



Define glaucoma.





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That's why glaucoma management concerns nothing but IOP-lowering maneuvers!



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Speaking of IOP...Let's drill down on the factors that determine it





Fill in the IOP equation below.







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Fill in the IOP equation below. What is its eponymous name? The equation

IOP = Aqueous Formation Rate (μL/min) Outflow Facility (μL/min/mmHg) + Episcleral Venous Pressure (mmHg)





Fill in the IOP equation below. What is its eponymous name? The Goldmann equation

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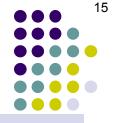
Note how the µL/min *cancel, leaving IOP in* mmHg



Fill in the IOP equation below. What is its eponymous name? The **Goldmann equation**

IOP = Aqueous Formation Rate (uL/min) + Episcleral Venous Outflow Facility (uL/min/mmHg) + Pressure (mmHg)

Episcleral venous pressure (EVP) normally measures about #w# mmHg (ie, the same as central venous pressure) in an upright pt.



Fill in the IOP equation below. What is its eponymous name? The **Goldmann equation**



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Episcleral venous pressure (EVP) normally measures about 8-12 mmHg (ie, the same as central venous pressure) in an upright pt. Looking at the Goldmann equation, you can see that, mathematically, it suggests EVP provides a baseline 'floor' value for IOP. That is, even if aqueous formation ceased (which would take the first term in the Goldmann equation down to zero), IOP should not fall below EVP; rather, it should be equal to zero plus whatever EVP was at the moment. Further, the Goldmann equation predicts that IOP should vary on a 1-to-1 basis with EVP—that is, each mmHg change in EVP should result in a mmHg change in IOP.



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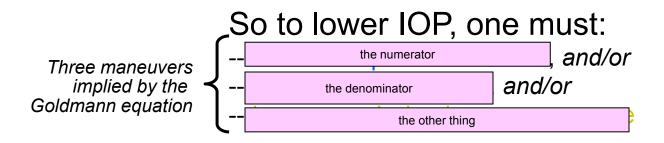
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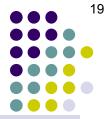


Fill in the IOP equation below. What is its eponymous name? The Goldmann equation









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Three maneuvers implied by the Goldmann equation So to lower IOP, one must: --decrease aqueous formation, and/or --increase outflow facility, and/or --decrease episcleral venous pressure



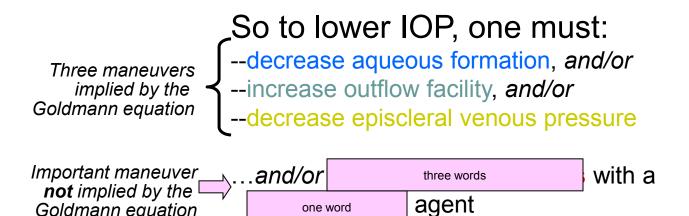
Goldmann equation

Glaucoma Overview



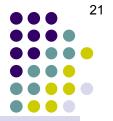
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one word





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Three maneuvers implied by the Goldmann equation

Important maneuver not implied by the Goldmann equation hyperosmotic agent





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Which classes of meds decrease aqueous formation?

So to lower IOP, one must:

--increase outflow facility, *and/or* --decrease episcleral venous pressure

...and/or dehydrate the vitreous with a hyperosmotic agent





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Which classes of meds decrease aqueous formation? --β blockers --CAls --α agonists

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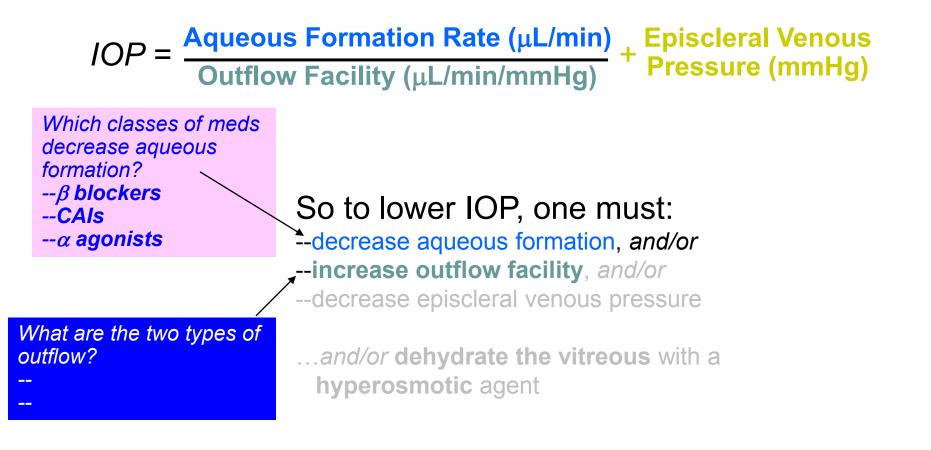
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...and/or **dehydrate the vitreous** with a **hyperosmotic** agent





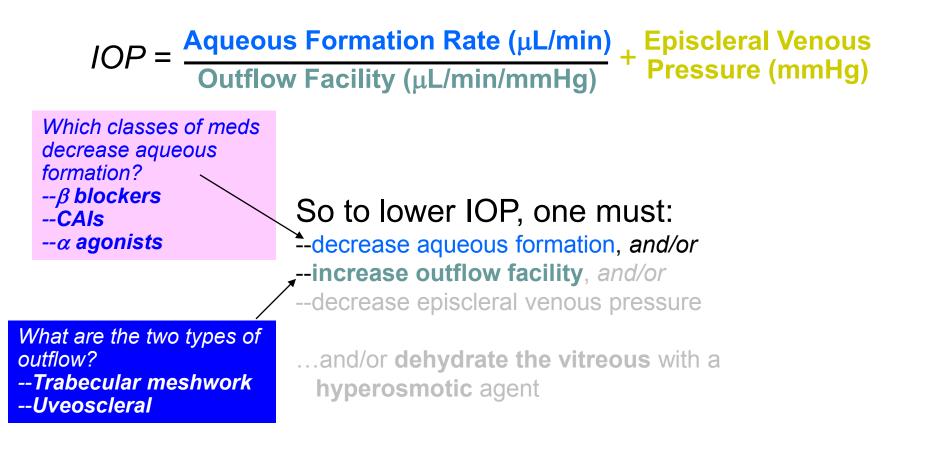
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Fill in the IOP equation below. What is its eponymous name? The **Goldmann equation**

Aqueous (μL/min) + Episcleral Venous Outflow Facility (μL/min/mmHg) + Pressure (mmHg) Which cla Obviously, aqueous-humor dynamics play a central

formation role in glaucoma. Let's delve into its production...

--β blockers --CAIs

--*α* agonists

What are the two types of outflow? --**Trabecular meshwork** --**Uveoscleral** So to lower IOP, one must: --decrease aqueous formation, and/or --increase outflow facility, and/or --decrease episcleral venous pressure

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What is the rate of aqueous formation?





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What is the rate of aqueous formation? 2-3 μL/min





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What is the rate of aqueous formation? 2-3 μL/min

What is the aqueous volume of the anterior chamber?





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What is the rate of aqueous formation? 2-3 μL/min

What is the aqueous volume of the anterior chamber? 200-300 μ L





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So then, what percent of AC volume is 'turned over' every minute?





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What is the rate of aqueous formation? 2-3 μL/min

What is the aqueous volume of the anterior chamber? 200-300 μL

So then, what percent of AC volume is 'turned over' every minute? About 1%





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Given this, how long does it take for the aqueous content of the AC to be fully replaced?





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What is the aqueous volume of the anterior chamber? 200-300 μ L

So then, what percent of AC volume is 'turned over' every minute? About 1%

Given this, how long does it take for the aqueous content of the AC to be fully replaced? Roughly 100 minutes





Fill in the IOP equation below. What is its eponymous name? The **Goldmann equation**



Speaking of aqueous formation...What specific tissue makes aqueous?

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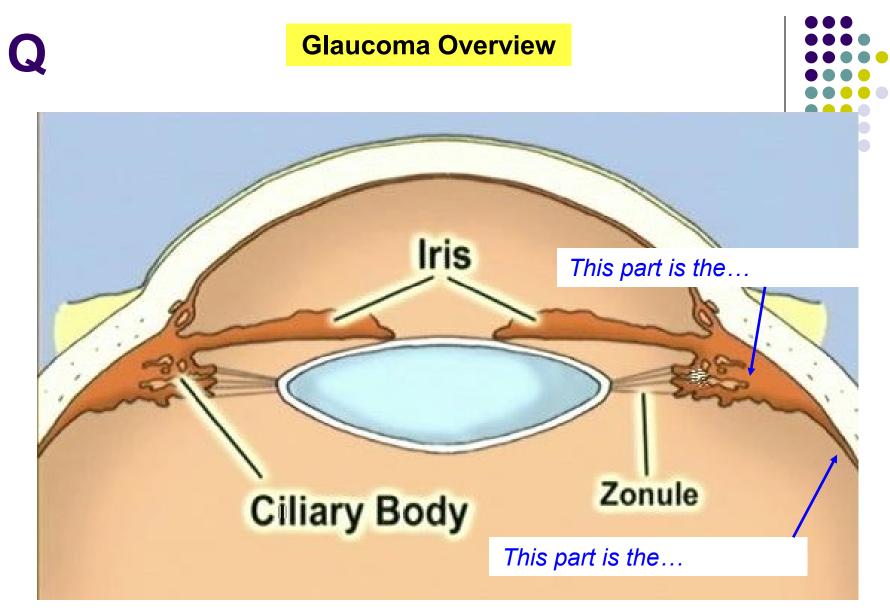
What is implied by the fact that aqueous is made by the 'nonpigmented' epithelium? The presence of a **pigmented** epithelium

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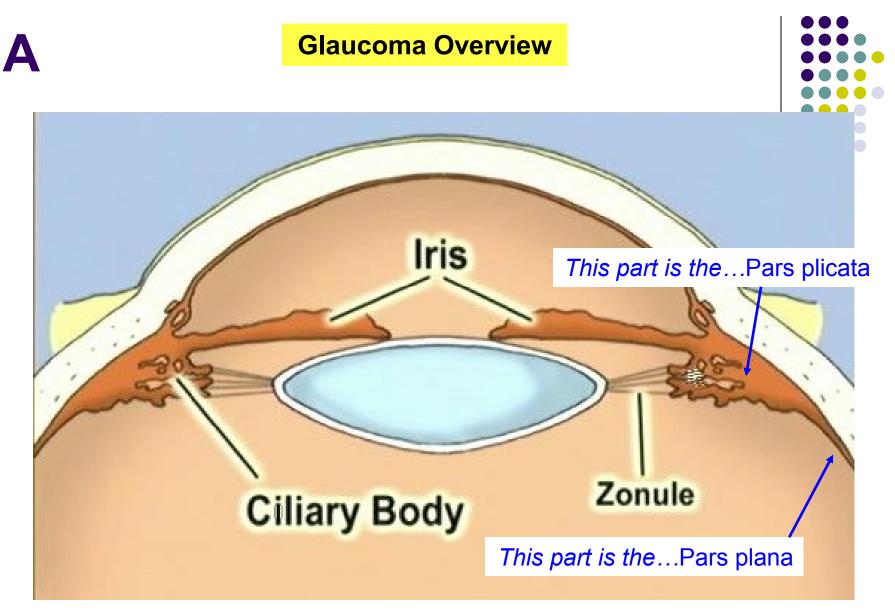
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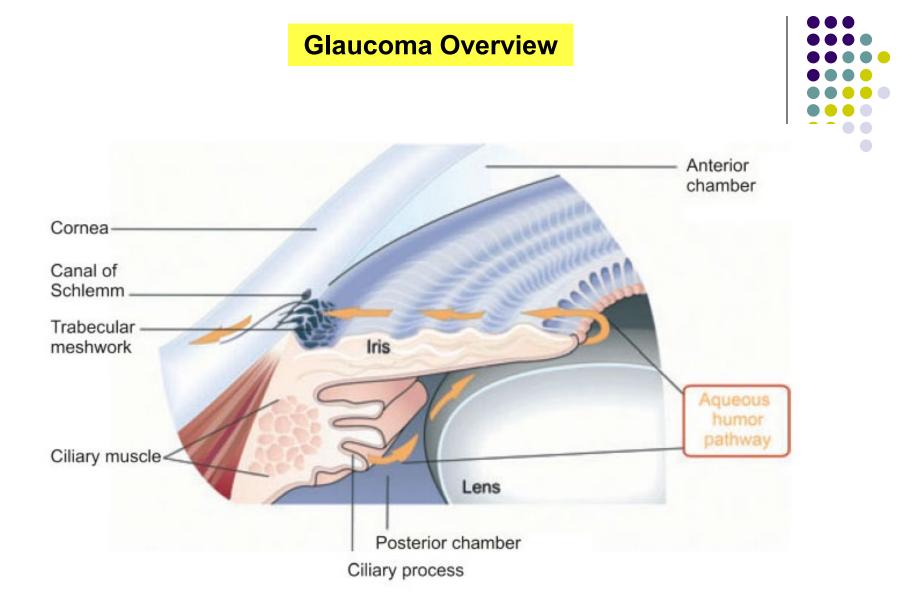
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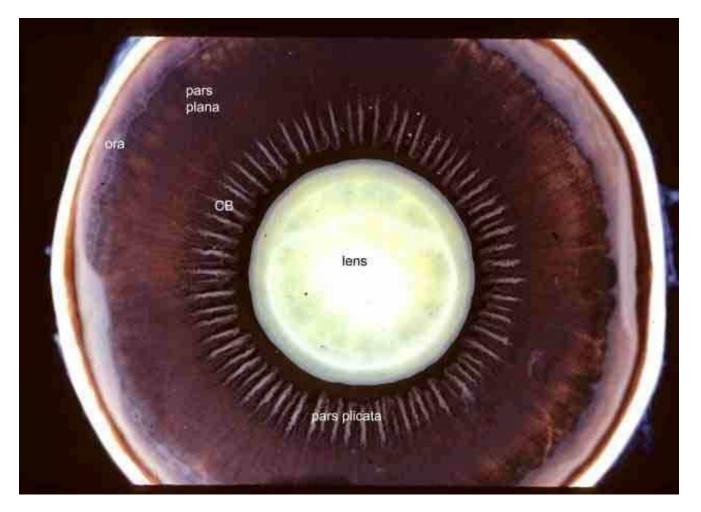
Ciliary body: One perspective, two questions



Ciliary body: One perspective, two questions



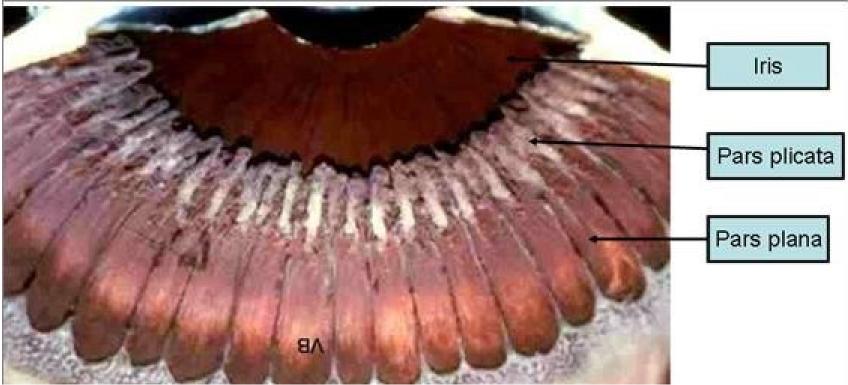
Ciliary body: Another perspective



Ciliary body: Another

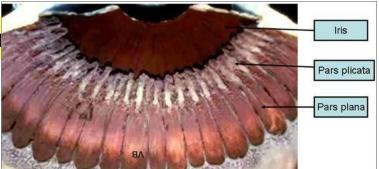


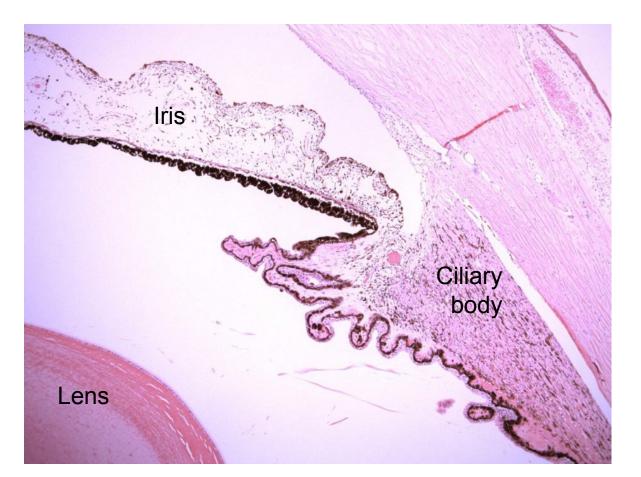




Ciliary body: Another

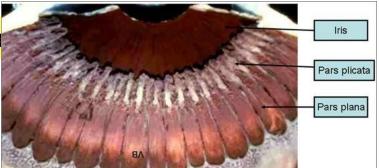






Now let's look at the CB epithelium. **Low power** photomicrograph.







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

Speaking of aqueous formation...What specific tissue makes aqueous? The nonpigmented epithelium of the pars plicata portion of the ciliary body

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ric Pars plicata Pars plana

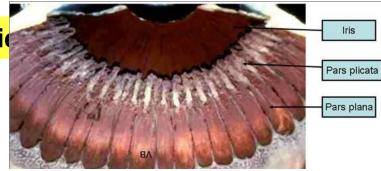
Now let's look at the CB epithelium. **High**.

No question—proceed when ready

Glaucoma Overvi

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From what embryonic tissue do the two epithelia of the CB derive?



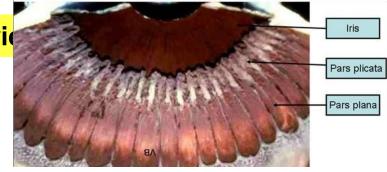


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From what embryonic tissue do the two epithelia of the CB derive? **Neuroectoderm**



Glaucoma Overvi

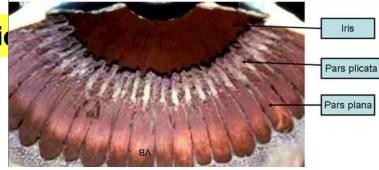
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What other portion of the eye derives from neuroectoderm? The retina (ie, the neurosensory retina + RPE)

How are the neurosensory retinal and RPE cells oriented with respect to one another?

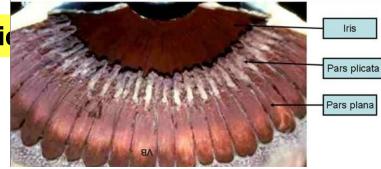
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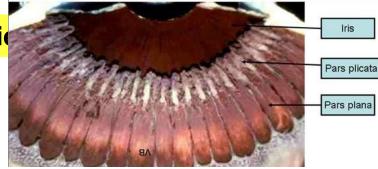
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Glaucoma Overvi

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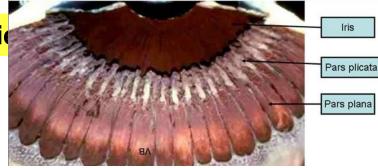
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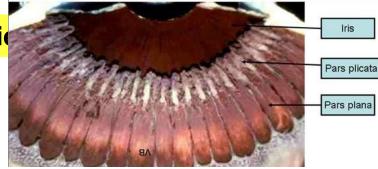
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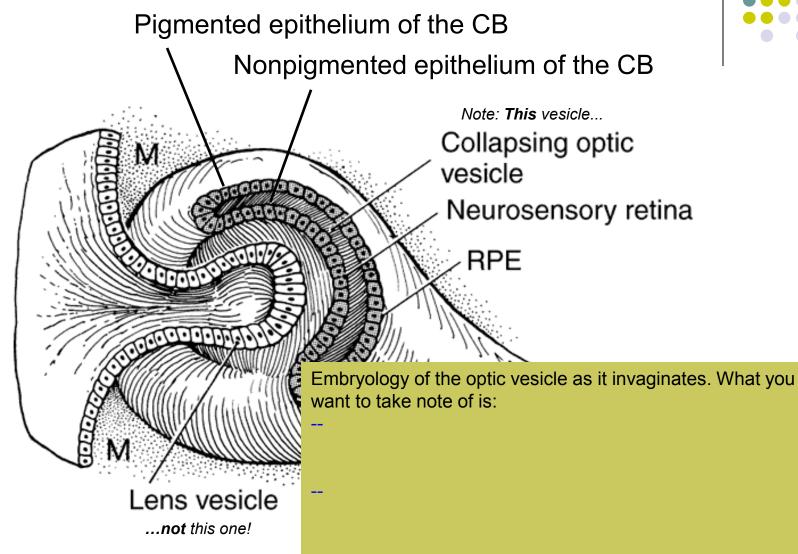
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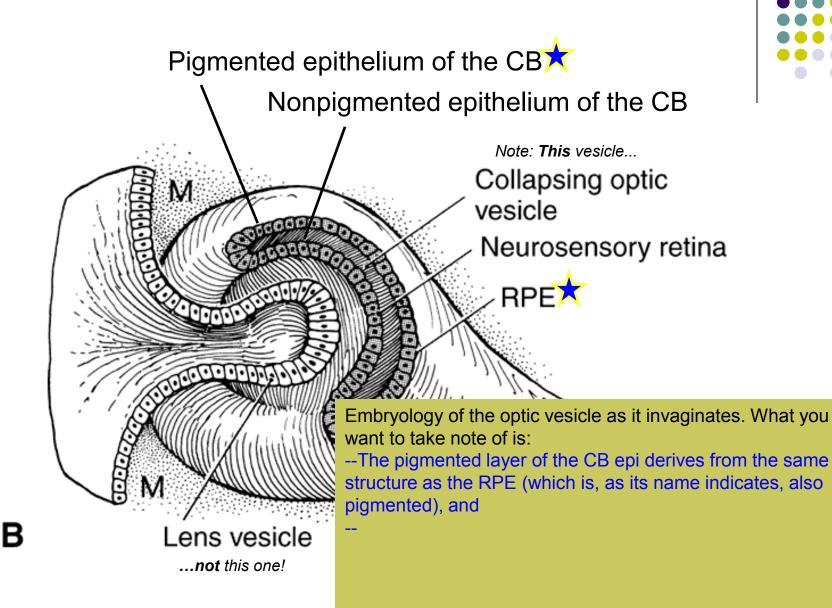
В

Glaucoma Overview

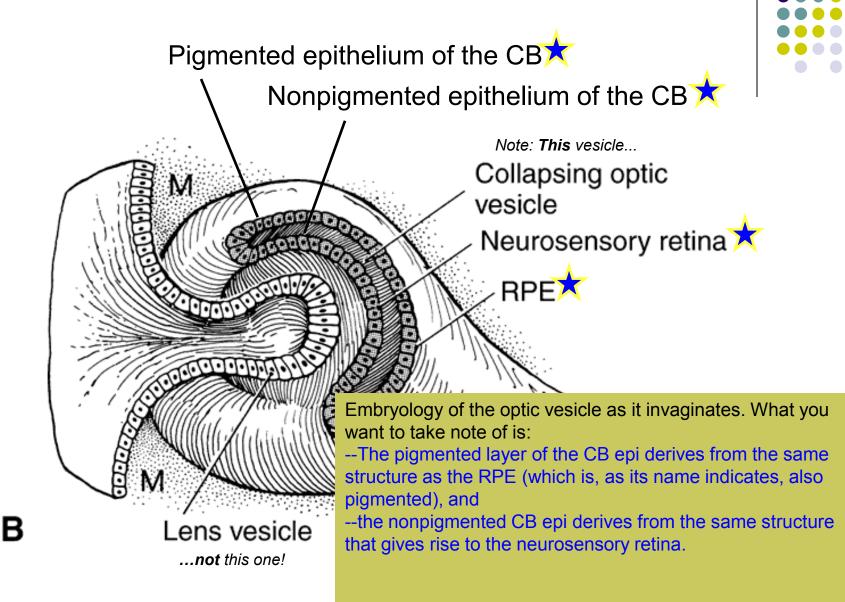








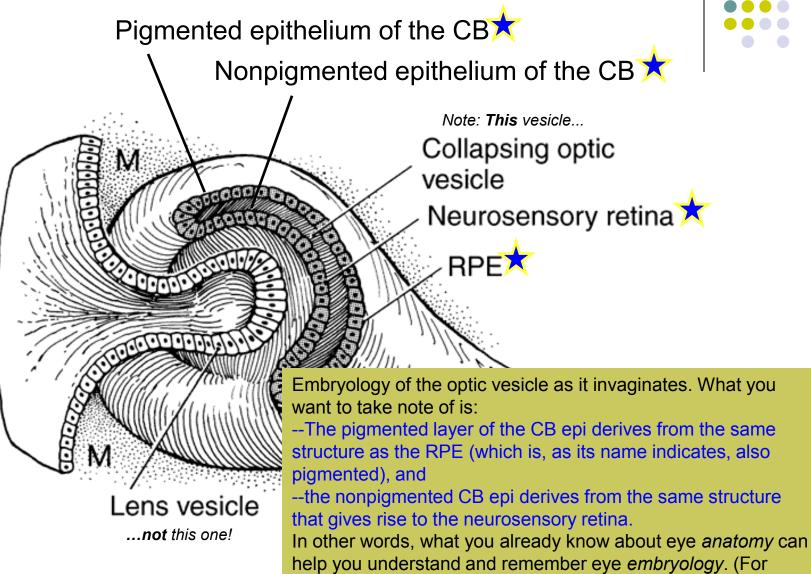






В

Glaucoma Overview



more, see the Embryology made simply ridiculous slide-set.)

Glaucoma Overvi

Which intraocular structure makes aqueous? The nonpigmented epithelium of the pars plicata portion of the ciliary body

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Hol up Dr Flynn. Looking at this photomicrograph, the pigmented epi layer appears to be the inner one. Did you make a mistake?

ells

(PE)

The same way--apex-to-apex

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The same way--apex-to-apex

Which CB epithelial layer is pigmented--the inner, or the outer? That's what I said--the outer!

Which portion of the retina is contiguous with the pigmented layer of the CB epithelium? **The RPE, ie the outer**





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The same way--apex-to-apex

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Which portion of the retina is contiguous with the pigmented layer of the CB epithelium? **The RPE, ie the outer**





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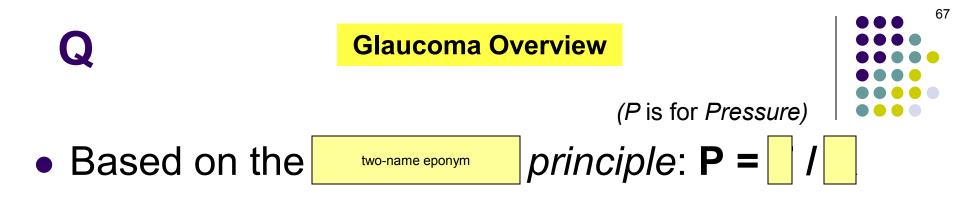
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Now let's look at IOP measurement via Goldmann applanation tonometry

So to lower IOP, one must: --decrease aqueous formation, and/or --increase outflow facility, and/or --decrease episcleral venous pressure

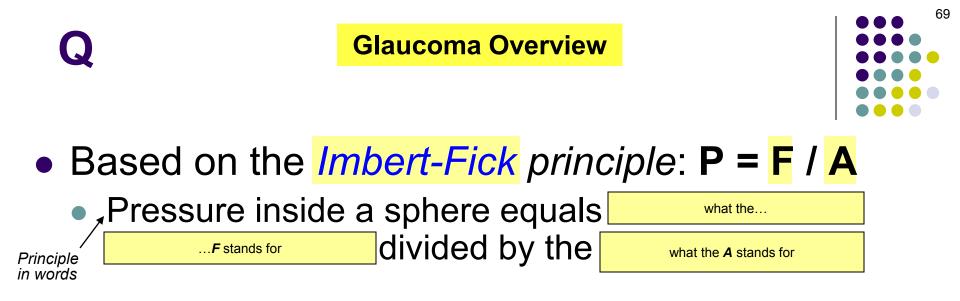
...and/or **dehydrate the vitreous** with a **hyperosmotic** agent







Based on the *Imbert-Fick* principle: P = F / A







Based on the *Imbert-Fick* principle: P = F / A

• Pressure inside a sphere equals force needed to I-F Principle flatten its surface divided by the area of flattening





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two words

, and (cornea is





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increase vs decrease

IOP reading





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For the Imbert-Fick principle to hold, the **only** force resisting applanation should be the pressure within the sphere. However, real objects such as the cornea have *intrinsic* resistance to deformation owing to their physical nature, ie, because they're made of 'stuff.'



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For the Imbert-Fick principle to hold, the **only** force resisting applanation should be the pressure within the sphere. However, real objects such as the cornea have *intrinsic* resistance to deformation owing to their physical nature, ie, because they're made of 'stuff.' This inherent structural resistance of the cornea will be additive to whatever pressure is inside the eye, thereby causing the pressure reading to be falsely **high**. (And the thicker the cornea is, the higher the reading will be.)





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 - Tear film→capillary attraction→

increase vs decrease **IOP** reading





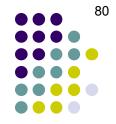
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On the other hand: The first ocular structure encountered by the applanator tip is the tear film. When contact with the tear film is made, a fluid bridge forms between the cornea and the tip. Surface tension of the water in this fluid bridge produces *capillary attraction*, which exerts a slight 'pull' on the applanator tip, drawing it toward the cornea.



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To be useful, an applanator-type device has to account for these factors. Fortunately, the brilliant Dr. Goldmann was (mostly) up to the challenge...





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 IOP reading
- Dr Goldmann realized if the diameter of the circle applanated by the device is _____ mm, capillary attraction and corneal thickness would cancel each other out (assuming CCT is ____ µm)

(CCT = Central corneal thickness)





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(We now know that CCT averages about 550, with wide variation among individuals)



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 - Goldmann believed CCT was ~520, with little variation
 - When the mires line up, the diameter of the applanated area is 3.06 mm No question—proceed when ready



Define glaucoma. A group of optic neuropathies that present with progressive **ONH** amage 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

We mentioned previously that glaucoma presents with "progressive ONH damage." Let's drill down on the structure of the ONH.



The optic nerves are composed of what?





The optic nerves are composed of what? The axons of retinal ganglion cells





The optic nerves are composed of what? The **axons of retinal ganglion cells**

How many fibers (axons) comprise an optic nerve?





The optic nerves are composed of what? The **axons of retinal ganglion cells**

How many fibers (axons) comprise an optic nerve? Depends upon which book you ask, but the answer **1.2M** works

Per the... *Glaucoma* book: 1.2-1.5M *Neuro* book: 1-1.2M *Fundamentals* book: "more than a million"





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Do they synapse in the region of the optic nerve head?





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Most? Where will the others synapse, and what are they responsible for?





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Anatomically speaking, the optic nerve is considered to have four portions. What are they?





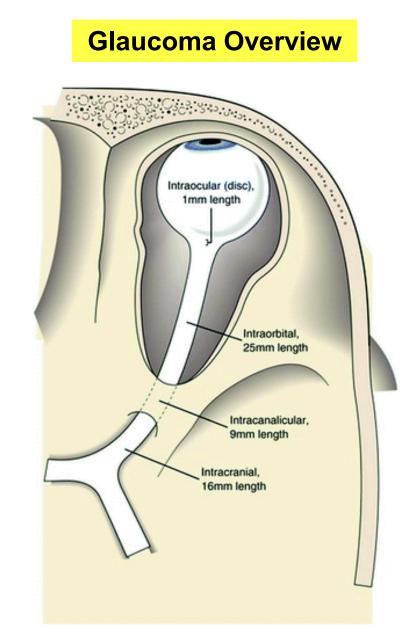
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Optic nerve portions (don't memorize the lengths)

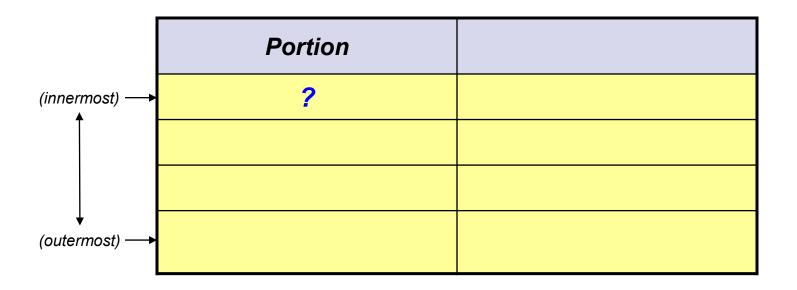


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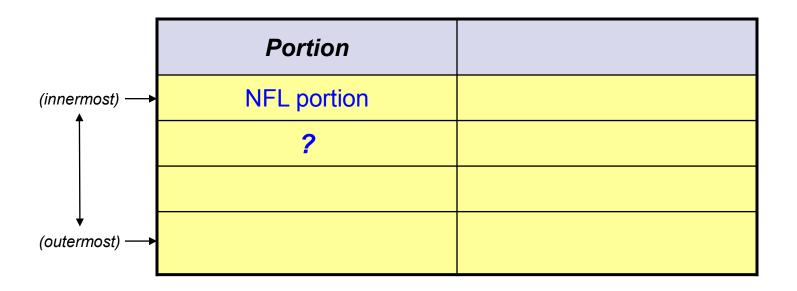


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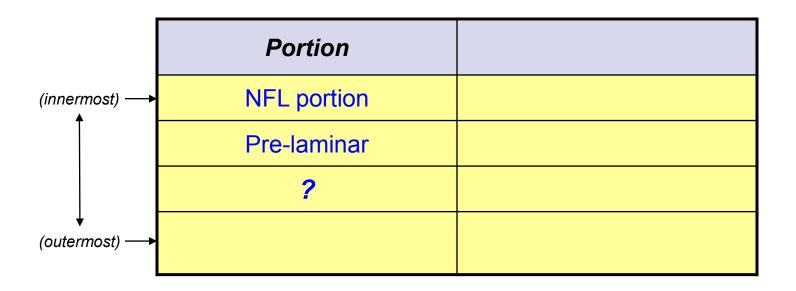


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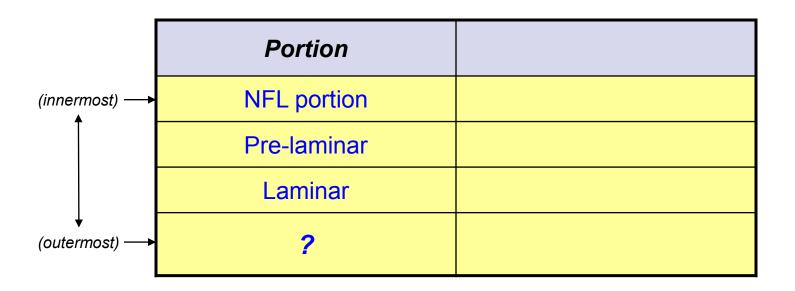


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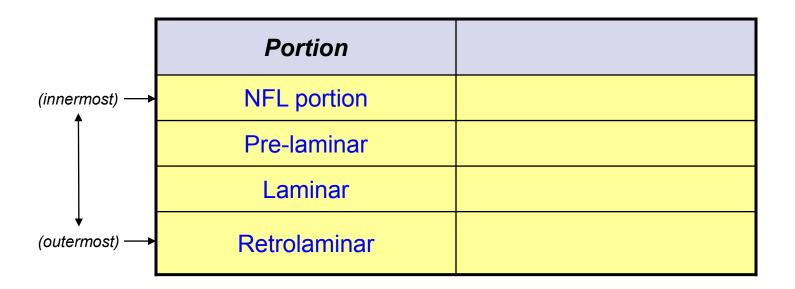
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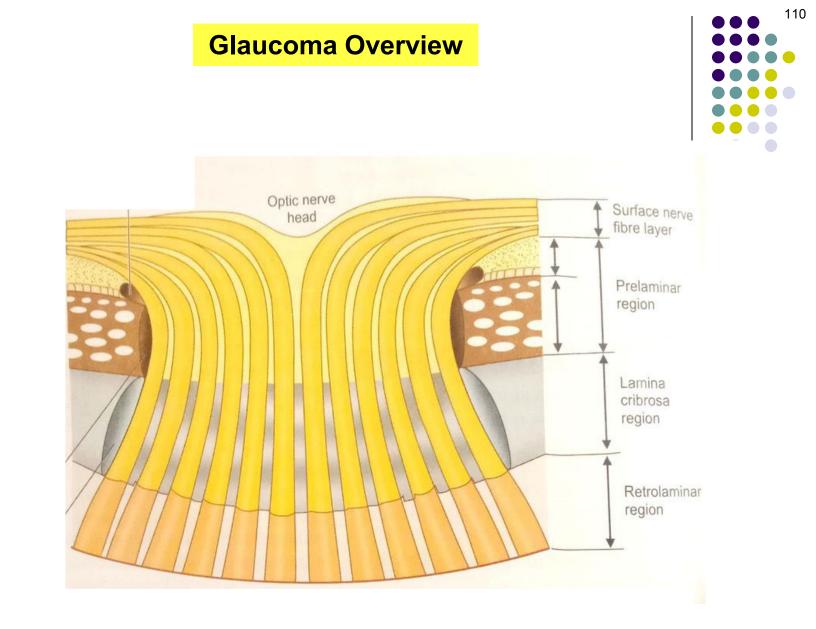
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Optic nerve head portions



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	Portion	Blood supply
(innermost) →	NFL portion	?
Ī	Pre-laminar	
	Laminar	
↓ (outermost)>	Retrolaminar	



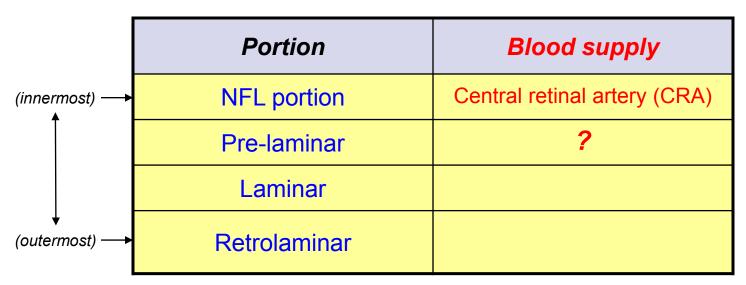


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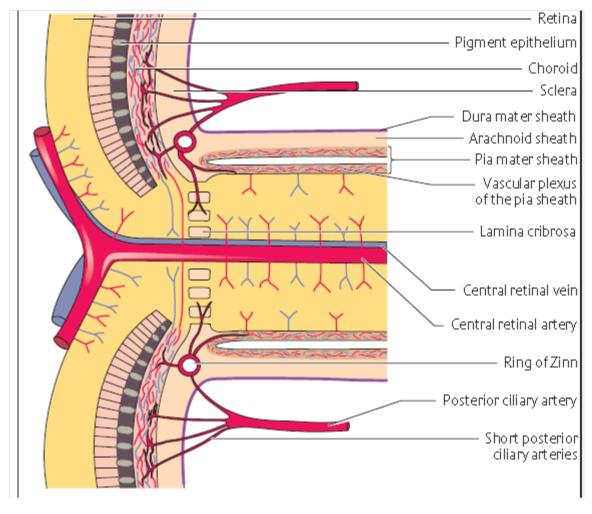
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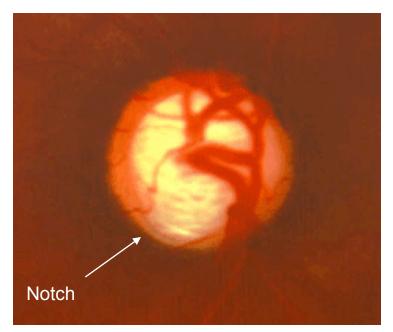




ONH: Blood supply



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. This leads to thinning at the poles (focal thinning is often referred to as a 'notch.')



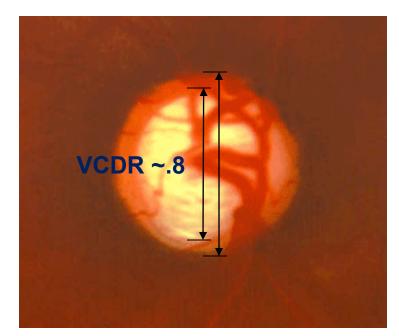


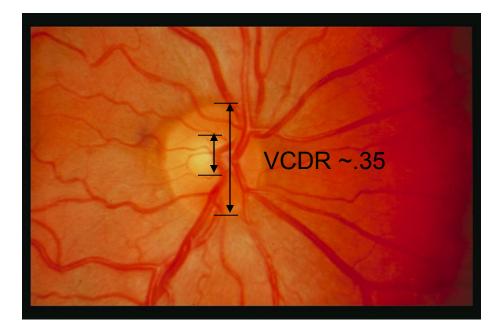
Glaucomatous ONH



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

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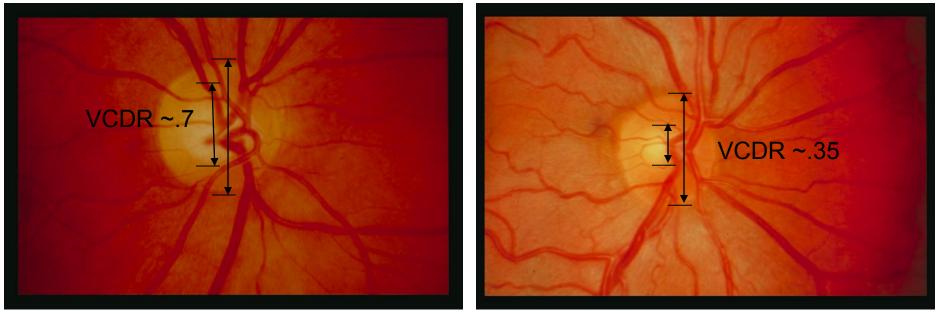




Glaucomatous ONH



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).

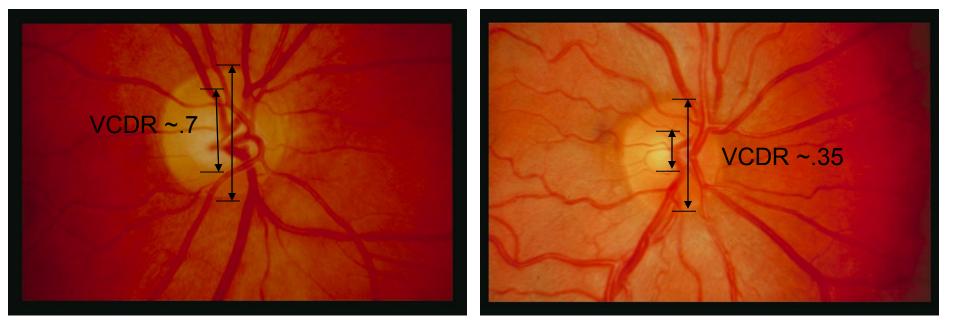


Normal ONH

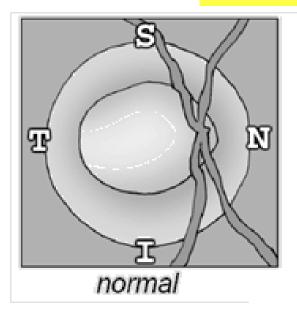


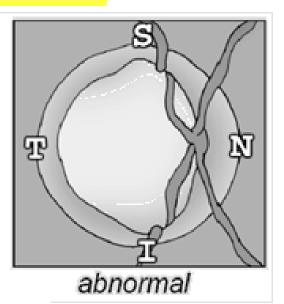
Note that the VCDR can be misleading in this regard, as it can be quite pronounced in some normal eyes (especially those with a large disc).

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.



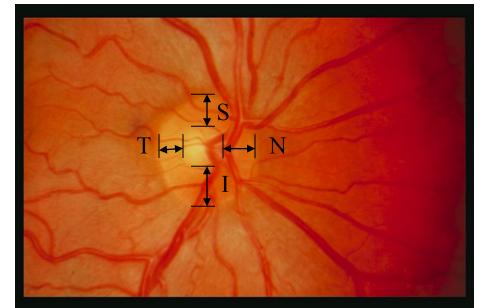
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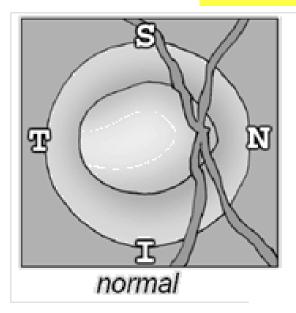


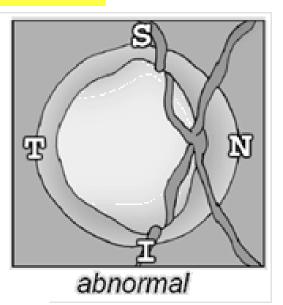




The nonglaucomatous neuroretinal rim tends to follow what's known as the *ISNT rule*: In decreasing order, the rim is thickest at its *Inferior*, Superior, *Nasal*, and *Temporal* portions. If an ONH's rim adheres to this rule, it *ISNT* glaucomatous.







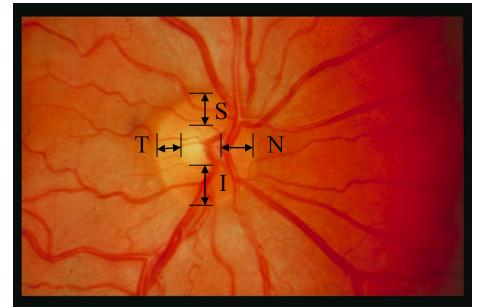


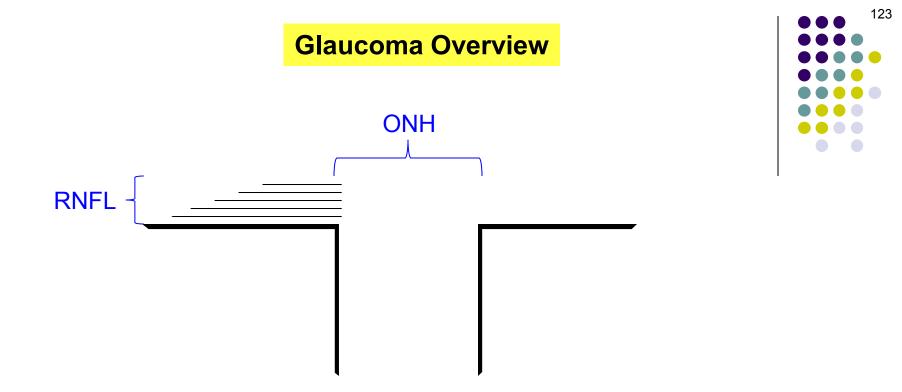
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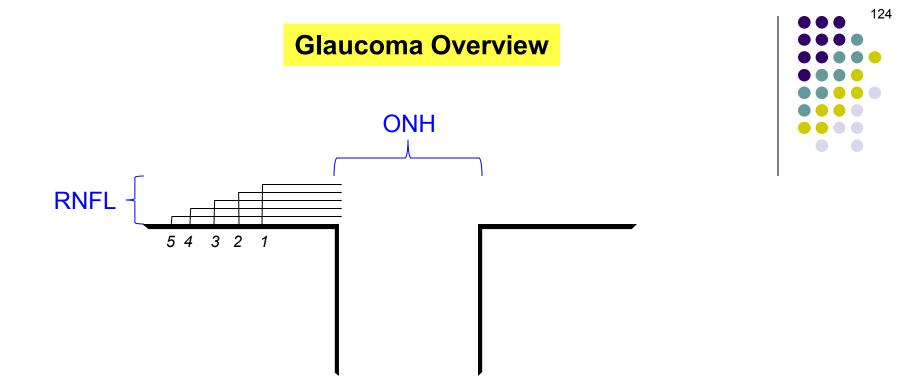
Note: Not all glaucoma docs find the ISNT rule to be helpful—YMMV. Ask!

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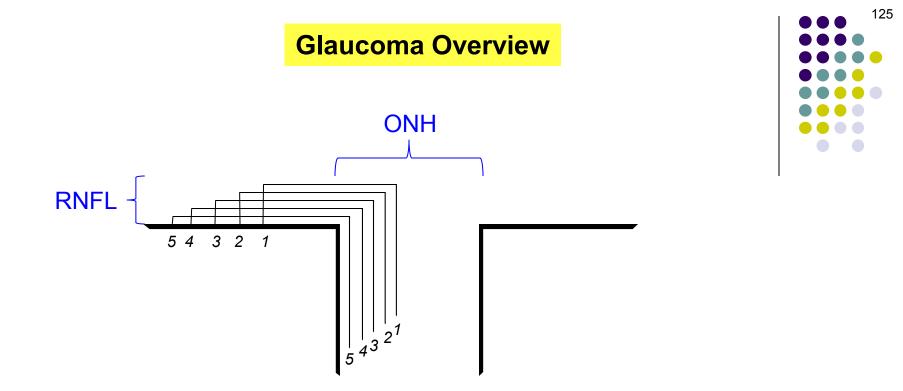


Now consider the ONH and retina in cross section. Note that the RNFL and ONH are both organized in a specific fashion:



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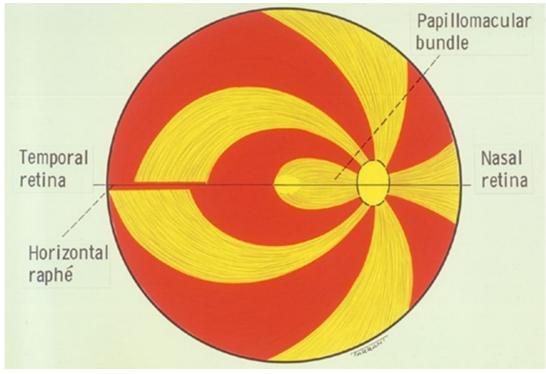
--The RNFL is stacked *vertically*, with fibers that originate at points distant from the ONH running at the bottom (ie, closer to the RPE); and

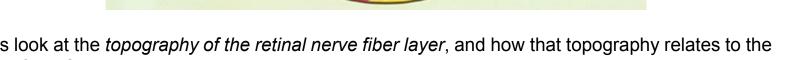


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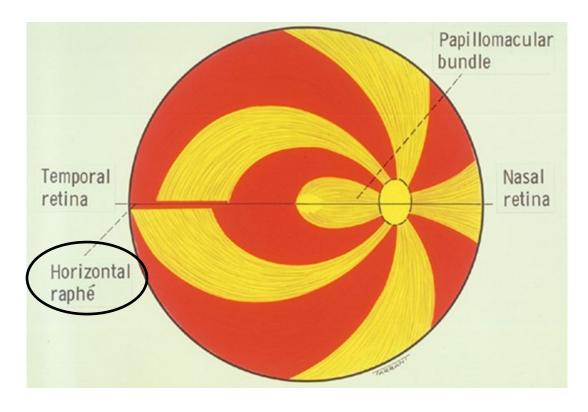
--The RNFL is stacked *vertically*, with fibers that originate at points distant from the ONH running at the bottom (ie, closer to the RPE); and

--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.





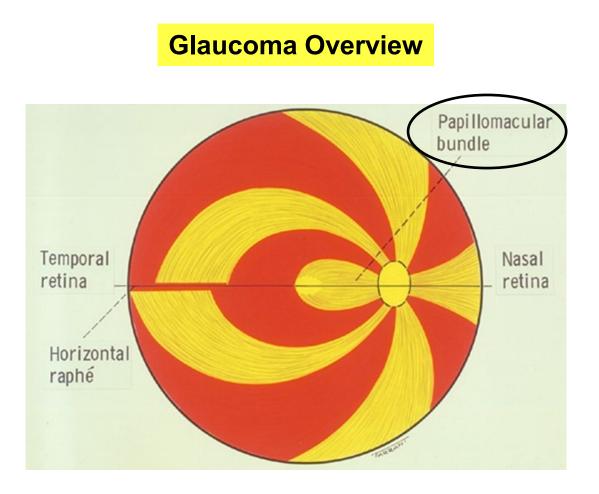
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First, take note of the *horizontal raphé*. Fibers do not cross this anatomic boundary—those superior to it join the superior ONH, and those inferior to it, the inferior ONH.

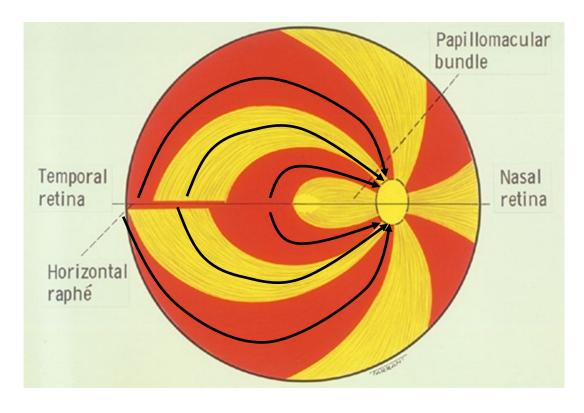




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Next, the *papillomacular (PM) bundle*—the swath of nerve fibers originating in the foveal region. Note how this bundle takes up the lion's share of the temporal ONH.





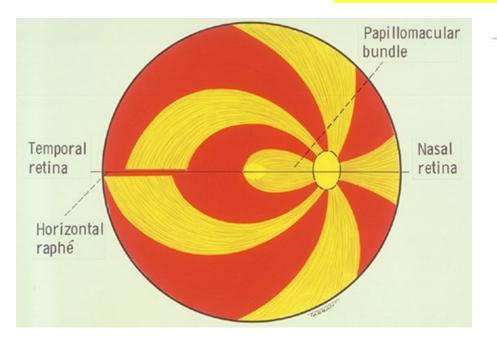
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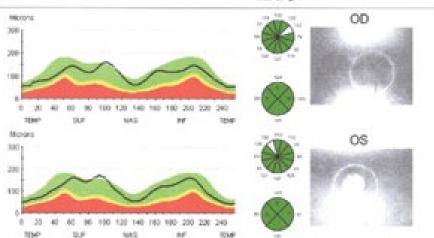
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Finally, note how the PM bundle impacts the structure of the ONH. Because the bundle takes up the temporal ONH, fibers from the temporal perifoveal region and beyond are forced to 'loop around' it, and end up joining the ONH near its superior and inferior poles.

No question—proceed when ready

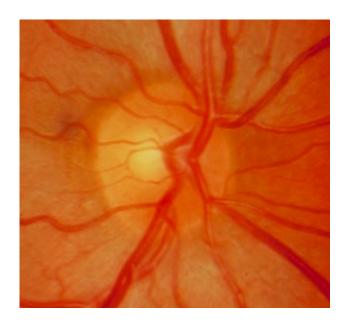


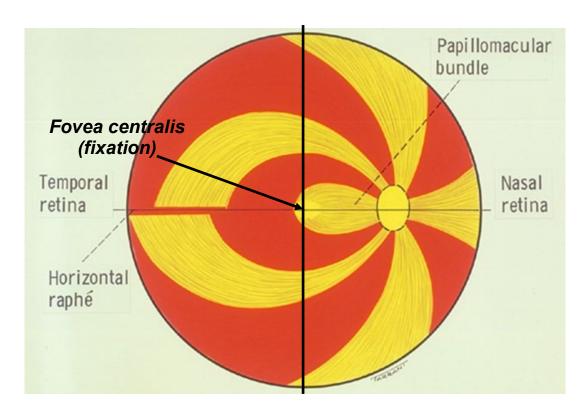


ScanLoAgth: 1

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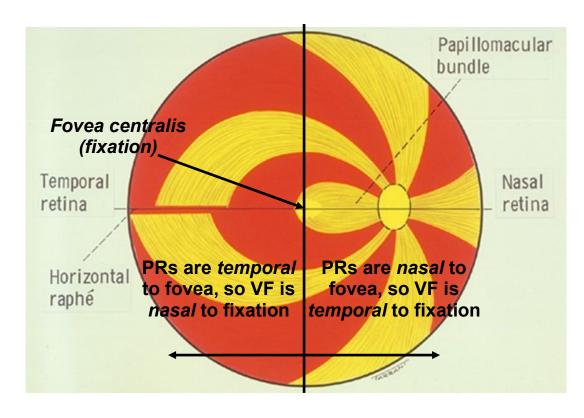
Because there are so many fibers at the superior and inferior poles, the normal ONH rim tends to be thicker at these sites. (This accounts for the relative proportions of the rim segments as captured by the *ISNT rule* described previously.)







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.



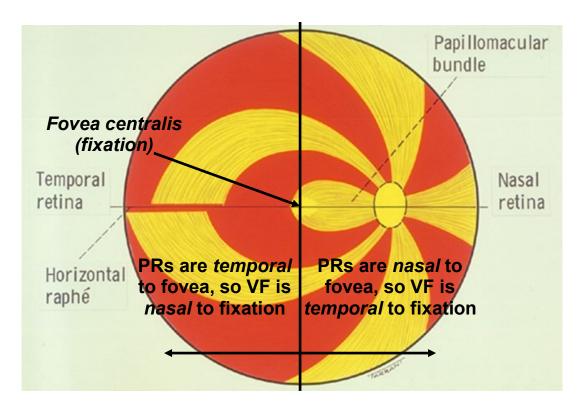


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Instead, it is identified via *visual field testing*. Fixation divides the VF into nasal and temporal fields, with the photoreceptors (PRs) responsible for the temporal VF being nasal to the vertical meridian, and those responsible for the nasal VF located temporal to it.

Q

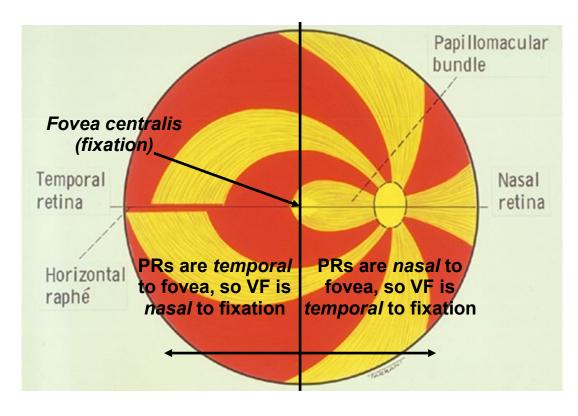
Glaucoma Overview





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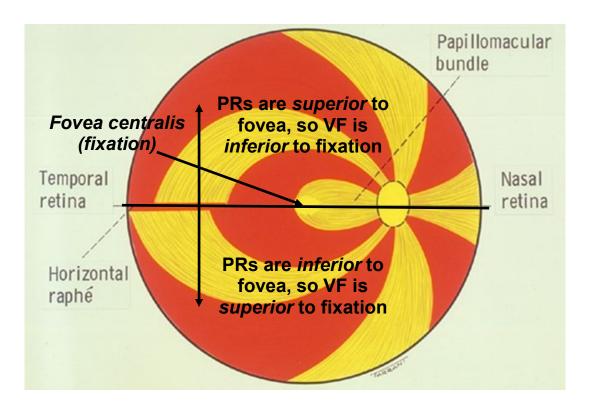
Instead, it is identified via viewel field testing. Fixation divides the V/F into peeel and temperal fields, with the If not within the retina, where is the anatomic location for the vertical meridian found in the visual fields?





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Instead, it is identified via viaual field testing. Eivetion divides the VE into need and temperal fields, with the *If not within the retina, where is the anatomic location for the vertical meridian found in the visual fields?* The optic chiasm. Recall that it is there that the visual field is divided vertically.



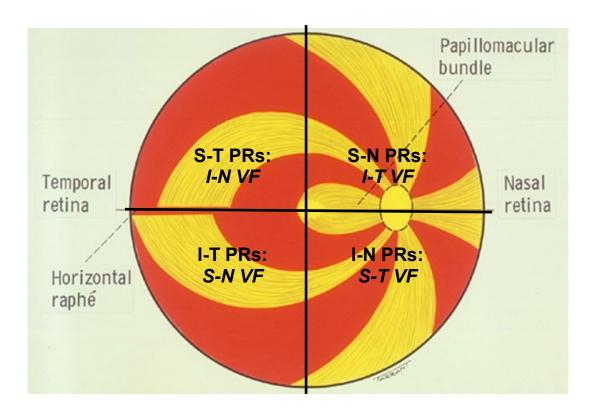


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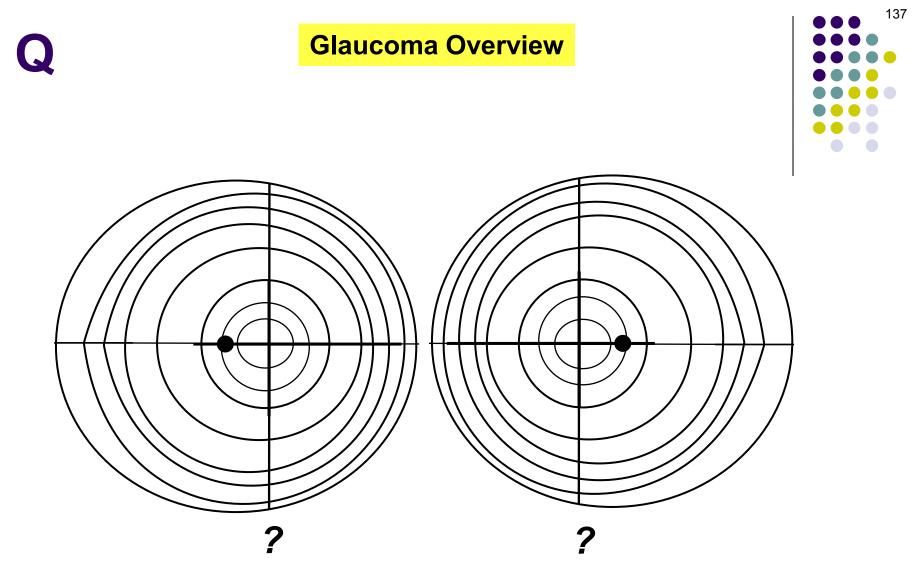
Finally, note that fixation also divides the VF into superior and inferior VFs. The corresponding portions of the retina related topographically to the horizontal raphé.

No question—proceed when ready

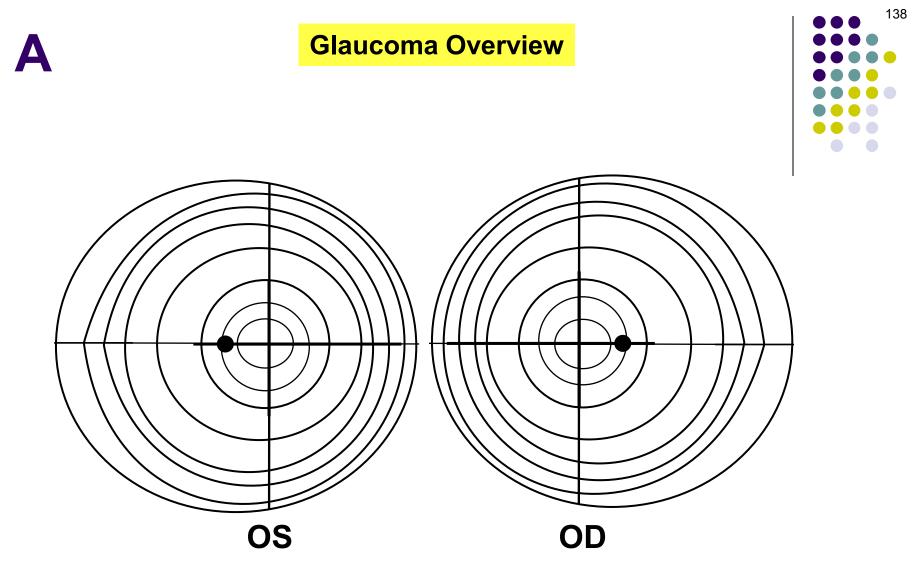




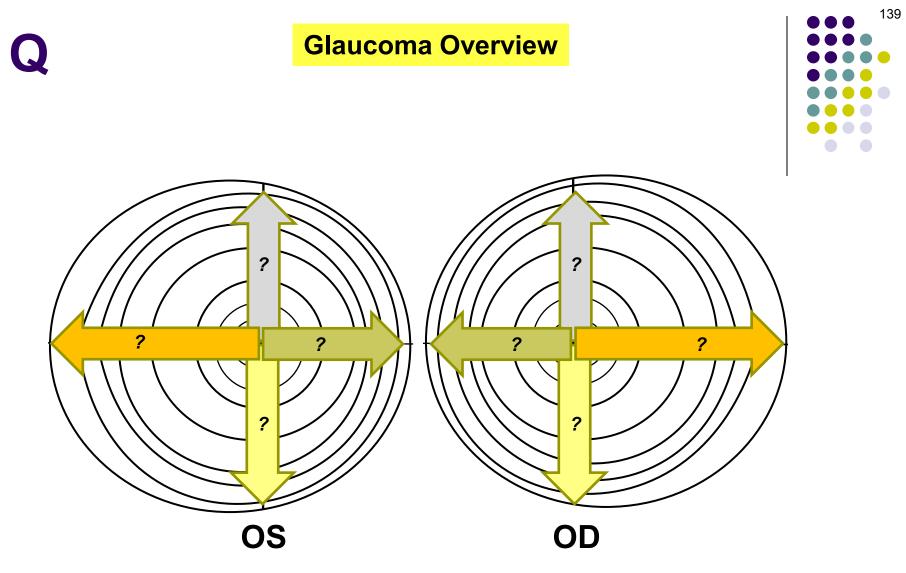
Putting it all together: The VF can be divided into four quadrants. Together, retinal topography and ONH structure dictate that each quadrants corresponds with a particular anatomic location on the ONH. This relationship is important to understand as it allows the clinician to determine whether VF changes correlate with structural changes in the ONH as detected via DFE and/or imaging technology.



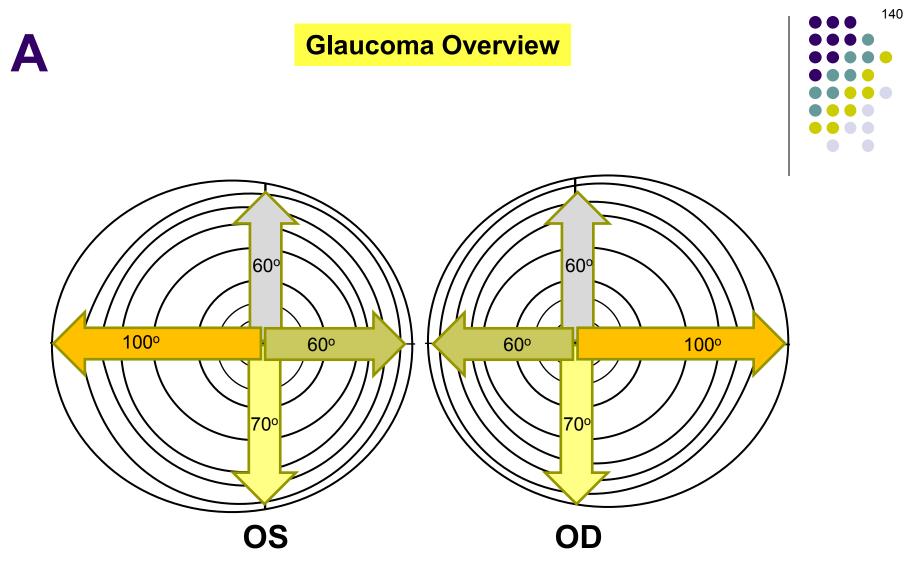
Here is a representation of the VF for each eye. Which is OD, and which OS?



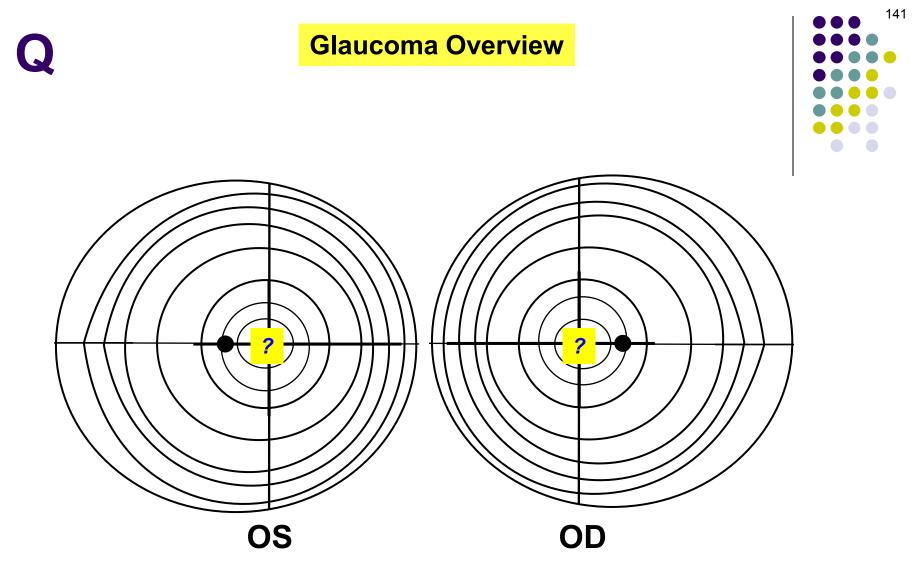
Here is a representation of the VF for each eye. Which is OD, and which OS? Remember, VFs are **not** drawn as if the pt is looking at you; they're drawn as if **you** are the pt!



Measured in degrees from fixation, how far does the normal VF extend superiorly, inferiorly, nasally and temporally? (Don't get too fixated on these specific numbers--different sources will give slightly different values.)



Measured in degrees from fixation, how far does the normal VF extend superiorly, inferiorly, nasally and temporally? (Don't get too fixated on these specific numbers--different sources will give slightly different values.)



Measured in degrees from fixation, how much of the VF is assessed via the automated perimetry machines found in most ophthalmology practices?







OS

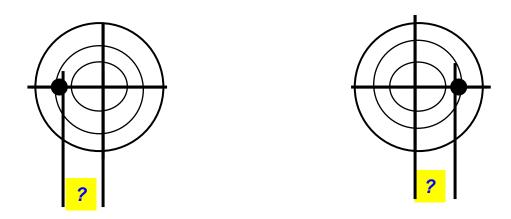
OD

Measured in degrees from fixation, how much of the VF is assessed via the automated perimetry machines found in most ophthalmology practices? The central 24 degrees

Q

Glaucoma Overview





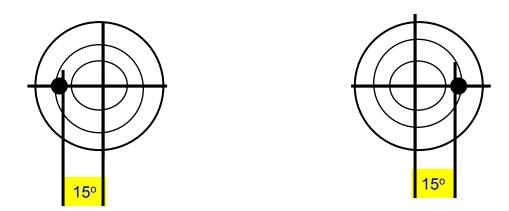
OS

OD

How far in degrees from fixation is the blind spot?







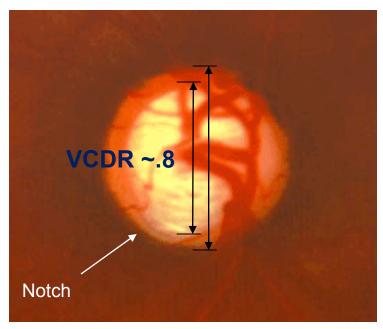
OS

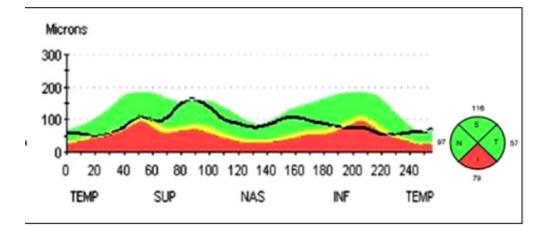
OD

How far in degrees from fixation is the blind spot? About 15 (again, don't get too hung up on that specific number.)



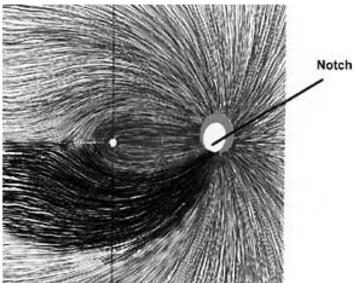
For reasons that have yet to be fully elucidated, glaucoma initially 'prefers' to damage the superior and inferior poles of the ONH. This leads to thinning at the poles (focal thinning is referred to as a 'notch.')



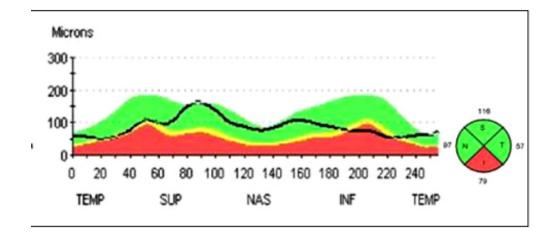


Glaucomatous ONH





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.

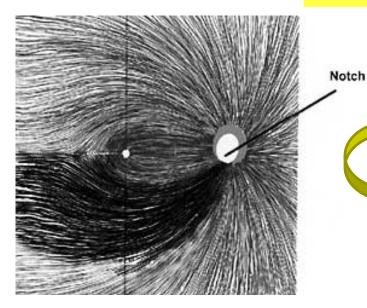


Glaucomatous ONH

VCDR~.8

Notch

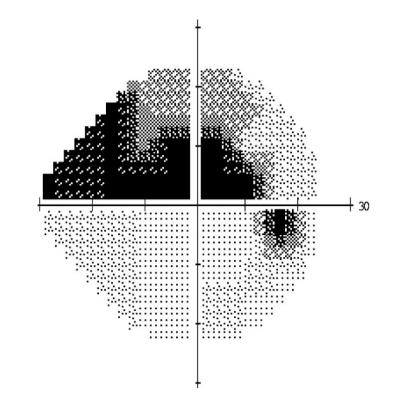




vccr.a.

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.') <u>Specifically, glaucoma tends initially to affect fibers that originate on the temporal side of the vertical meridian.</u>

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



Glaucomatous ONH



Define glaucoma. A greup of optic neuropatives 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

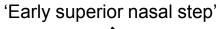
In addition to being the strongest risk factor for glaucoma, **IOP** has another quality that renders it unique—what is it? It is the only risk factor that is **modifiable** in a manner proven to influence the risk of glaucoma progression

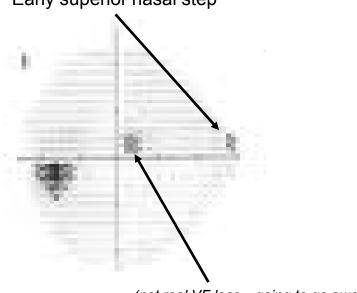
It was noted initially that glaucoma presents with "characteristic VF loss." That's what we're getting at here. Let's take a detailed look at the way glaucomatous VF defects appear and progress.



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.

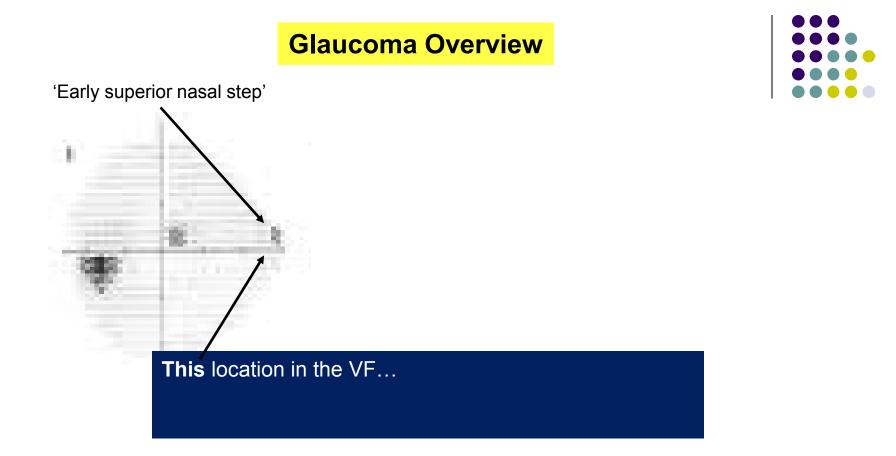






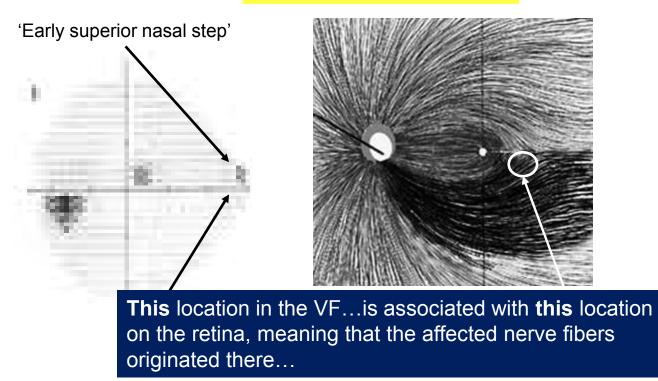
(not real VF loss—going to go away)

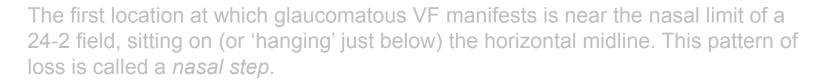
The first location at which glaucomatous VF manifests is near the nasal limit of a 24-2 field, sitting on (or 'hanging' just below) the horizontal midline. This pattern of loss is called a *nasal step*.



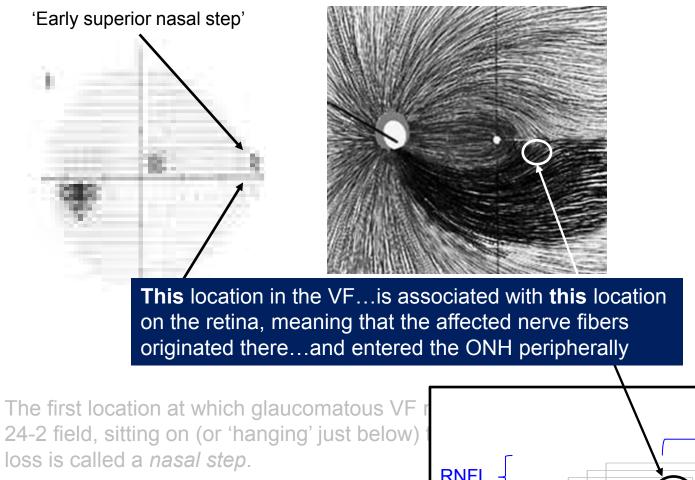
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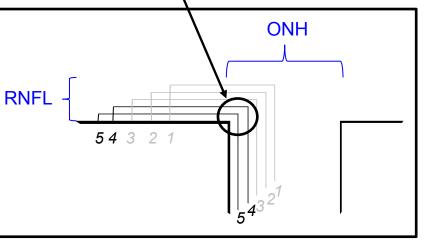








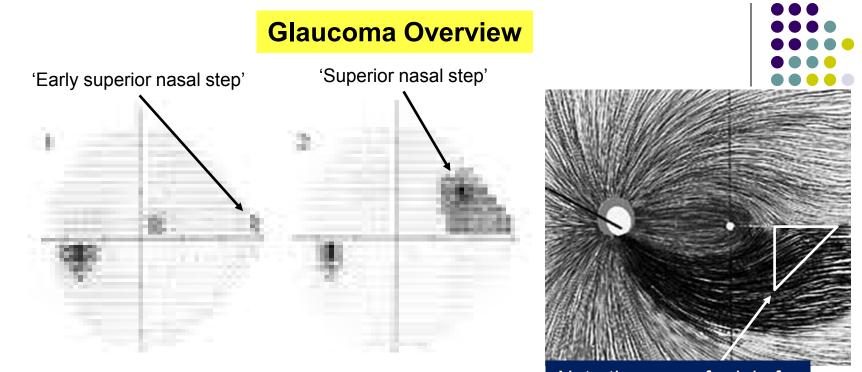
No question—proceed when ready



Glaucoma Overview 'Superior nasal step' 'Early superior nasal step' (told ya)



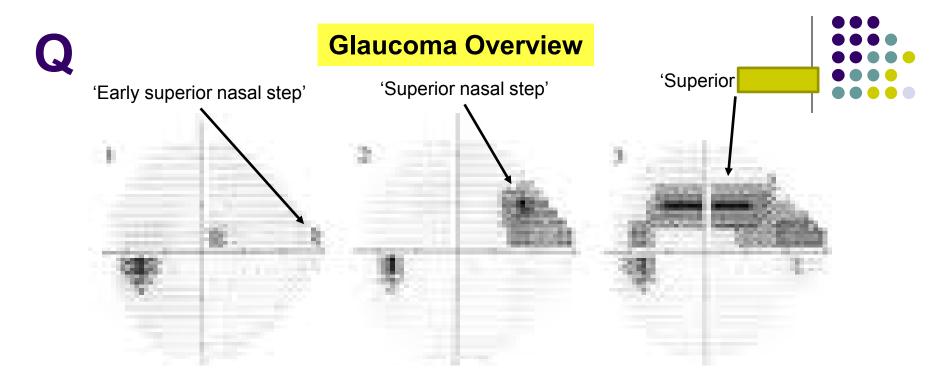
If left untreated, the nasal step will gradually enlarge.



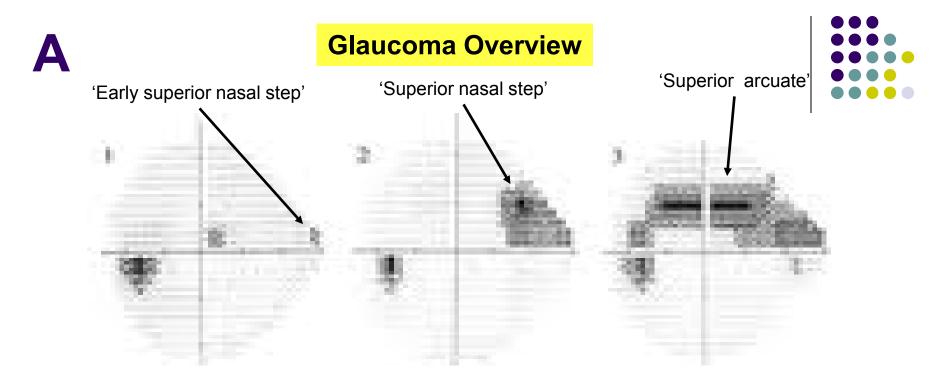
Note the area of origin for affected fibers has grown

If left untreated, the nasal step will gradually enlarge.

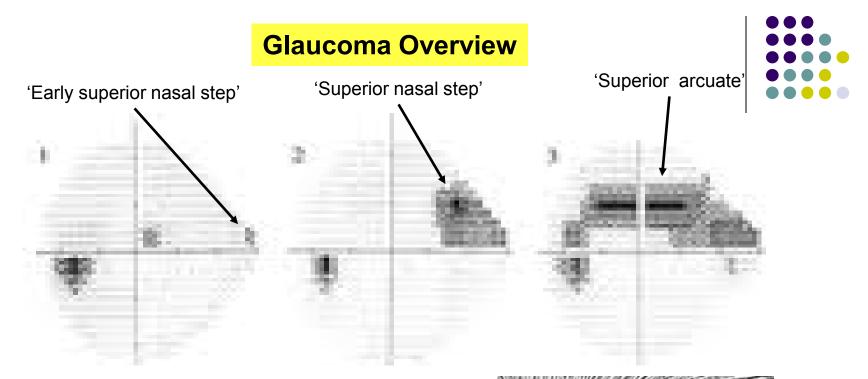
No question—proceed when ready



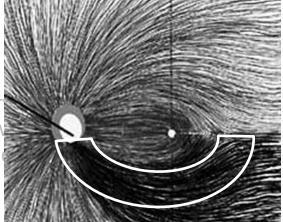
As glaucoma damage progresses, further loss of nerve fibers joining at that portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an



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*.

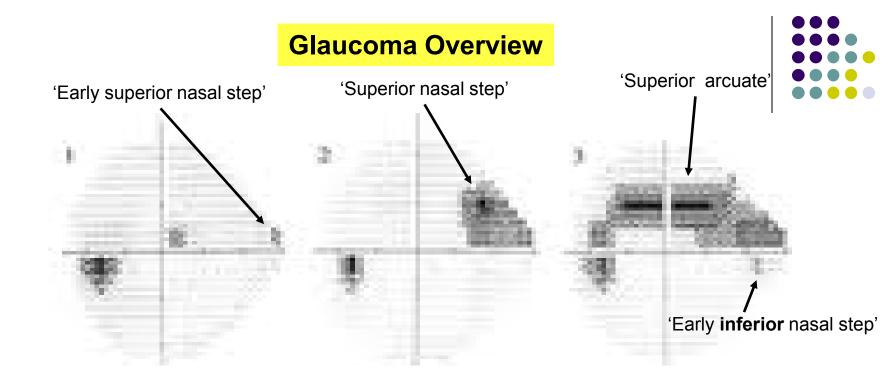


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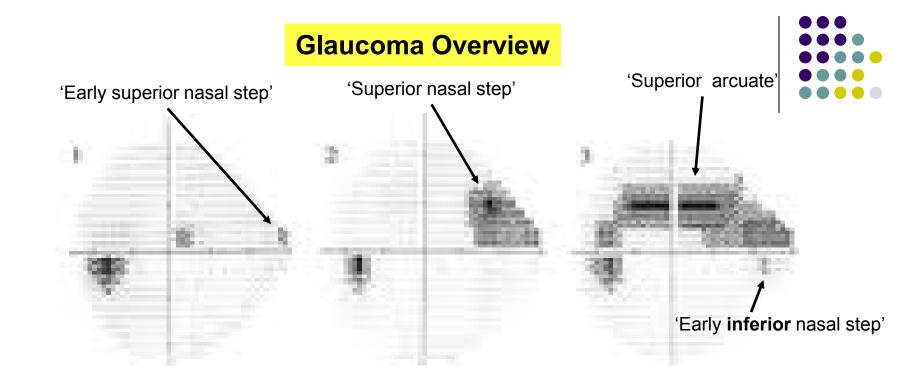
Note the area of origin for affected fibers now extends all the way to the ONH itself

No question—proceed when ready

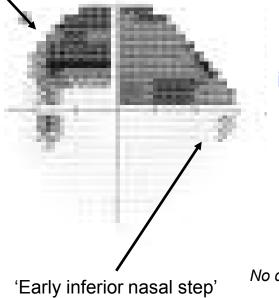


As glaucoma damage progresses, further loss of nerve fibers joining at that portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an *arcuate*.

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

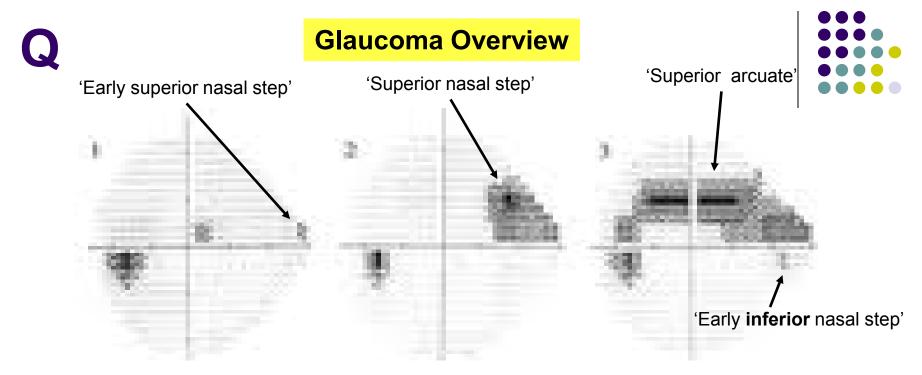


'Advanced arcuate'

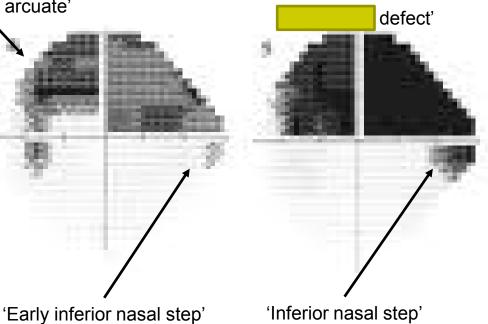


If left unchecked, an arcuate will expand into the surrounding portion of the VF.

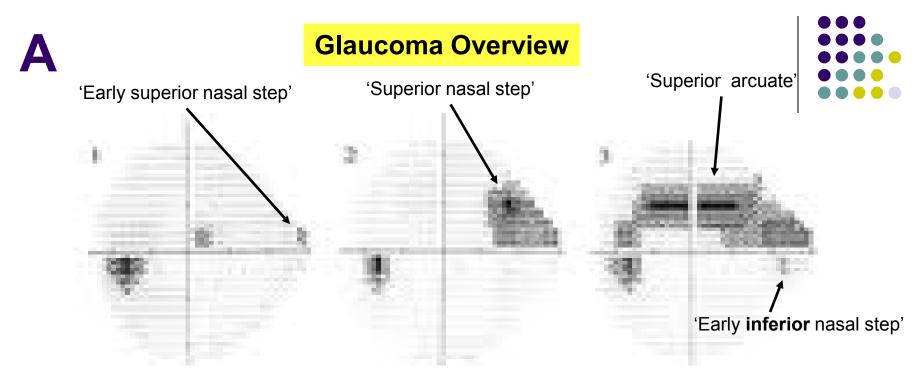
No question—proceed when ready



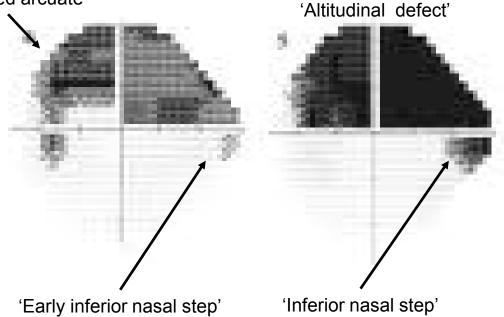
'Advanced arcuate'



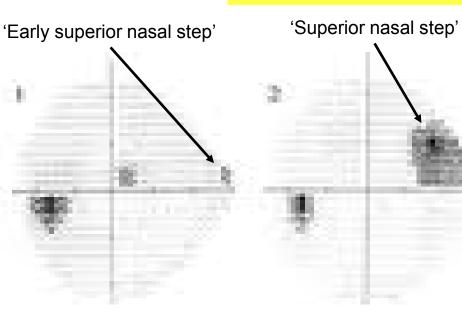
Once an arcuate has expanded sufficiently, it becomes an *defect*. The superior visual field is now all but gone.



'Advanced arcuate'

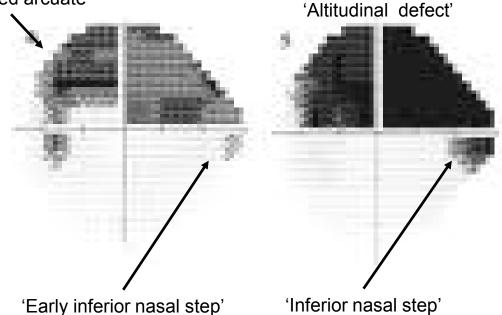


Once an arcuate has expanded sufficiently, it becomes an *altitudinal defect*. The superior visual field is now all but gone.



'Early **inferior** nasal step'

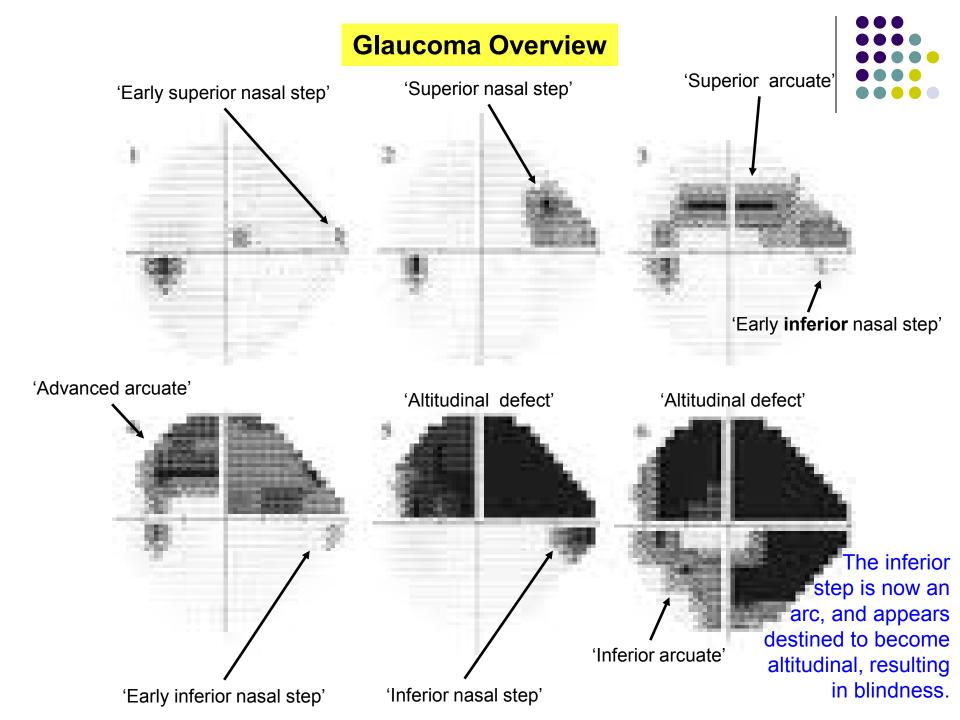
'Advanced arcuate'

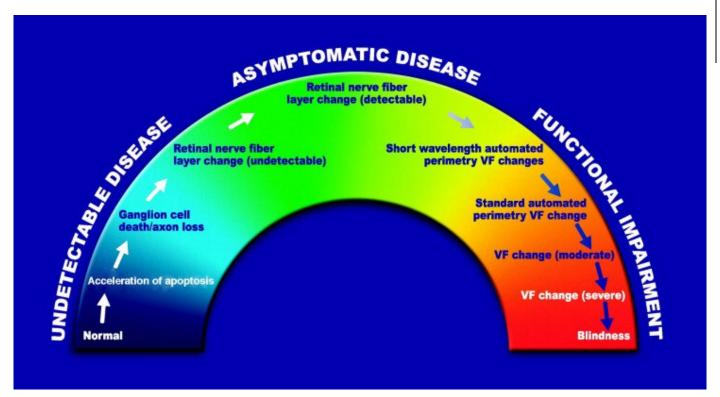


Once an arcuate has expanded sufficiently, it becomes an *altitudinal defect*. The superior visual field is now all but gone. Note the inferior nasal step continues to enlarge.

'Superior arcuate'

No question—proceed when ready

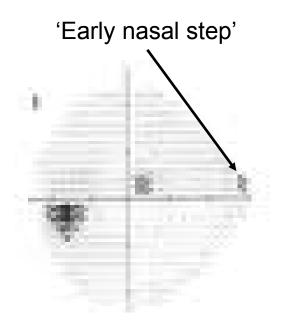




A note on the 'Glaucoma Continuum'

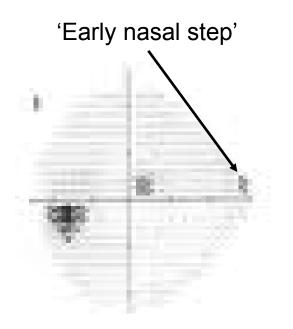
Glaucoma is a progressive condition, passing from undetectable early disease to asymptomatic-but-detectable (via RNFL imaging) disease to functional (ie, marked by VF loss) disease, the last stage of which is severe vision loss and blindness.

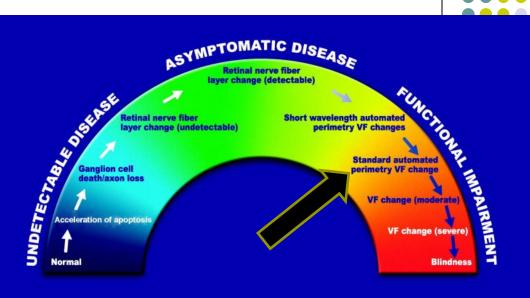




In this regard, a word on the notion of 'early' glaucoma. We previously described the above VF defect as an 'early' nasal step.



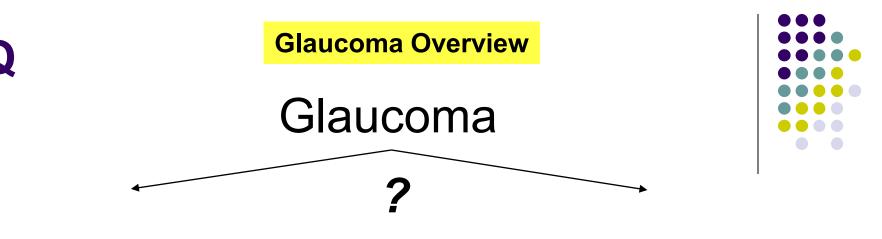




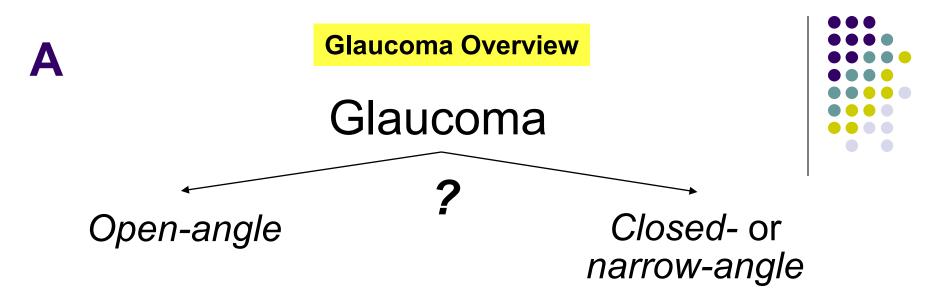
<u>In this regard, a word on the notion of 'early' glaucoma</u>. We previously described the above VF defect as an 'early' nasal step. But take note of the point along the glaucoma continuum at which such a VF defect occurs clearly, it doesn't qualify as 'early' disease with respect to the continuum.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



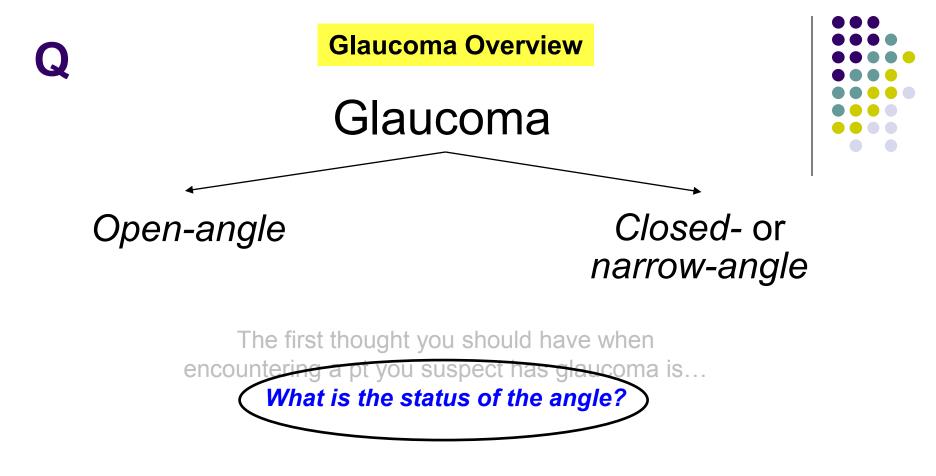
Finally, let's look briefly at how one should think through the new glaucoma case sitting in your exam chair



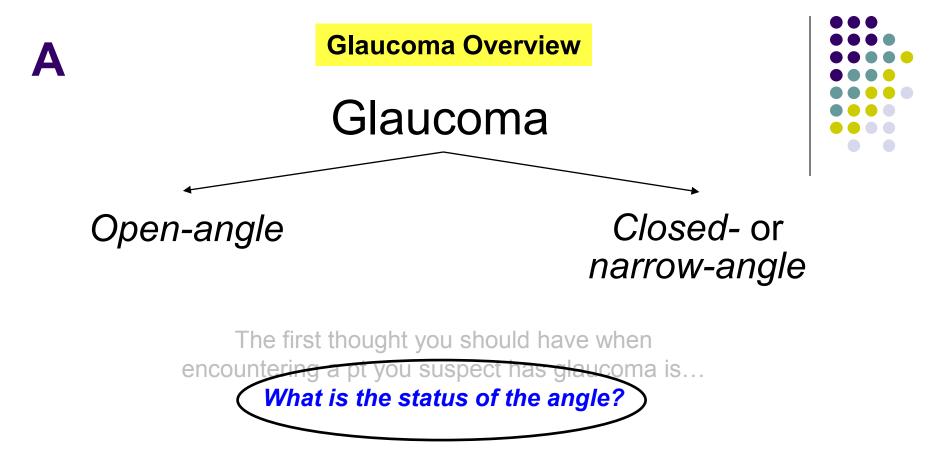
The first thought you should have when encountering a pt you suspect has glaucoma is...



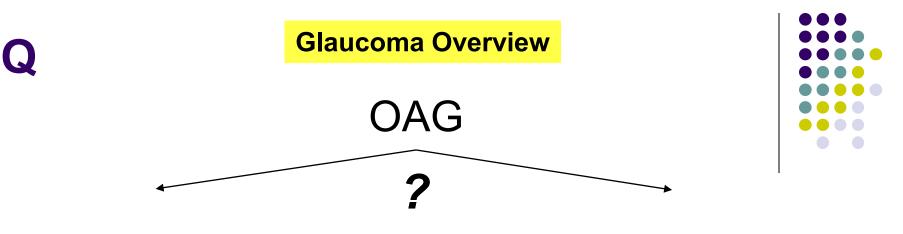
The first thought you should have when encountering a pt you suspect has glaucoma is... *What is the status of the angle?*



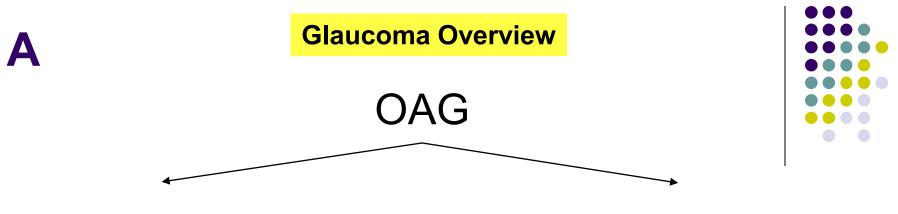
How does one determine the status of the angle?



How does one determine the status of the angle? Gonioscopy. Don't assume your glaucoma pt has open angles—prove it by gonioing them!



Once you have determined a pt has open-angle glaucoma, the next 'first thought' is to ask...

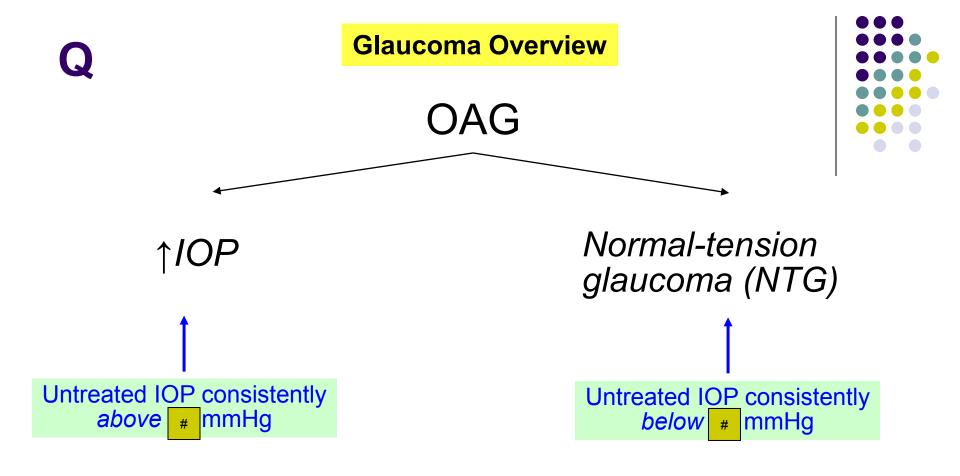


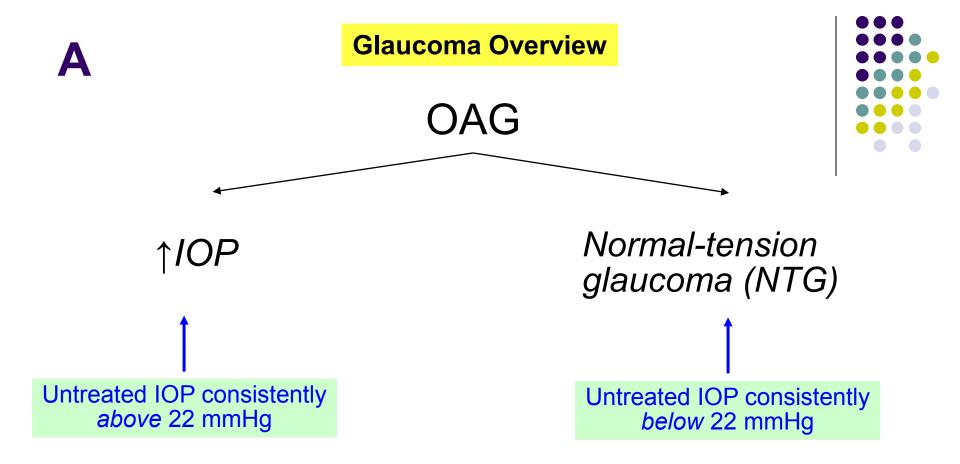
↑IOP

Normal-tension glaucoma (NTG)

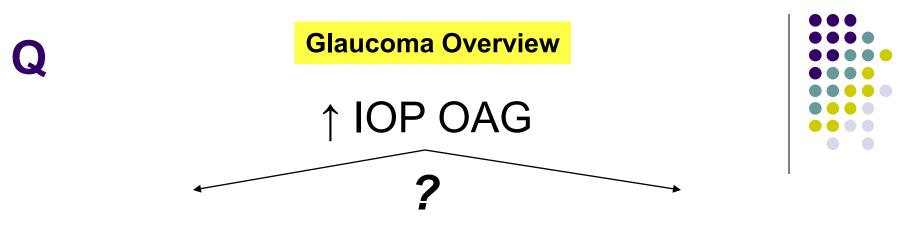
Once you have determined a pt has open-angle glaucoma, the next 'first thought' is to ask...

Is it high-pressure OAG, or low (aka normal) tension OAG?

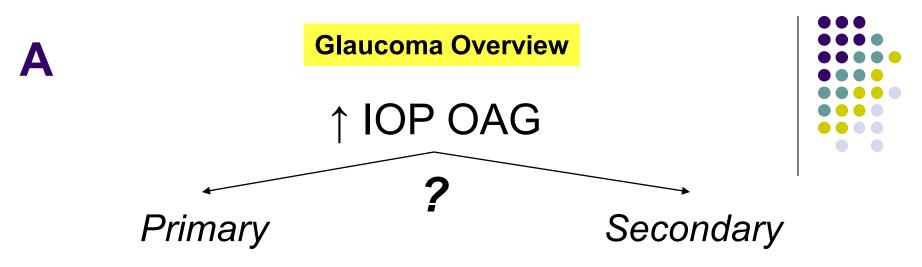




(Note that this distinction is somewhat controversial, as some glaucomalogists contend NTG is **not** a separate condition.)

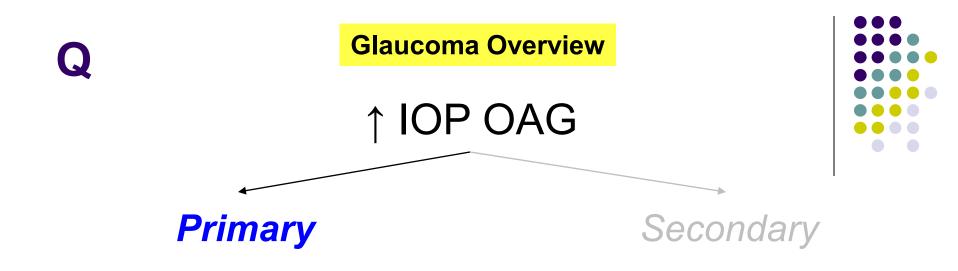


Once you have determined a pt has high-pressure open-angle glaucoma, the next 'first thought' is to ask...

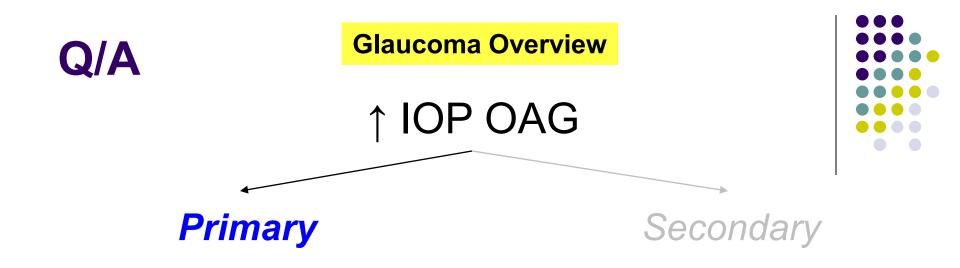


Once you have determined a pt has high-pressure open-angle glaucoma, the next 'first thought' is to ask...

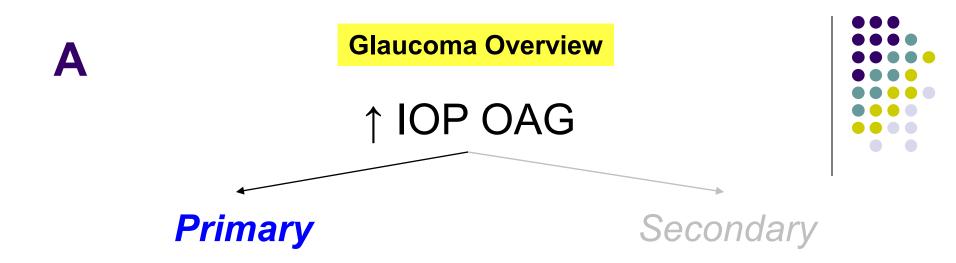
Is it primary open-angle glaucoma (POAG), or secondary OAG?

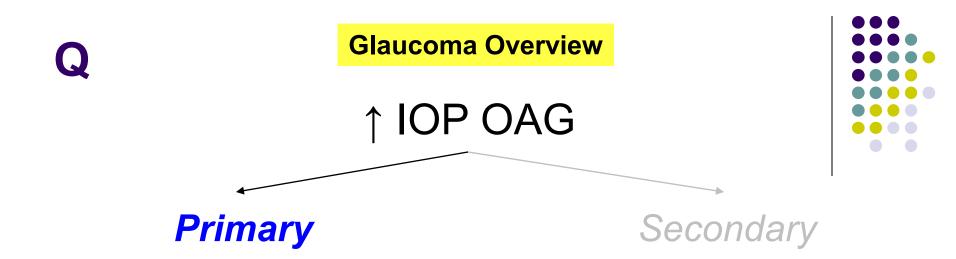


How prevalent is POAG in the US?

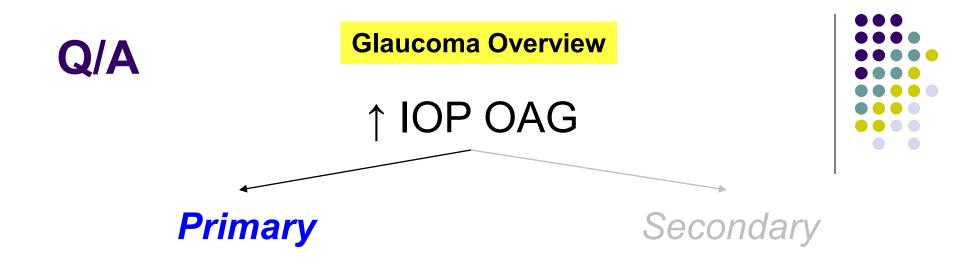


How prevalent is POAG in the US? Very. It affects about 6 of the over-40 population.

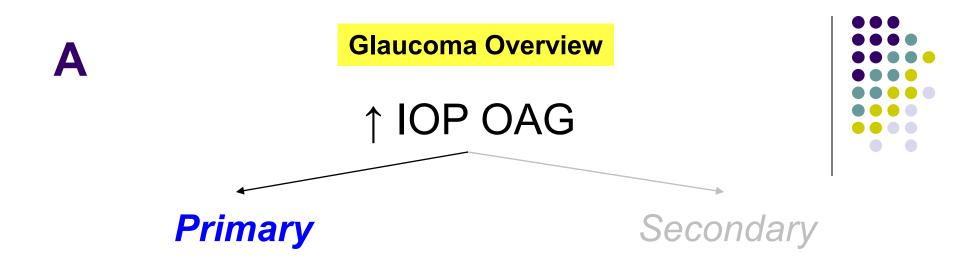




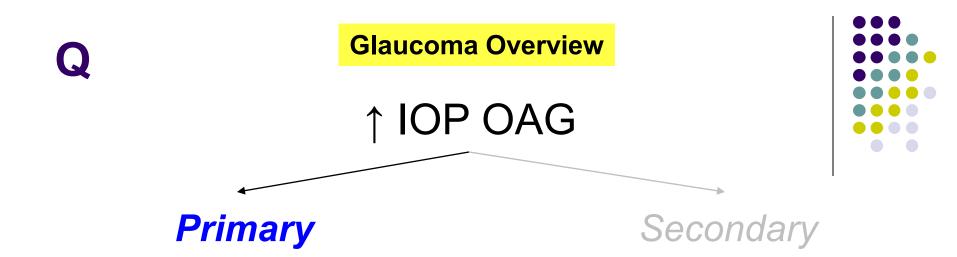
Where does POAG rank worldwide as a cause of blindness?



Where does POAG rank worldwide as a cause of blindness? It is second only to

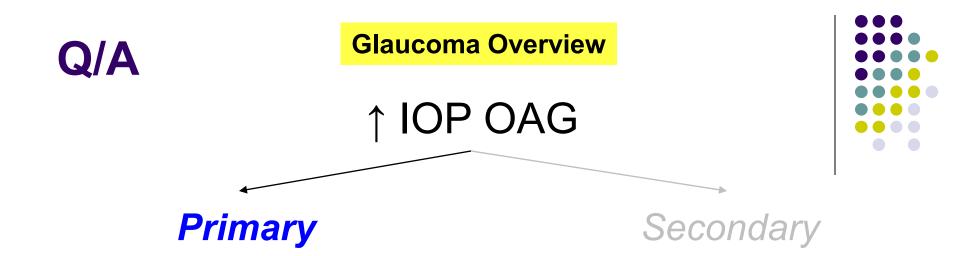


Where does POAG rank worldwide as a cause of blindness? It is second only to cataract



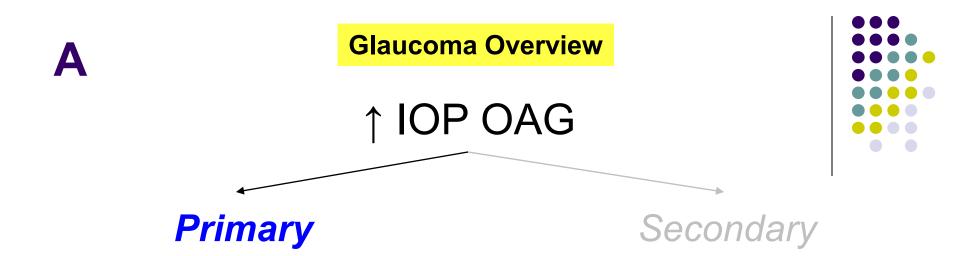
Where does POAG rank worldwide as a cause of blindness? It is second only to cataract

Is there a racial predilection?



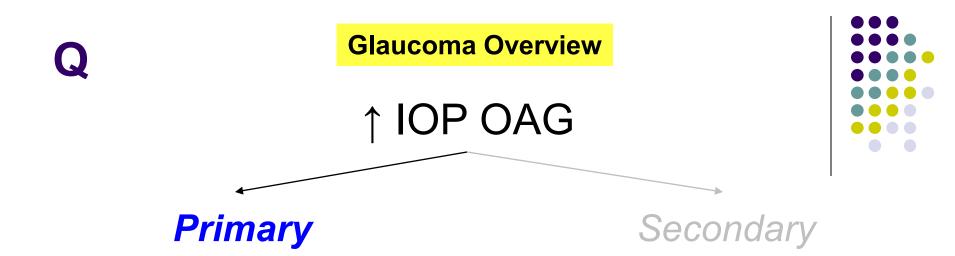
Where does POAG rank worldwide as a cause of blindness? It is second only to cataract

Is there a racial predilection? Yes, individuals of and heritage are at a 4x greater risk than are (and their relative risk of going blind is even higher than that)



Where does POAG rank worldwide as a cause of blindness? It is second only to cataract

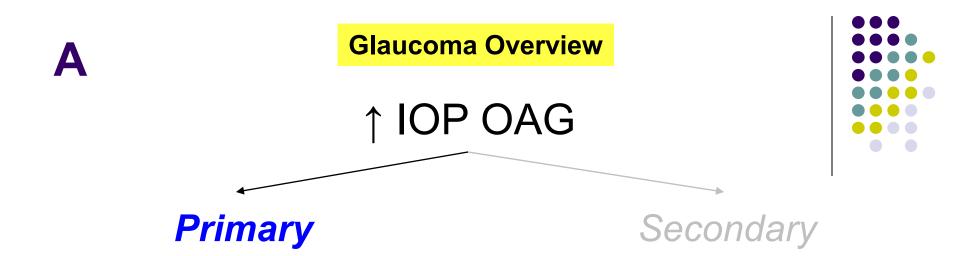
Is there a racial predilection? Yes, individuals of black and Hispanic heritage are at a 4x greater risk than are whites (and their relative risk of going blind is even higher than that)



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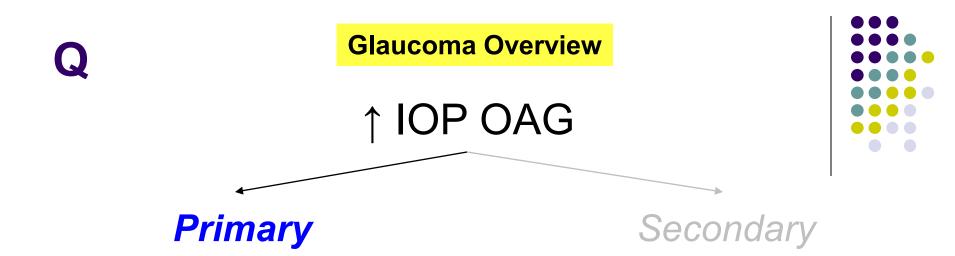
Is age a risk factor?



Where does POAG rank worldwide as a cause of blindness? It is second only to cataract

Is there a racial predilection? Yes, individuals of black and Hispanic heritage are at a 4x greater risk than are whites (and their relative risk of going blind is even higher than that)

Is age a risk factor? Yes, POAG rates increase dramatically with age

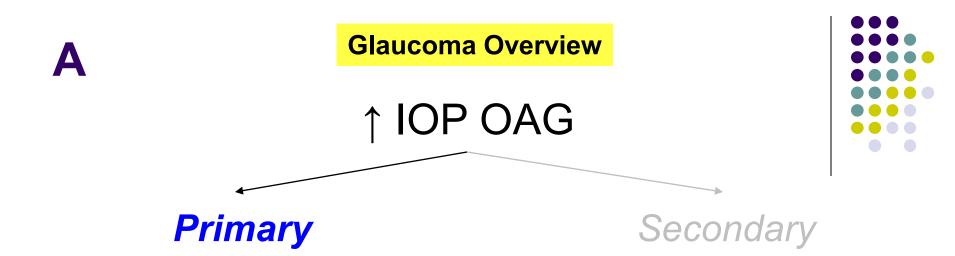


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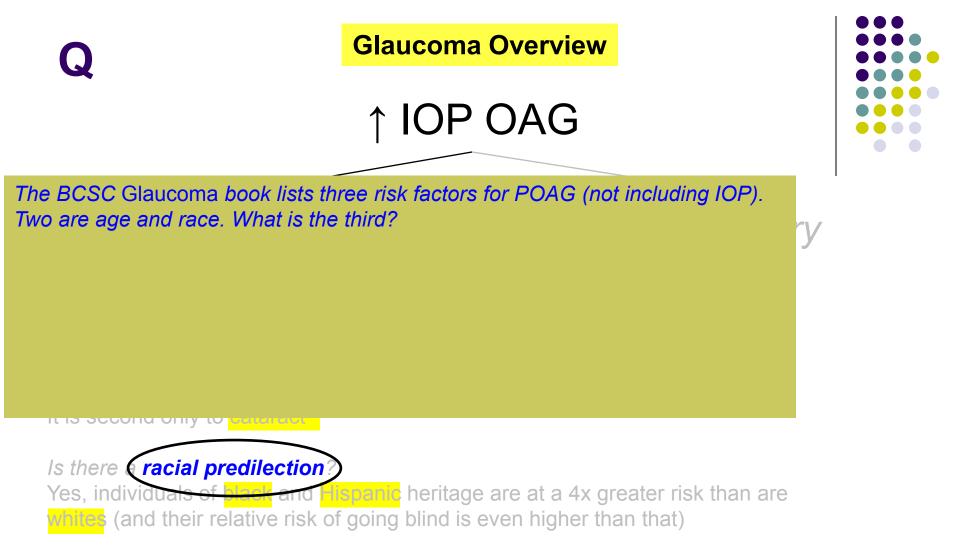
What is the #1 risk factor?



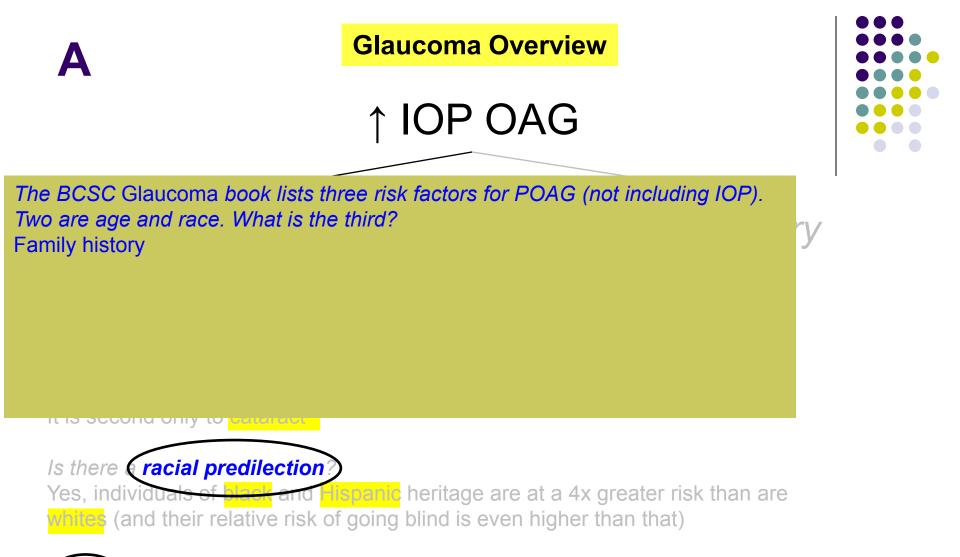
Where does POAG rank worldwide as a cause of blindness? It is second only to cataract

Is there a racial predilection? Yes, individuals of black and Hispanic heritage are at a 4x greater risk than are whites (and their relative risk of going blind is even higher than that)

Is age a risk factor? Yes, POAG rates increase dramatically with age



Yes, POAG rates increase dramatically with age



Yes, POAG rates increase dramatically with age



--?

↑ IOP OAG

The BCSC Glaucoma book lists three risk factors for POAG (not including IOP). Two are age and race. What is the third? Family history

While not listed in the section on risk factors, the Glaucoma book alludes to two other variables as being well-established as significant risk factors for POAG. What are they?

It is second only to paralable

Is there **(racial predilection**?)

Yes, individuals of <mark>sleek</mark> and <mark>Hispanic</mark> heritage are at a 4x greater risk than are whites (and their relative risk of going blind is even higher than that)

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↑ IOP OAG

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While not listed in the section on risk factors, the Glaucoma book alludes to two other variables as being well-established as significant risk factors for POAG. What are they?

--Central corneal thickness (CCT)

--Ocular perfusion pressure (OPP)

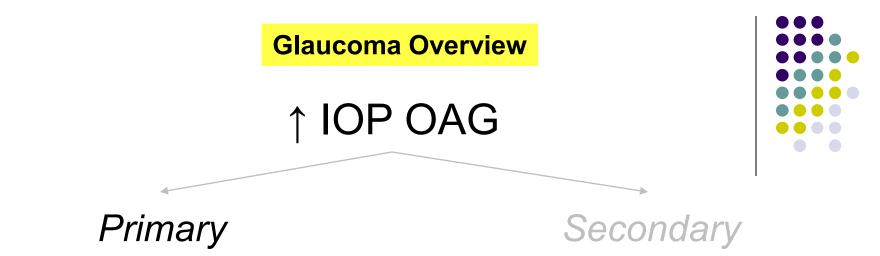
It is second only to patalact

Is there **(racial predilection**?)

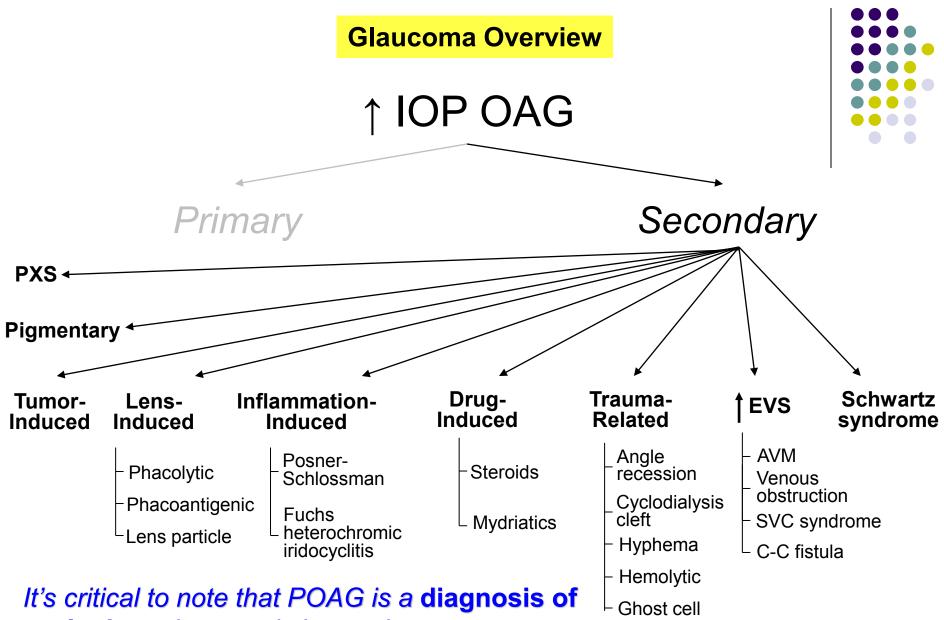
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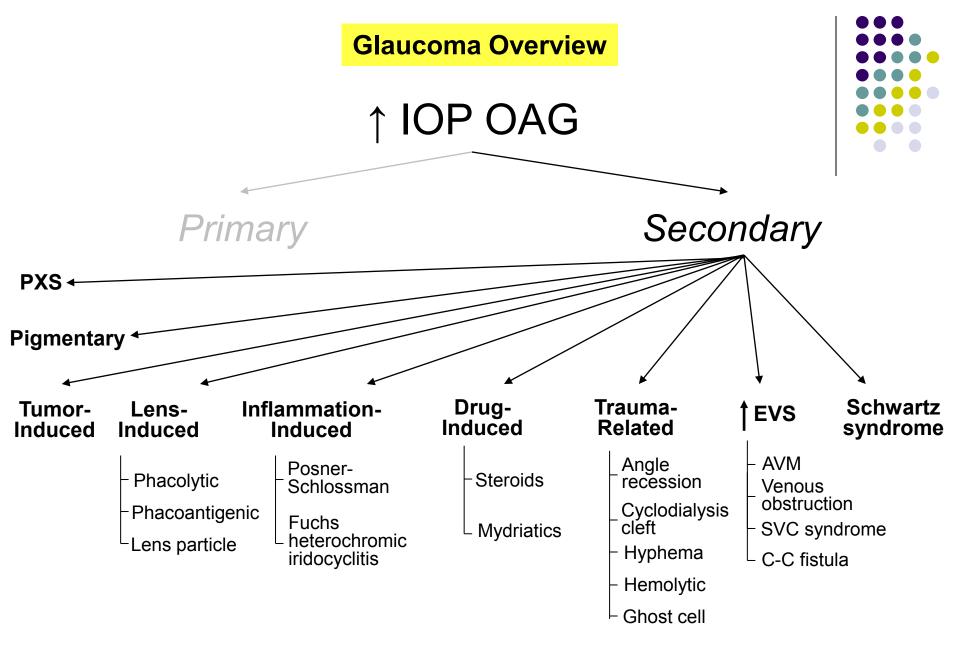




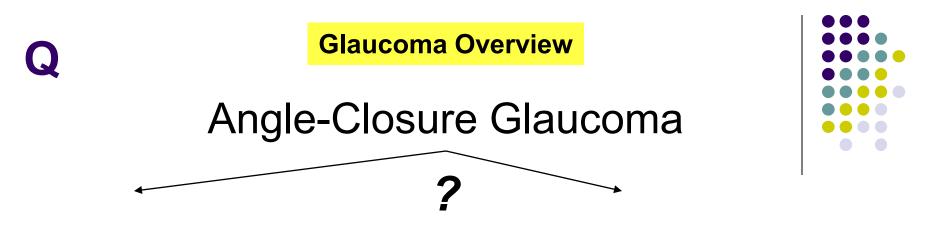
It's critical to note that POAG is a diagnosis of exclusion—



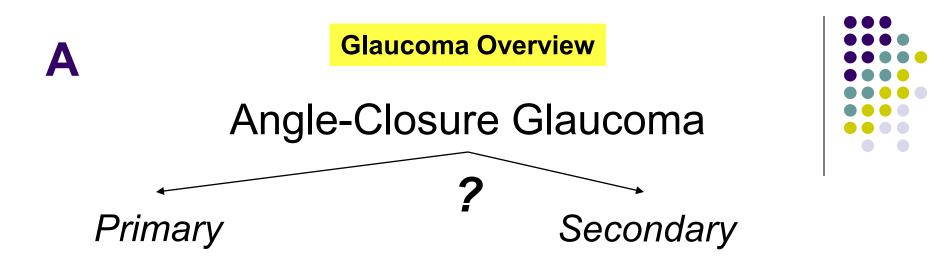
exclusion—*it can only be made once secondary causes of OAG have been ruled out!*



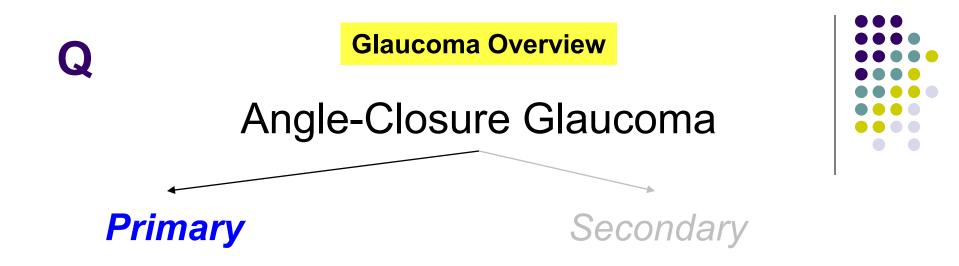
(Most of these conditions are addressed in detail in other slide-sets—see the Table of Contents)

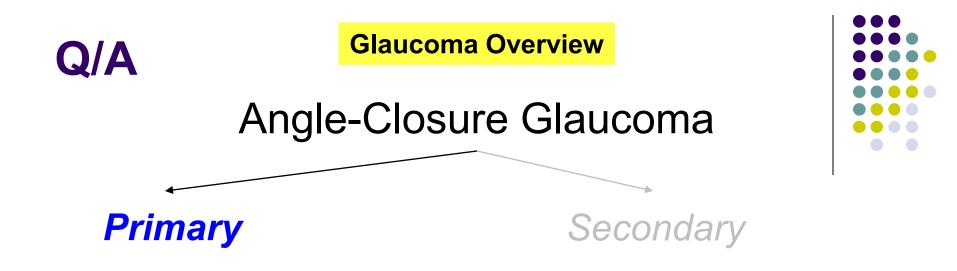


Once you have determined a pt has angle-closure glaucoma, the next 'first thought' is to ask...

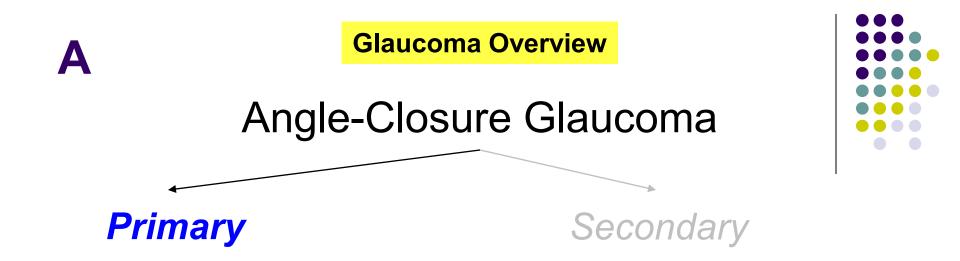


Once you have determined a pt has angle-closure glaucoma, the next 'first thought' is to ask... *is it primary or secondary?*

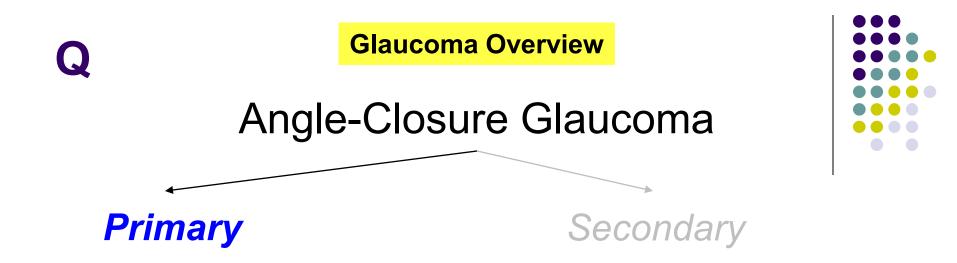




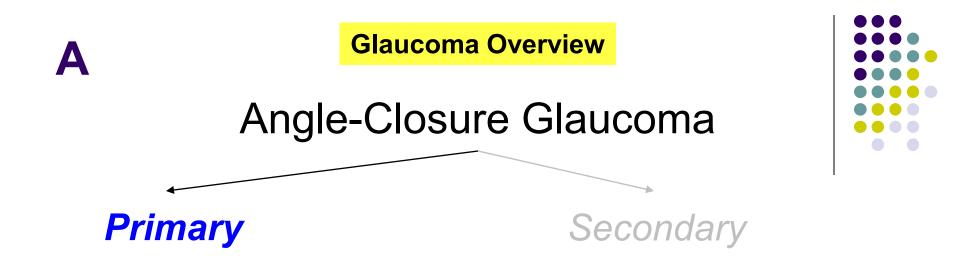
Is there a racial predilection regarding the risk of PACG? Yes, individuals of heritage have the highest known risk of PACG



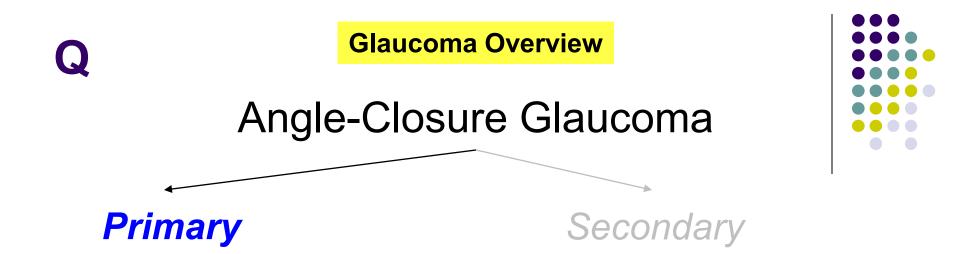
Is there a racial predilection regarding the risk of PACG? Yes, individuals of Inuit heritage have the highest known risk of PACG



Yes, individuals of Inuit heritage have the highest known risk of PACG--their relative risk has been estimated to be as high as #× that of whites.

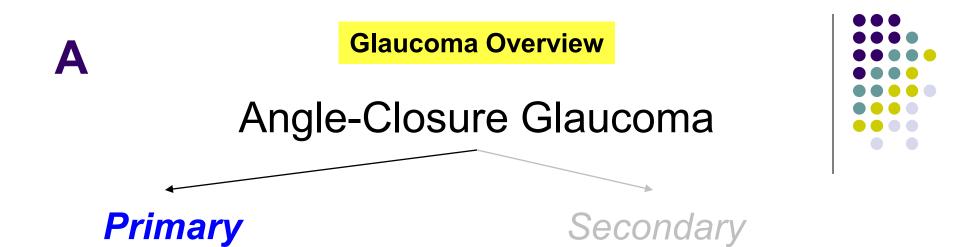


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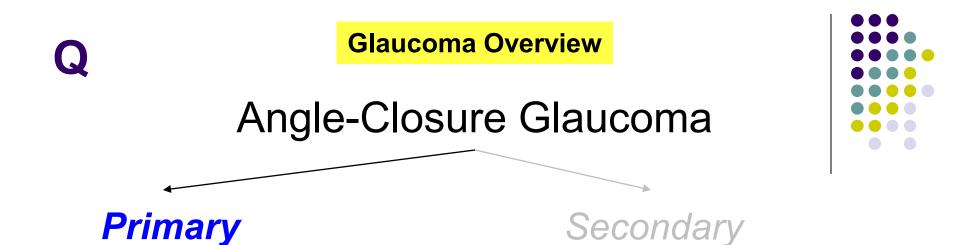
What about people of Asian descent?



Yes, individuals of Inuit heritage have the highest known risk of PACG--their relative risk has been estimated to be as high as 40x that of whites.

What about people of Asian descent?

Their relative risk is somewhere between that of the Inuit and whites

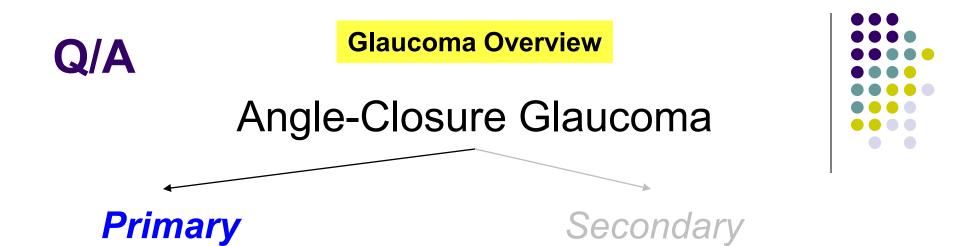


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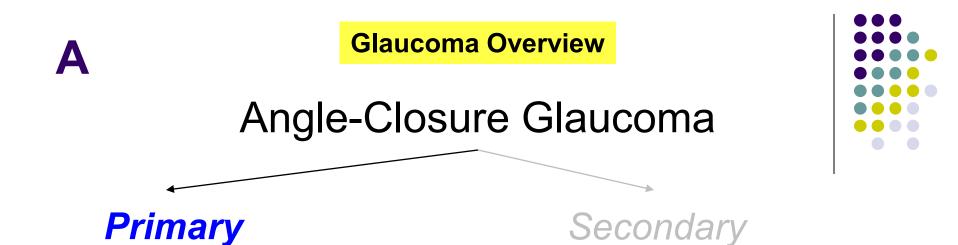


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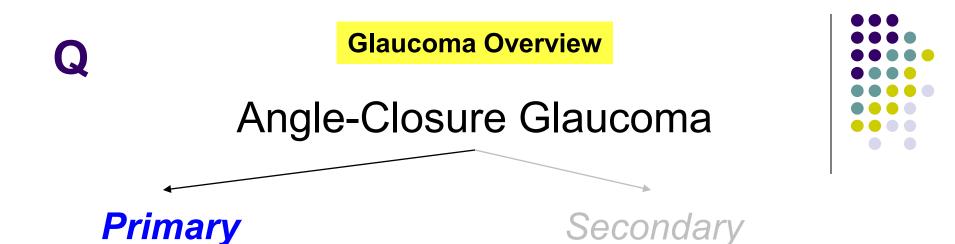
Is age a risk factor? Yes, the incidence with age



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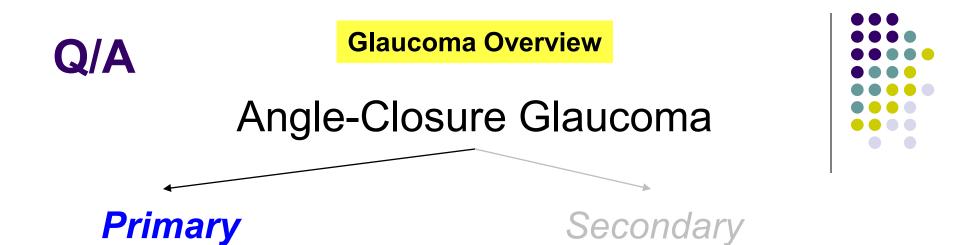


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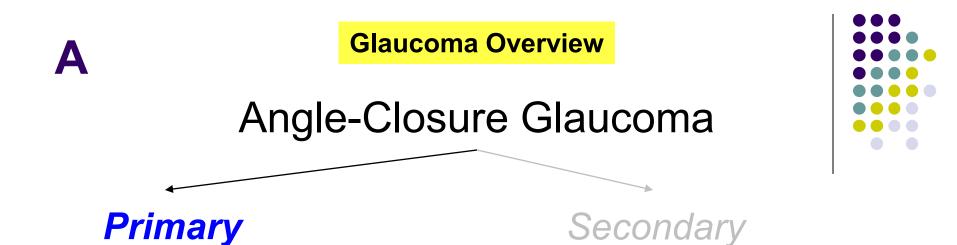


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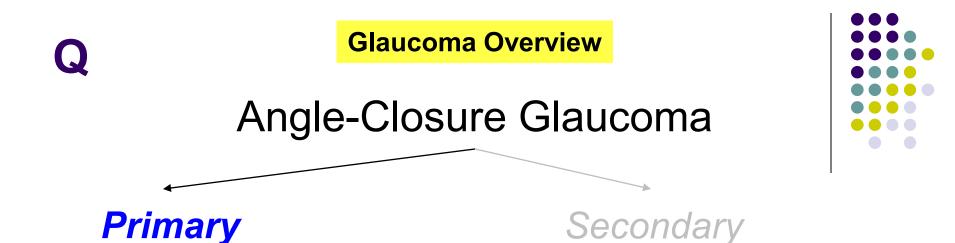


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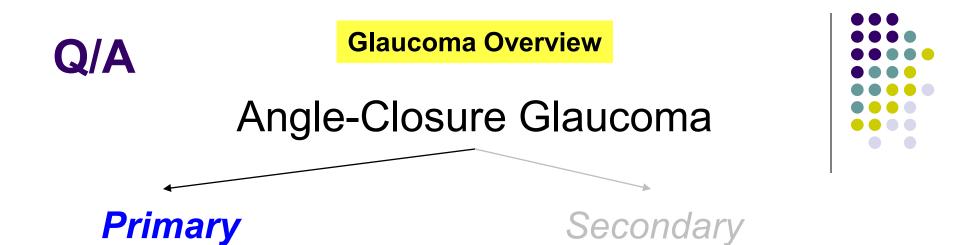
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Is refraction a risk factor?



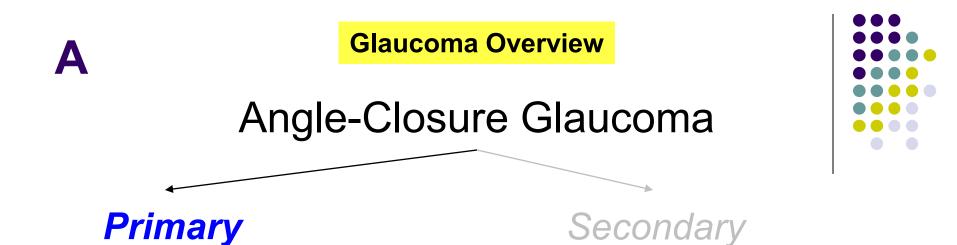
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Is gender a risk factor? Yes, women are at higher risk

Is refraction a risk factor? Yes; PACG is more likely to occur in



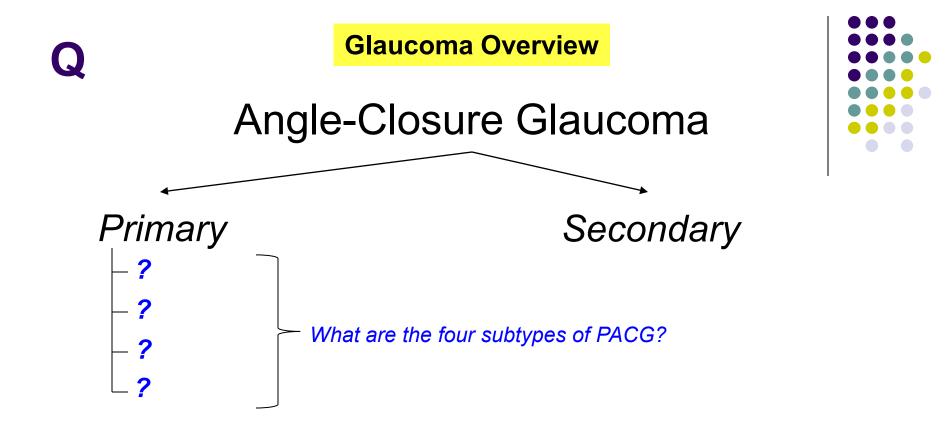
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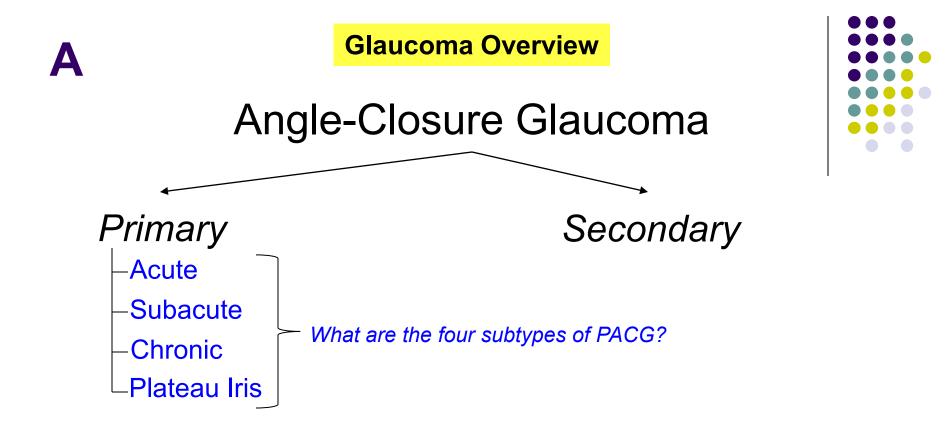
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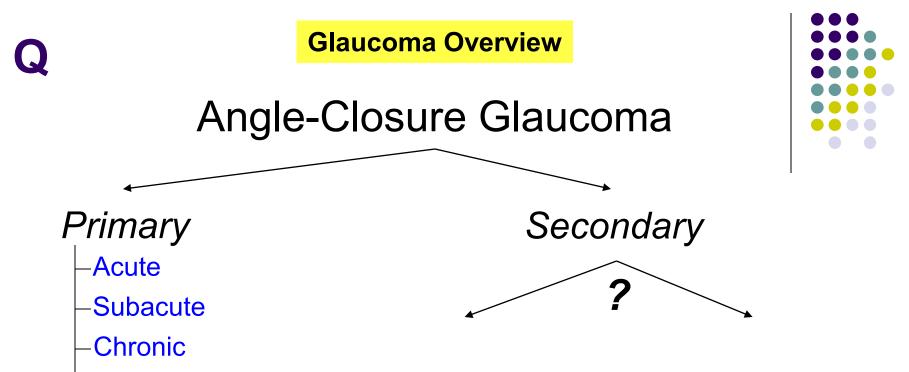
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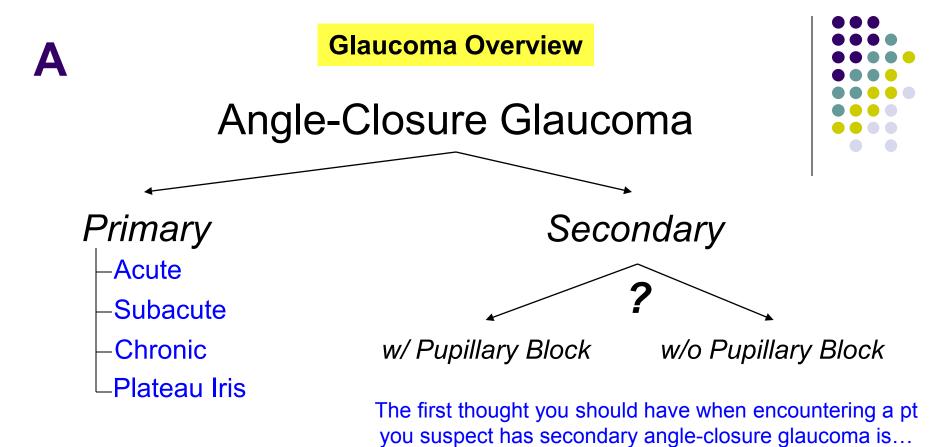




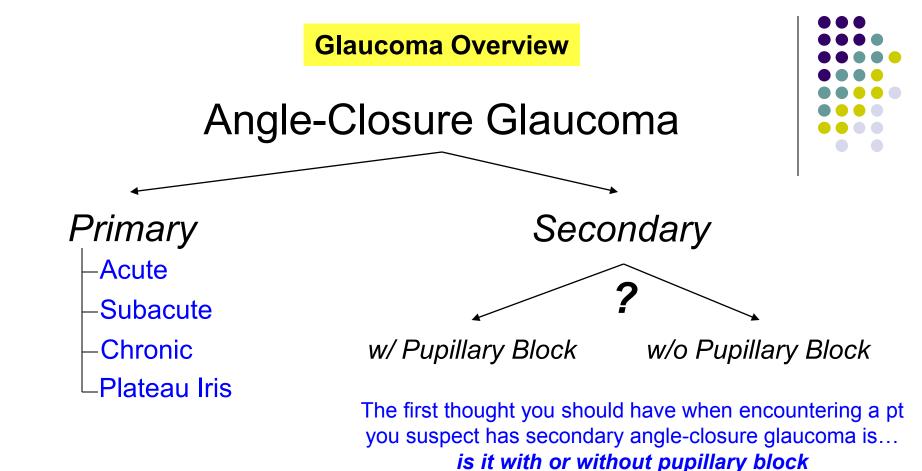


Plateau Iris

The first thought you should have when encountering a pt you suspect has secondary angle-closure glaucoma is...



is it with or without pupillary block



More information is available regarding the various forms of angle-closure glaucoma, check the Table of Contents to find it