Q

Open-angle Glaucoma: *Primary*

Define glaucoma.

A

Open-angle Glaucoma: *Primary*

Define glaucoma.

A group of optic neuropathies that present with characteristic patterns of ONH damage and VF loss

Q

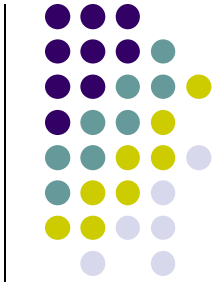
Open-angle Glaucoma: *Primary*

Define glaucoma.
A group of optic neuropathies that present with characteristic patterns of ONH damage and VF loss

Why isn't elevated IOP mentioned above?

A

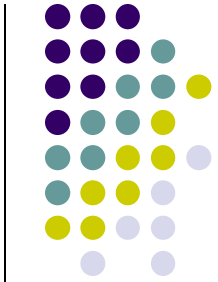
Open-angle Glaucoma: *Primary*



Define glaucoma.
A group of optic neuropathies that present with characteristic patterns of ONH damage and 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

Open-angle Glaucoma: *Primary*

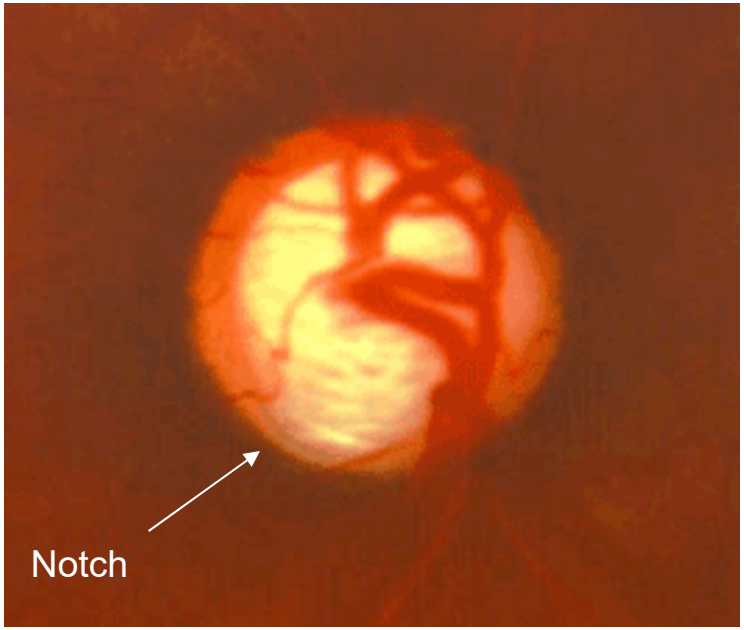


Re characteristic ONH damage in glaucoma:

Open-angle Glaucoma: *Primary*

Re characteristic ONH damage in glaucoma:

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 referred to as a 'notch.')



Glaucomatous ONH

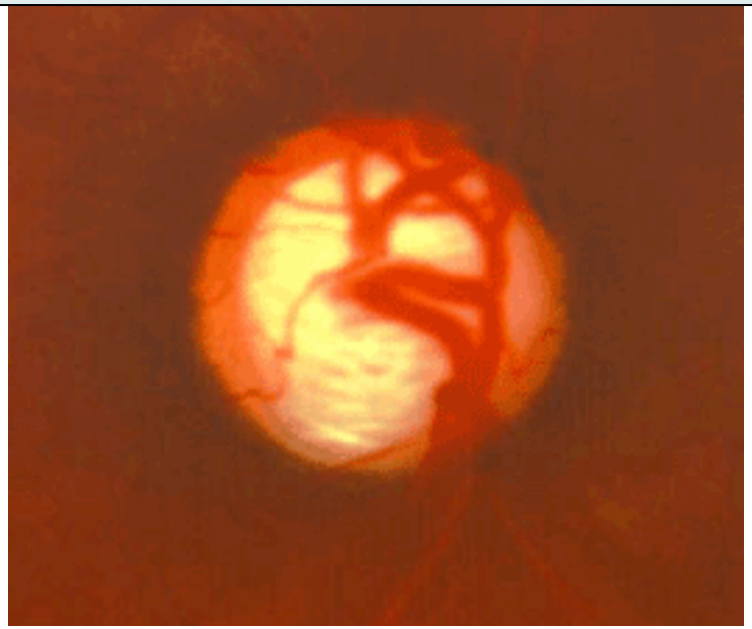
Normal ONH

Open-angle Glaucoma: *Primary*

Re characteristic ONH damage in glaucoma:

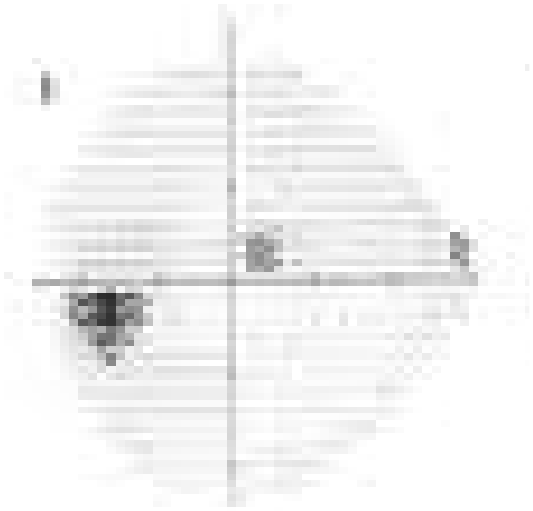
For reasons that have yet to be fully elucidated, glaucomatous optic neuropathy tends to damage the superior and inferior poles of the ONH preferentially and early. This leads to thinning at the poles (focal thinning is often referred to as a 'notch.')

For more on ONH damage in glaucoma, see slide-set G0

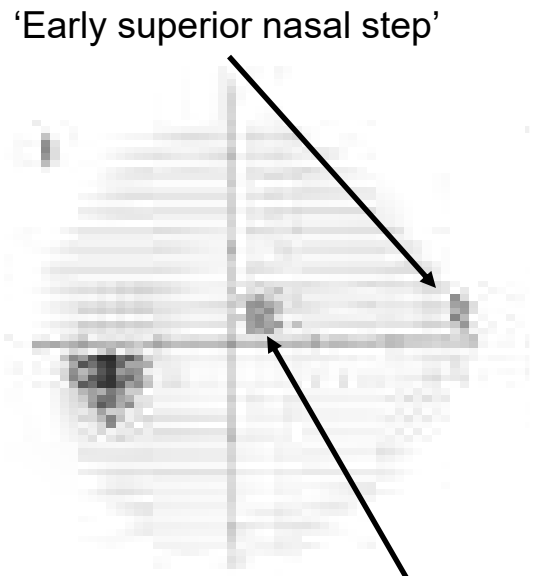


Glaucomatous ONH

Normal ONH

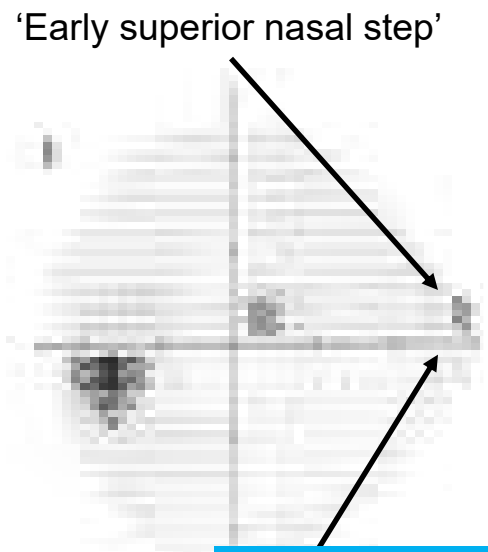


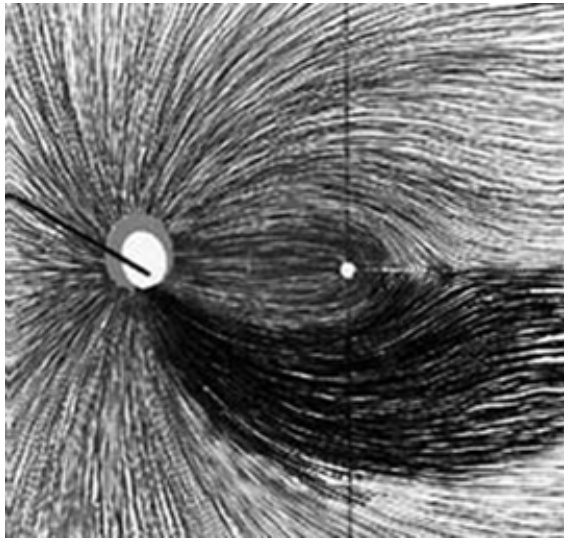
Re characteristic VF loss in glaucoma:



Re characteristic VF loss in glaucoma:

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*.

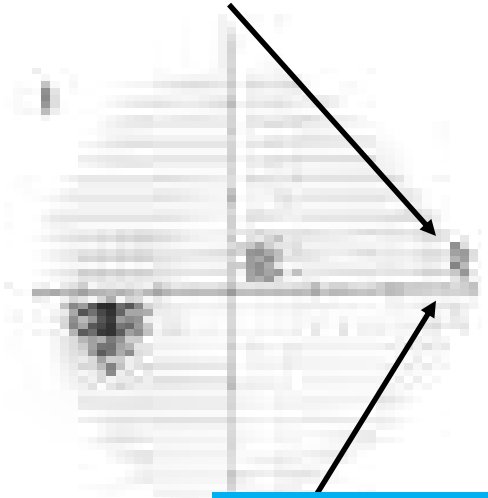


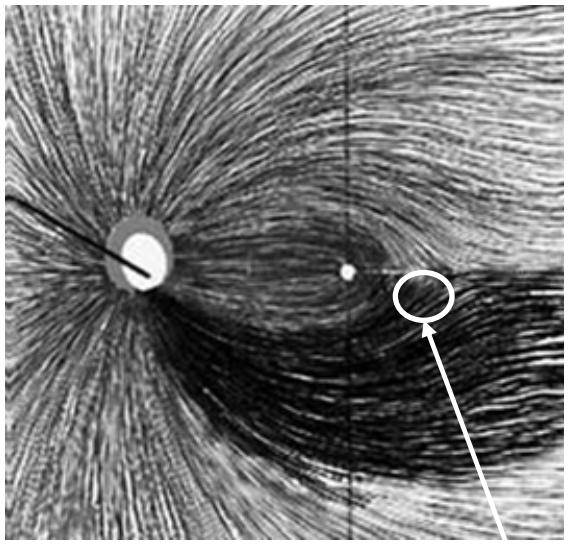


This location in the VF…

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*.

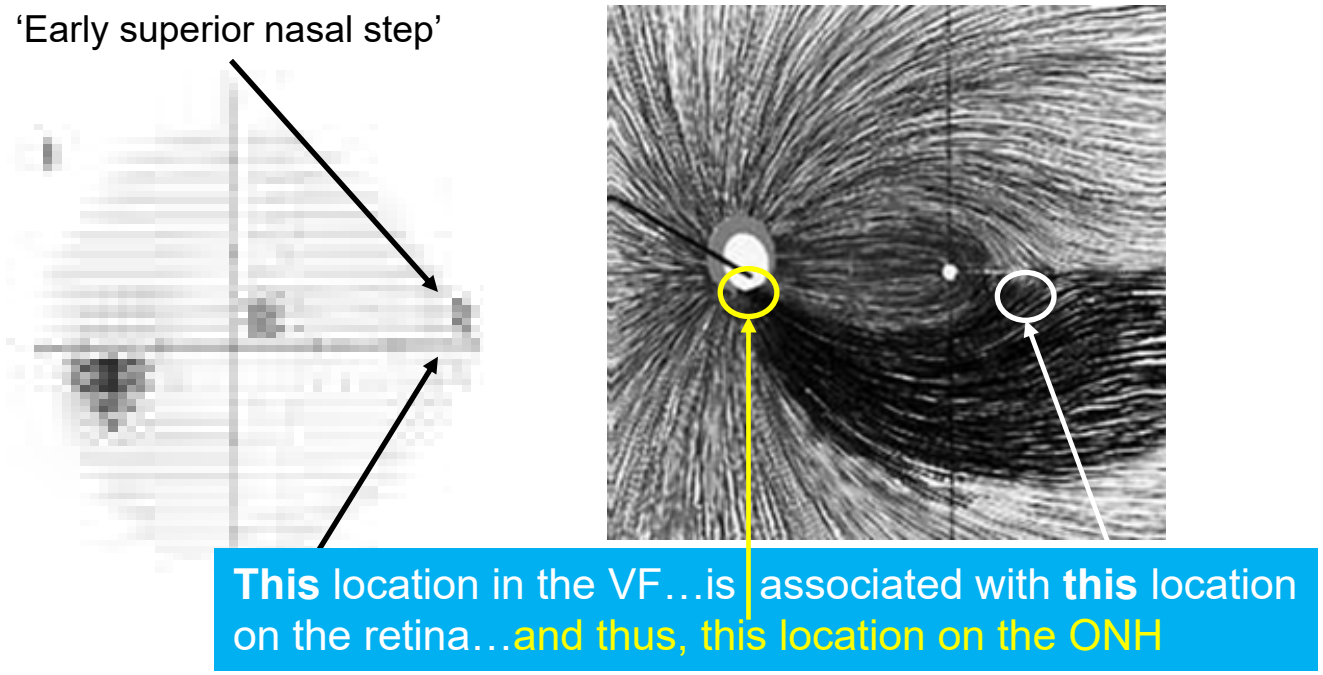
‘Early superior nasal step’





This location in the VF…is associated with **this** location on the retina…

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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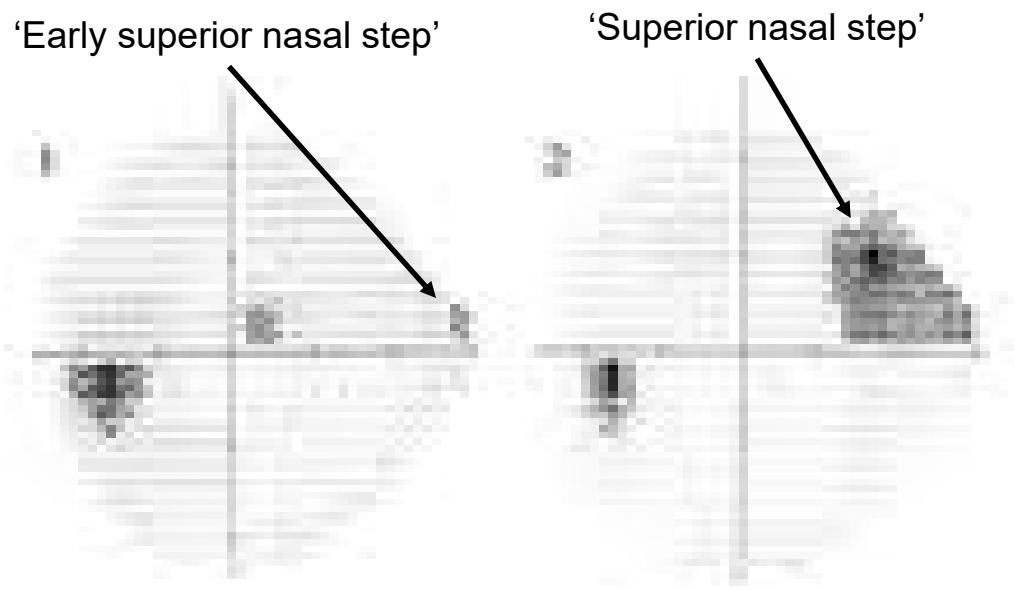
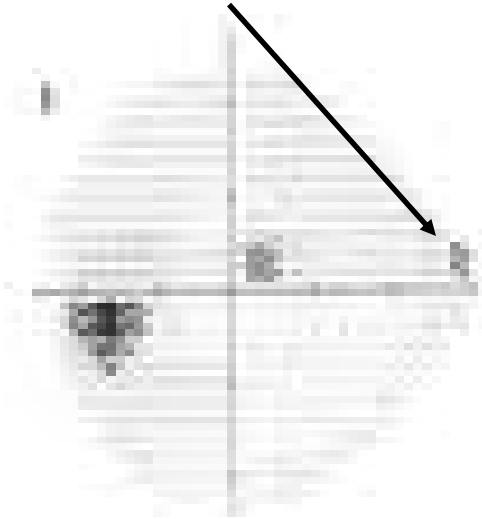
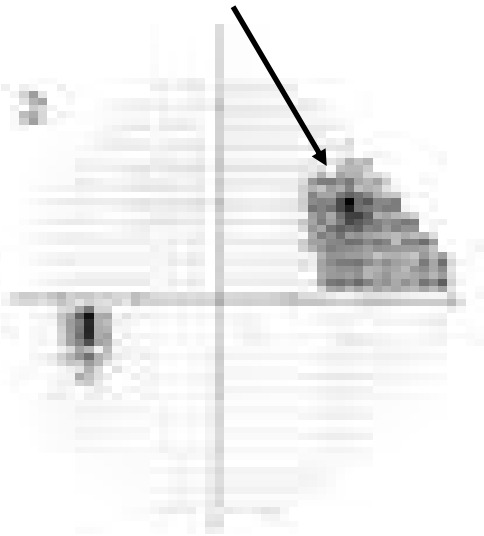


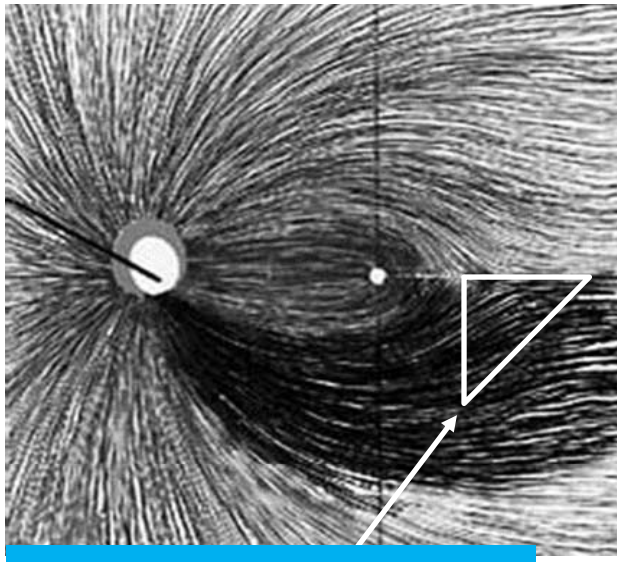
If left untreated, the nasal step will gradually enlarge.

‘Early superior nasal step’



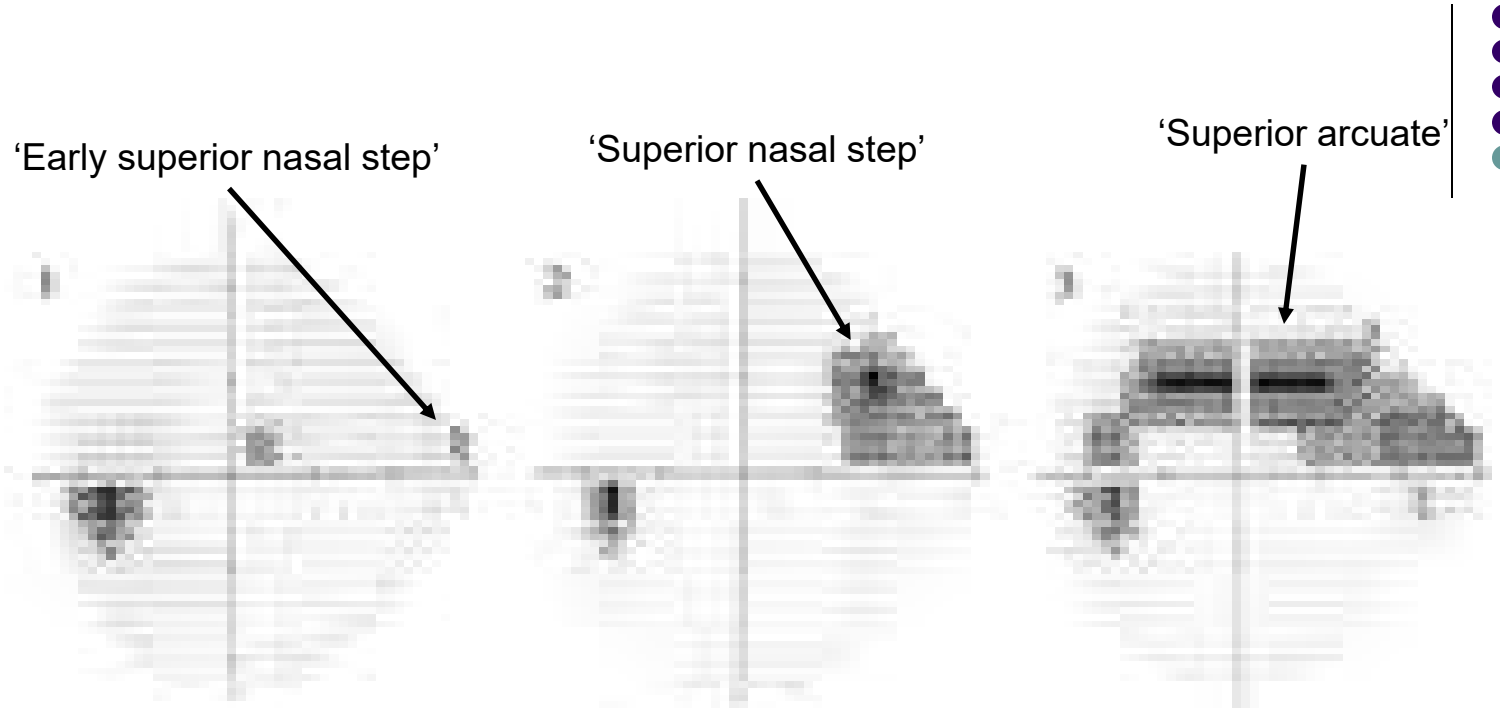
‘Superior nasal step’



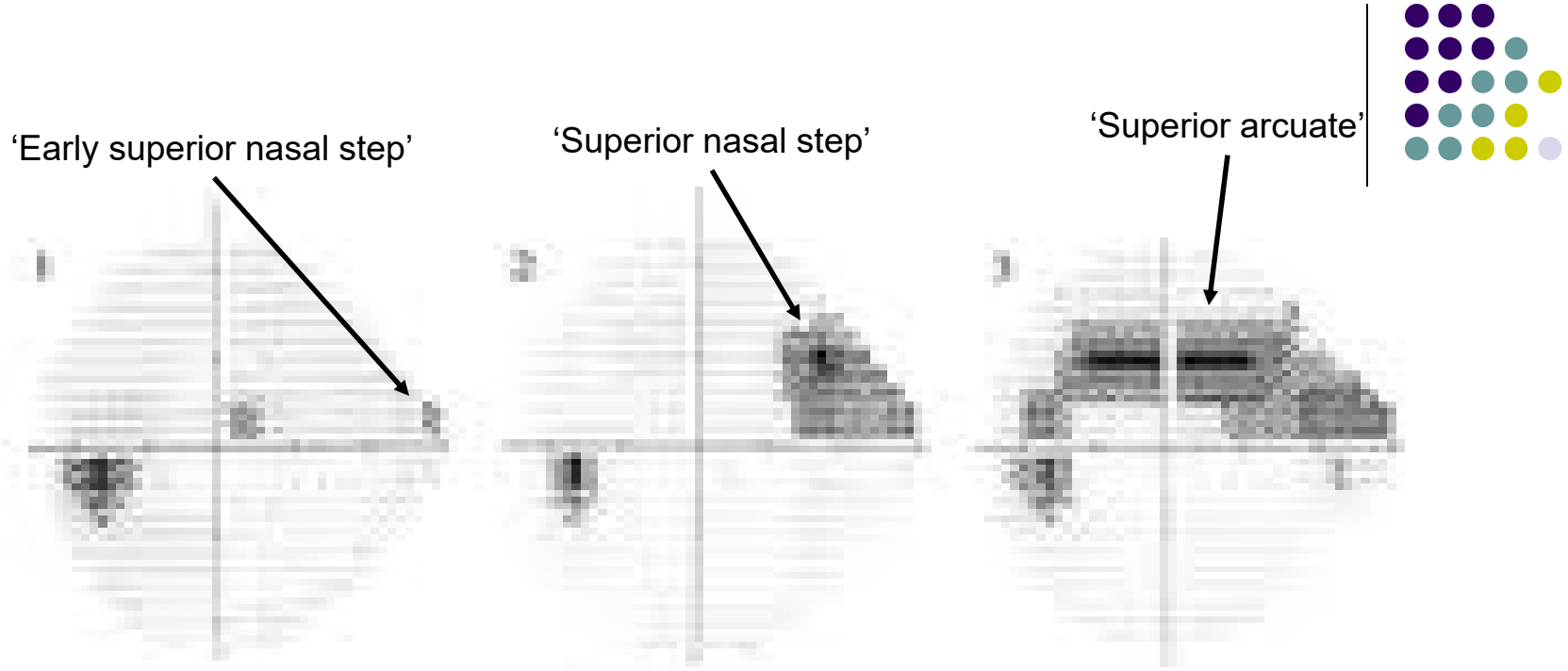


Note the area of origin for affected fibers has grown

If left untreated, the nasal step will gradually enlarge.



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



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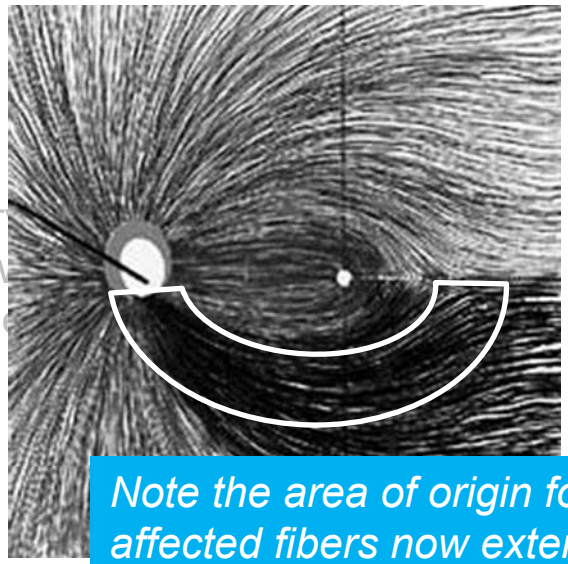
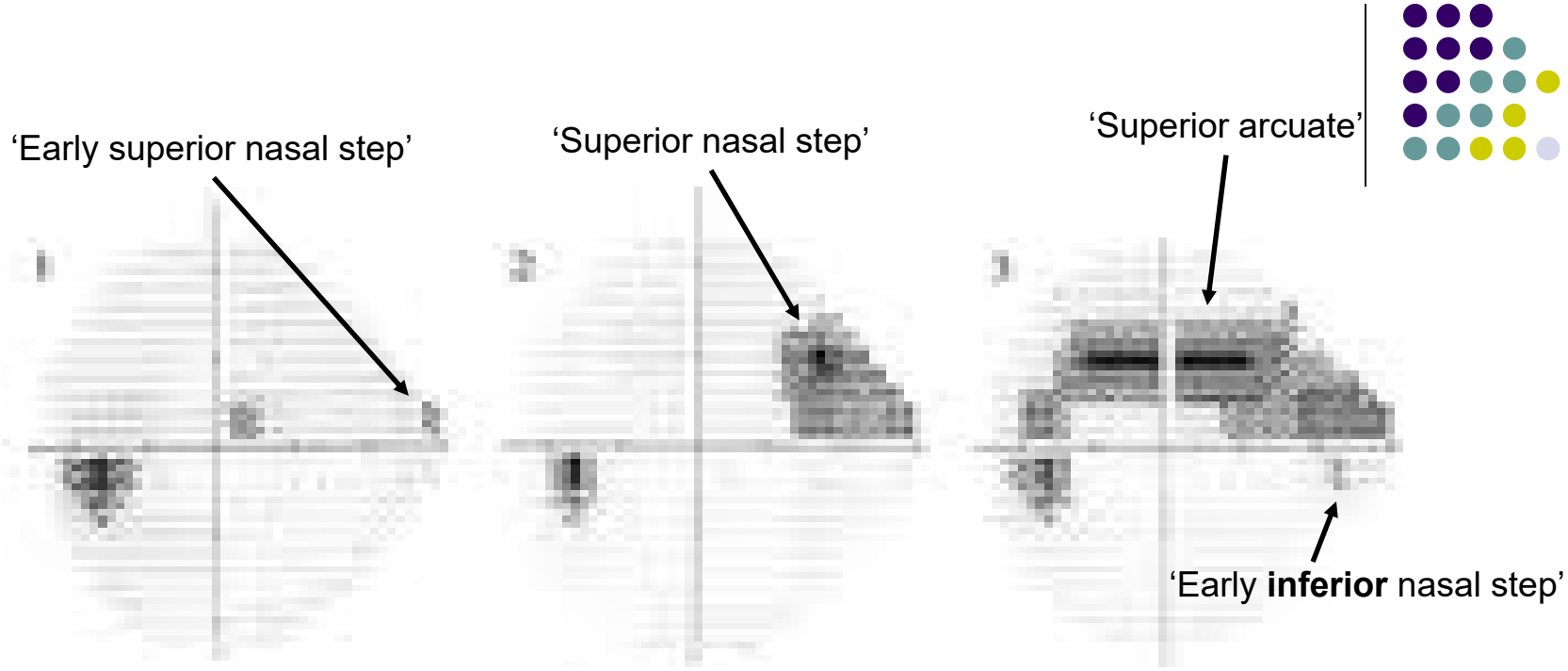
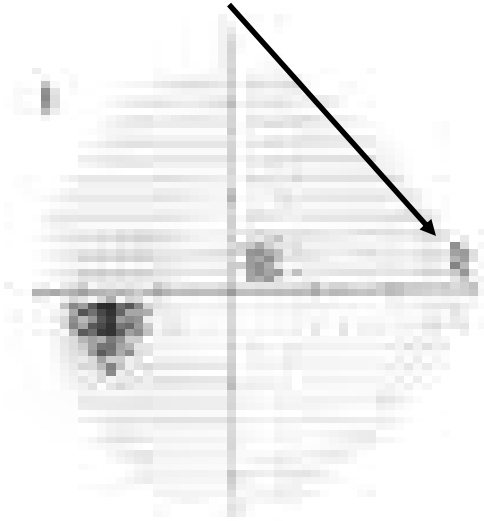


Note the area of origin for affected fibers now extends all the way to the ONH itself

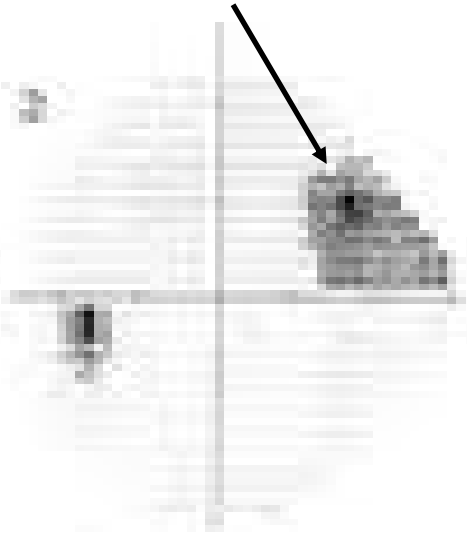


As glaucoma damage progresses, further loss of nerve fibers joining at that portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an *arcuate*.

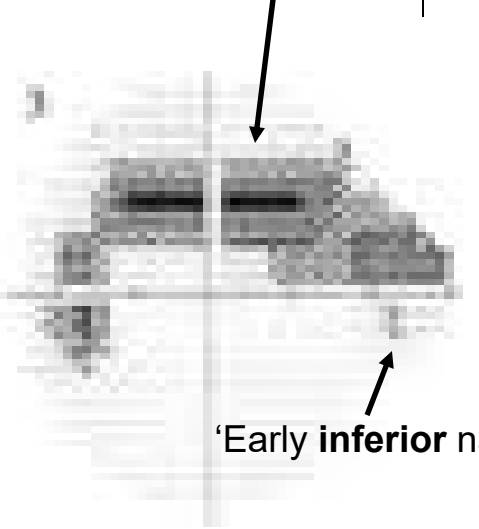
Note also that an early *inferior* nasal step is now present.



‘Early superior nasal step’



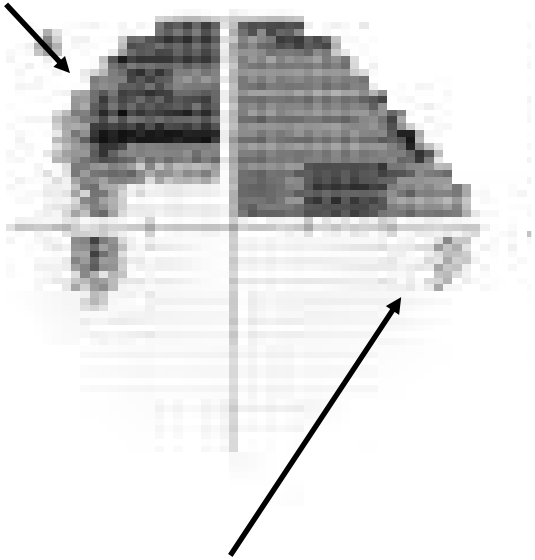
‘Superior nasal step’



‘Superior arcuate’

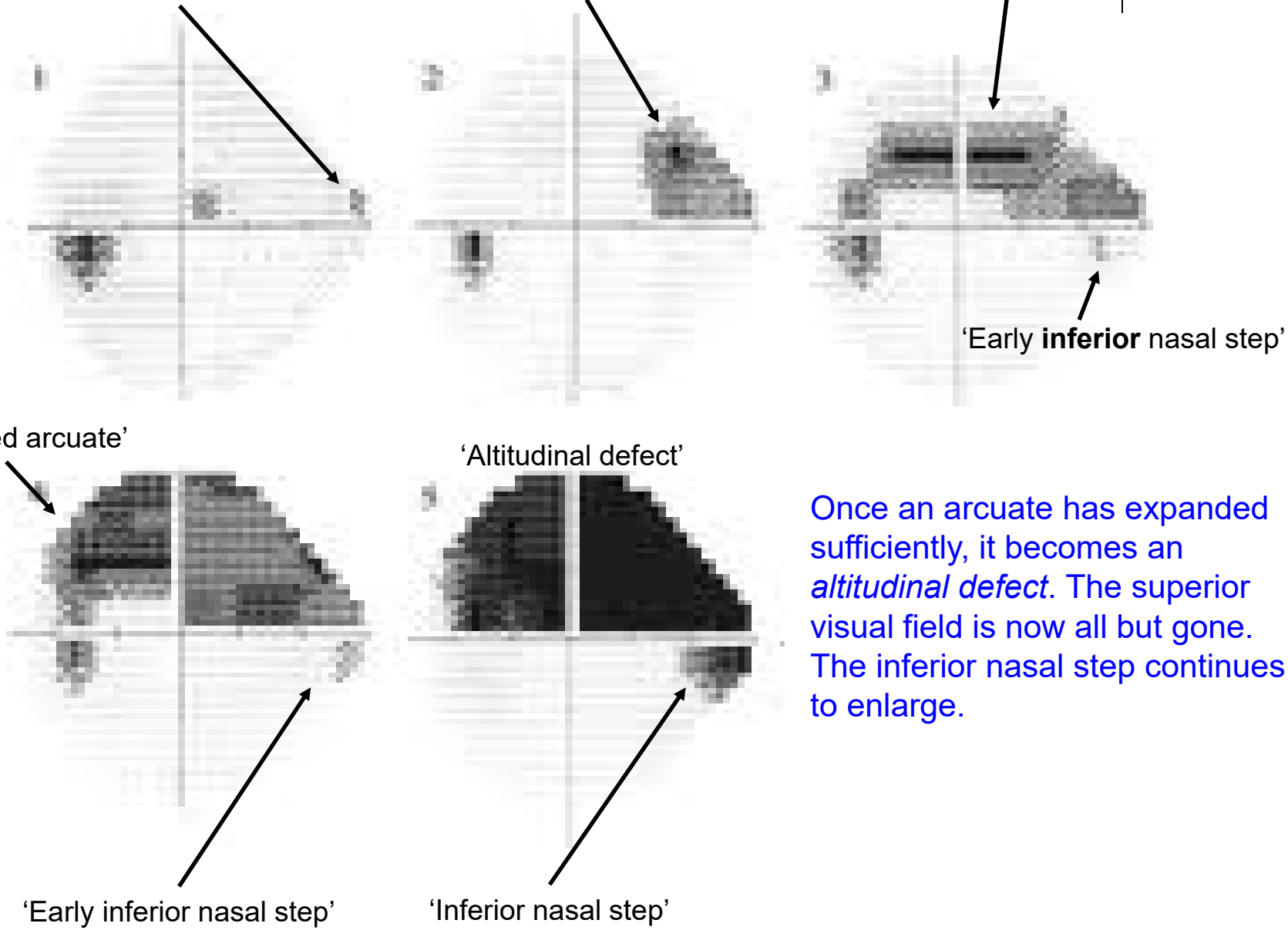
‘Early **inferior** nasal step’

‘Advanced arcuate’

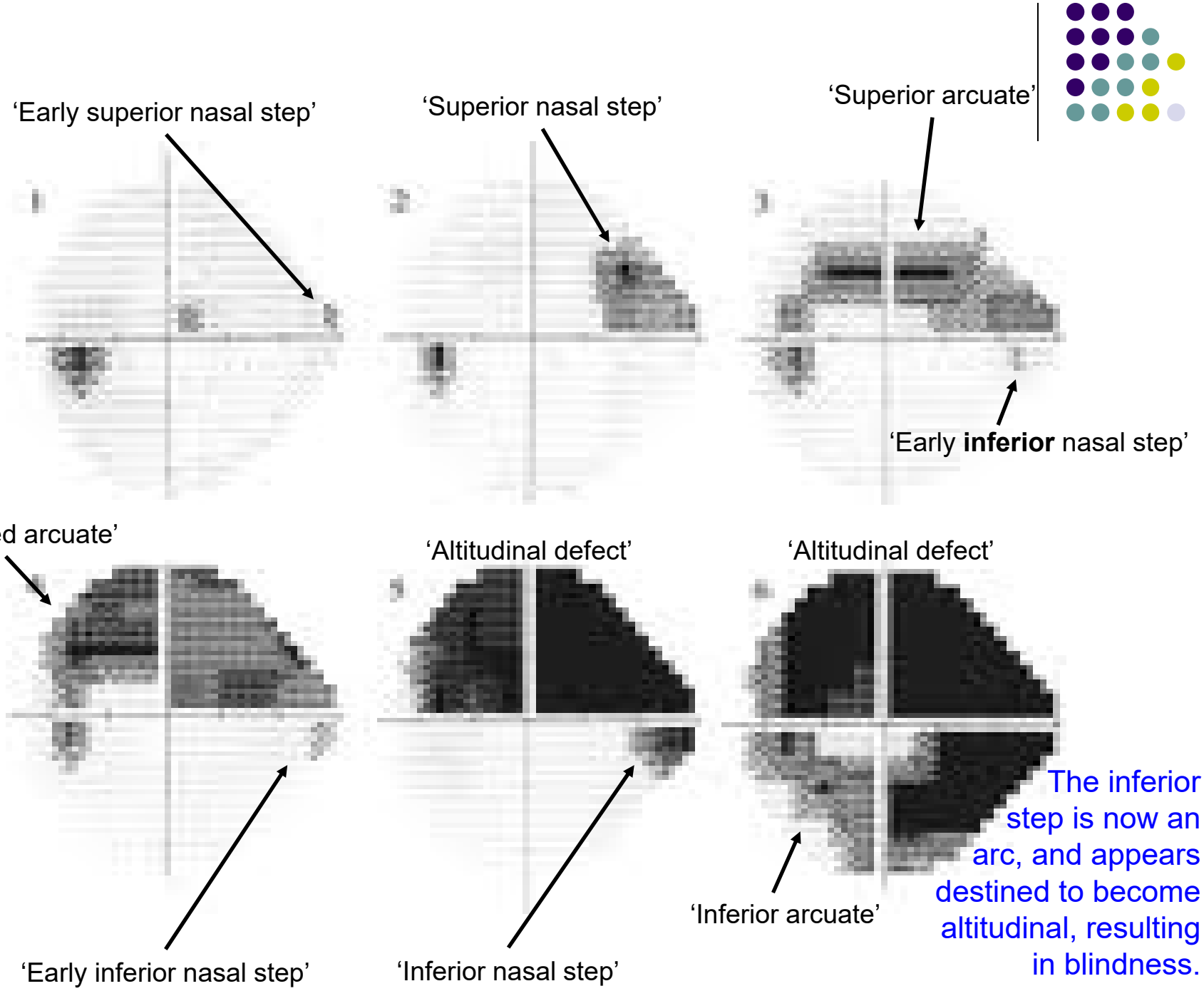


‘Early inferior nasal step’

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



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.


'Early superior nasal step'
'Superior nasal step'
'Superior arcuate'
'Early **inferior** nasal step'
'Advanced arcuate'
'Altitudinal defect'
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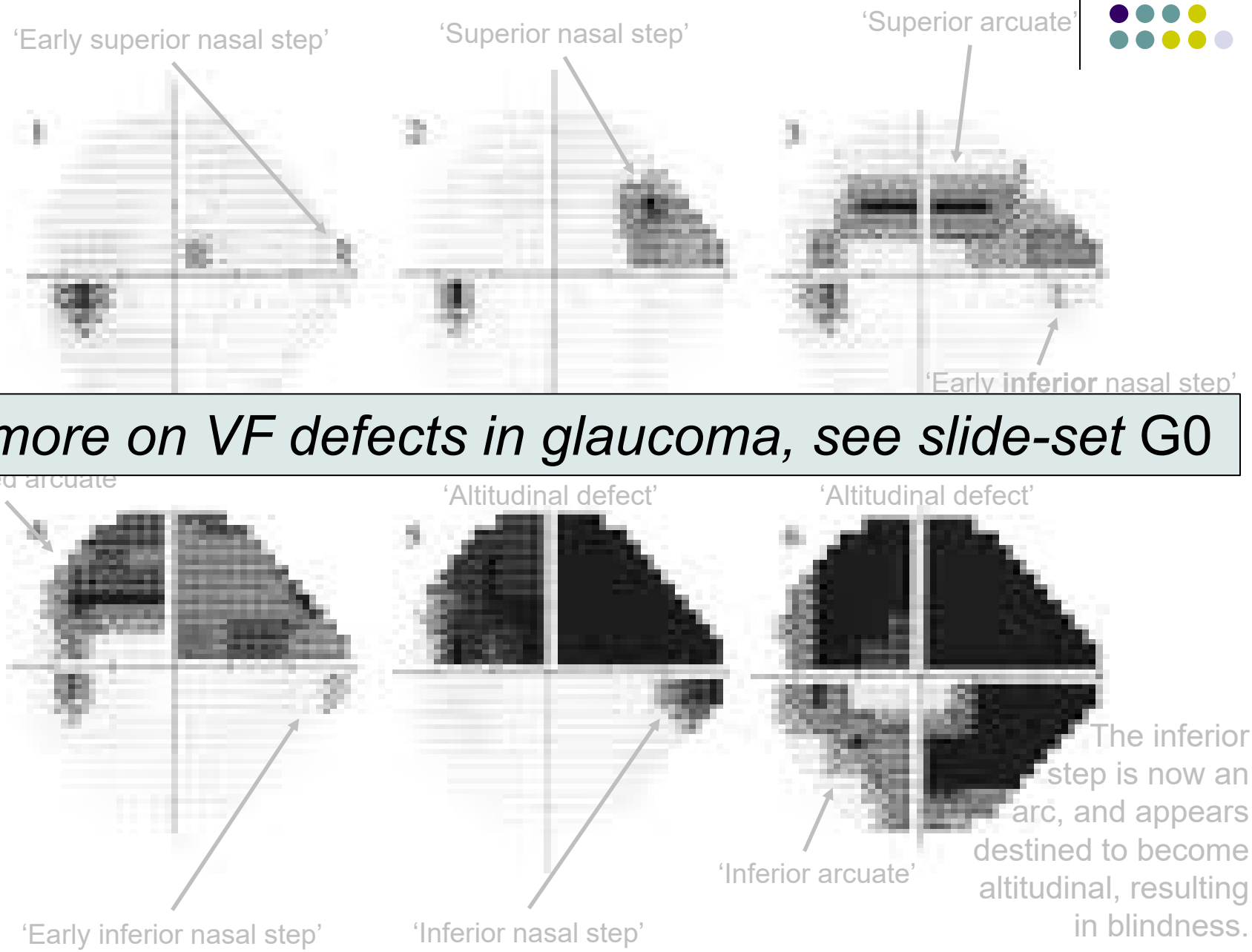
'Inferior nasal step'
'Inferior arcuate'
The inferior step is now an arc, and appears destined to become altitudinal, resulting in blindness.


'Early superior nasal step'
'Superior nasal step'
'Superior arcuate'
'Early inferior nasal step'
For more on VF defects in glaucoma, see slide-set G0
Advanced arcuate
'Altitudinal defect'
'Altitudinal defect'
'Early inferior nasal step'
'Inferior nasal step'
'Inferior arcuate'
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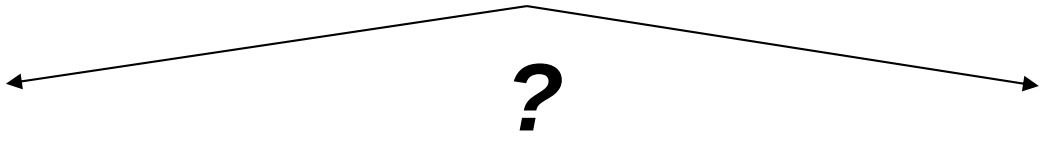
Q

Open-angle Glaucoma: *Primary*

Glaucoma

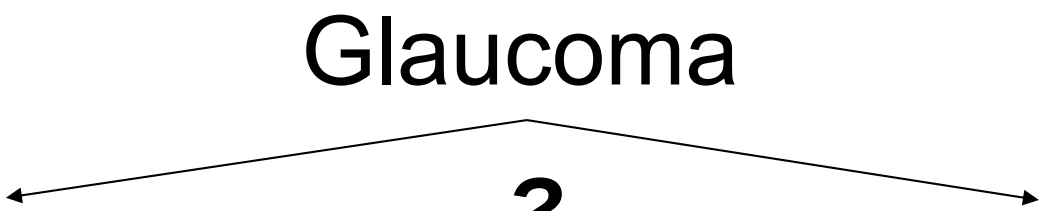


?

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

A

Open-angle Glaucoma: *Primary*



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

What is the status of the angle?

Q

Open-angle Glaucoma: *Primary*

Glaucoma

Open-angle

Closed- or *narrow-angle*

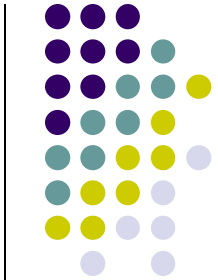
The first thought you should have when encountering a pt you suspect has glaucoma is…

What is the status of the angle?

How does one determine the status of the angle?

A

Open-angle Glaucoma: *Primary*



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?

How does one determine the status of the angle?

Gonioscopy. Don't assume your glaucoma pt has open angles—**prove** it by gonioing them!

Open-angle Glaucoma: *Primary*

Glaucoma

Open-angle

Closed-* or *narrow-angle

Angle-closure glaucoma *is covered in multiple slide-sets; see the* Table of Contents

How does one determine the status of the angle?

Gonioscopy. Don't assume your glaucoma pt has open angles—**prove** it by gonioing them!

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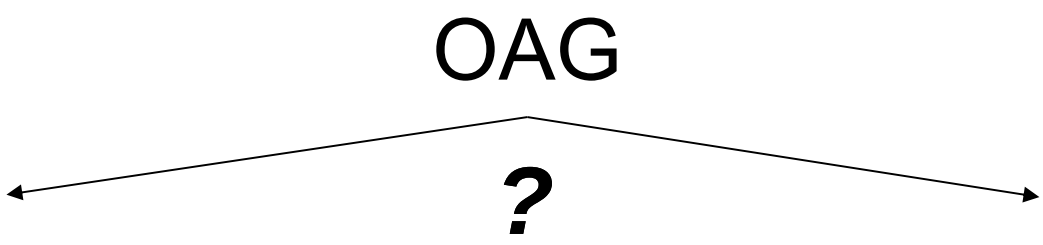
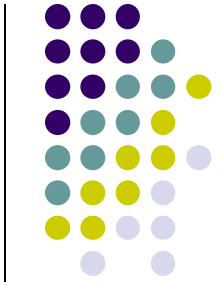
Open-angle Glaucoma: *Primary*

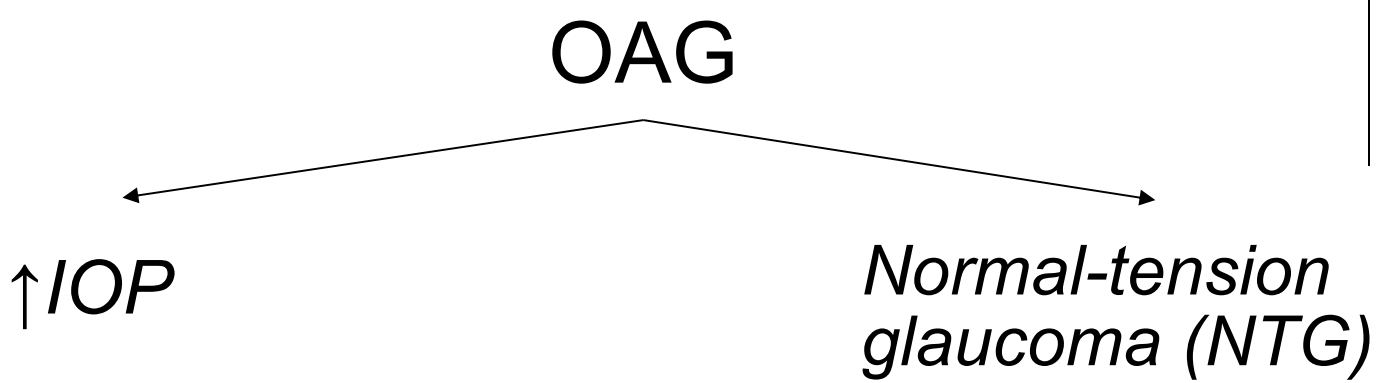




Once you have determined a pt has open-angle glaucoma,
the next ‘first thought’ is to ask…

A

Open-angle Glaucoma: *Primary*



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?

Q

Open-angle Glaucoma: *Primary*

OAG

↑*IOP*

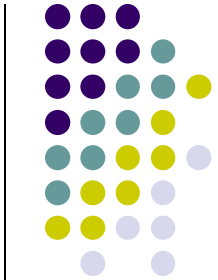
Untreated IOP consistently *above* # mmHg

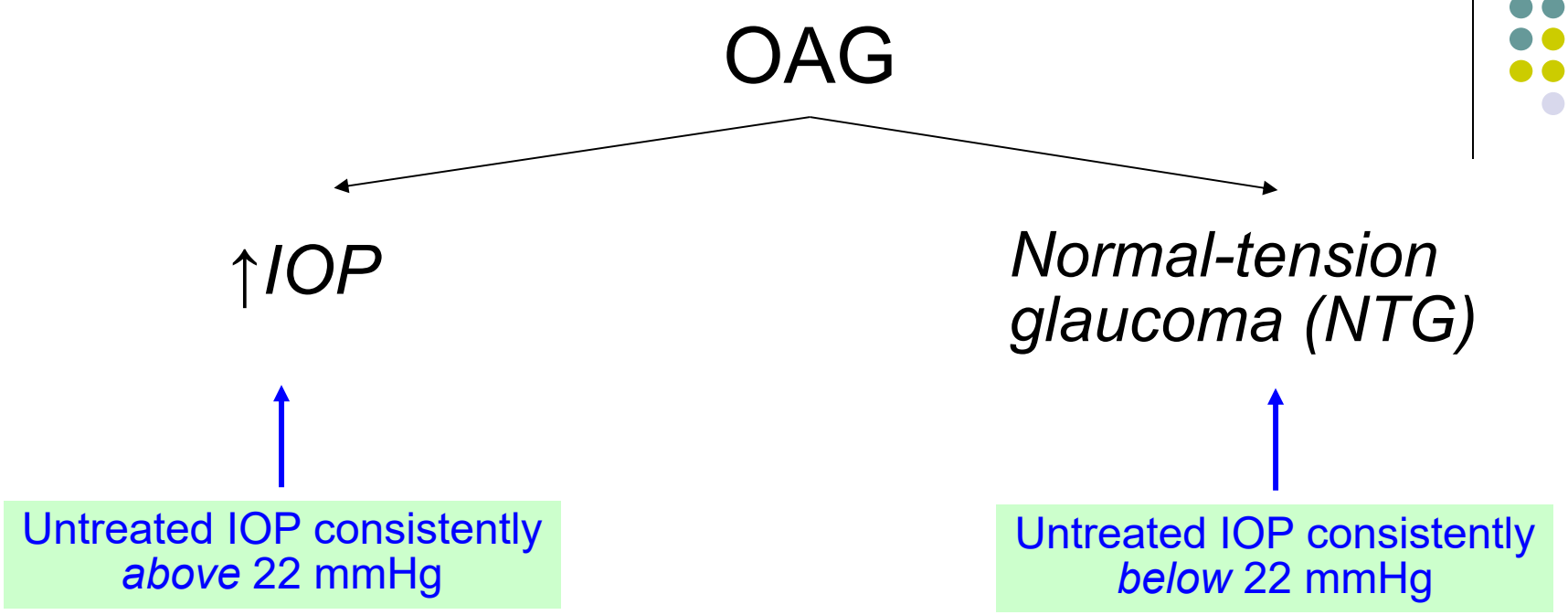
Normal-tension glaucoma (NTG)

Untreated IOP consistently *below* # mmHg

A

Open-angle Glaucoma: *Primary*





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

Open-angle Glaucoma: *Primary*

OAG

↑*IOP*

Normal-tension glaucoma (NTG)

Untreated IOP consistently *above* 22 mmHg

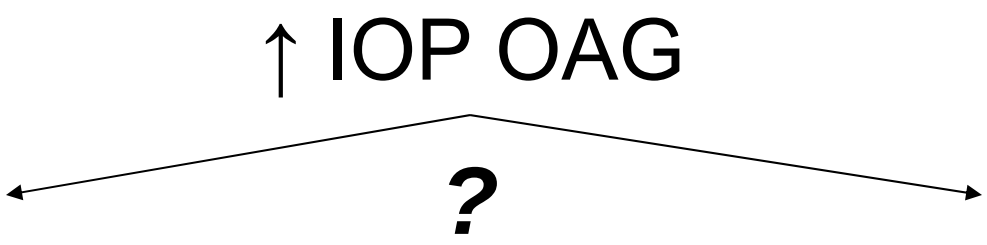
Untreated IOP consistently *below* 22 mmHg

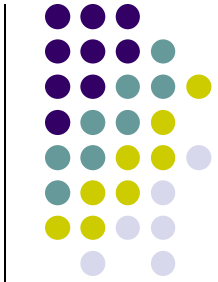
(Note that this di *comalogists contend NTG is*

Normal-tension glaucoma *is covered in its own slide-set (*G21*)*

Q

Open-angle Glaucoma: *Primary*

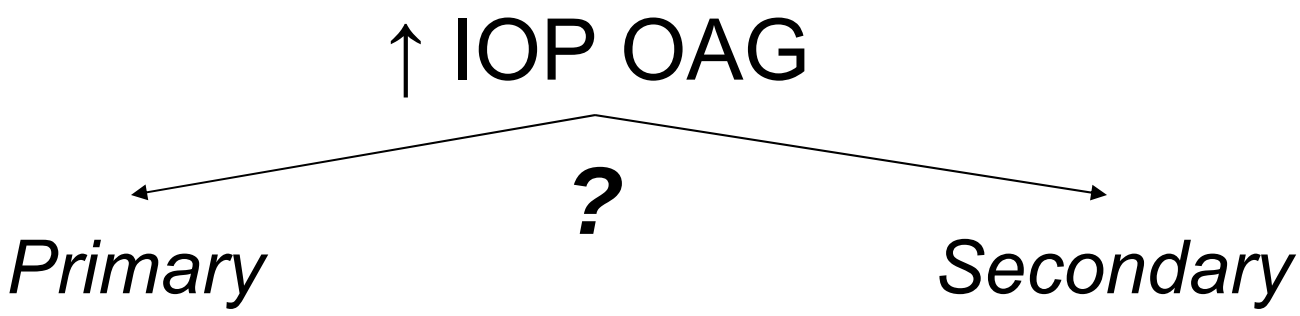




Once you have determined a pt has high-pressure open-angle glaucoma, the next 'first thought' is to ask…

A

Open-angle Glaucoma: *Primary*

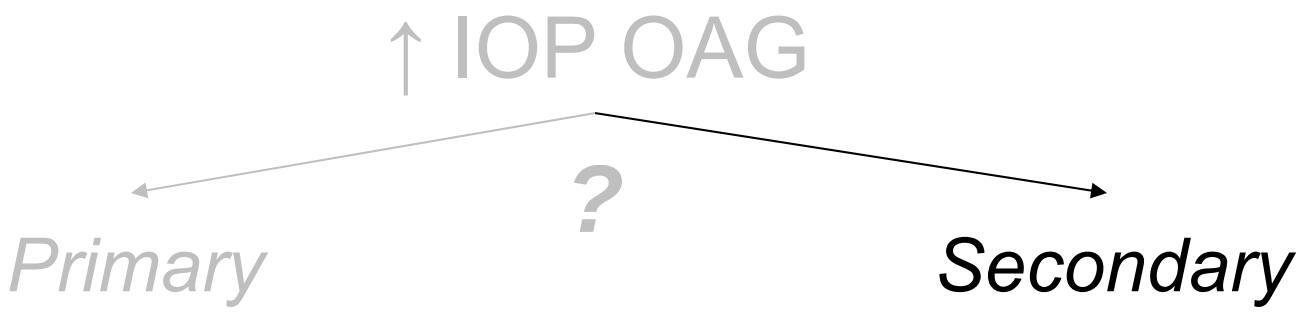


Once you have determined a pt has high-pressure open-angle glaucoma, the next 'first thought' is to ask…

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

Q

Open-angle Glaucoma: *Primary*



Once you have determined a pt has high-pressure open-angle glaucoma,
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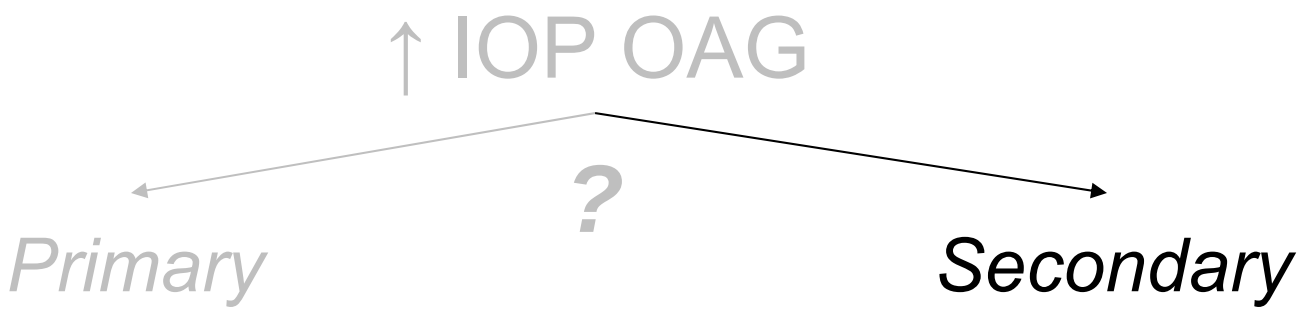
What does it mean to say a case of glaucoma is 'secondary'*?*

A

Open-angle Glaucoma: *Primary*



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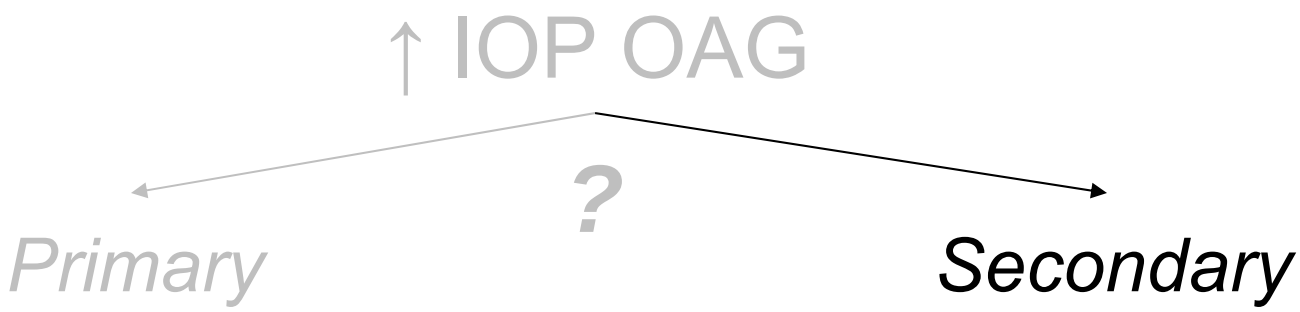
What does it mean to say a case of glaucoma is 'secondary'*?*
It means a specific factor causing the glaucoma has been identified

Q

Open-angle Glaucoma: *Primary*



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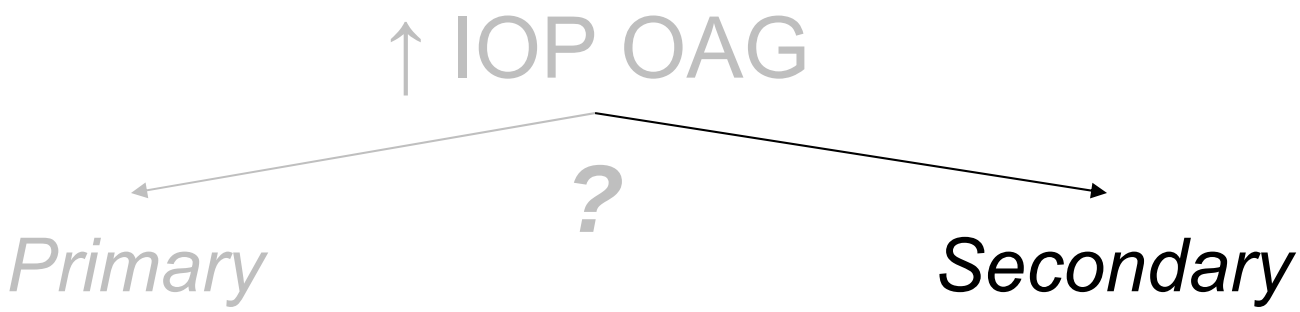
What are some of these specific factors?

Q

Open-angle Glaucoma: *Primary*



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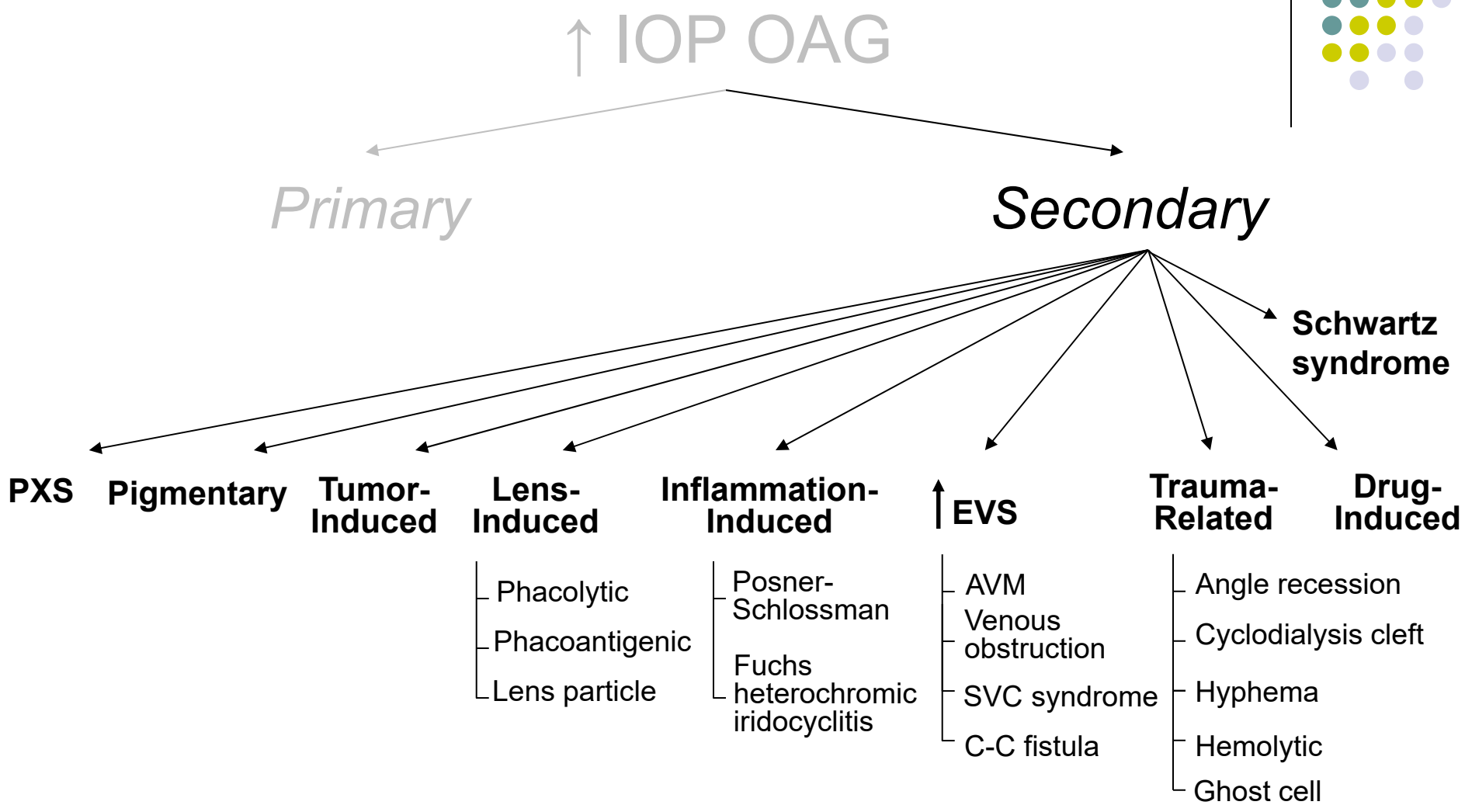
What does it mean to say a case of glaucoma is 'secondary'*?*
It means a specific factor causing the glaucoma has been identified

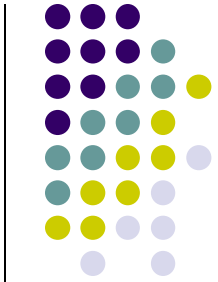
What are some of these specific factors?
Brace yourself…

Open-angle Glaucoma: *Primary*



(All are addressed in detail in other slide-sets—see the Table of Contents.*)*

Open-angle Glaucoma: *Primary*



↑ IOP OAG

Primary

Secondary

Note that primary open-angle glaucoma (POAG) is a diagnosis of exclusion—*it can only be made by first determining that the angle is open, and then ruling out the myriad causes of* ***secondary*** *OAG*

Schwartz syndrome

PXS

Pigmentary

Tumor-Induced

Lens-Induced

- Phacolytic
- Phacoantigenic
- Lens particle

Inflammation-Induced

- Posner-Schlossman
- Fuchs heterochromic iridocyclitis

PXS

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

Trauma-Related

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

Drug-Induced

Open-angle Glaucoma: *Primary*

Glaucoma

So, you see a pt with ONH and VF loss consistent with glaucomatous optic neuropathy. This can appropriately be referred to as ‘glaucoma.’

(No question—proceed when ready)

Open-angle Glaucoma: *Primary*

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Open-angle Glaucoma: *Primary*

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Likewise, before calling it *primary* open angle glaucoma (POAG), you must first consider and rule out the myriad causes of *secondary* OAG.

(No question—proceed when ready)

Open-angle Glaucoma: *Primary*

Primary Open-Angle Glaucoma (POAG)

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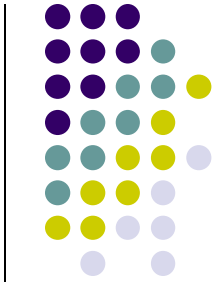
But before calling it *open-angle* glaucoma (OAG), you must first gonio the pt and determine affirmatively that the angle is open. Don't use the label **OA**G until you've done so!
Likewise, before calling it *primary* open angle glaucoma (POAG), you must first consider and rule out the myriad causes of *secondary* OAG. Don't use the label **P**OAG until you've done so!

(No question—proceed when ready)

Q

Open-angle Glaucoma: *Primary*



Besides IOP, what are the "important" (per the Glaucoma *book) risk factors for POAG development and/or progression?*

--?

--IOP

--?

--?

--?

--?

A

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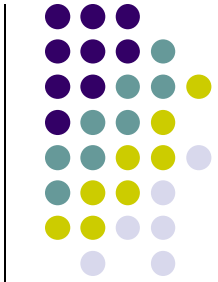

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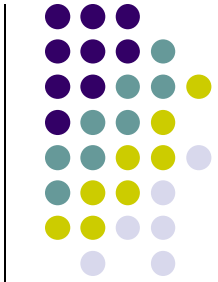

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What are the "other" (ditto) risk factors?

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--***?***

A

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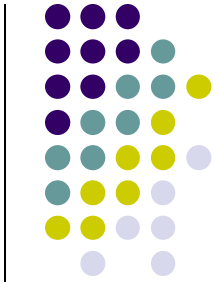

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--Low cerebrospinal fluid (CSF) pressure
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Q

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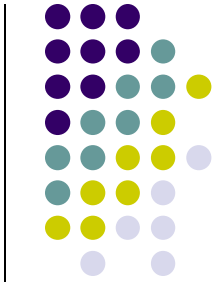

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With regards to race, who is at higher risk for POAG in the US?

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Q/A

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With regards to race, who is at higher risk for POAG in the US?

Individuals of [redacted] and [redacted] heritage are at a 4x greater risk than are [redacted]

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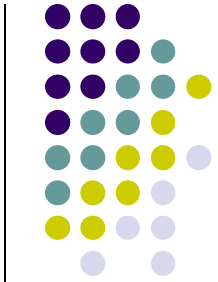

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With regards to race, who is at higher risk for POAG in the US?
Individuals of black and Hispanic heritage are at a 4x greater risk than are whites

What are the "other" (ditto) risk factors?
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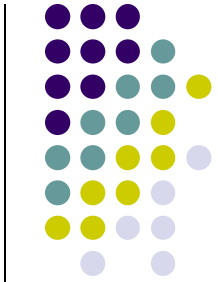

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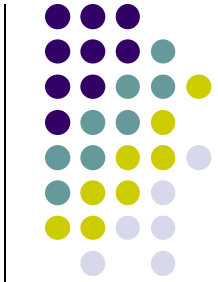

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What about the risk of going blind from POAG?
These same folk are at a 4x higher risk of that as well

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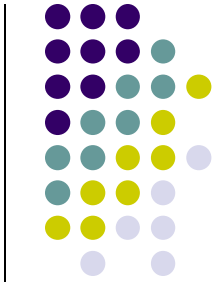

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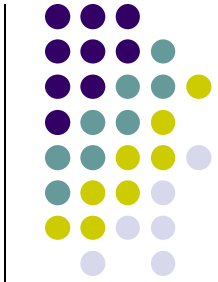

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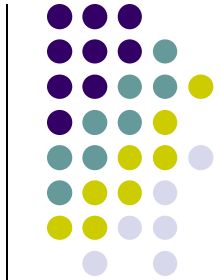

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That's why glaucoma treatment turns on IOP-lowering maneuvers!

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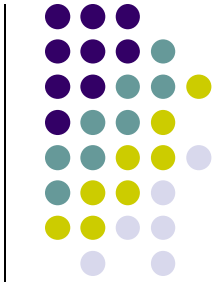

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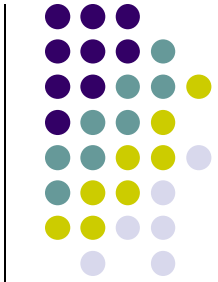

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For what racial group is age a particularly impactful risk factor?

Q/A

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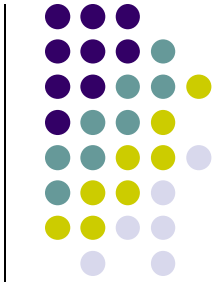

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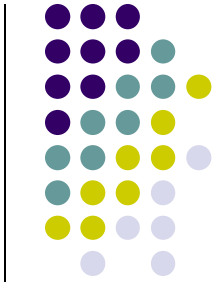

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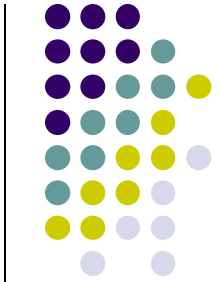

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Q

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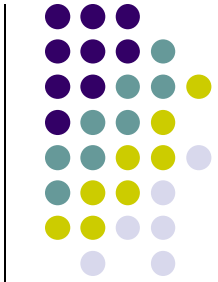

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How might this work physiologically?

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No—thin CCT is a risk factor even after accounting for its effect on IOP measurement, ie, it's an independent risk factor

How might this work physiologically?
No one knows for certain, but it might be that thinness of the CCT reflects structural characteristics of the eyewall that make the ONH vulnerable to glaucomatous damage

Q

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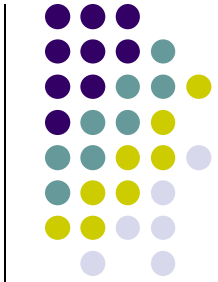

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Speaking of structural characteristics of the eyewall that make the ONH vulnerable to glaucomatous damage…What does the term hysteresis *refer to?*

--Low ocular perfusion pressure (OPP)

--Low cerebrospinal fluid (CSF) pressure

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It refers to the fact that changes in the physical property of a structure may lag behind changes in the forces that determine it.

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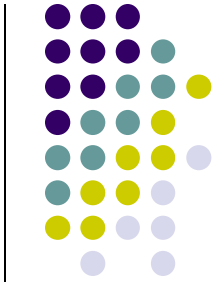

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Wiggity what? Can you unpack that with respect to the cornea, please?

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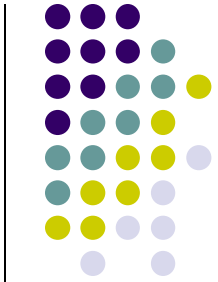

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Sure—flip to the next slide…

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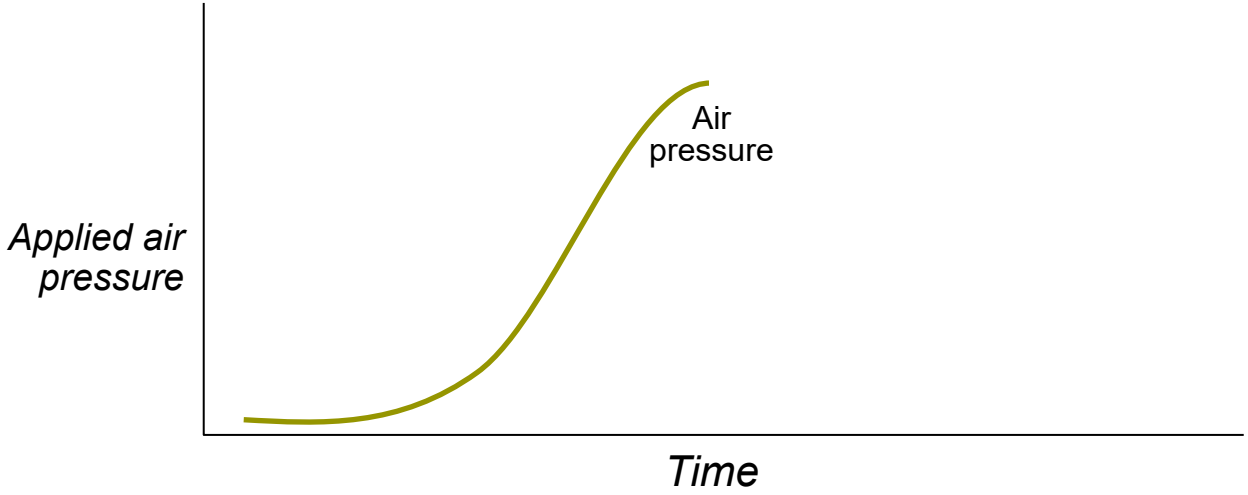

Consider: A column of air is directed at the cornea, and its pressure is ramped up over time. As the pressure increases, it causes the cornea to flatten more and more (not depicted on graph).

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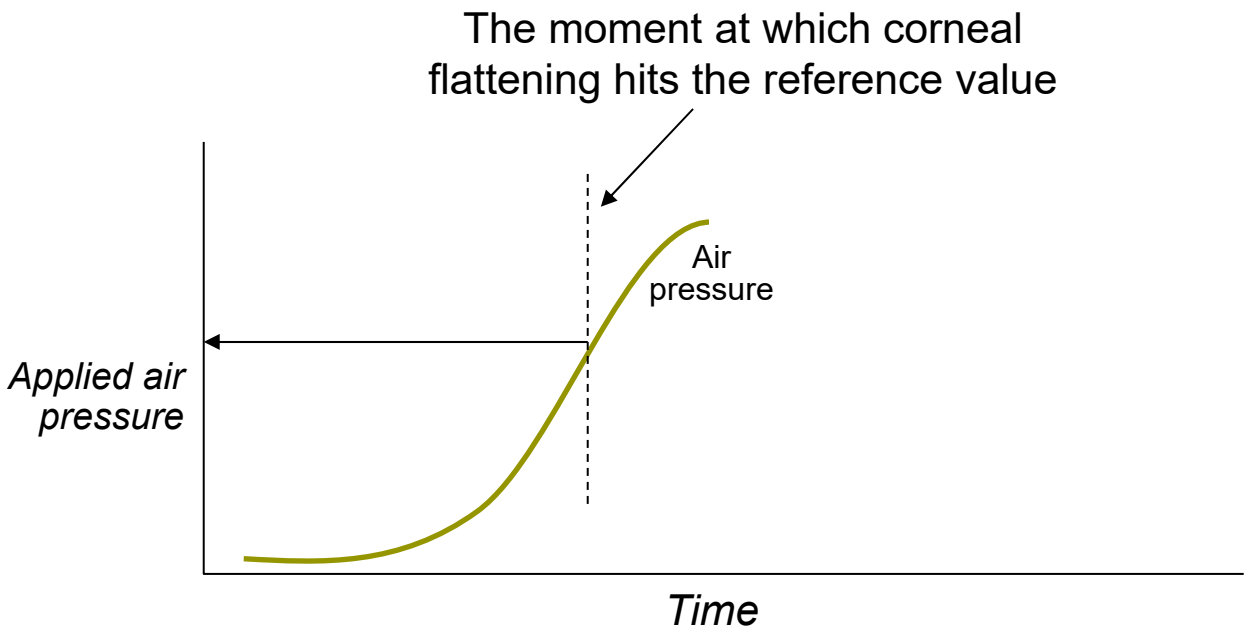

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At some point, corneal flattening reaches a predetermined reference value. The amount of air pressure required to produce this level of flattening is noted.

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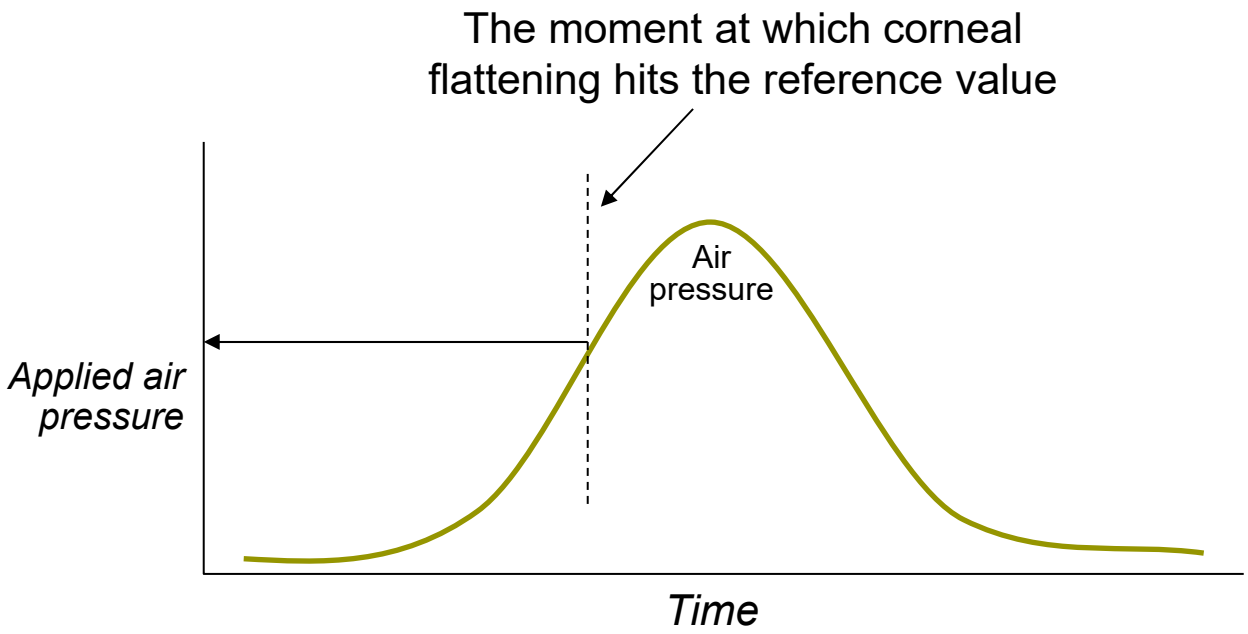
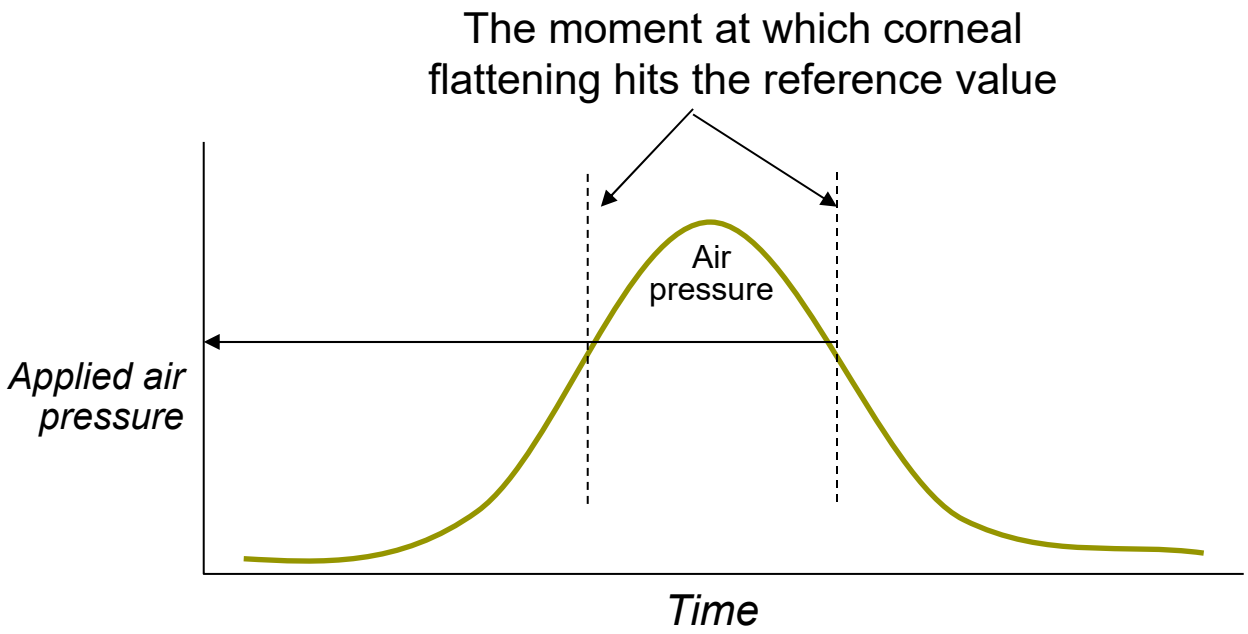


Consider: A column of air is directed at the cornea, and its pressure is ramped up over time. As the pressure increases, it causes the cornea to flatten more and more (not depicted on graph).
At some point, corneal flattening reaches a predetermined reference value. The amount of air pressure required to produce this level of flattening.is noted.
Now the amount of air pressure is ramping back down. As the pressure drops, the cornea will proceed to round back out to its normal shape (again, not depicted on graph).

Open-angle Glaucoma: *Primary*



Consider: A column of air is directed at the cornea, and its pressure is ramped up over time. As the pressure increases, it causes the cornea to flatten more and more (not depicted on graph).
At some point, corneal flattening reaches a predetermined reference value. The amount of air pressure required to produce this level of flattening.is noted.
Now the amount of air pressure is ramping back down. As the pressure drops, the cornea will proceed to round back out to its normal shape (again, not depicted on graph).
If the cornea was perfectly elastic, it would reach the reference level of flattening on the way ‘out’ at the same air-pressure level that produced it on the way ‘in.’

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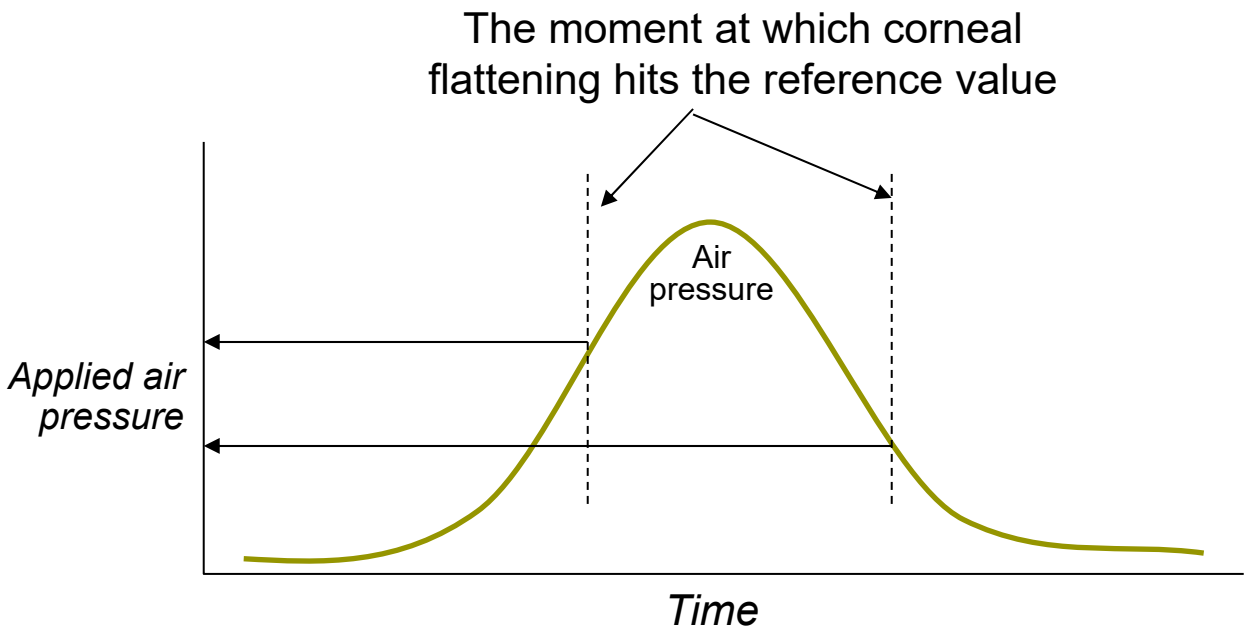

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But that's not what happens. Instead, the cornea 'lags' behind, and doesn't achieve the reference level of flattening until the air pressure has dropped *past* that which was required to produce it while the air pressure was ramping up.

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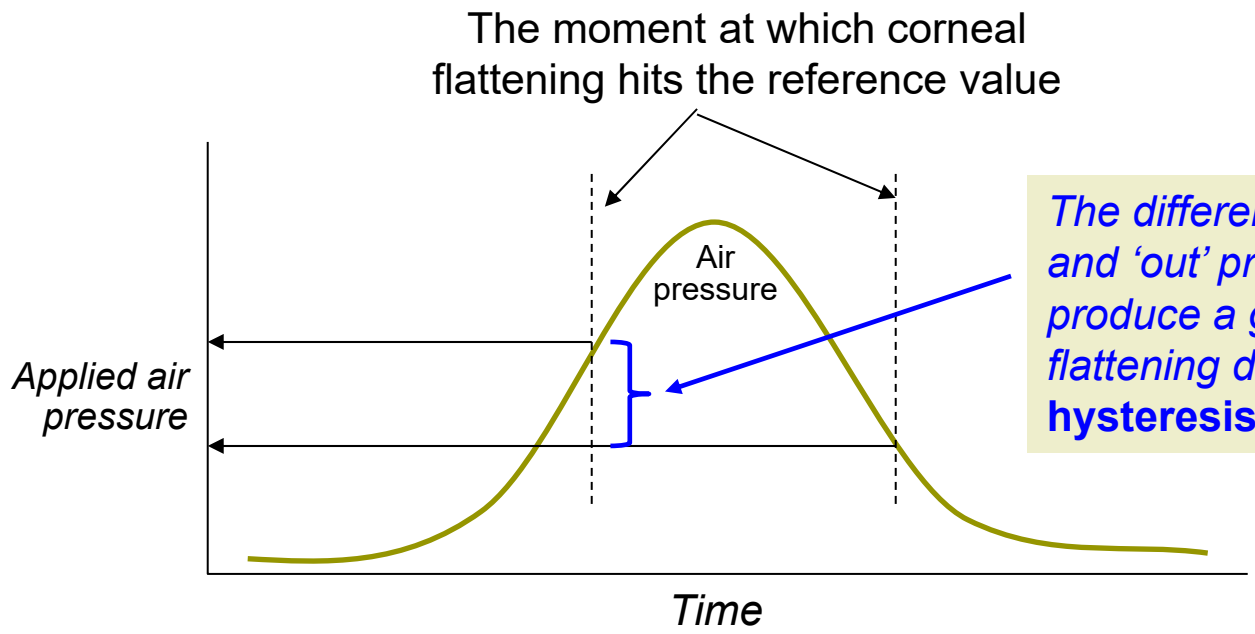

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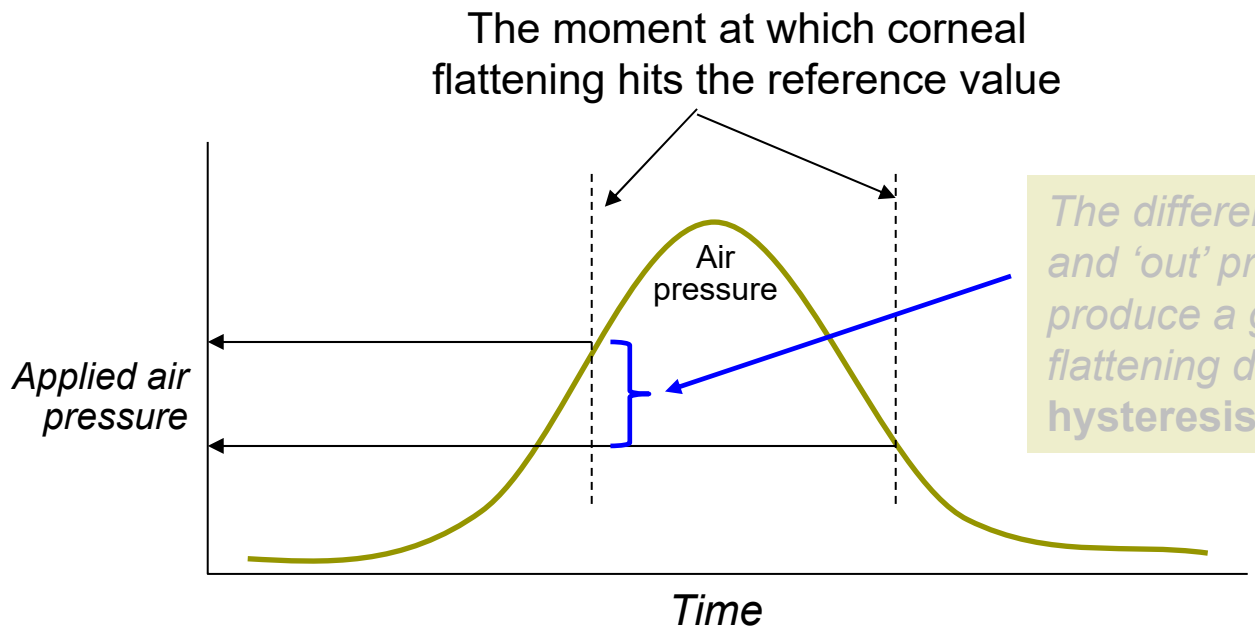

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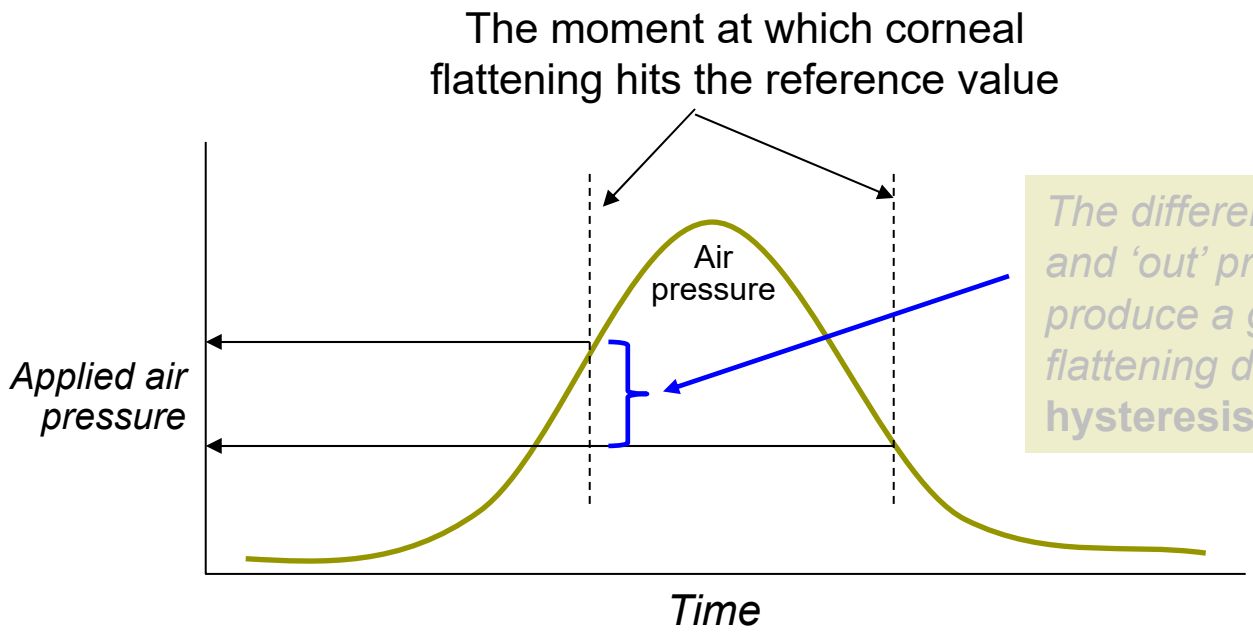

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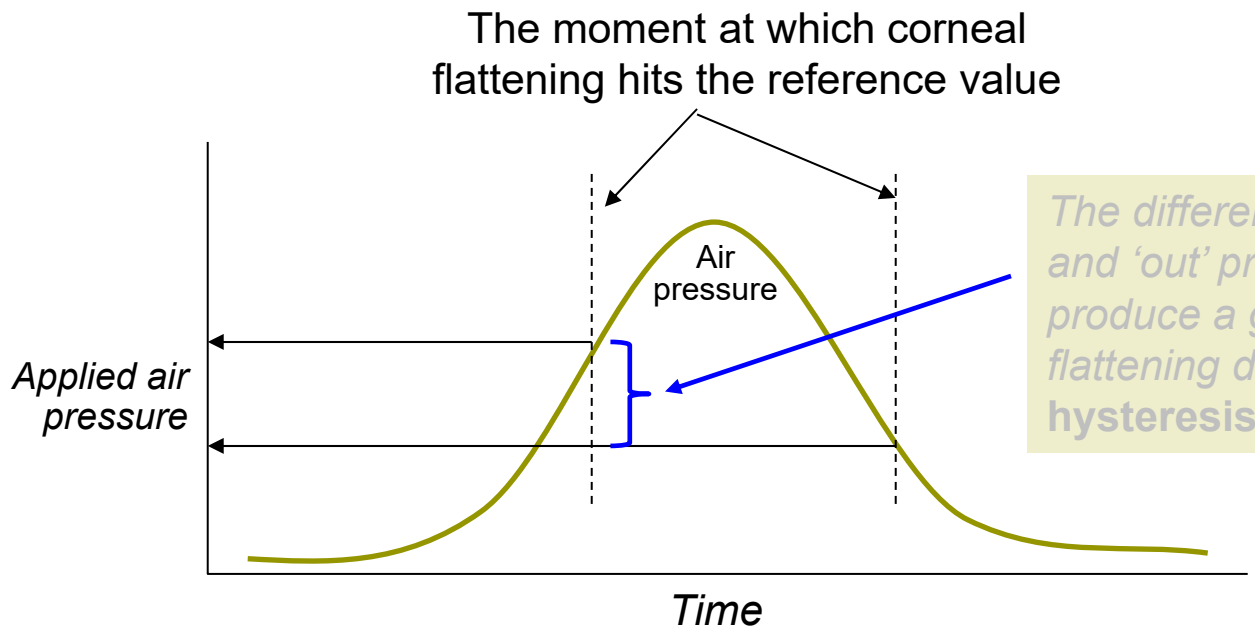

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OK, but how does low corneal hysteresis increase glaucoma risk? As with thin CCT, no one knows for certain. But also like thin CCT, the assumption is low hysteresis reflects structural properties of the eyewall that render the ONH vulnerable to glaucomatous damage.

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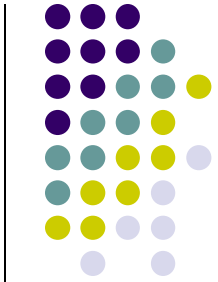

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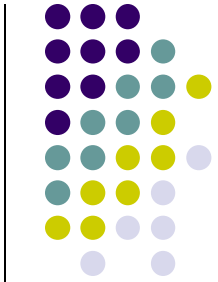

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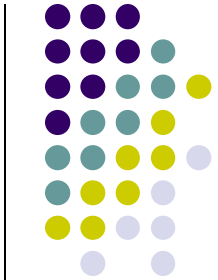

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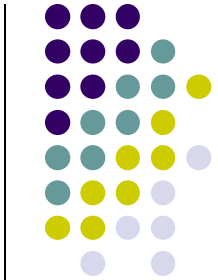

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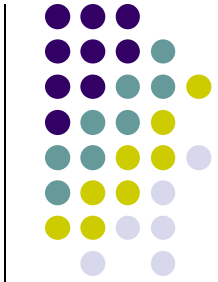

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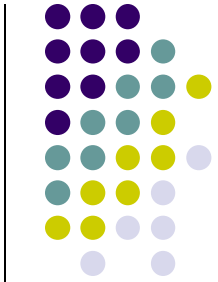

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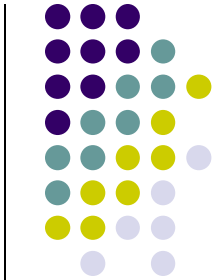

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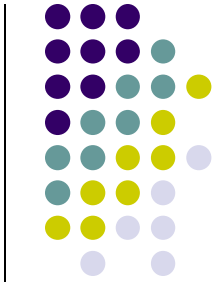

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What may occur is that the increased OPP associated with HTN conveys a reduced risk of POAG until the deleterious vascular effects of HTN (ie, atherosclerosis and other changes) damages the microcirculation of the ONH to the extent that the deleterious effect of these changes outweighs the advantage conveyed by increased OPP

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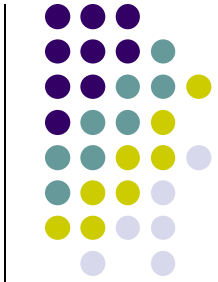

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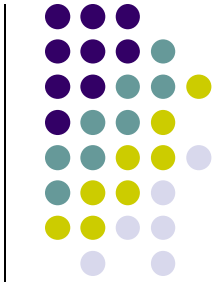

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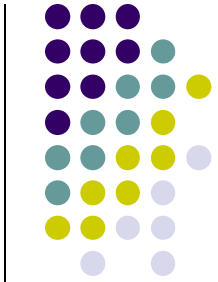

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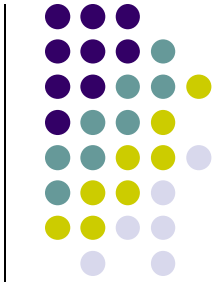

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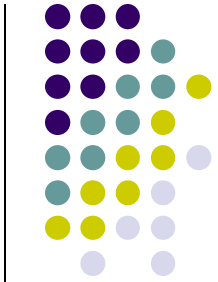

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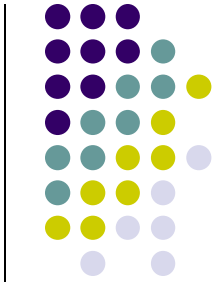

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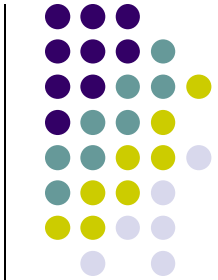
Q

Open-angle Glaucoma: *Primary*

Where does POAG rank worldwide as a cause of blindness?



Q/A

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Q

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Q/A

Open-angle Glaucoma: *Primary*

Where does POAG rank worldwide as a cause of blindness?
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How prevalent is POAG in the US?
Very. Almost % of the over-40 US population– # people—have POAG.

A

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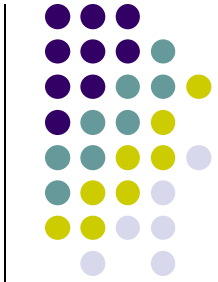

Where does POAG rank worldwide as a cause of blindness?
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How prevalent is POAG in the US?
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

Q

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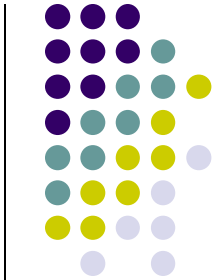

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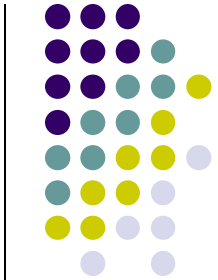

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Because of its stealthy nature. It is insidious in onset, and progresses very slowly. It causes no discomfort. Further, visual acuity isn't affected until late in the dz process, often when the pt is already functionally blind from field loss.

A

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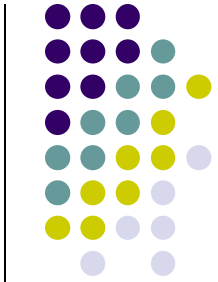

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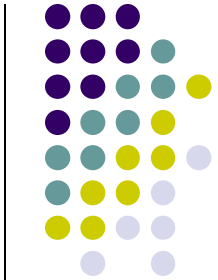
Open-angle Glaucoma: *Primary*



Where does POAG rank worldwide as a cause of blindness?
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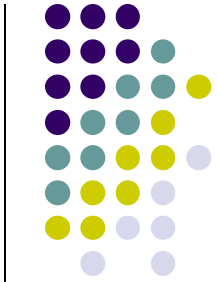

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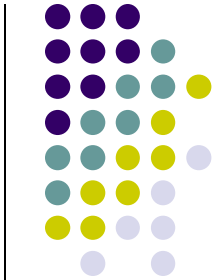

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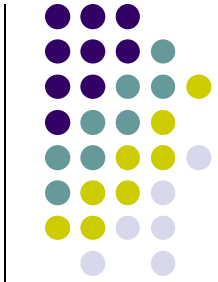

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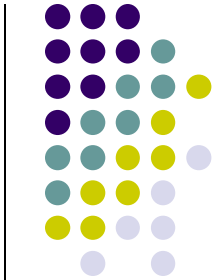

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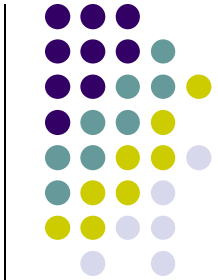

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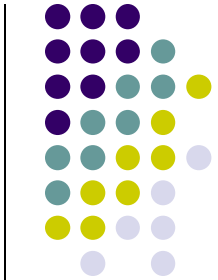

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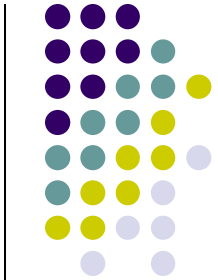

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--Topical meds
--Laser surgery
--Incisional surgery

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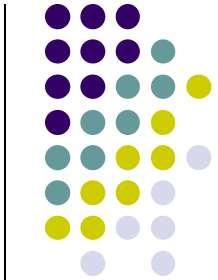

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How

For details on glaucoma clinical trials, see slide-set G19

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