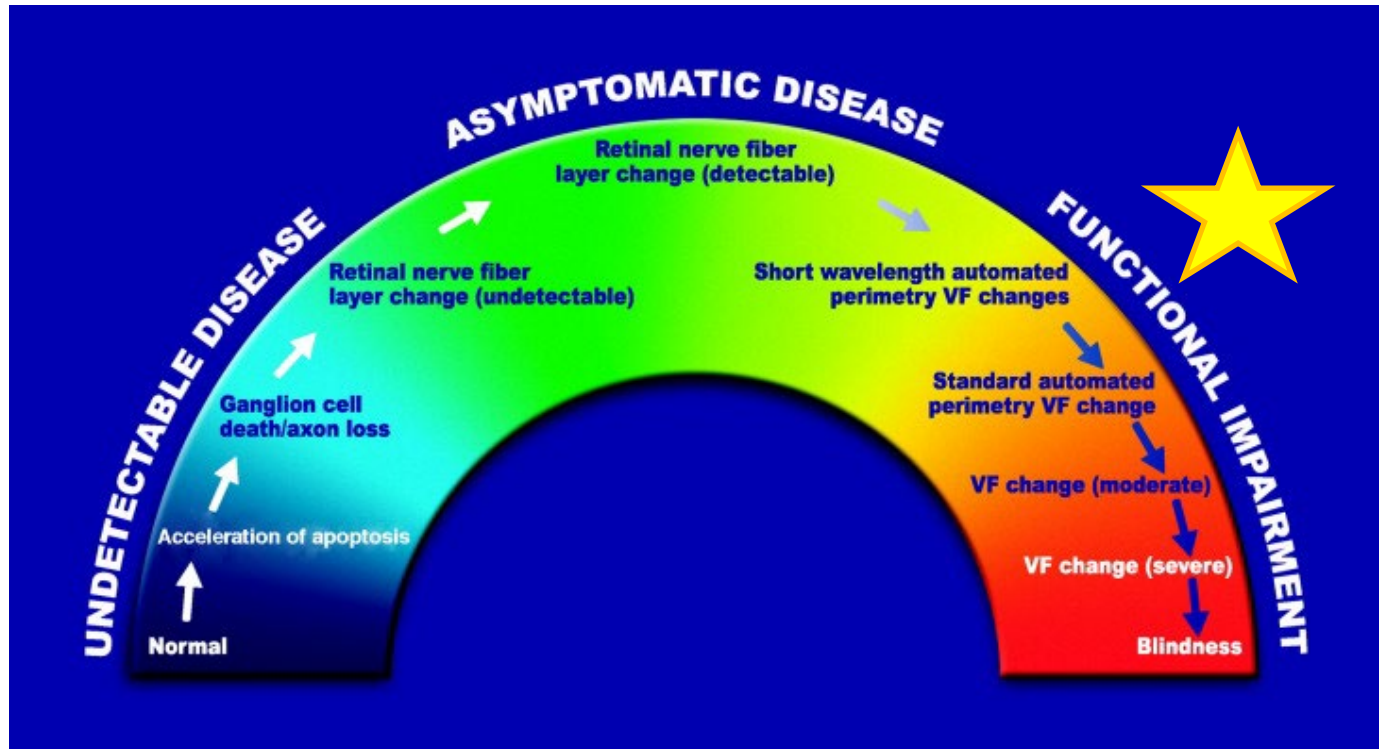
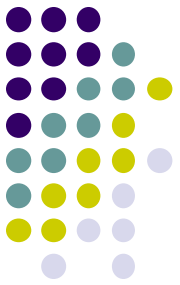
Glaucoma Overview



The 'Glaucoma Continuum'

Glaucoma is a progressive condition, passing from undetectable early disease...
to asymptomatic-but-detectable (via RNFL imaging) disease...

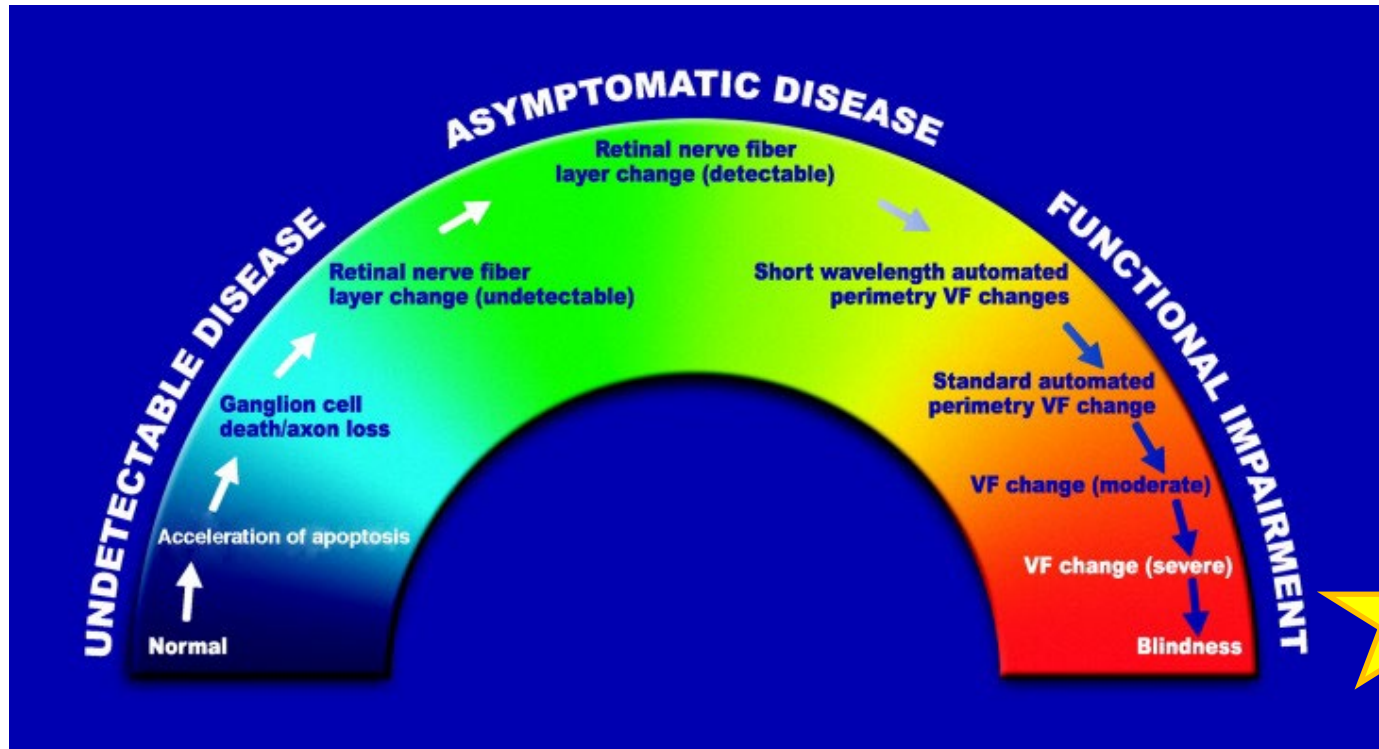
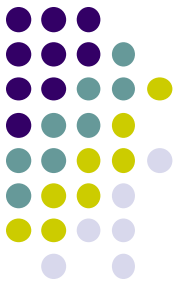
Glaucoma Overview



The 'Glaucoma Continuum'

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...

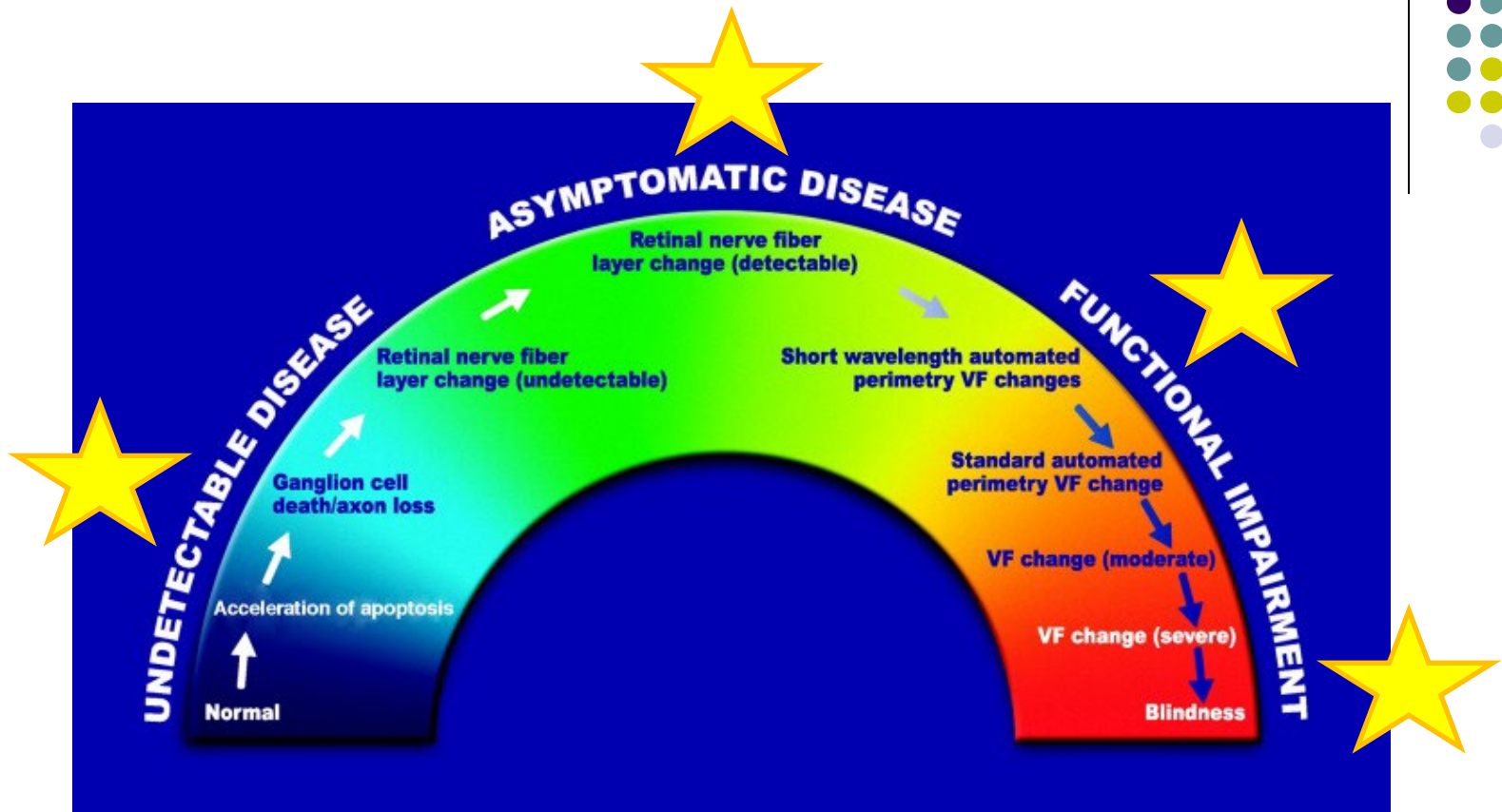
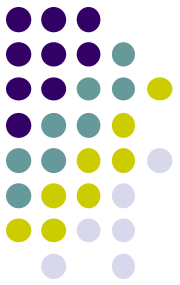
Glaucoma Overview



The 'Glaucoma Continuum'

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.

Glaucoma Overview

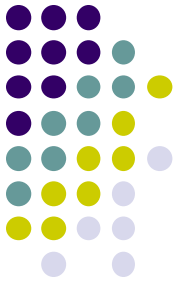


The 'Glaucoma Continuum'

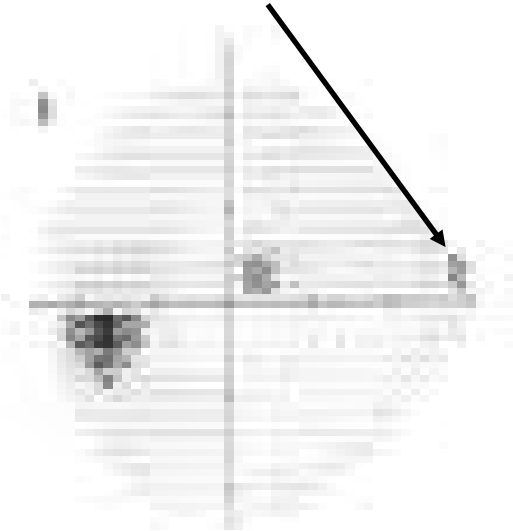
Glaucoma is a progressive condition, passing from undetectable early disease... to asymptomatic-but-detectable (via RNFL imaging) disease... to functional (ie, marked by VF loss) disease... to severe vision loss and blindness.

This stepwise pattern of progression has been coined the **glaucoma continuum**.

Glaucoma Overview



‘Early nasal step’

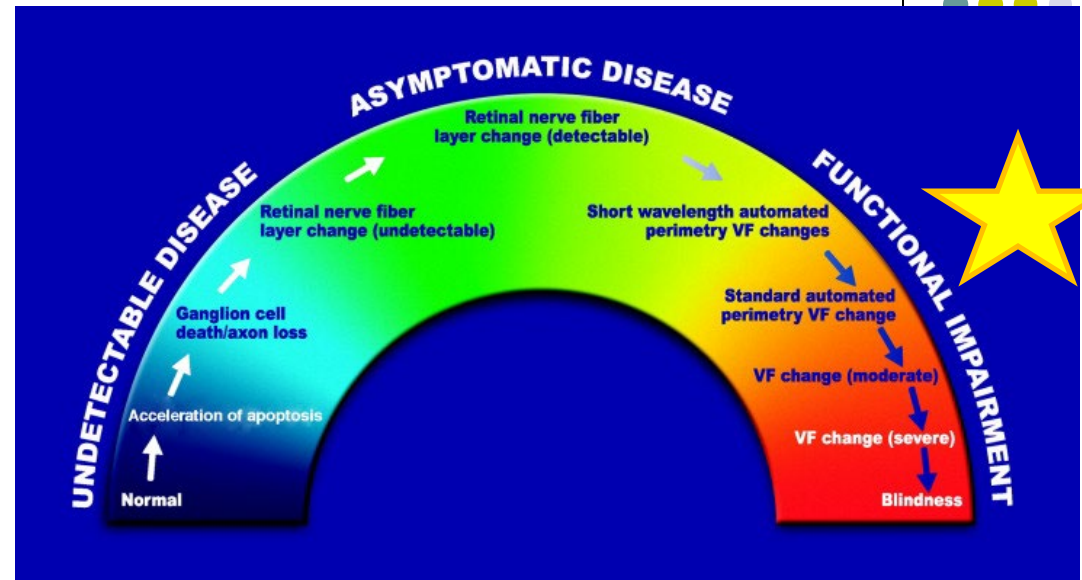
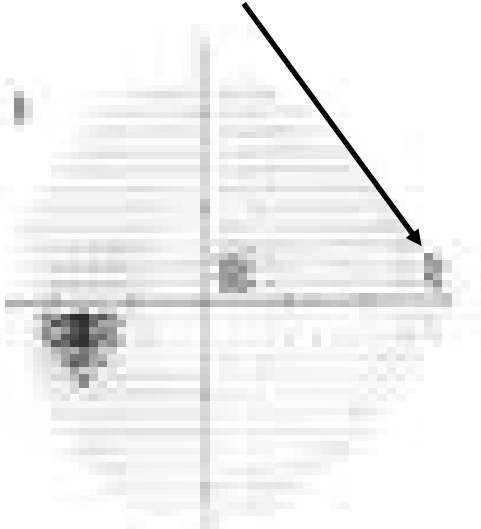


In this regard, a word on the notion of ‘early’ glaucoma. We previously described the above VF defect as an ‘early’ nasal step.

Glaucoma Overview

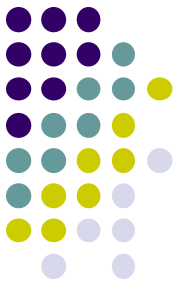


‘Early nasal step’

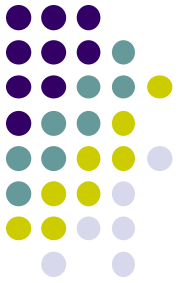


In this regard, a word on the notion of ‘early’ glaucoma. We previously described the above VF defect as an ‘early’ nasal step. But take note of the point along the glaucoma continuum at which such a VF defect occurs—clearly, it doesn’t qualify as ‘early’ disease with respect to the continuum. *Don’t mistake early VF changes for early disease!*

Glaucoma Overview



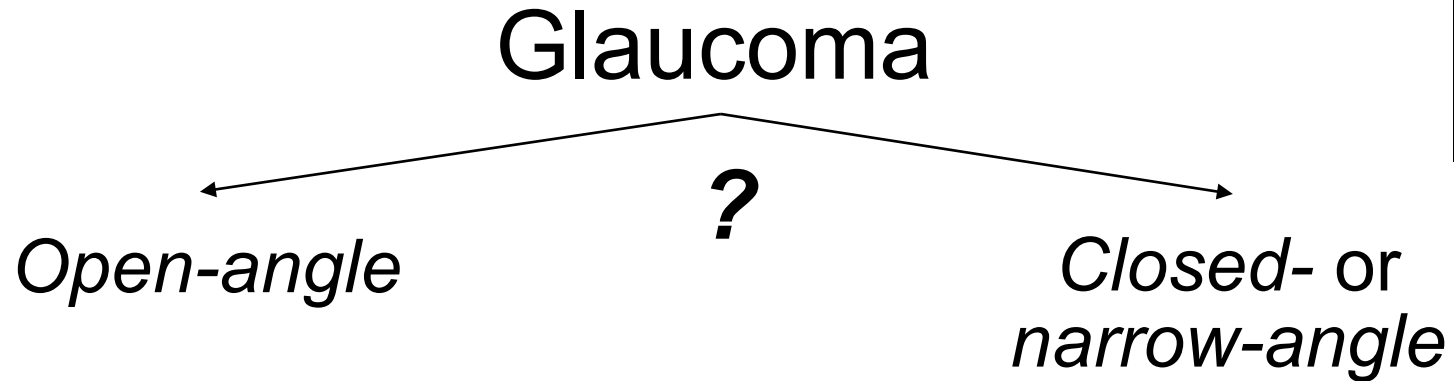
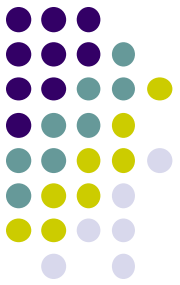
Finally, let's look briefly at how one should think through the new glaucoma case sitting in your exam chair



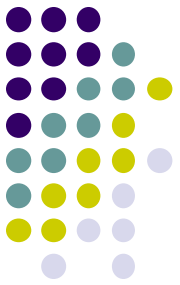
Glaucoma



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



The first thought you should have when
encountering a pt you suspect has glaucoma is...
What is the status of the angle?



Glaucoma

Open-angle

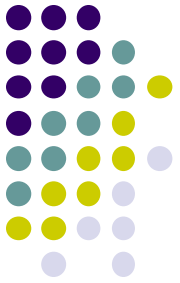
*Closed- or
narrow-angle*

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

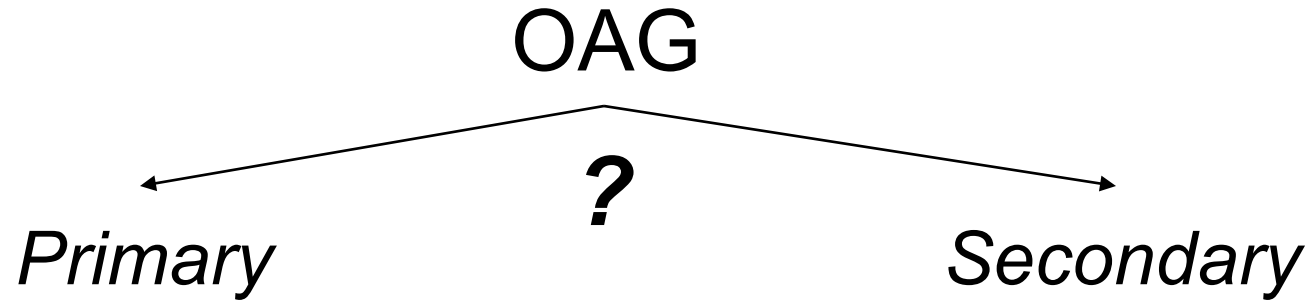
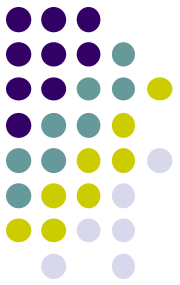


OAG



If you have determined a pt has open-angle glaucoma,
your next question is...

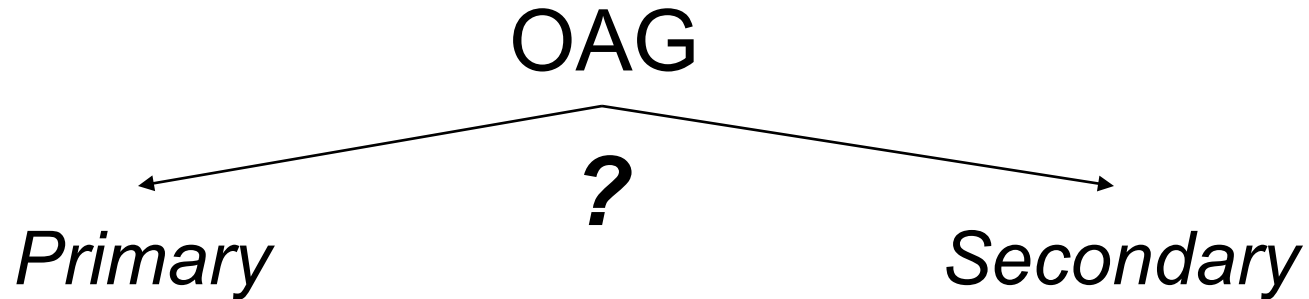
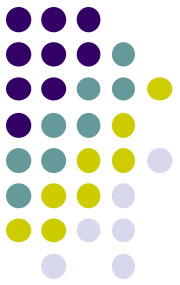
Glaucoma Overview



If you have determined a pt has open-angle glaucoma,
your next question is...

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

Glaucoma Overview

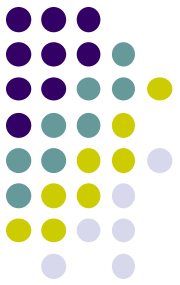


If you have determined a pt has open-angle glaucoma,
your next question is...

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.*

Glaucoma Overview



OAG

Primary

Secondary

PXS

Pigmentary

Tumor-Induced

Lens-Induced

Inflammation-Induced

Drug-Induced

Trauma-Related

↑ EVS

Schwartz syndrome

- Phacolytic
- Phacoantigenic
- Lens particle

- Posner-Schlossman
- Fuchs heterochromic iridocyclitis

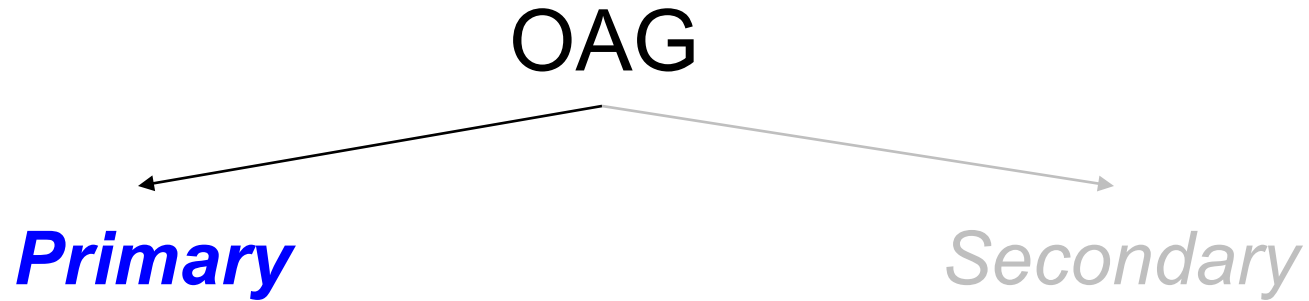
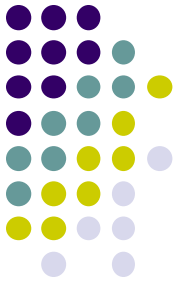
- Steroids
- Mydriatics

- Angle recession
- Cyclodialysis cleft
- Hyphema
- Hemolytic
- Ghost cell

- AVM
- Venous obstruction
- SVC syndrome
- C-C fistula

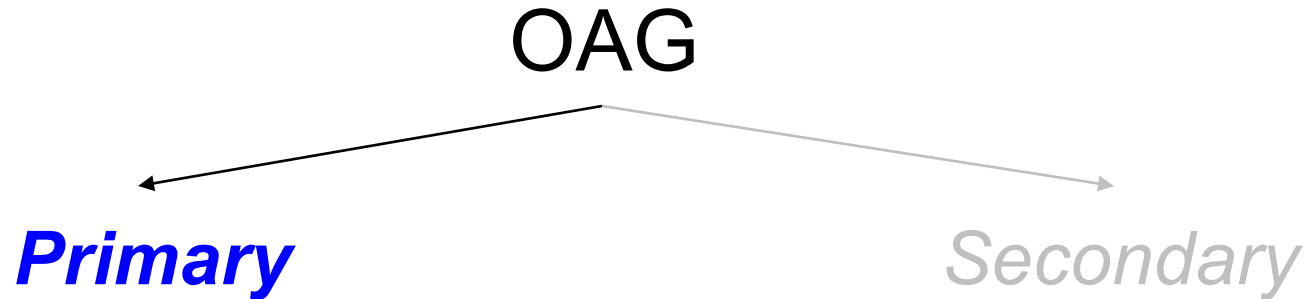
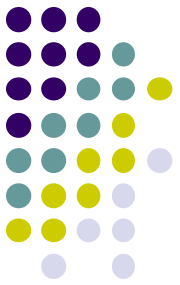
These are the causes of secondary OAG that must be ruled out. *Do not memorize the chart*—learning the 2ndry OAGs is a task for another day. For now, just be aware they exist.

Glaucoma Overview



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.

Glaucoma Overview

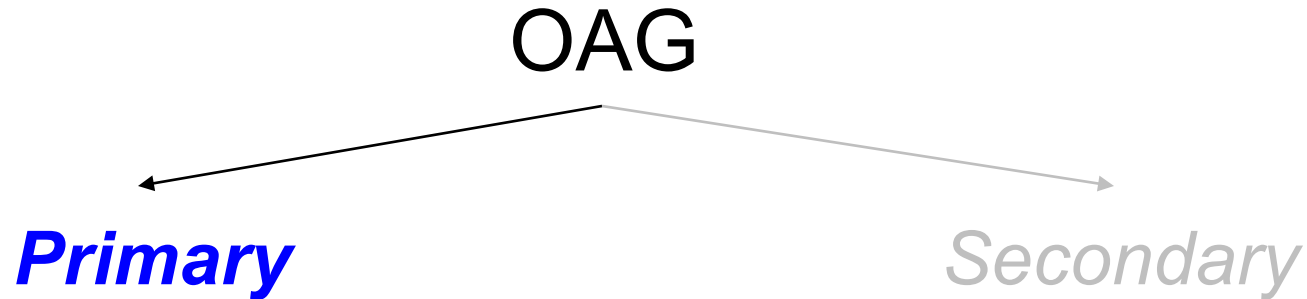
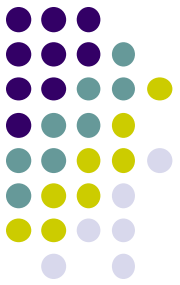


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The BCSC *Glaucoma* book emphasizes five risk factors for POAG (other than IOP):

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.

Glaucoma Overview

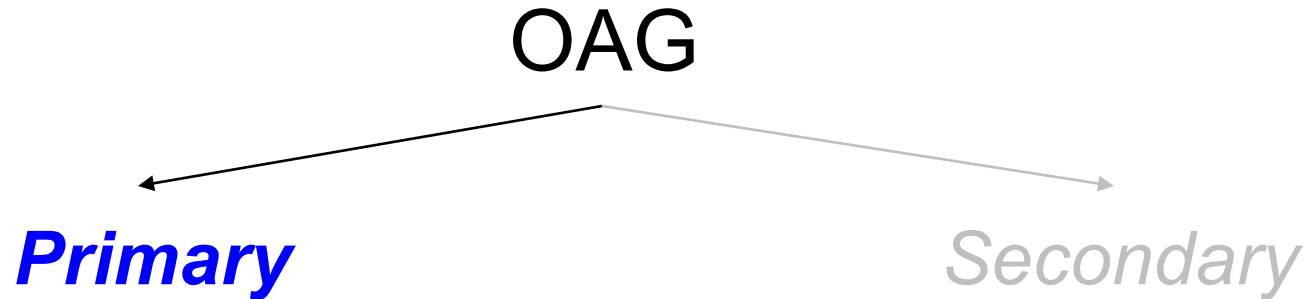
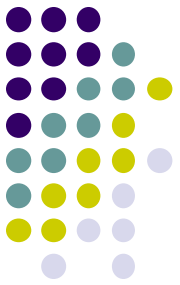


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2. **Age** is a strong risk factor for both having POAG, and for POAG progression.
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Glaucoma Overview

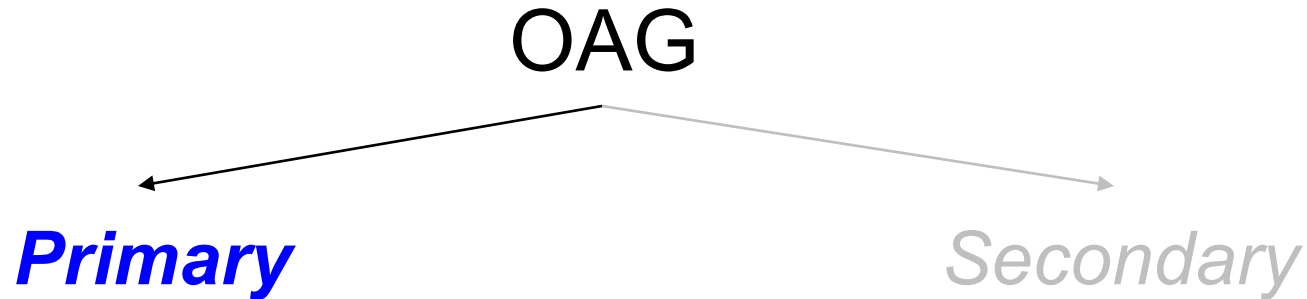
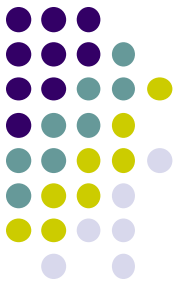


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- 4.
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Glaucoma Overview

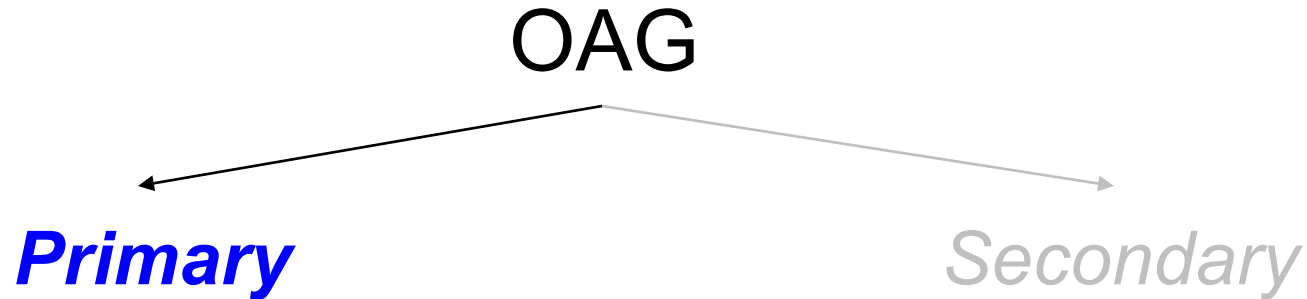
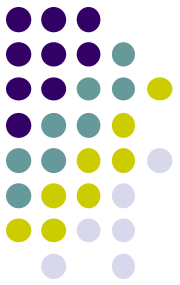


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2. **Age** is a strong risk factor for both having POAG, and for POAG progression.
3. **Family history** of a first-degree relative is significant.
4. **Myopia** has been determined to be a risk factor by most (but not all) studies looking at the subject.
- 5.

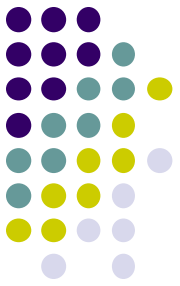
Glaucoma Overview



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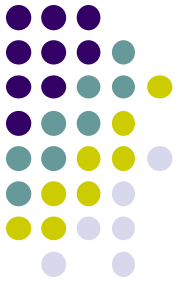
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3. **Family history** of a first-degree relative is significant.
4. **Myopia** has been determined to be a risk factor by most (but not all) studies looking at the subject.
5. **Thin central cornea** was established as a risk factor in the *Ocular Hypertension Treatment Study* (OHTS).

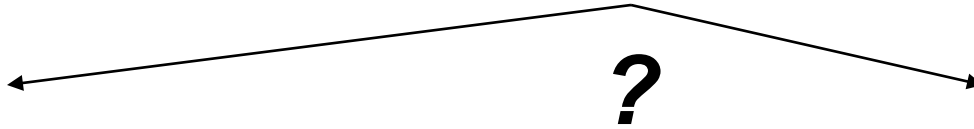


Angle-Closure Glaucoma

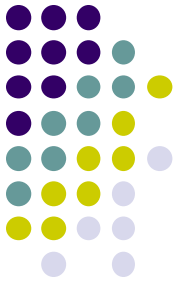
Next we will turn our attention to angle-closure glaucoma



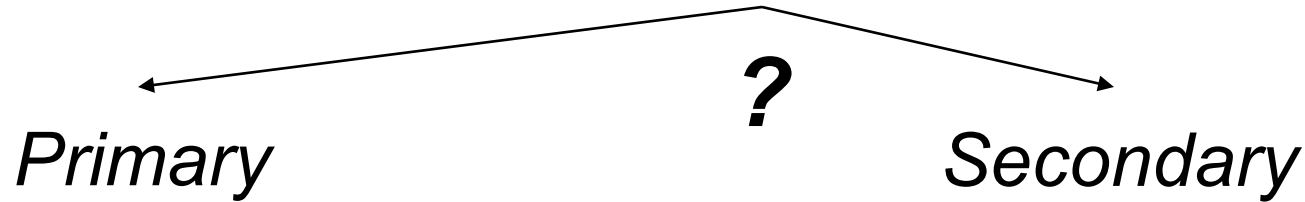
Angle-Closure Glaucoma



If you have determined a pt has angle-closure glaucoma,
your next question is:

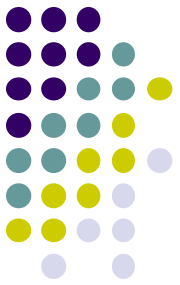


Angle-Closure Glaucoma



If you have determined a pt has angle-closure glaucoma,
your next question is:

Is it primary angle closure, or secondary?



Angle-Closure Glaucoma

Primary

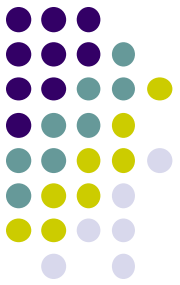
Secondary

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.

Secondary Angle Closure Glaucoma



w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

- Phacomorphic
- Ectopia lentis

Aphakic/
Pseudophakic

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.

'Push'

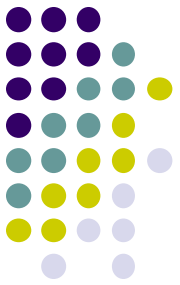
- Aqueous misdirection
- ERD/choroidal effusion
- Retinal surgery
- Nanophthalmos
- Drug-induced
- PFV

'Pull'

- NVG
- ICE
- Flat AC
- Epithelial/
fibrous
ingrowth

Both/Either

- Inflammation
- Tumor



Angle-Closure Glaucoma

Primary

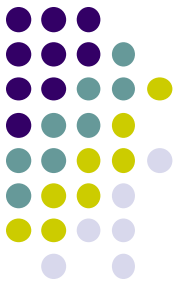
Secondary

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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**:



Angle-Closure Glaucoma

Primary

Secondary

If you have determined a pt has angle-closure glaucoma,
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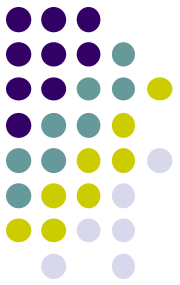
Is it primary angle closure, or secondary?

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

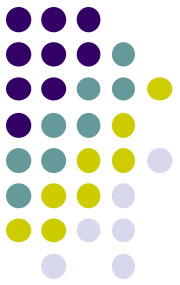


Angle-Closure Glaucoma

Primary (PACG)

Secondary

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.

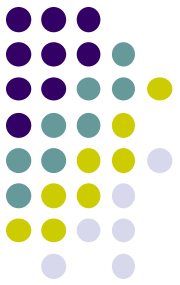


Angle-Closure Glaucoma

Primary (PACG)

Secondary

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.)

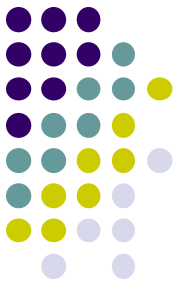


Angle-Closure Glaucoma

←
Primary (PACG)

→
Secondary

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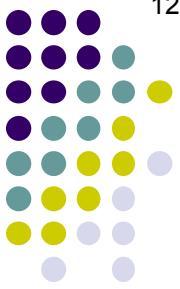
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Glaucoma Overview



That's it! Go through this slide-set a couple of times (at least) until you feel like you have a handle on it. [When you're ready, do slide-set G0, which covers this material in a Q&A format \(and more detail\).](#)