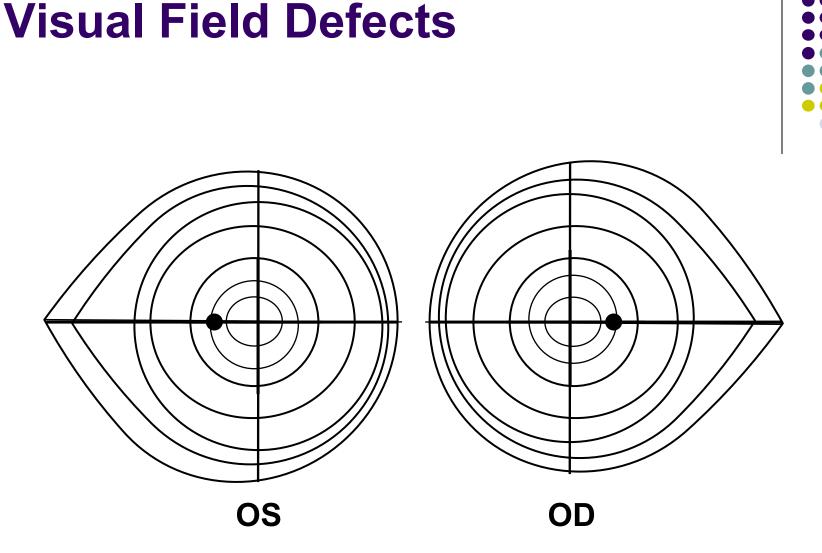
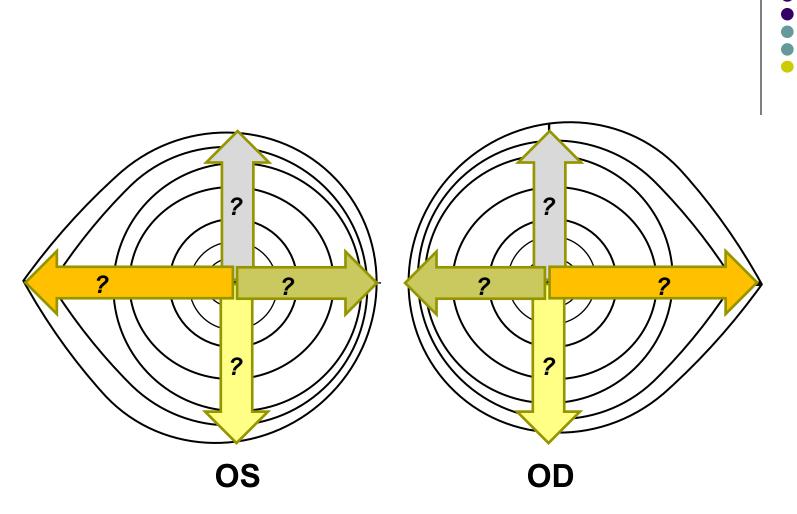


1

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

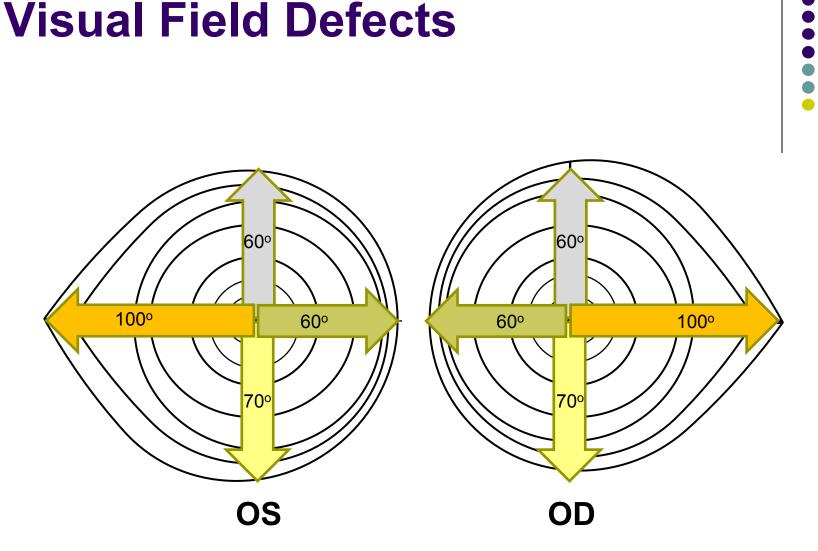


Here is a representation of the VF for each eye. Which is OD, and which OS? Remember, VFs are **not** drawn as if the pt is looking at you; they're drawn as if **you** are the pt!



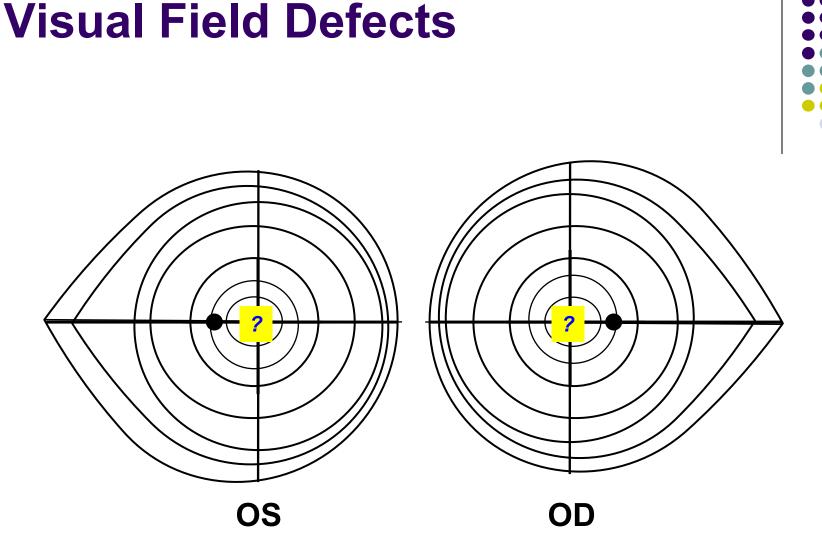
3

Measured in degrees from fixation, how far does the normal VF extend superiorly, inferiorly, nasally and temporally?

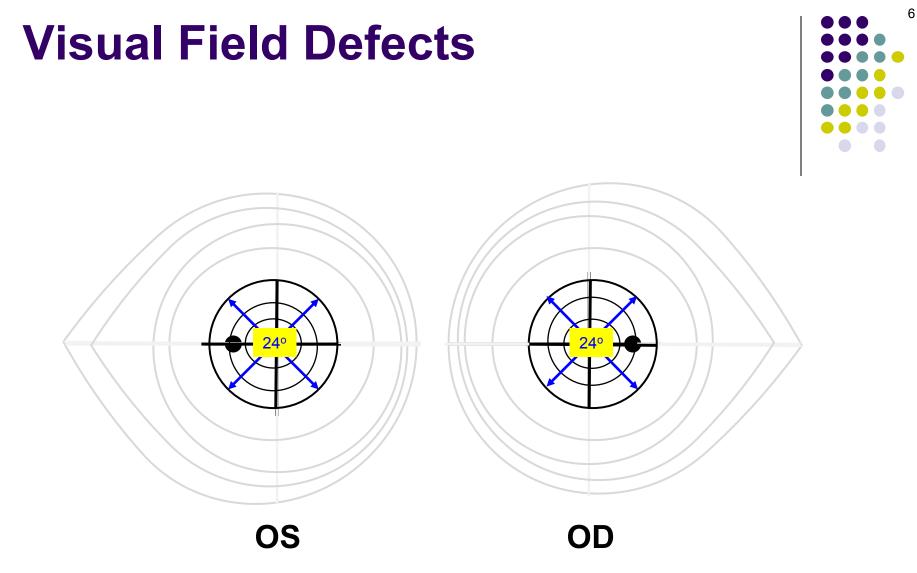


4

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.)

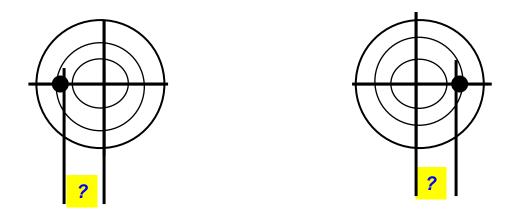


Measured in degrees from fixation, how much of the VF is assessed via the automated perimetry machines found in most ophthalmology practices?



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**



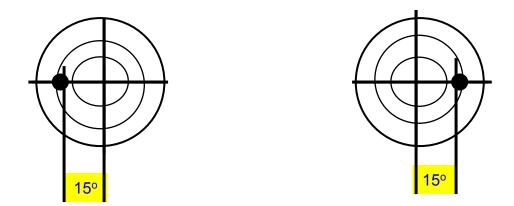


OS

OD

How far in degrees from fixation is the blind spot?





OS

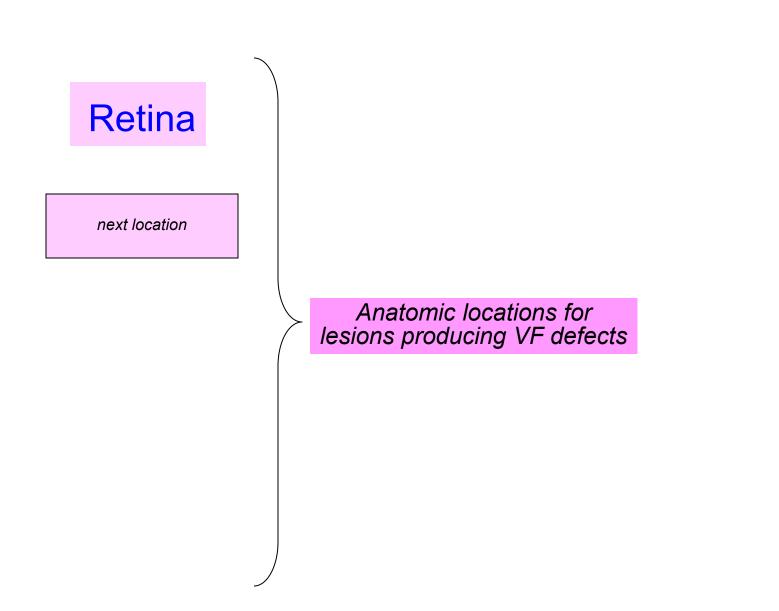
OD

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

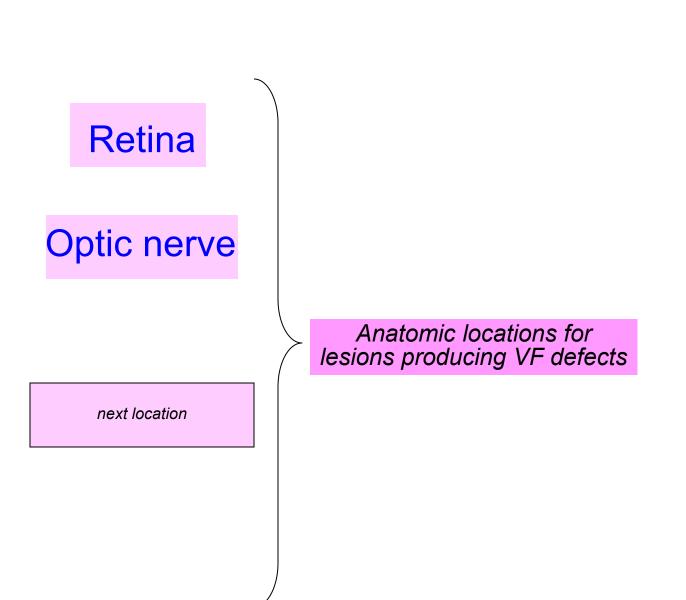


most anterior location

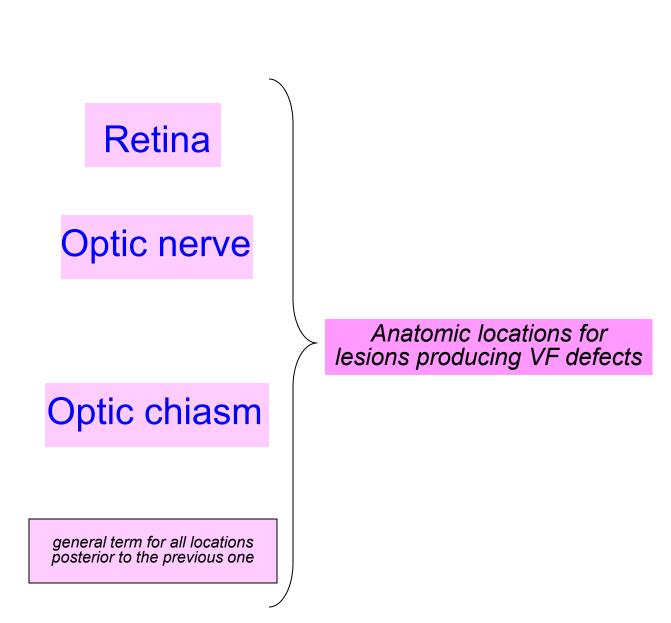
Anatomic locations for lesions producing VF defects



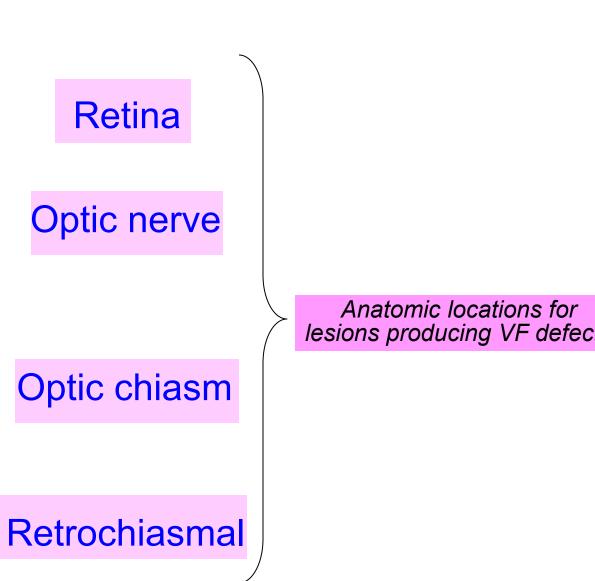






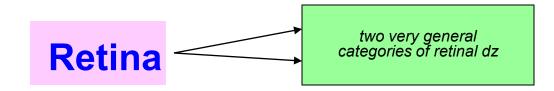








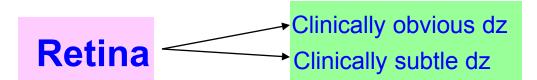
lesions producing VF defects



Optic nerve

Optic chiasm





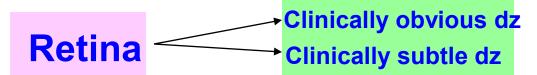


Optic chiasm

Retrochiasmal

15





What is meant by clinically obvious vs clinically subtle retinal dz?

Optic nerve

Optic chiasm





What is meant by clinically obvious vs clinically subtle retinal dz? In clinically obvious disease, the retina will appear abnormal on DFE, whereas in clinically subtle disease it will look normal

Optic chiasm

Optic nerve





Optic nerve

What is meant by clinically obvious vs clinically subtle retinal dz? In clinically obvious disease, the retina will appear abnormal on DFE, whereas in clinically subtle disease it will look normal

What is an example of... ...clinically obvious disease?

Optic chiasm





Optic nerve

What is meant by clinically obvious vs clinically subtle retinal dz? In clinically obvious disease, the retina will appear abnormal on DFE, whereas in clinically subtle disease it will look normal

What is an example of... ...clinically obvious disease? 'Typical' retinitis pigmentosa

Optic chiasm





What is meant by clinically obvious vs clinically subtle retinal dz? In clinically obvious disease, the retina will appear abnormal on DFE, whereas in clinically subtle disease it will look normal

What is an example of... ...clinically obvious disease? 'Typical' retinitis pigmentosa ---clinically subtle disease?

Optic chiasm

Optic nerve



Retina Clinically obvious dz (eg...RP)

What is meant by clinically obvious vs clinically subtle retinal dz? In clinically obvious disease, the retina will appear abnormal on DFE, whereas in clinically subtle disease it will look normal

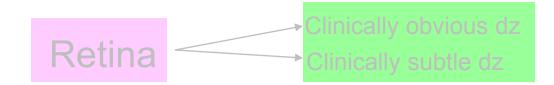
What is an example of...

...clinically obvious disease? 'Typical' retinitis pigmentosa ---clinically subtle disease? Cancer-associated retinopathy

Optic chiasm

Optic nerve





Optic nerve

Let's take a brief aside to cover optic nerve fundamentals before we address optic nerve VF defects

Optic chiasm



The optic nerves are composed of what?



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



	e optic nerv			
(The	e axons of	retinal	ganglion	cells)

How many fibers (axons) comprise an optic nerve?



The optic nerves	are composed of what?
The axons of ret	inal ganglion cells

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

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



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

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



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

Do they synapse in the region of the optic nerve head? No



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

Do they synapse in the region of the optic nerve head? No

Where will they synapse?



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

Do they synapse in the region of the optic nerve head? No

Where will they synapse? Most will synapse in the lateral geniculate nucleus (LGN)



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

Do they synapse in the region of the optic nerve head? No

Where will they synapse? **Most** will synapse in the lateral geniculate nucleus (LGN)

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



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

Do they synapse in the region of the optic nerve head? No

Where will they synapse? **Most** will synapse in the lateral geniculate nucleus (LGN)

Most? Where will the others synapse, and what are they responsible for? Most of the others are involved in the pupillary light reflex; they peel off just prior to reaching the LGN, heading instead to the pretectum of the dorsal midbrain to synapse in the pretectal nuclei



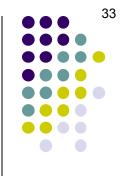
The optic nerves are composed of what? The axons of retinal ganglion cells

Do they synapse in the region of the optic nerve head? No

Where will they synapse? Most will synapse in the lateral geniculate nucleus (LGN)

Most? Where will the others synapse, and what are they responsible for? Most of the others are involved in the pupillary light reflex; they peel off just prior to reaching the LGN, heading instead to the pretectum of the dorsal midbrain to synapse in the pretectal nuclei

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



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

Do they synapse in the region of the optic nerve head? No

Where will they synapse? Most will synapse in the lateral geniculate nucleus (LGN)

Most? Where will the others synapse, and what are they responsible for? Most of the others are involved in the pupillary light reflex; they peel off just prior to reaching the LGN, heading instead to the pretectum of the dorsal midbrain to synapse in the pretectal nuclei

'Most'? Where will the others synapse, and what are they responsible for? The hypothalamus, where they are involved in modulating circadian responses



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

Do they synapse in the region of the optic nerve head? No

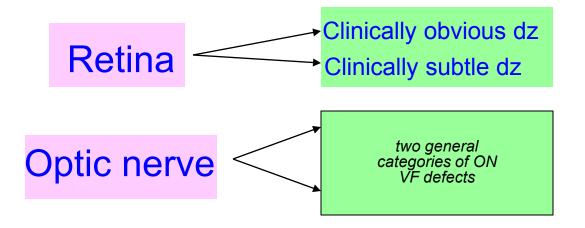
Where will they synapse? Most will synapse in the lateral geniculate nucleus (LGN)

For a more in-depth look at the optic nerve, see slide-set FELT6

Most? Where will the others synapse, and what are they responsible for? Most of the others are involved in the pupillary light reflex; they peel off just prior to reaching the LGN, heading instead to the pretectum of the dorsal midbrain to synapse in the pretectal nuclei

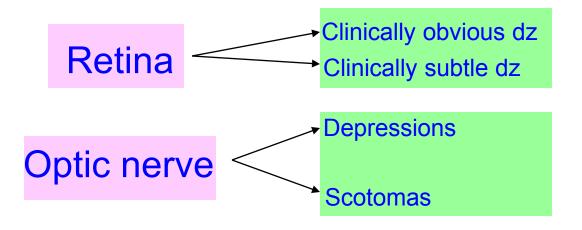
'Most'? Where will the others synapse, and what are they responsible for? The hypothalamus, where they are involved in modulating circadian responses





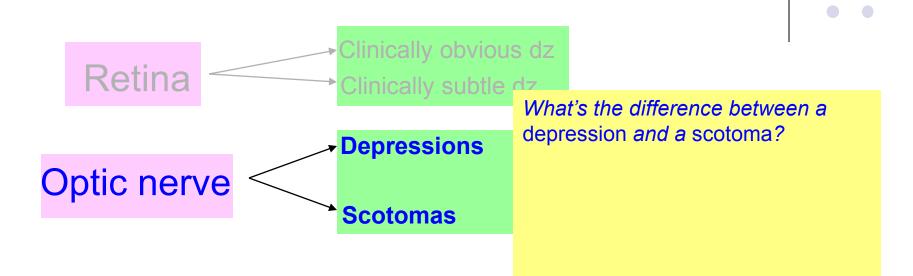


Optic chiasm





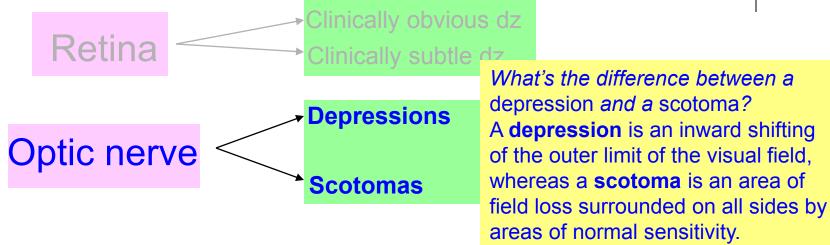
Optic chiasm



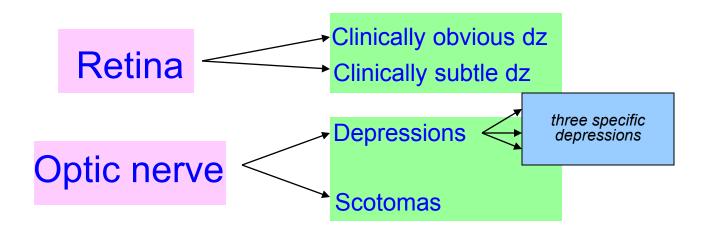






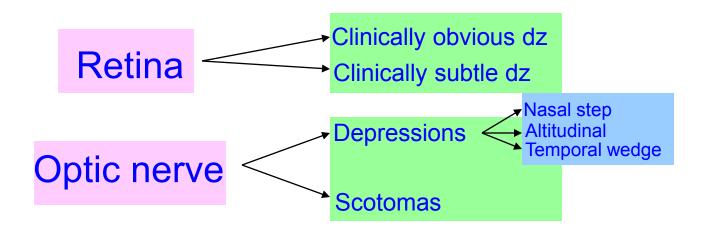


Optic chiasm



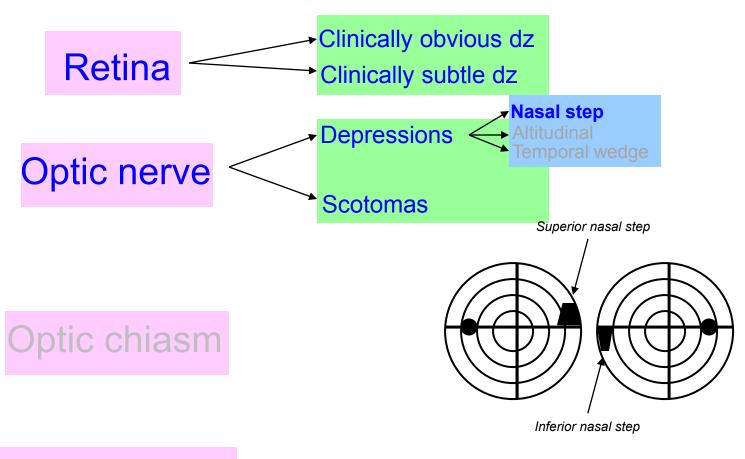


Optic chiasm





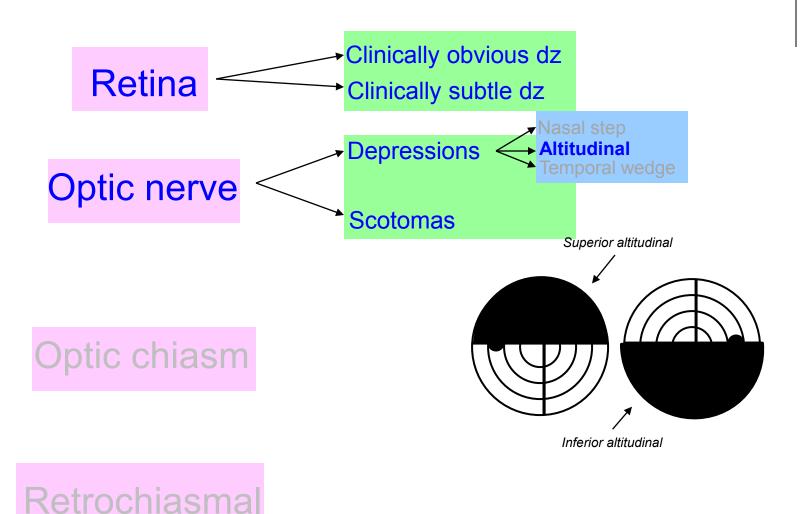
Optic chiasm



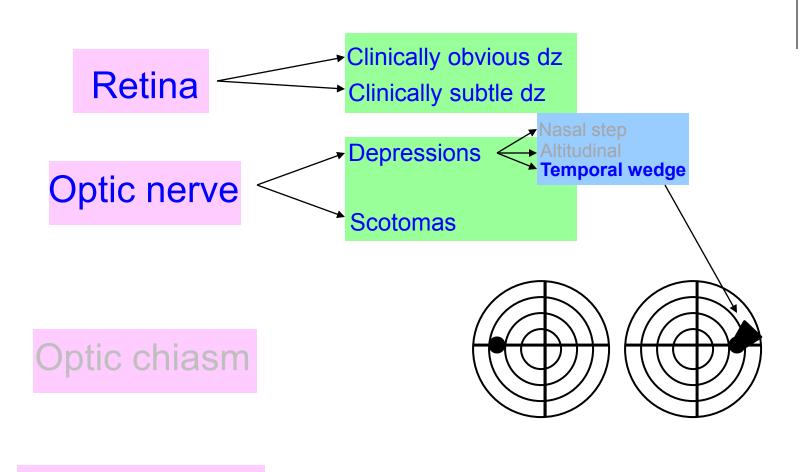


Retrochiasmal

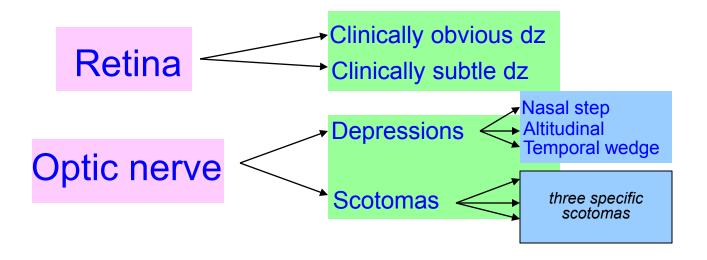
42







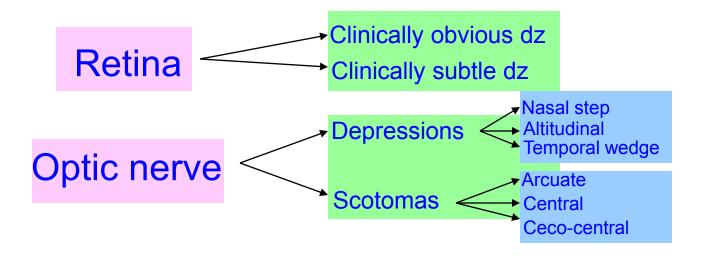








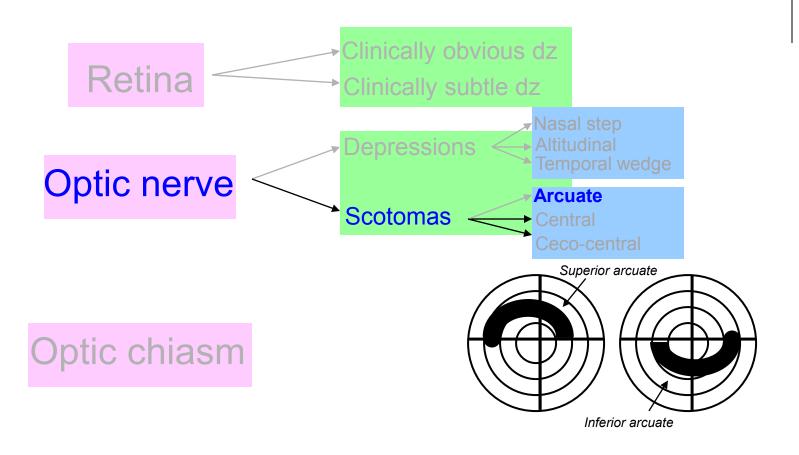




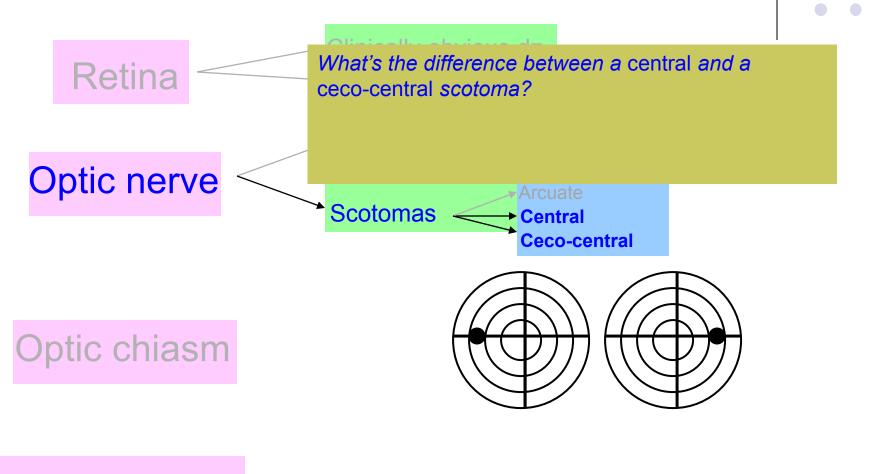




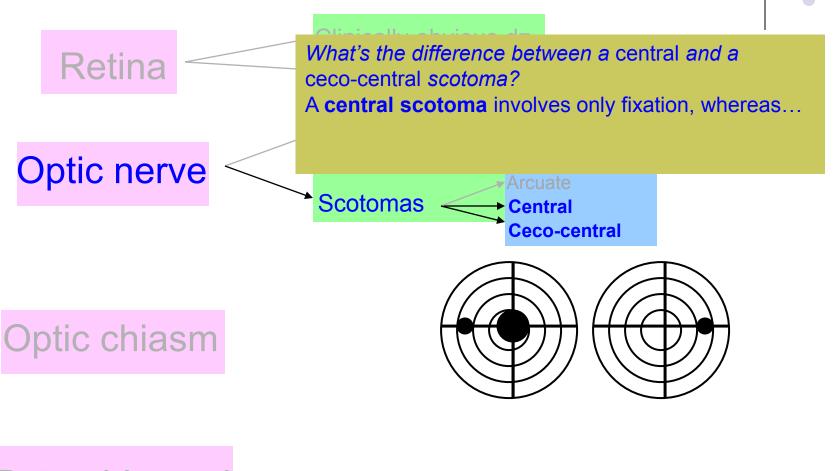


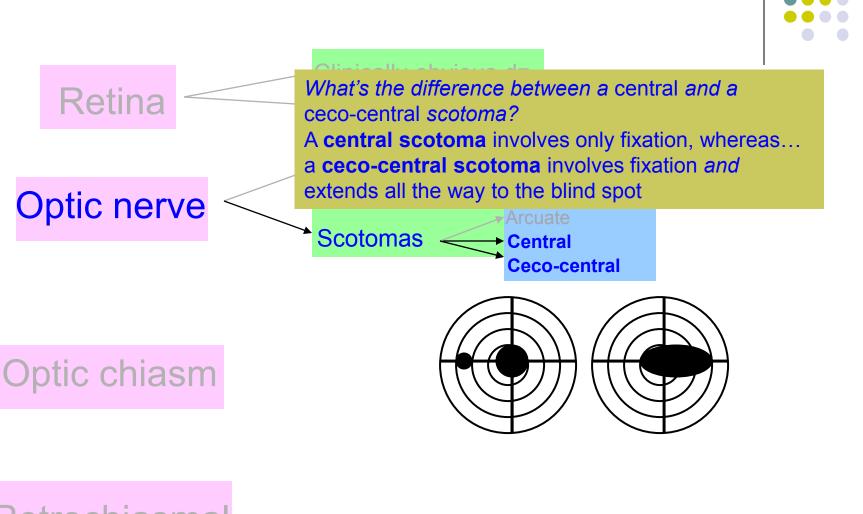






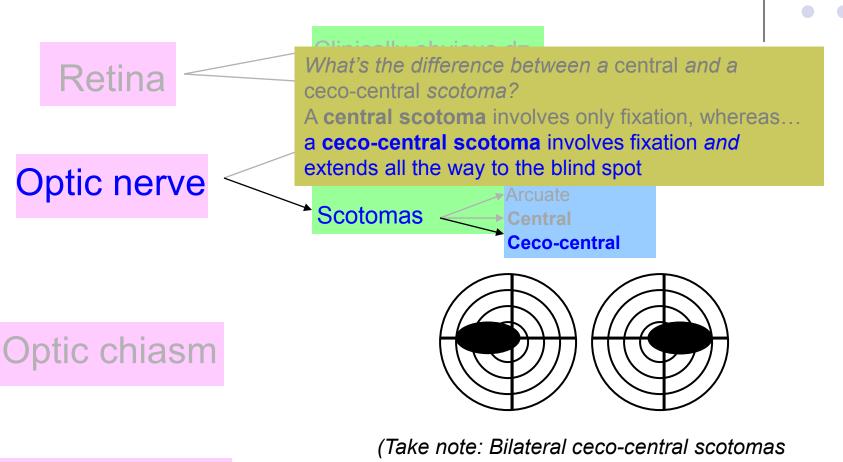






Retrochiasmal

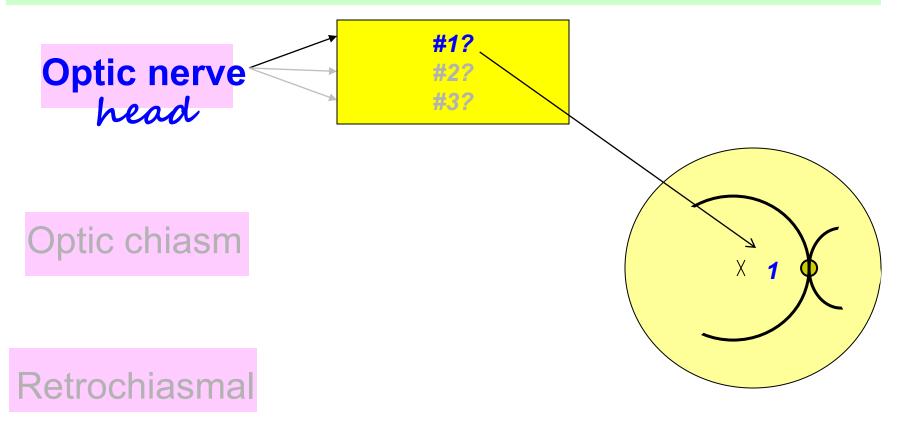
50



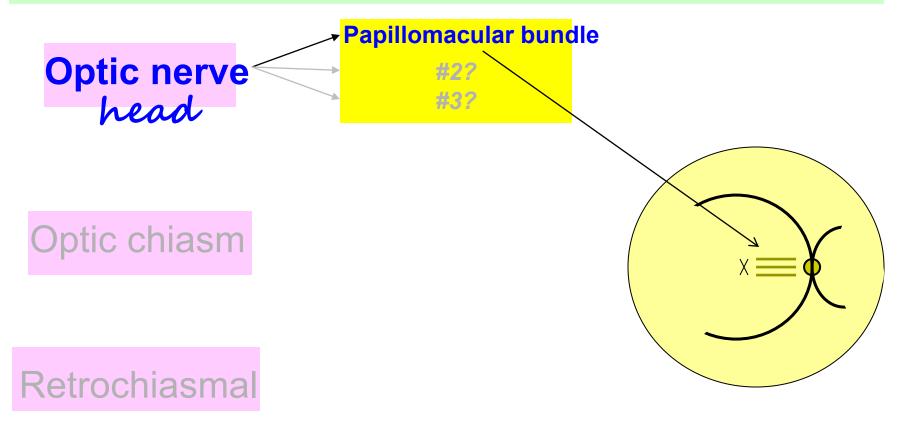
Retrochiasmal

could be mistaken for bitemporal VF loss!)

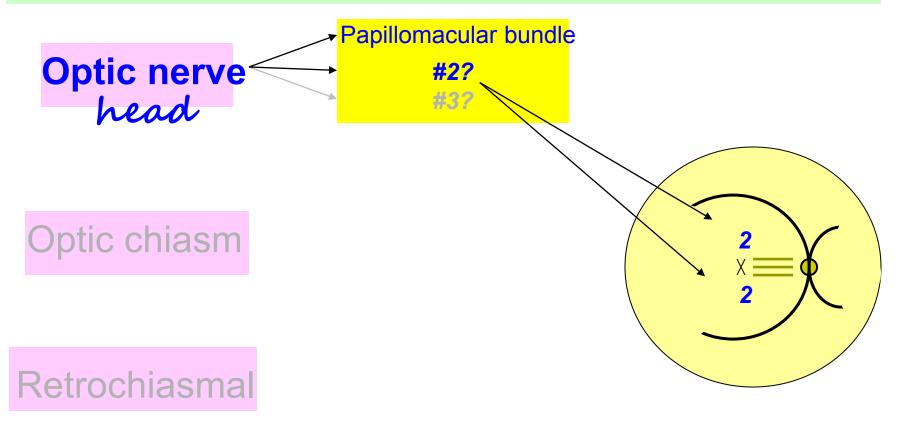




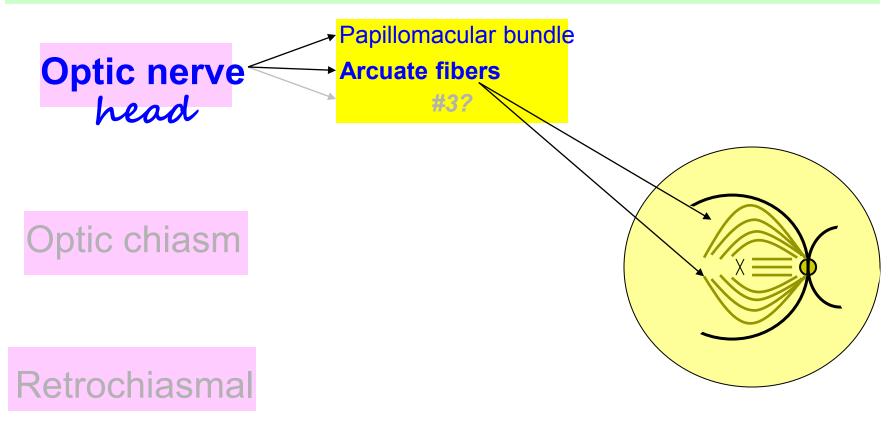




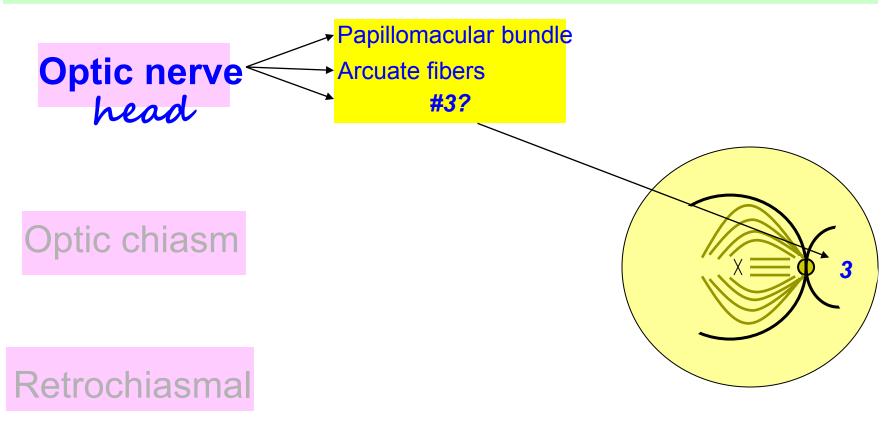




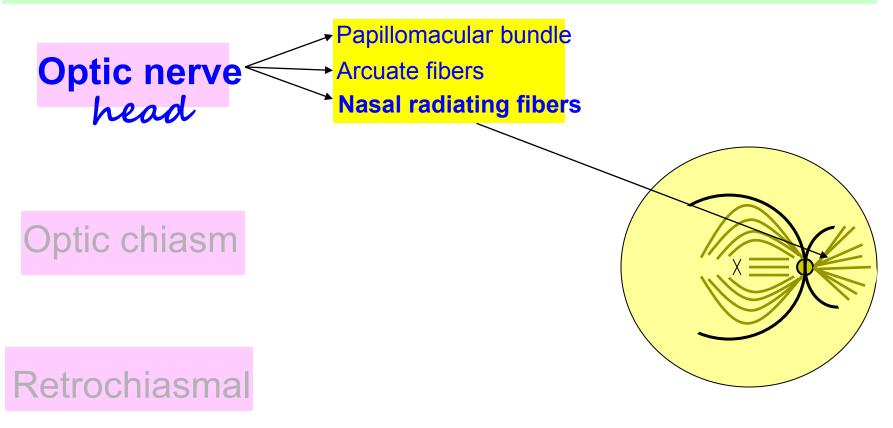














Another way to think about the optic nerve is with respect to its topography at the optic nerve head. Specifically, the retinal nerve fibers composing the optic nerve can be divided into three groups:



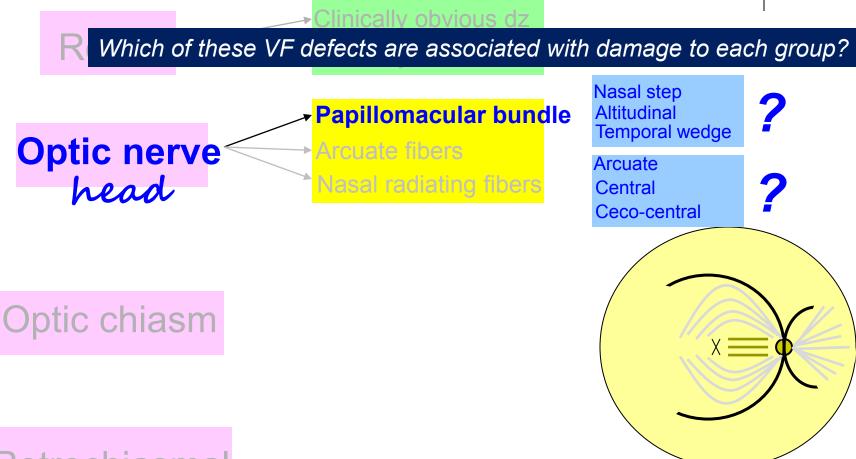
Papillomacular bundle
Arcuate fibers
Nasal radiating fibers

Optic chiasm

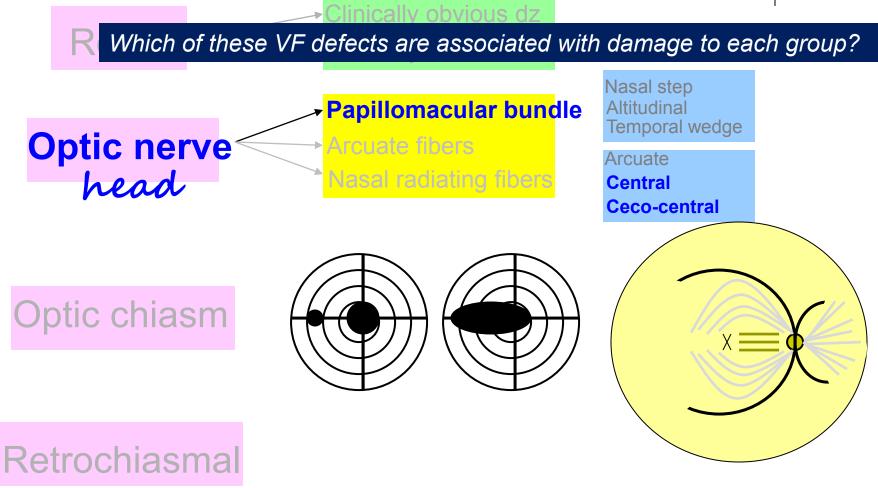
The basic topography of the RNFL looks a lot like a fish!











Optic nerve head

Optic chiasm



61

Temporal wedge

Arcuate Central

Ceco-central

Which sorts of optic neuropathy are implicated if a P-M bundle VF defect is present?

Clinically obvious dz

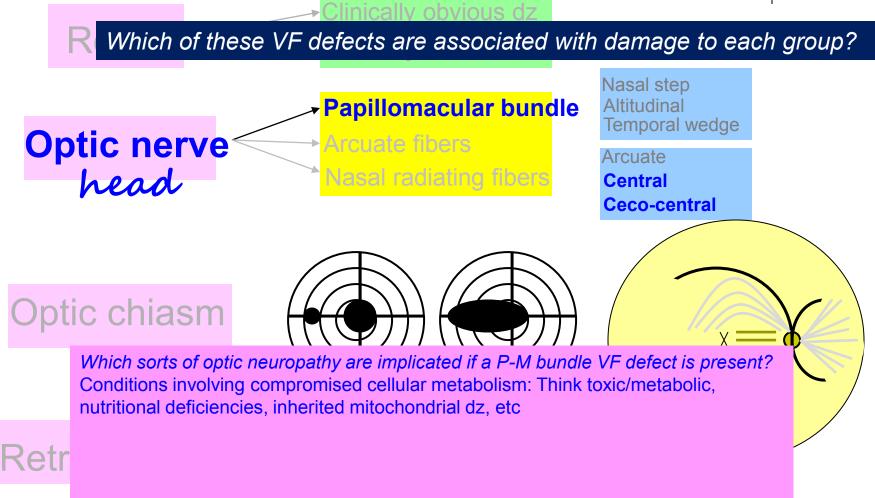
Papillomacular bundle

Nasal radiating fibers

Arcuate fibers



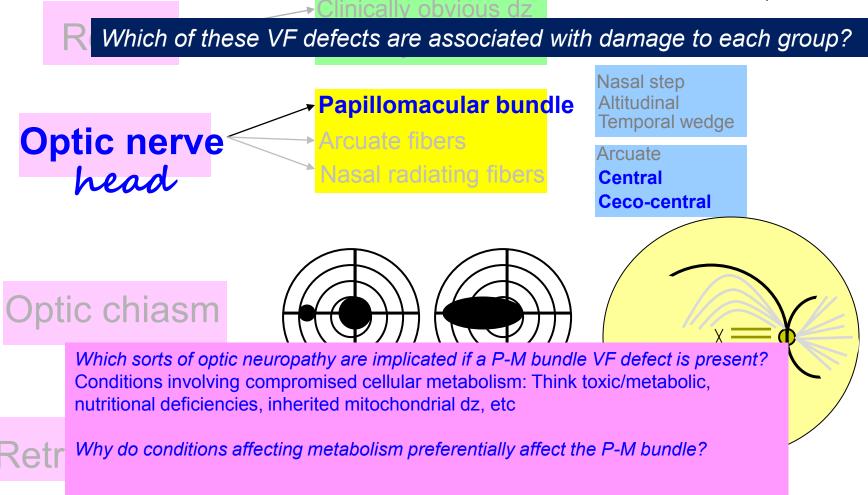




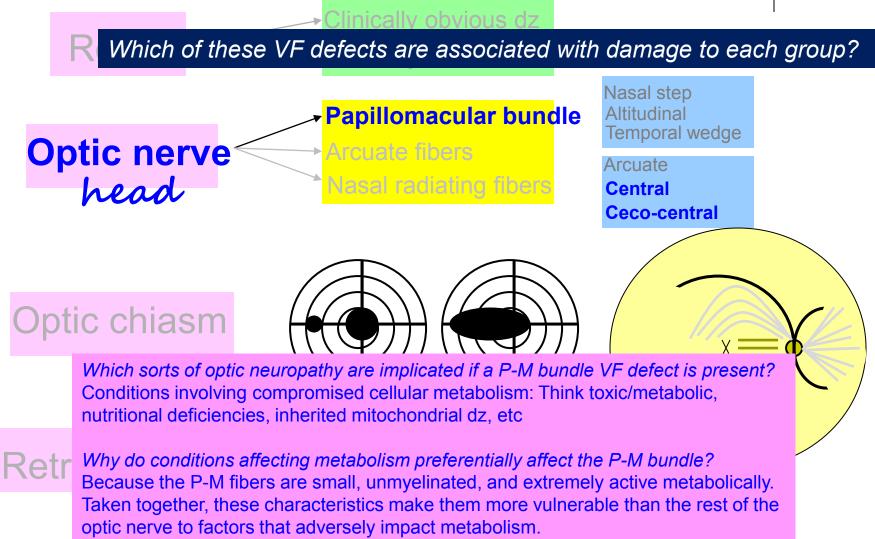
62

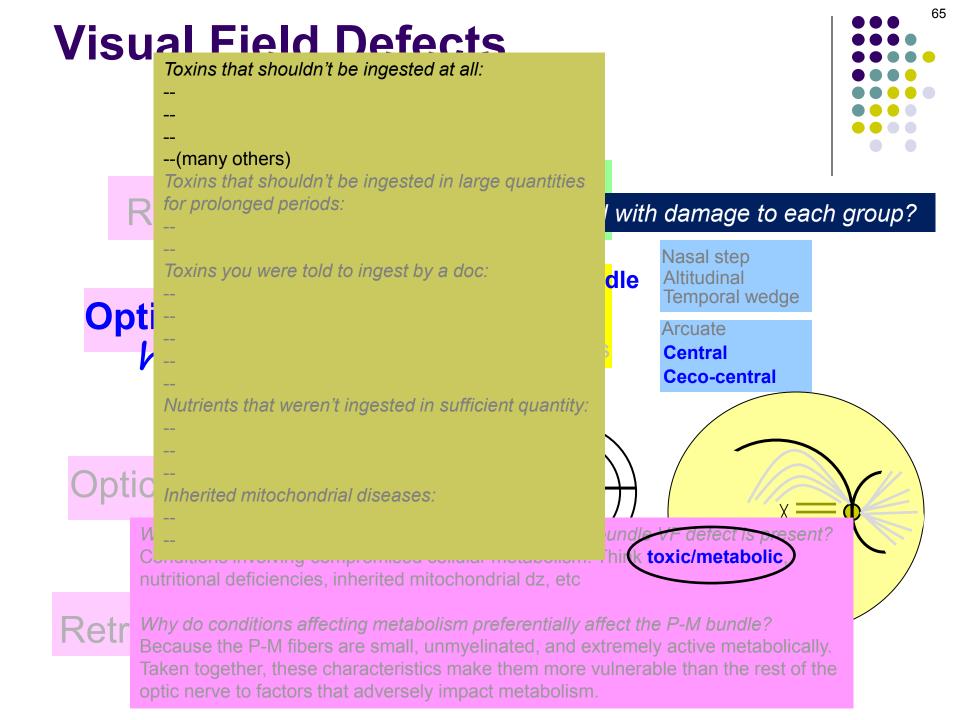


63











--(many others)

Opt

for prolonged periods:





letect is present?

Nasal step

Altitudinal

Arcuate Central

hirk toxic/metabolic.

Temporal wedge

Ceco-central

dle

Toxins you were told to ingest by a doc:

Nutrients that weren't ingested in sufficient quantity:

Toxins that shouldn't be ingested in large quantities

Inherited mitochondrial diseases:

nutritional deficiencies, inherited mitochondrial dz, etc

Retr *Why do conditions affecting metabolism preferentially affect the P-M bundle?* Because the P-M fibers are small, unmyelinated, and extremely active metabolically. Taken together, these characteristics make them more vulnerable than the rest of the optic nerve to factors that adversely impact metabolism.

Toxins that shouldn't be ingested at all: --Methanol --Ethylene glycol --Lead (in children) --(many others) Toxins that shouldn't be ingested in large quantities for prolonged periods: Toxins you were told to ingest by a doc: Opt Nutrients that weren't ingested in sufficient quantity: Inherited mitochondrial diseases:

with damage to each group?

resent?

dle Nasal step Altitudinal Temporal wedge

> Arcuate Central

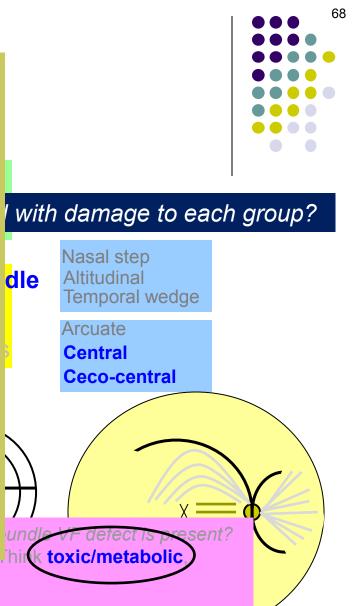
hirk toxic/metabolic.

Ceco-central

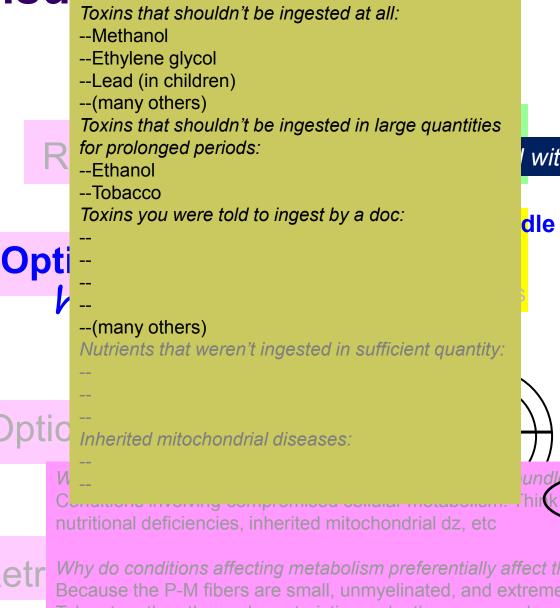
nutritional deficiencies, inherited mitochondrial dz, etc

Retr *Why do conditions affecting metabolism preferentially affect the P-M bundle?* Because the P-M fibers are small, unmyelinated, and extremely active metabolically. Taken together, these characteristics make them more vulnerable than the rest of the optic nerve to factors that adversely impact metabolism.

Toxins that shouldn't be ingested at all: --Methanol --Ethylene glycol --Lead (in children) --(many others) Toxins that shouldn't be ingested in large quantities for prolonged periods: --Ethanol --Tobacco Toxins you were told to ingest by a doc: dle Opt Nutrients that weren't ingested in sufficient quantity: Inherited mitochondrial diseases: nutritional deficiencies, inherited mitochondrial dz, etc

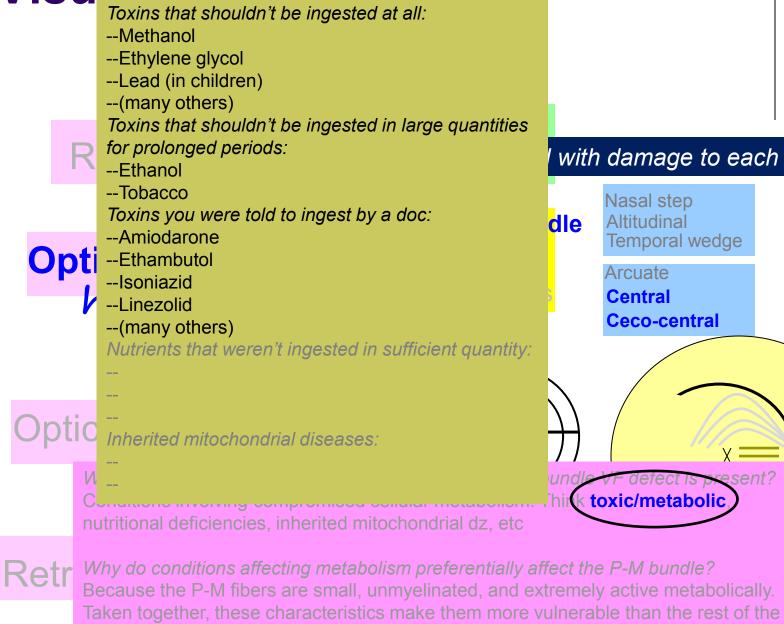


Retr Why do conditions affecting metabolism preferentially affect the P-M bundle? Because the P-M fibers are small, unmyelinated, and extremely active metabolically. Taken together, these characteristics make them more vulnerable than the rest of the optic nerve to factors that adversely impact metabolism.

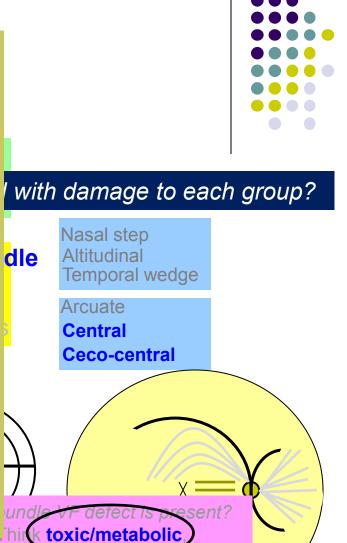




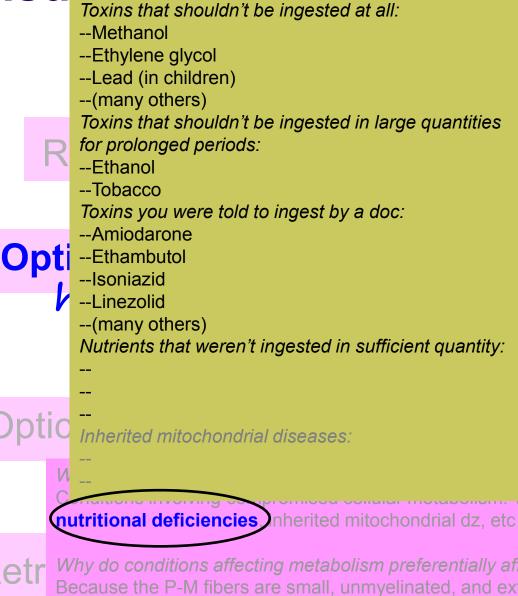
Retr *Why do conditions affecting metabolism preferentially affect the P-M bundle?* Because the P-M fibers are small, unmyelinated, and extremely active metabolically. Taken together, these characteristics make them more vulnerable than the rest of the optic nerve to factors that adversely impact metabolism.



optic nerve to factors that adversely impact metabolism.



70





with damage to each group?

Nasal step **Altitudinal** dle

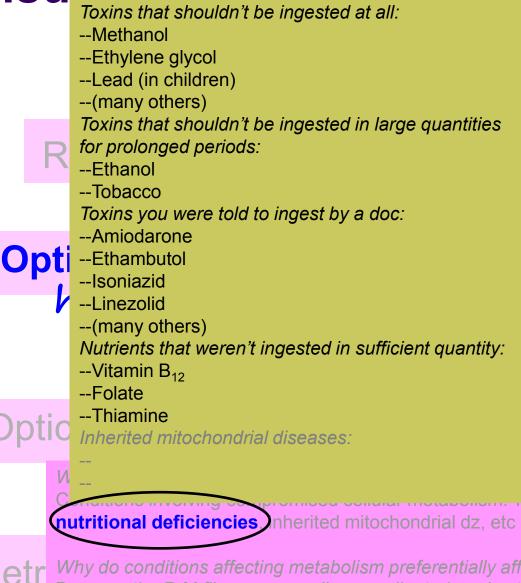
Temporal wedge

Arcuate Central

Ceco-central

undle VF defect is present? hink toxic/metabolic.

Retr Why do conditions affecting metabolism preferentially affect the P-M bundle? Because the P-M fibers are small, unmyelinated, and extremely active metabolically. Taken together, these characteristics make them more vulnerable than the rest of the optic nerve to factors that adversely impact metabolism.





with damage to each group?

Nasal step **Altitudinal** dle

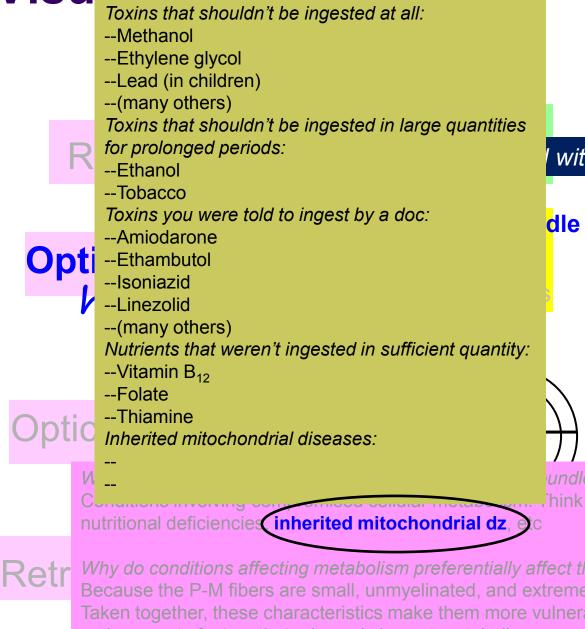
Temporal wedge

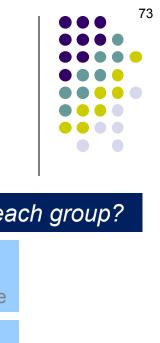
Arcuate Central

Ceco-central

undle VF defect is present? hink toxic/metabolic.

Retr Why do conditions affecting metabolism preferentially affect the P-M bundle? Because the P-M fibers are small, unmyelinated, and extremely active metabolically. Taken together, these characteristics make them more vulnerable than the rest of the optic nerve to factors that adversely impact metabolism.





with damage to each group?

Nasal step **Altitudinal**

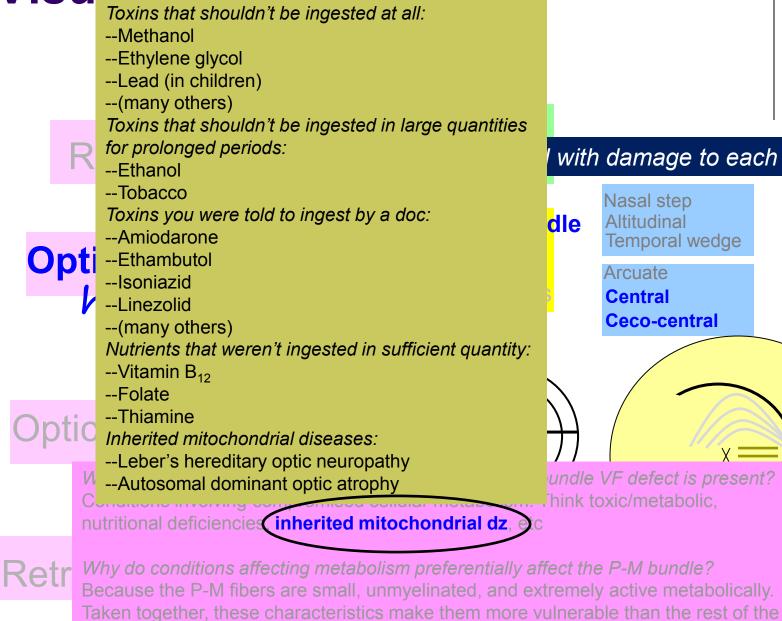
Temporal wedge

Arcuate Central

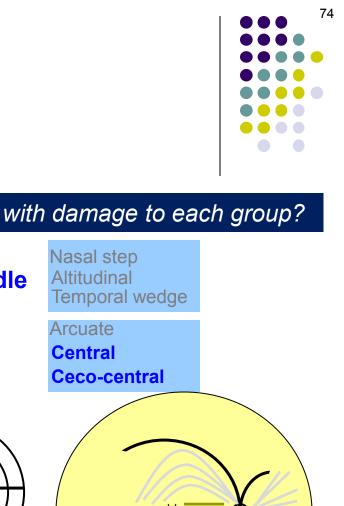
Ceco-central

undle VF defect is present? hink toxic/metabolic,

Why do conditions affecting metabolism preferentially affect the P-M bundle? Because the P-M fibers are small, unmyelinated, and extremely active metabolically. Taken together, these characteristics make them more vulnerable than the rest of the optic nerve to factors that adversely impact metabolism.



optic nerve to factors that adversely impact metabolism.

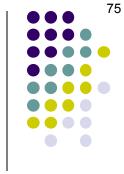


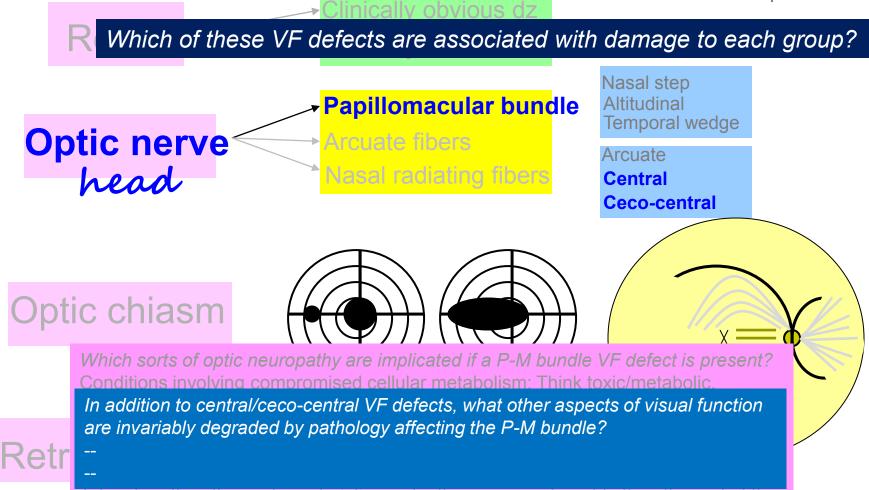
undle VF defect is present? hink toxic/metabolic,

Arcuate

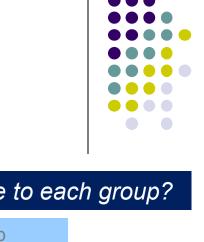
Central

dle

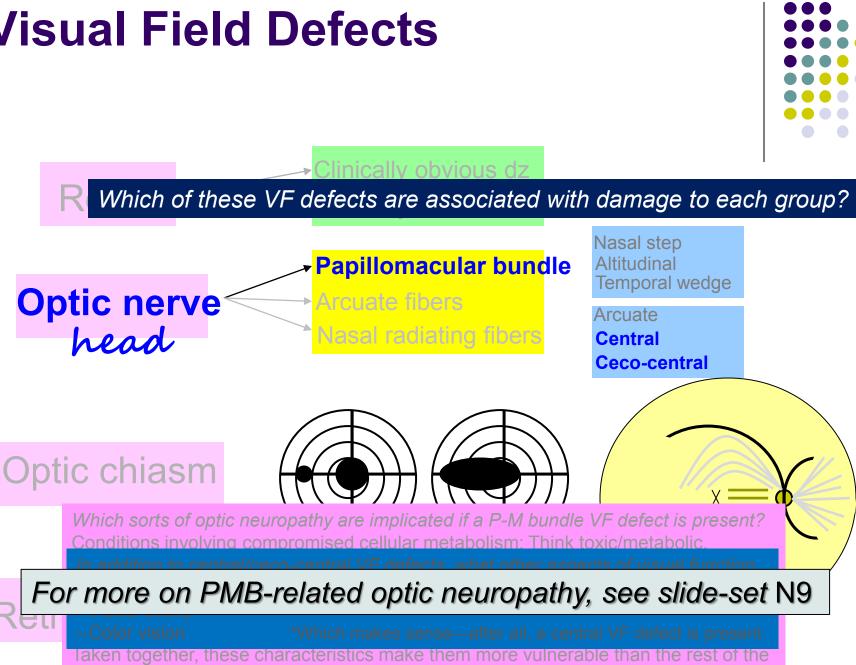




Taken together, these characteristics make them more vulnerable than the rest of the optic nerve to factors that adversely impact metabolism.



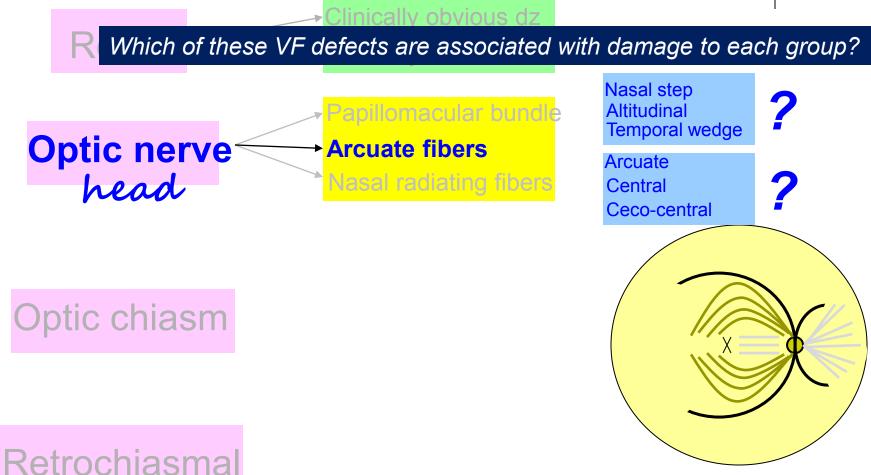
linically obvious dz Which of these VF defects are associated with damage to each group? Nasal step Papillomacular bundle Altitudinal Temporal wedge Optic nerve head Arcuate fibers Arcuate Nasal radiating fibers Central **Ceco-central Optic chiasm** Which sorts of optic neuropathy are implicated if a P-M bundle VF defect is present? Conditions involving compromised cellular metabolism: Think toxic/metabolic In addition to central/ceco-central VF defects, what other aspects of visual function are invariably degraded by pathology affecting the P-M bundle? --Visual acuity* Retr --Color vision *Which makes sense—after all, a central VF defect is present Taken together, these characteristics make them more vulnerable than the rest of the optic nerve to factors that adversely impact metabolism.



77

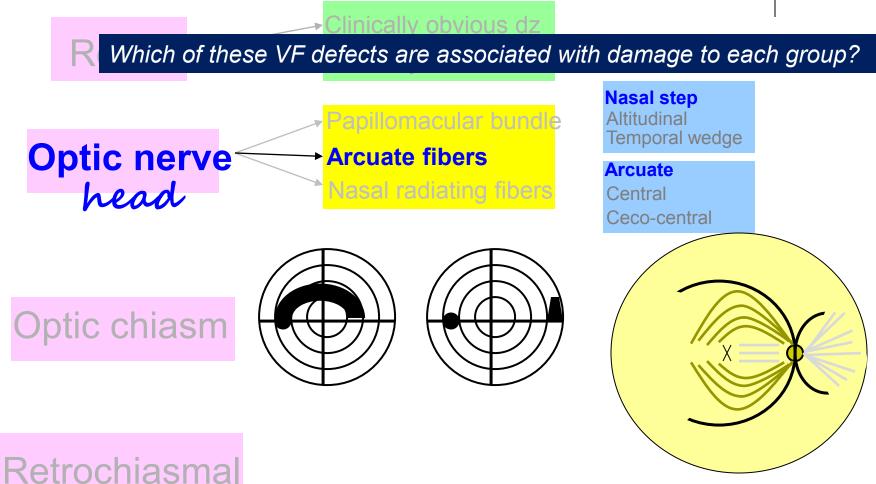
optic nerve to factors that adversely impact metabolism.





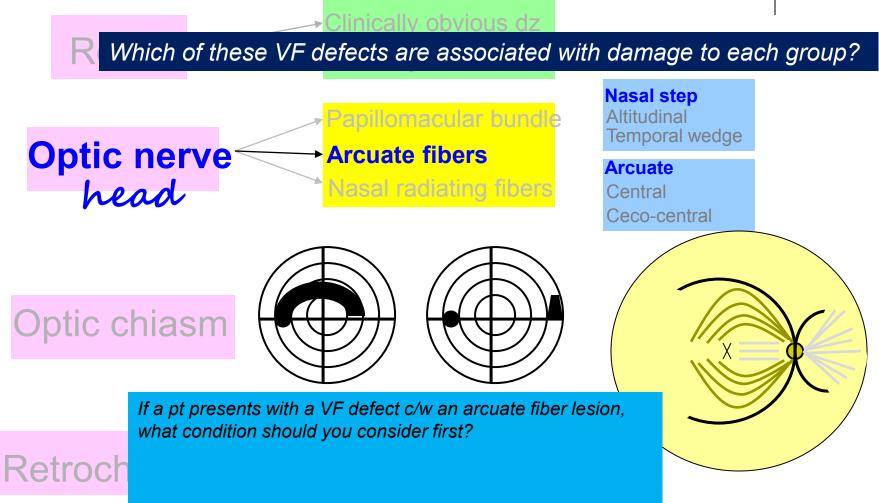
78



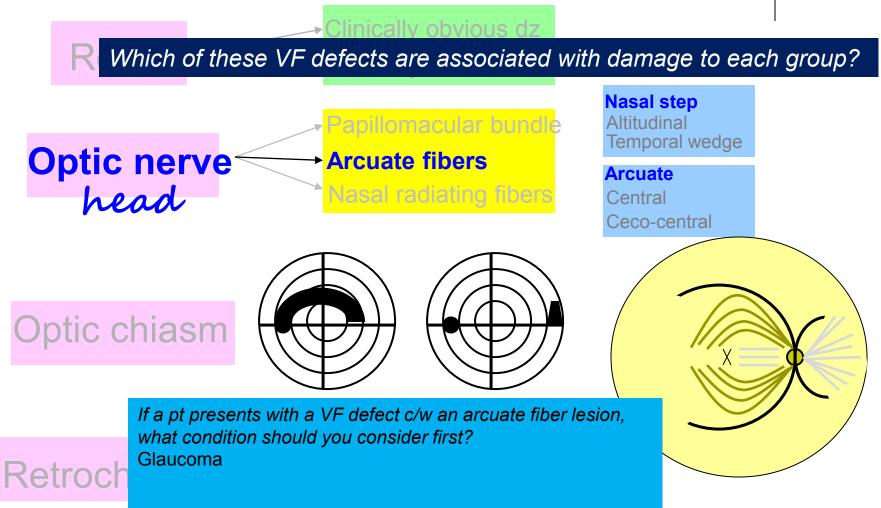


79



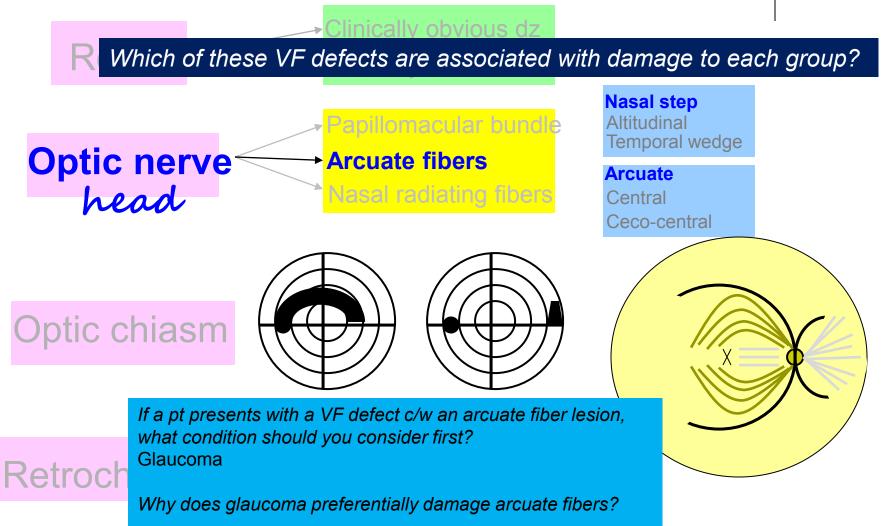




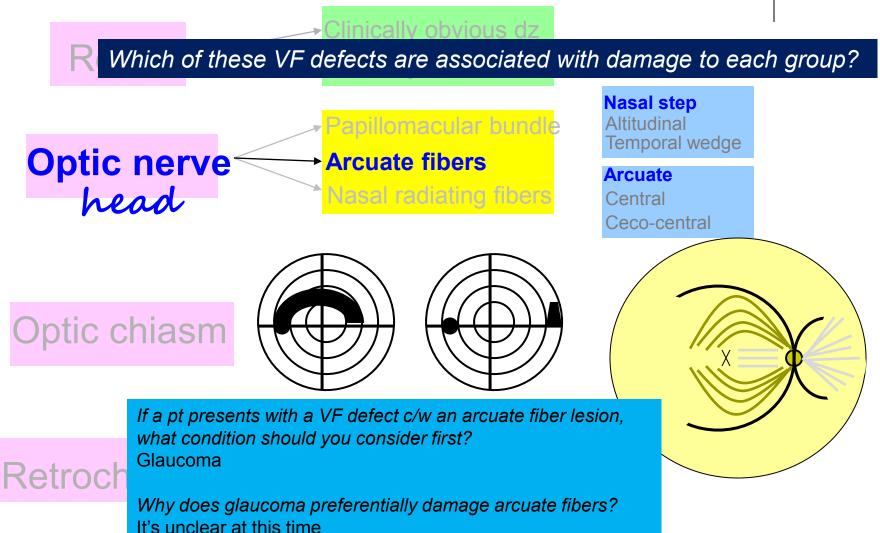


81

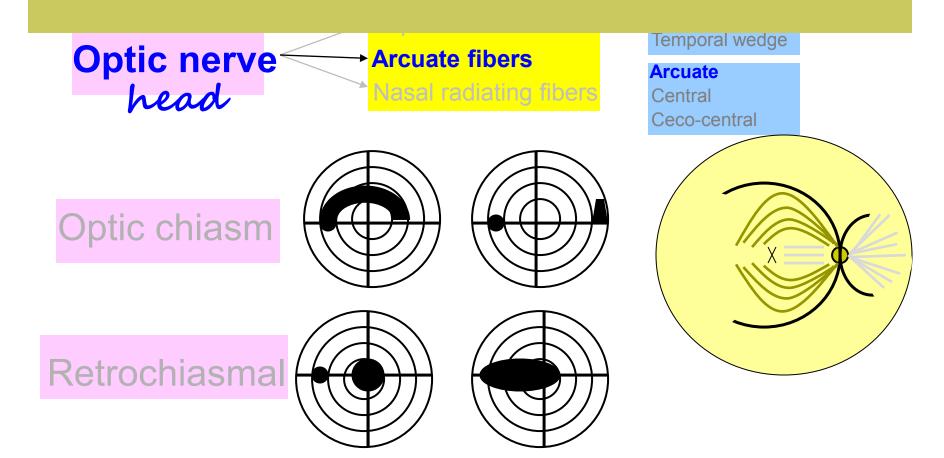


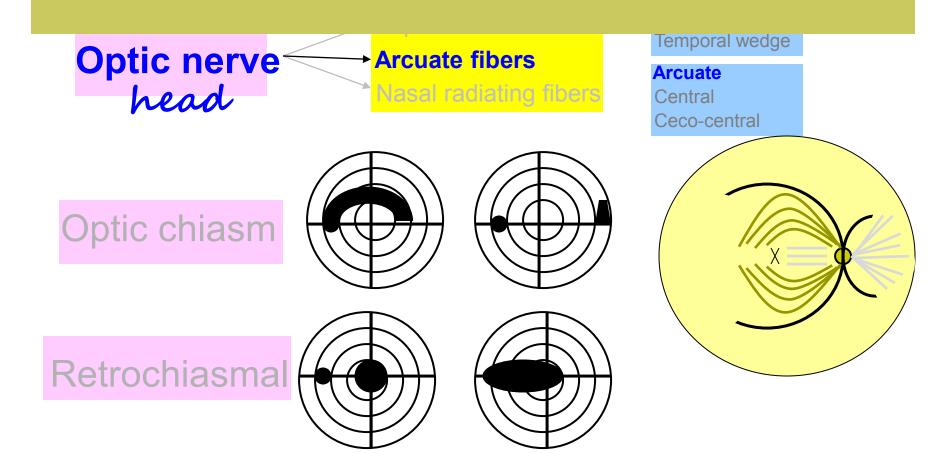




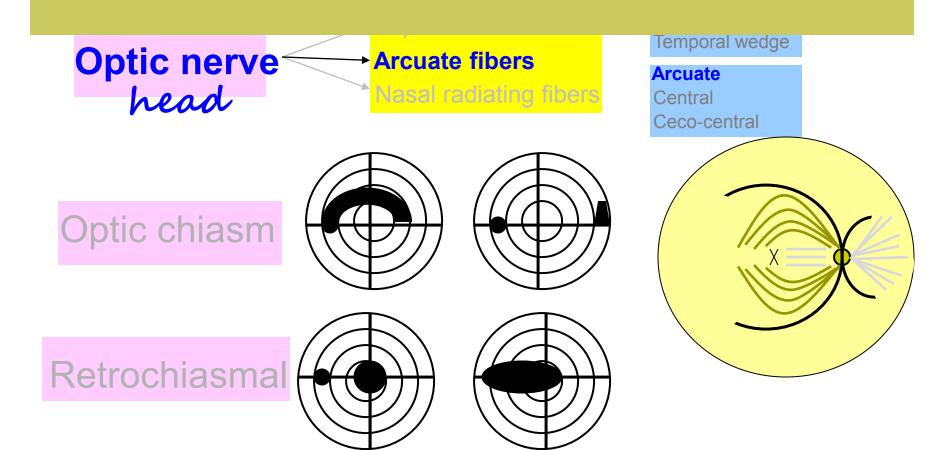


Compare the distribution of arcuate-fiber defects with those associated with a P-M bundle dysfunction. What important difference do you see?





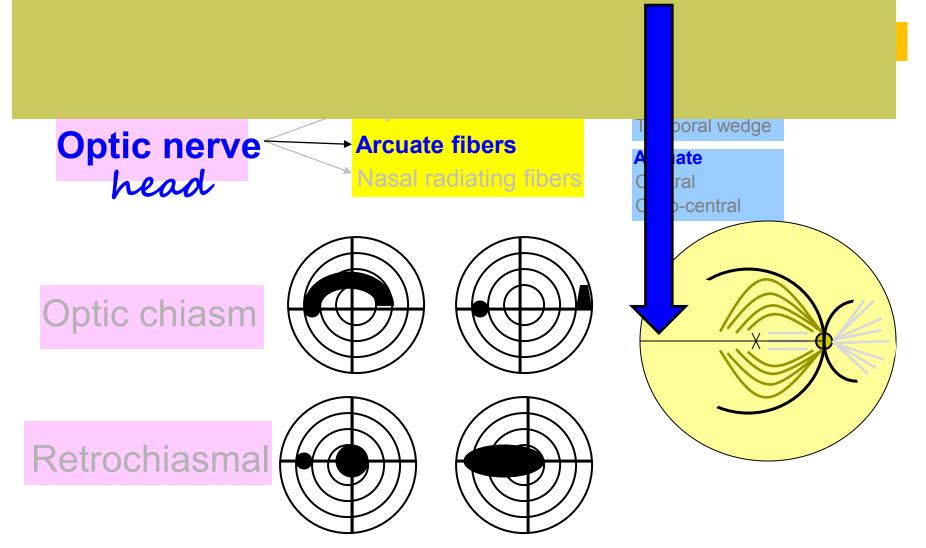
Why not?



87

Why not?

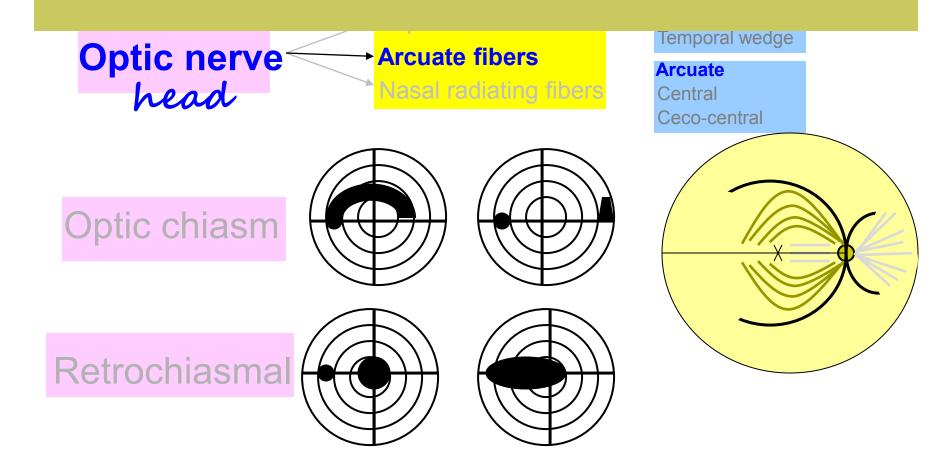
Because fibers on the temporal side of the ONH approach, but do **not** cross, the horizontal midline. The arcuate fibers arc around the P-M bundle, and meet along a **horizontal demarcation line**.



88

Why not?

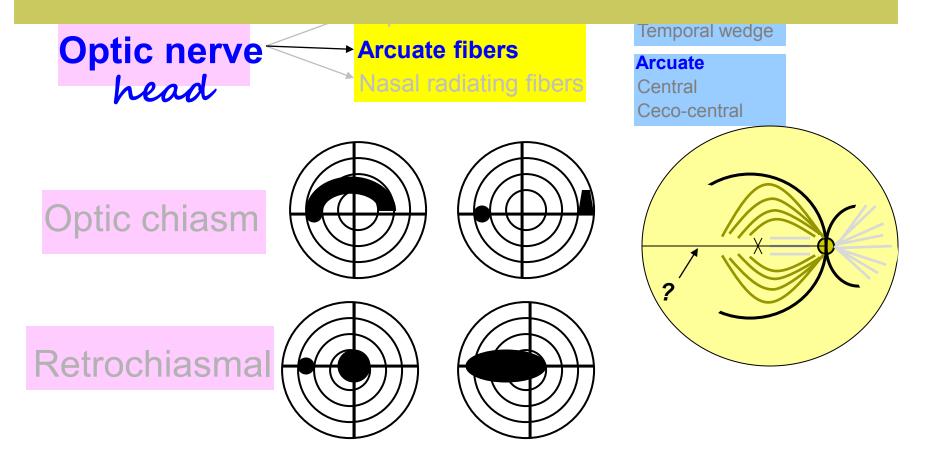
Because fibers on the temporal side of the ONH approach, but do **not** cross, the horizontal midline. The arcuate fibers arc around the P-M bundle, and meet along a horizontal demarcation line. Thus, damage to these fibers always result in VF defects that are limited to either the superior *or* the inferior portion of the field.



Why not?

Because fibers on the temporal side of the ONH approach, but do **not** cross, the horizontal midline. The arcuate fibers arc around the P-M bundle, and meet along a horizontal demarcation line. Thus, damage to these fibers always result in VF defects that are limited to either the superior *or* the inferior portion of the field.

What is this horizontal demarcation line called?

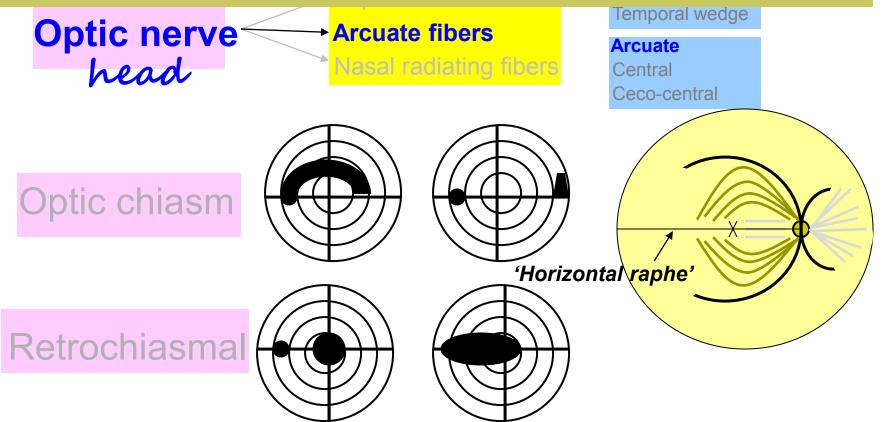


90

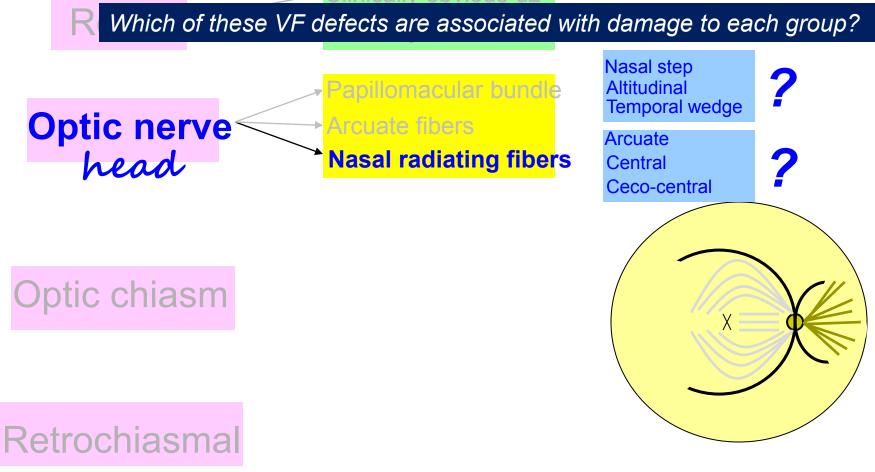
Why not?

Because fibers on the temporal side of the ONH approach, but do **not** cross, the horizontal midline. The arcuate fibers arc around the P-M bundle, and meet along a horizontal demarcation line. Thus, damage to these fibers always result in VF defects that are limited to either the superior *or* the inferior portion of the field.

What is this horizontal demarcation line called? The **horizontal raphe**

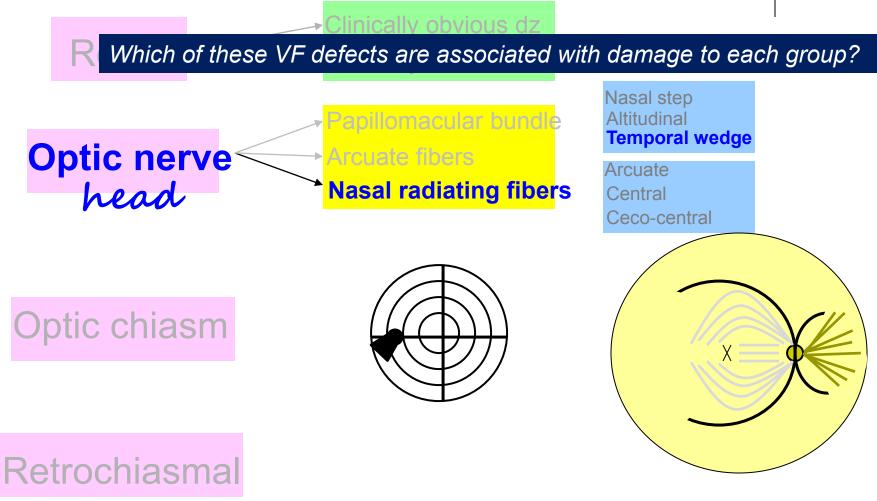


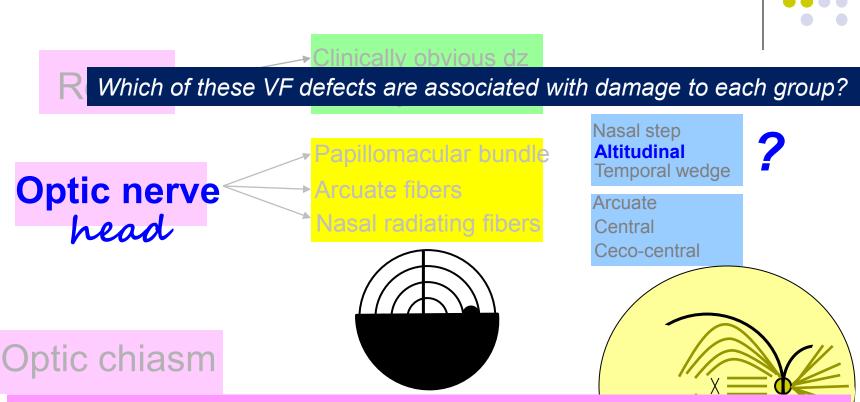




Clinically obvious dz

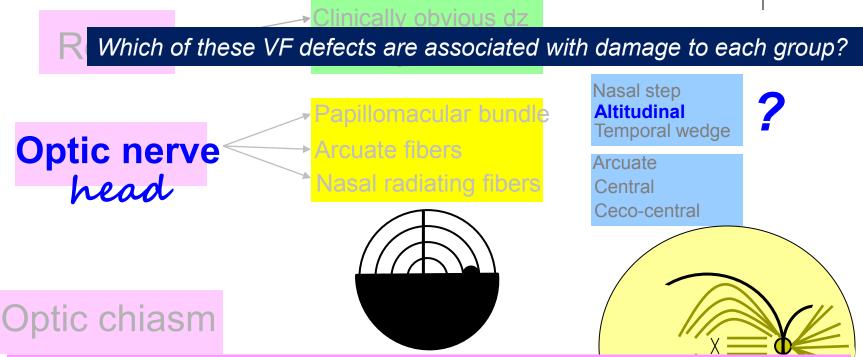




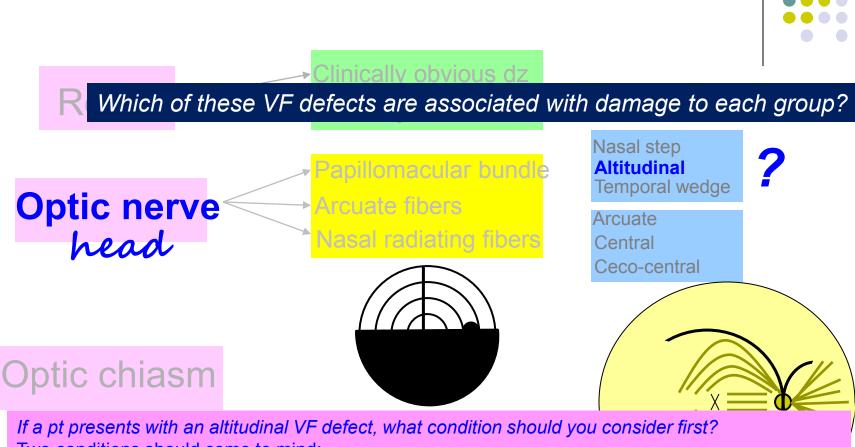


If a pt presents with an altitudinal VF defect, what condition should you consider first?





If a pt presents with an altitudinal VF defect, what condition should you consider first? Two conditions should come to mind:

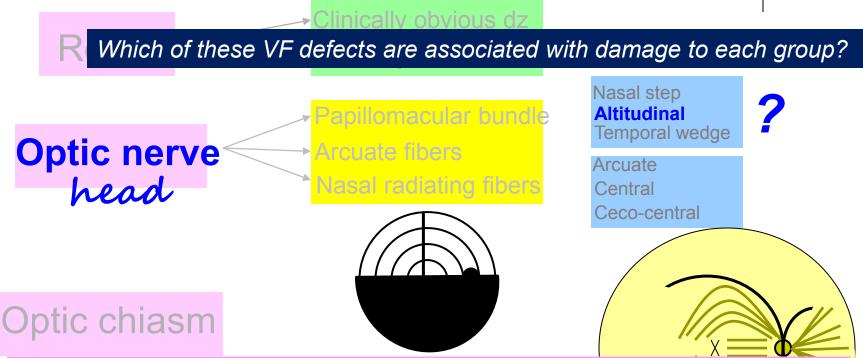


95

Two conditions should come to mind: --If the pt is a _____age and condition _____, it's likely nonarteritic anterior ischemic optic neuropathy (NAION)

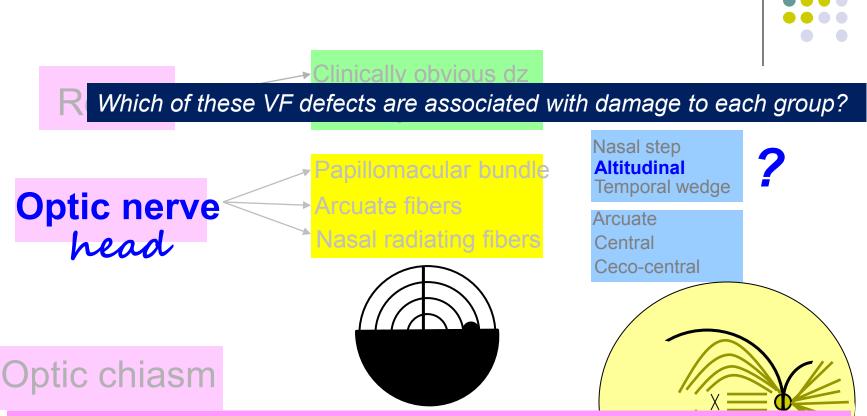
--If the pt has glaucoma, it likely represents advanced glaucomatous optic neuropathy





If a pt presents with an altitudinal VF defect, what condition should you consider first? Two conditions should come to mind:

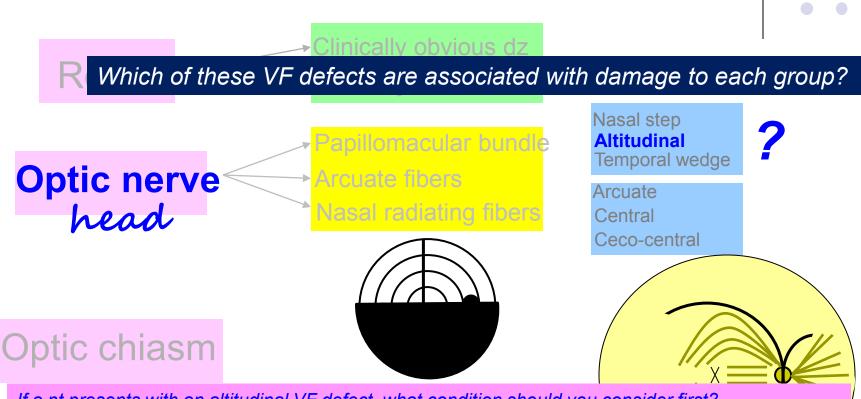
--If the pt is a 50+ vasculopath, it's likely nonarteritic anterior ischemic optic neuropathy (NAION) --If the pt has glaucoma, it likely represents advanced glaucomatous optic neuropathy



If a pt presents with an altitudinal VF defect, what condition should you consider first? Two conditions should come to mind:

--If the pt is a 50+ vasculopath, it's likely nonarteritic anterior ischemic optic neuropathy (NAION) --If the pt has glaucoma, it likely represents advanced glaucomatous optic neuropathy

How can you differentiate between these two conditions?

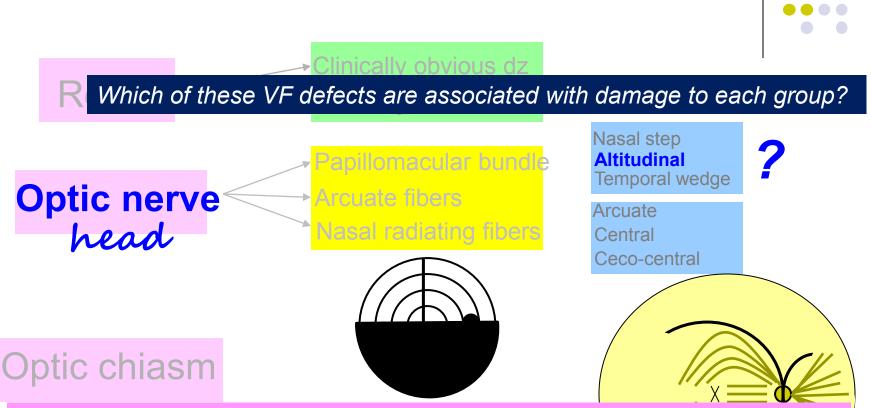


If a pt presents with an altitudinal VF defect, what condition should you consider first? Two conditions should come to mind:

--If the pt is a 50+ vasculopath, it's likely nonarteritic anterior ischemic optic neuropathy (NAION) --If the pt has glaucoma, it likely represents advanced glaucomatous optic neuropathy

How can you differentiate between these two conditions?

There are a number of ways, but the most straightforward would be to inspect the ONH, which will be one word in NAION, and two words in advanced glaucoma

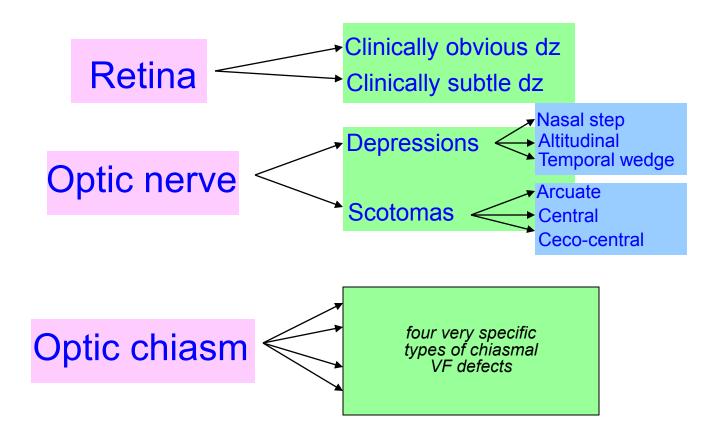


If a pt presents with an altitudinal VF defect, what condition should you consider first? Two conditions should come to mind:

--If the pt is a 50+ vasculopath, it's likely nonarteritic anterior ischemic optic neuropathy (NAION) --If the pt has glaucoma, it likely represents advanced glaucomatous optic neuropathy

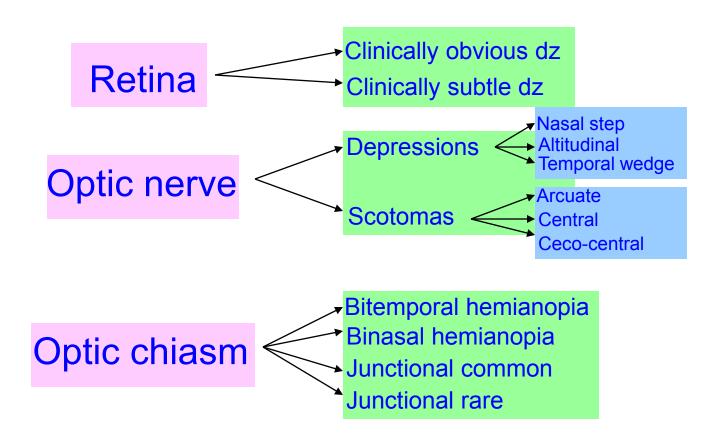
How can you differentiate between these two conditions?

There are a number of ways, but the most straightforward would be to inspect the ONH, which will be edematous in NAION, and severely cupped in advanced glaucoma



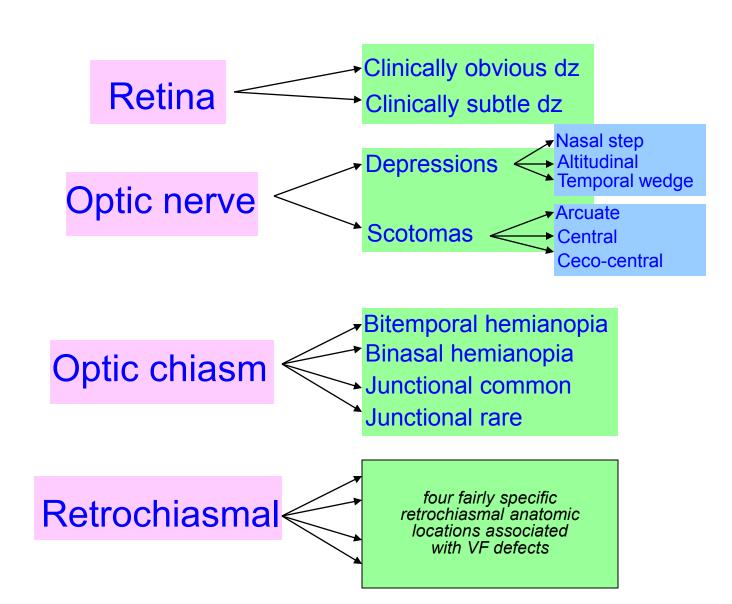


Retrochiasmal

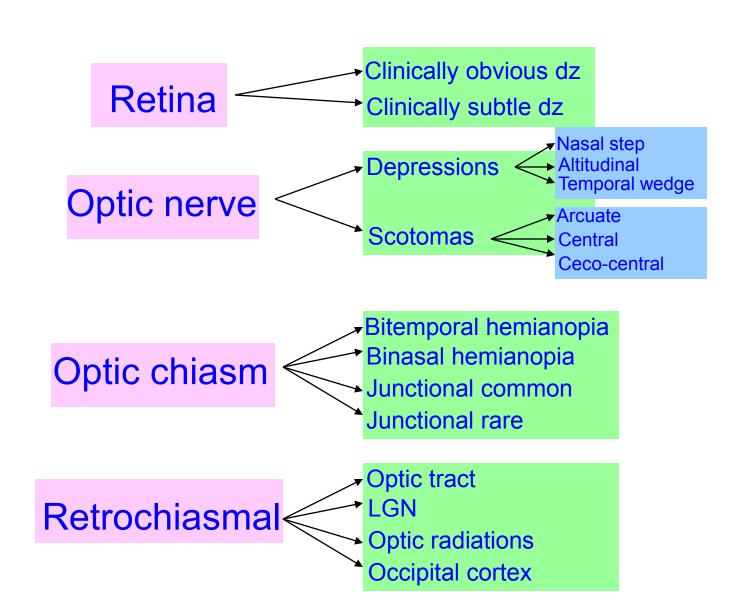




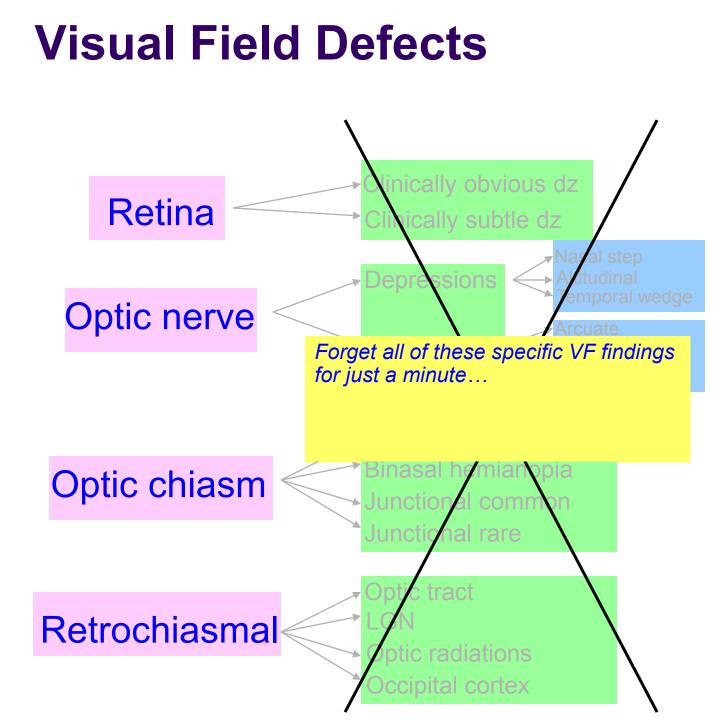
Retrochiasmal

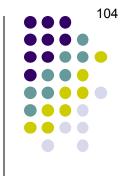


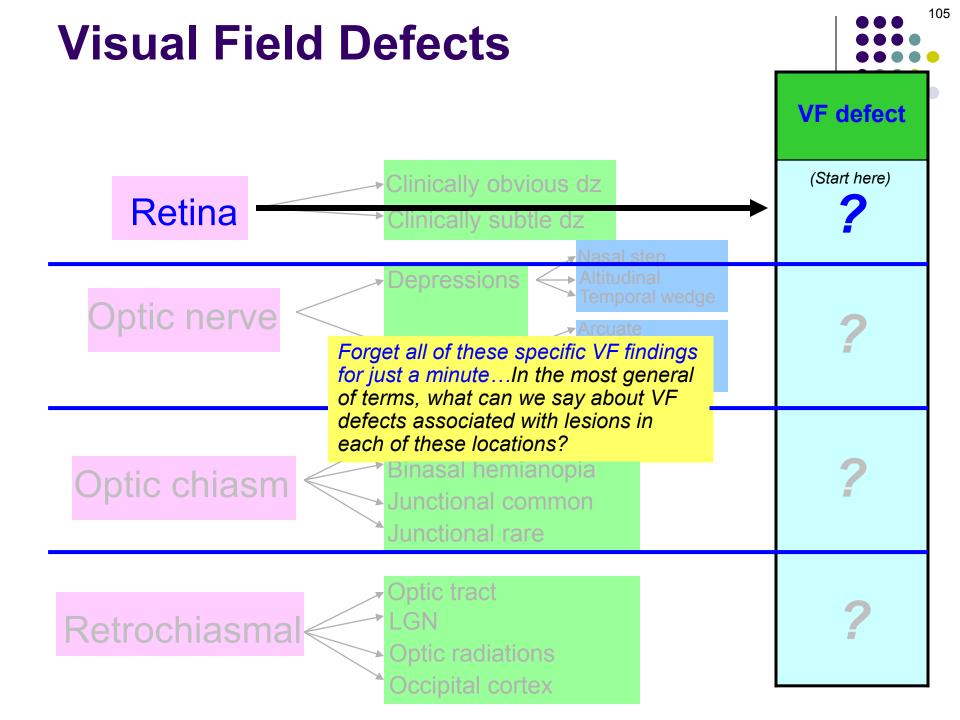


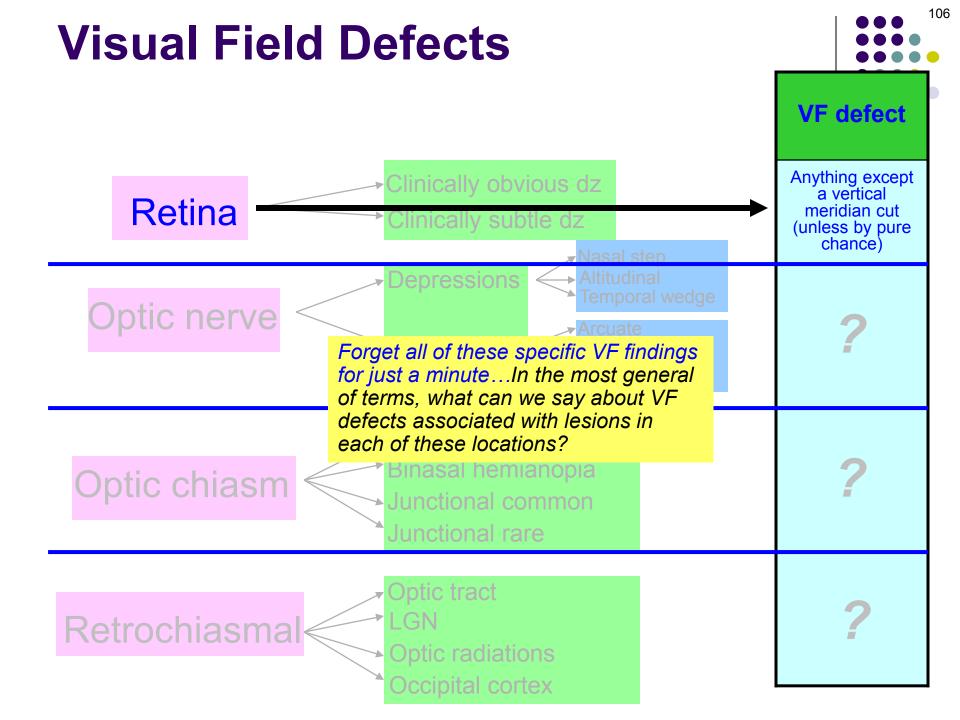


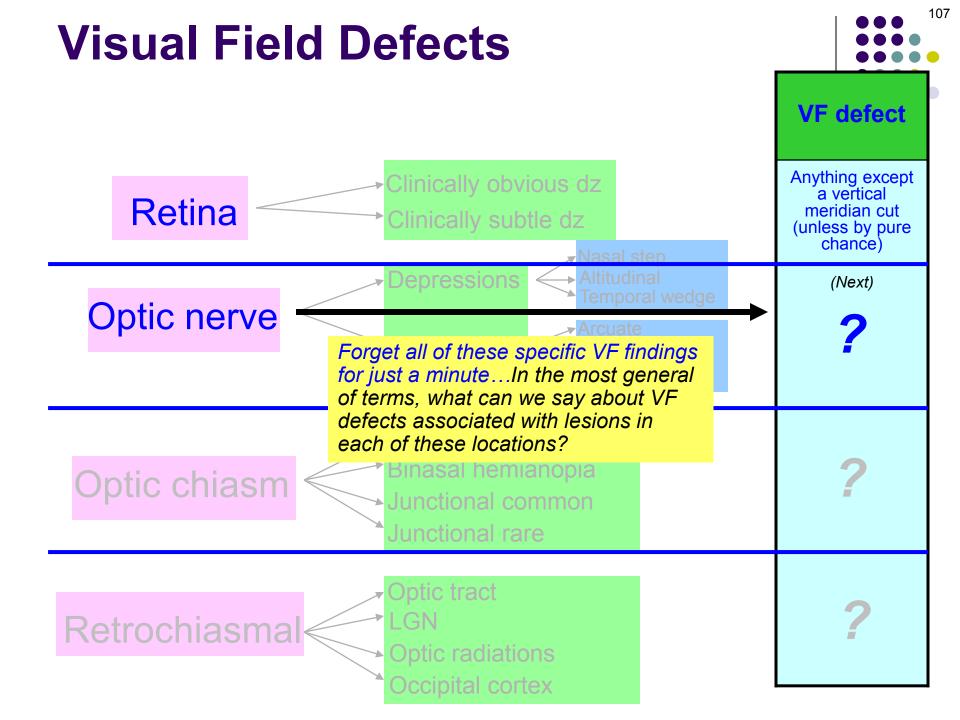


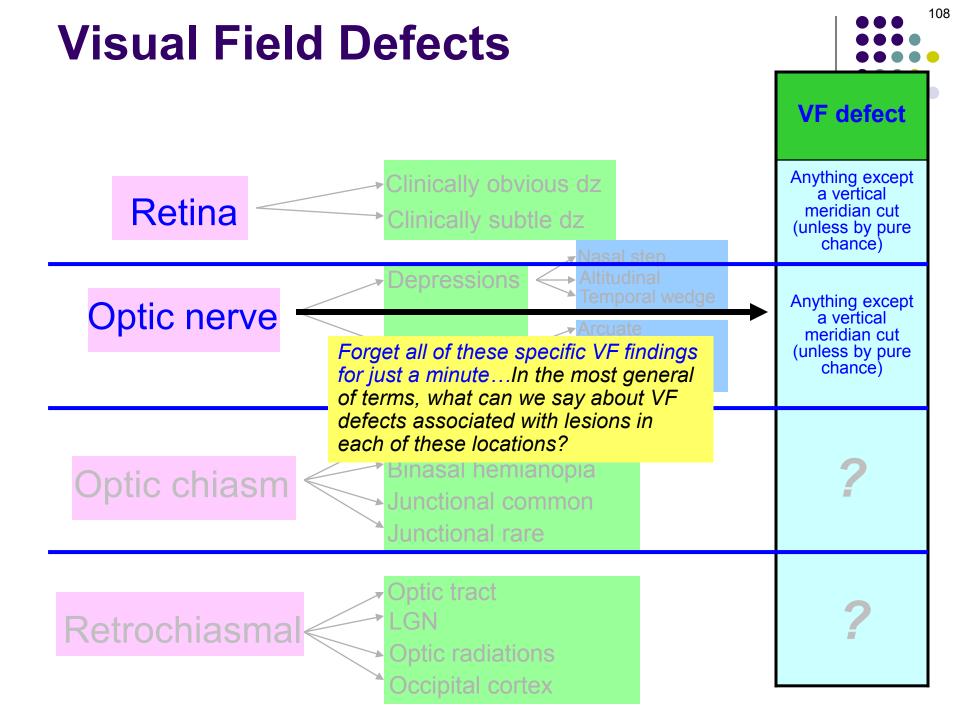


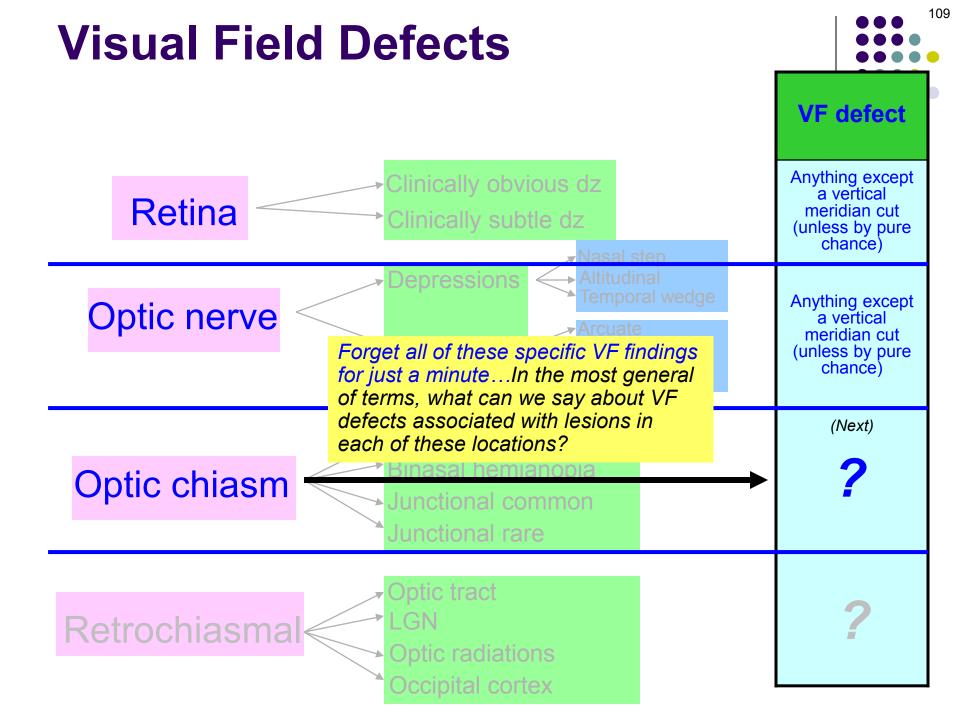


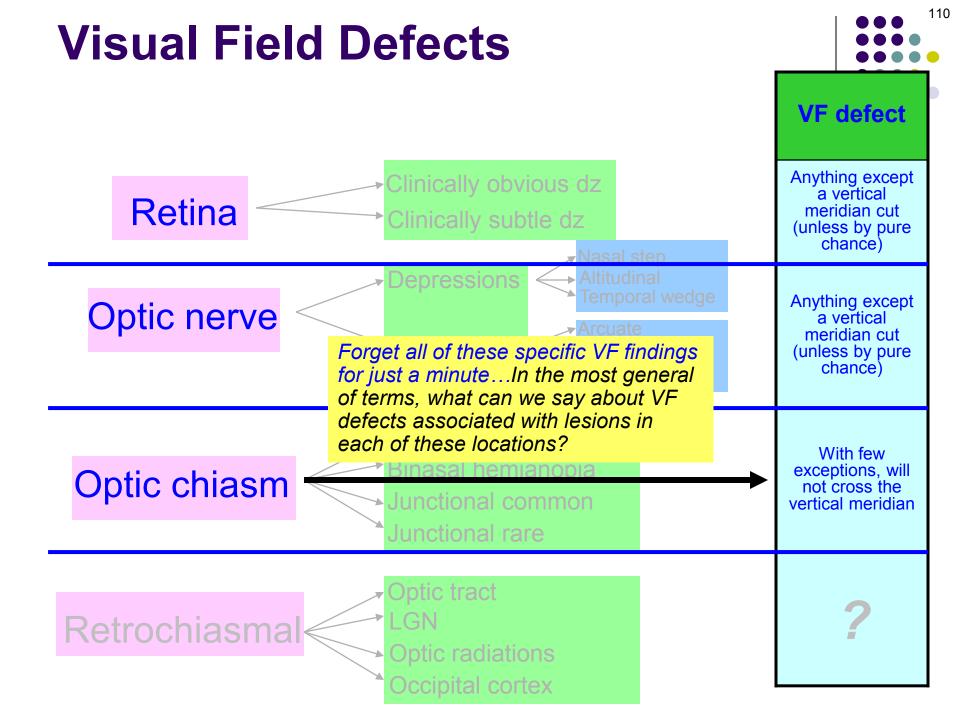


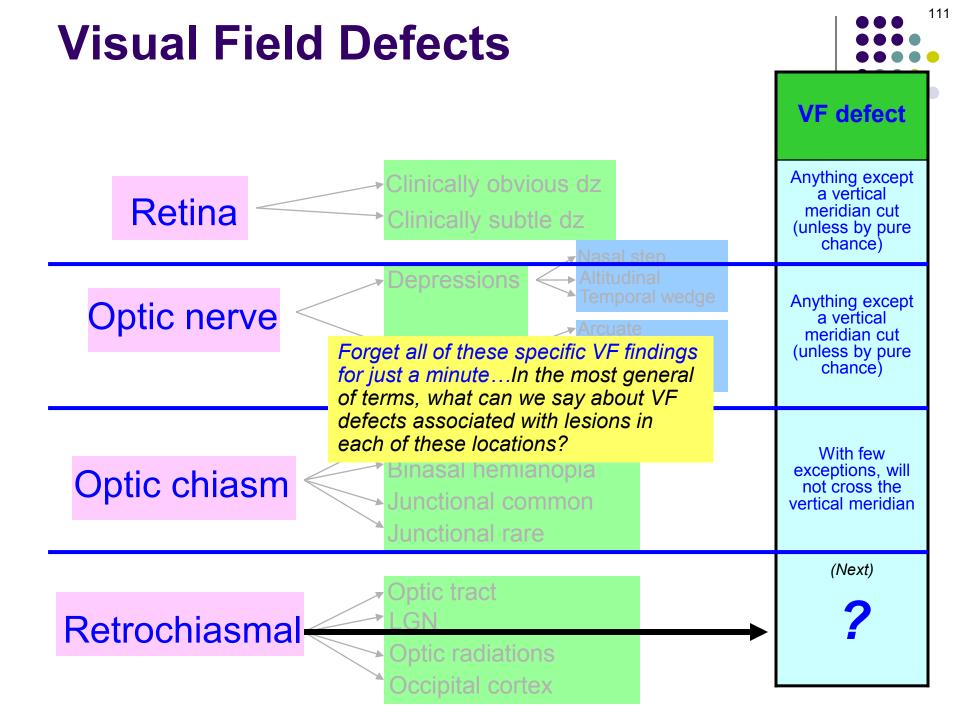


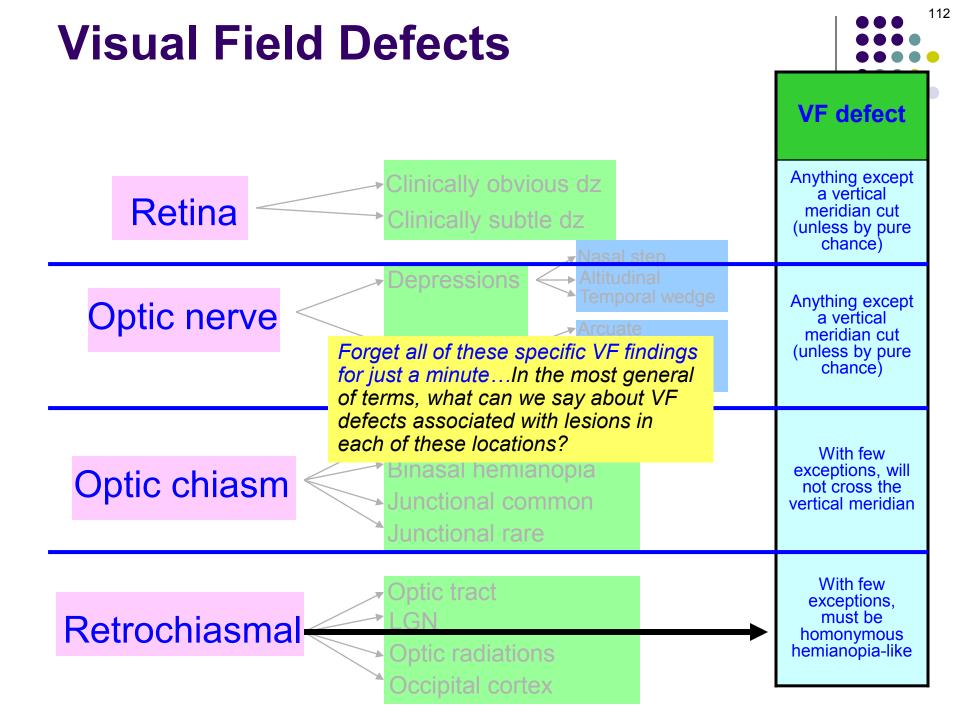


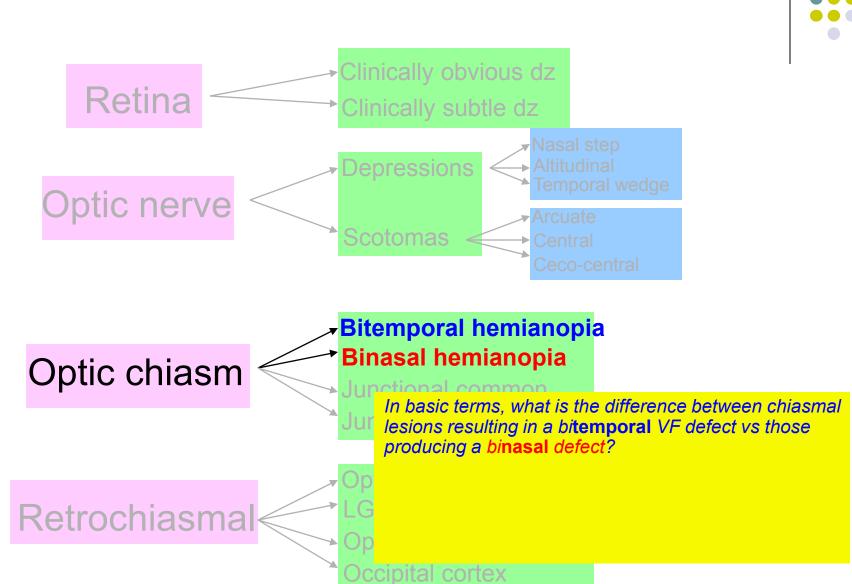






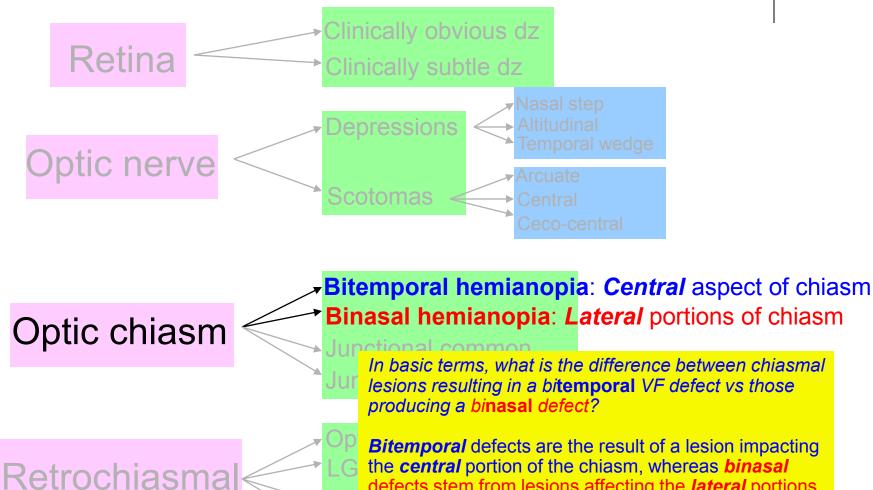






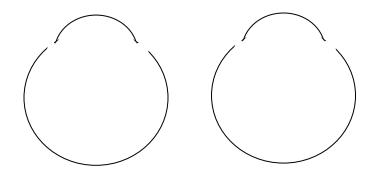






the central portion of the chiasm, whereas binasal defects stem from lesions affecting the *lateral* portions of the chiasm





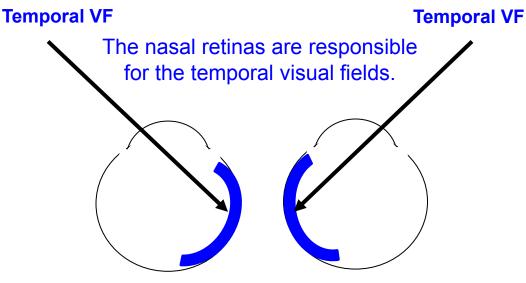
Here's why:

Bitemporal hemianopia: Central aspect of chiasm

Binasal hemianopia: Lateral portions of chiasm

In basic terms, what is the difference between chiasmal lesions resulting in a bi**temporal** VF defect vs those producing a bi**nasal** defect?



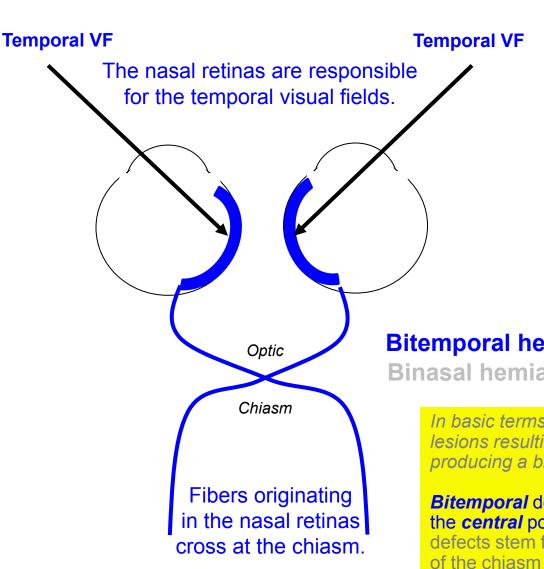


Here's why:

Bitemporal hemianopia: Central aspect of chiasm

Binasal hemianopia: Lateral portions of chiasm

In basic terms, what is the difference between chiasmal lesions resulting in a bitemporal VF defect vs those producing a binasal defect?





Bitemporal hemianopia: Central aspect of chiasm

Here's why:

Binasal hemianopia: Lateral portions of chiasm

In basic terms, what is the difference between chiasmal lesions resulting in a bitemporal VF defect vs those producing a binasal defect?



Temporal VF

Temporal VF

The nasal retinas are responsible for the temporal visual fields.

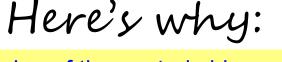
Optic

Chiasm

Fibers originating

in the nasal retinas

cross at the chiasm.

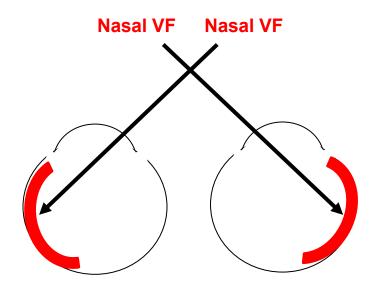


So a lesion of the central chiasm will bag these fibers, and thus tend to cause bitemporal defects

Bitemporal hemianopia: Central aspect of chiasm

Binasal hemianopia: Lateral portions of chiasm

In basic terms, what is the difference between chiasmal lesions resulting in a bi**temporal** VF defect vs those producing a bi**nasal** defect?

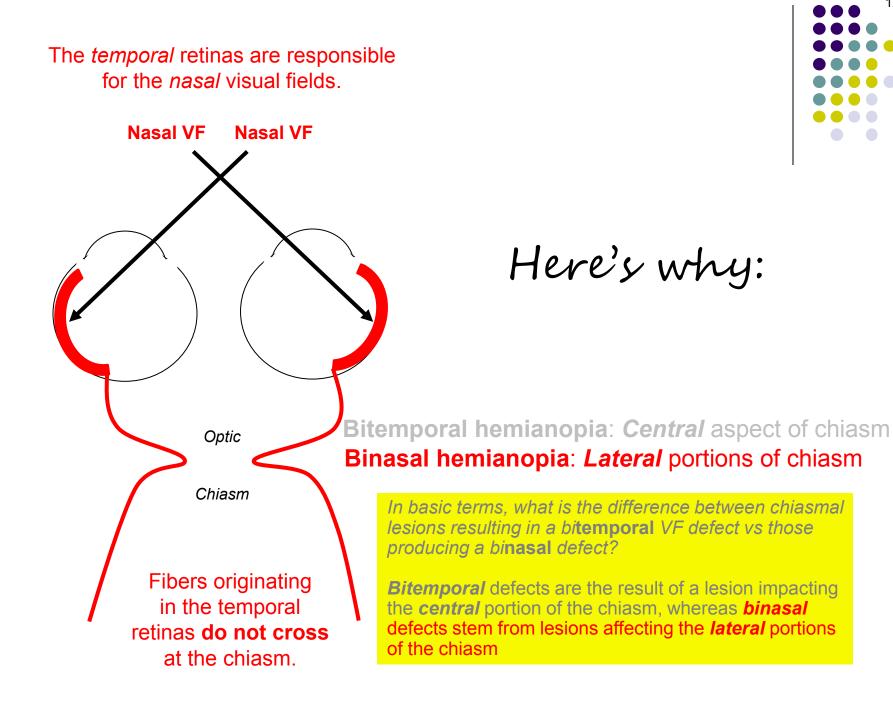




Here's why:

Bitemporal hemianopia: *Central* aspect of chiasm Binasal hemianopia: *Lateral* portions of chiasm

In basic terms, what is the difference between chiasmal lesions resulting in a bi**temporal** VF defect vs those producing a bi**nasal** defect?





Nasal VF Nasal VF Bitem Optic Chiasm Fibers originating in the temporal retinas do not cross

at the chiasm.



Here's why:

So lesions of the central chiasm will miss these fibers...

Binasal hemianopia: Lateral portions of chiasm

In basic terms, what is the difference between chiasmal lesions resulting in a bi**temporal** VF defect vs those producing a bi**nasal** defect?

Nasal VF Nasal VF Optic Chiasm Fibers originating in the temporal retinas do not cross at the chiasm.

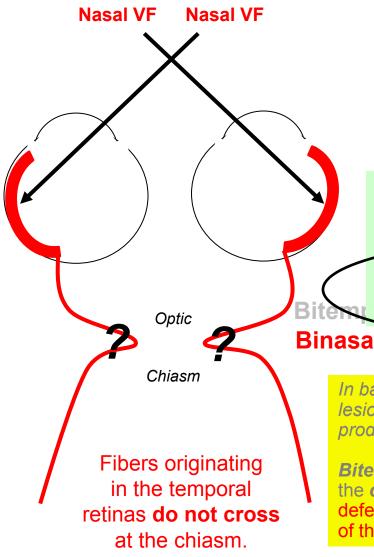


Here's why:

So lesions of the central chiasm will miss these fibers...But lesions of the **lateral** chiasm will bag them, thereby causing binasal defects (note that **two** lesions are **Bitem**; required to do this)

Binasal hemianopia: Lateral portions of chiasm

In basic terms, what is the difference between chiasmal lesions resulting in a bi**temporal** VF defect vs those producing a bi**nasal** defect?



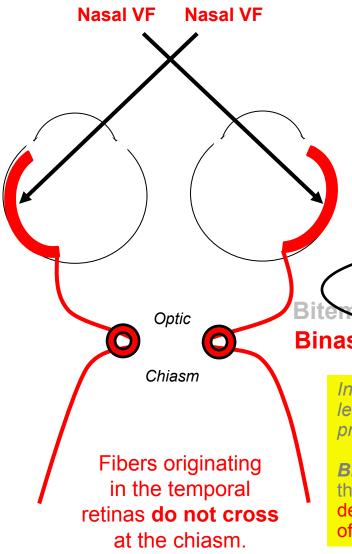
What structures are located at the lateral aspects of the chiasm?

Here's why:

So lesions of the central chiasm will miss these fibers...But lesions of the **lateral** chiasm will bag them, thereby causing binasal defects (note that **two** lesions are required to do this)

Binasal hemianopia: Lateral portions of chiasm

In basic terms, what is the difference between chiasmal lesions resulting in a bitemporal VF defect vs those producing a binasal defect?



What structures are located at the lateral aspects of the chiasm? The internal carotid arteries

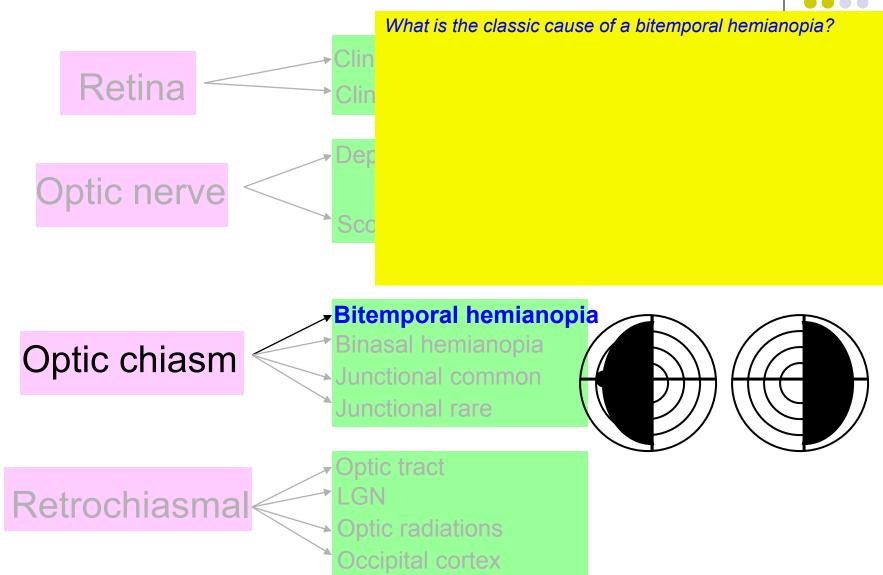
Here's why:

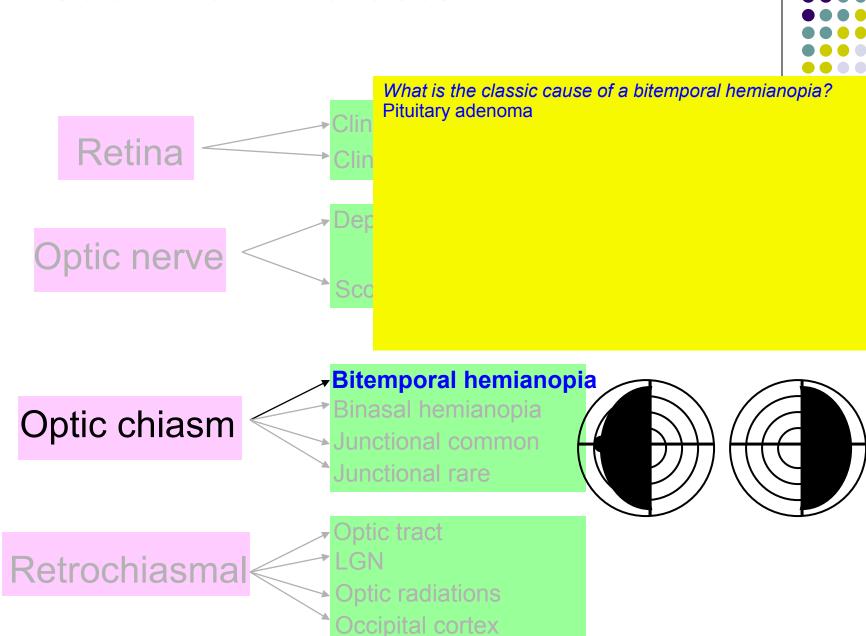
So lesions of the central chiasm will miss these fibers...But lesions of the **lateral** chiasm will bag them, thereby causing binasal defects (note that **two** lesions are required to do this)

Binasal hemianopia: Lateral portions of chiasm

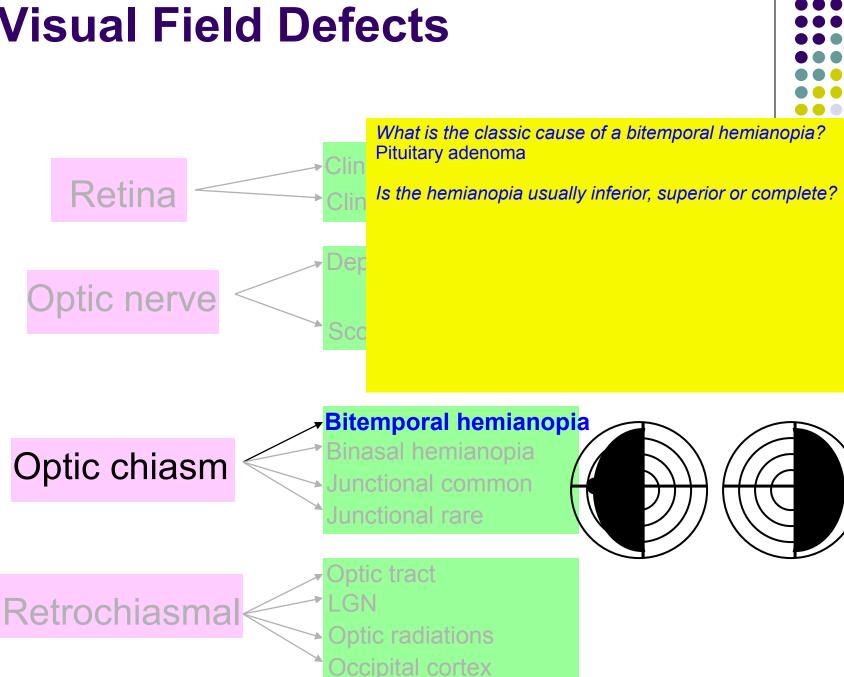
In basic terms, what is the difference between chiasmal lesions resulting in a bitemporal VF defect vs those producing a binasal defect?



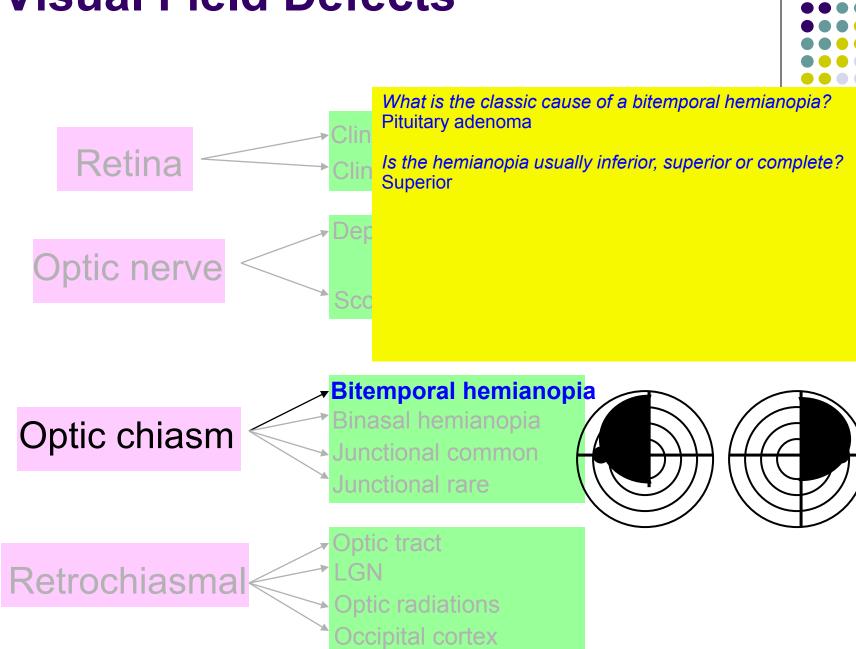


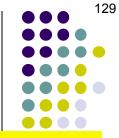


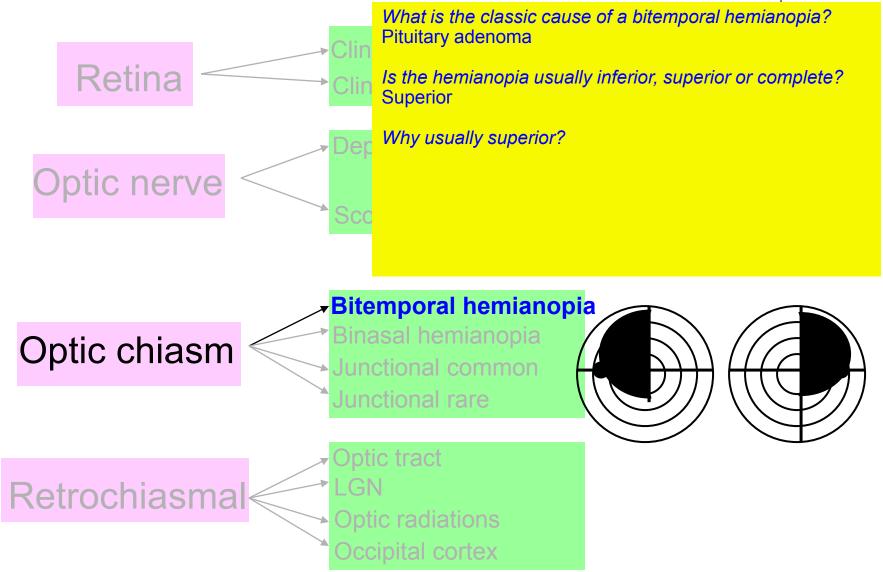
126

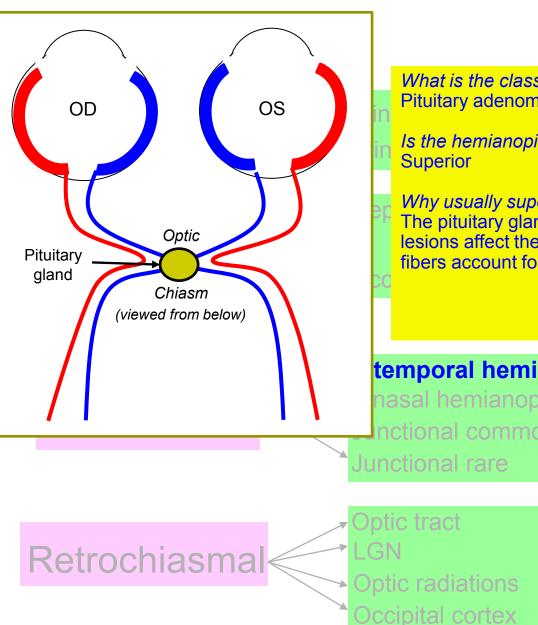


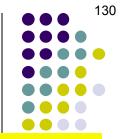
127











What is the classic cause of a bitemporal hemianopia? Pituitary adenoma

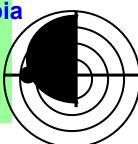
Is the hemianopia usually inferior, superior or complete?

Why usually superior?

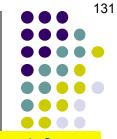
The pituitary gland is **below** the chiasm, therefore, pituitary lesions affect the inferior chiasmal fibers primarily. These fibers account for the **superior** visual field.

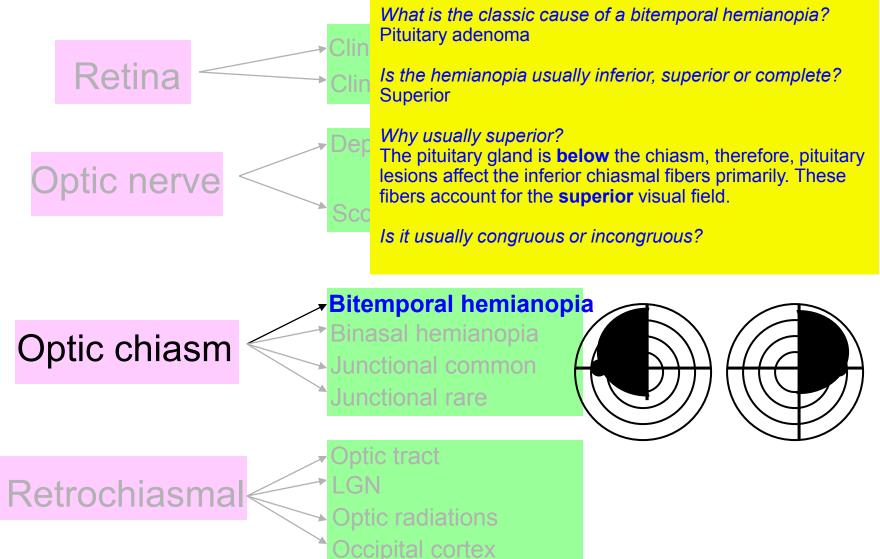
temporal hemianopia

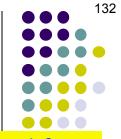
nasal hemianopia Inctional common

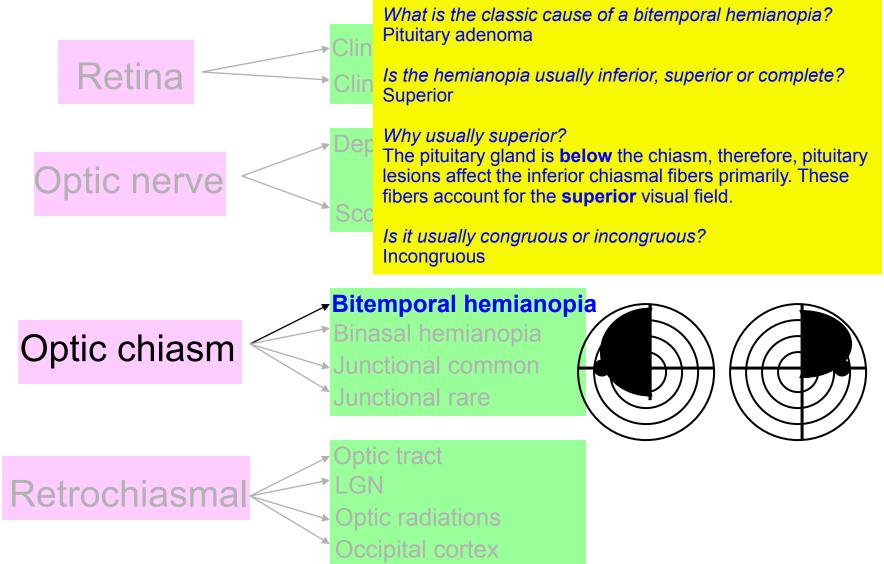




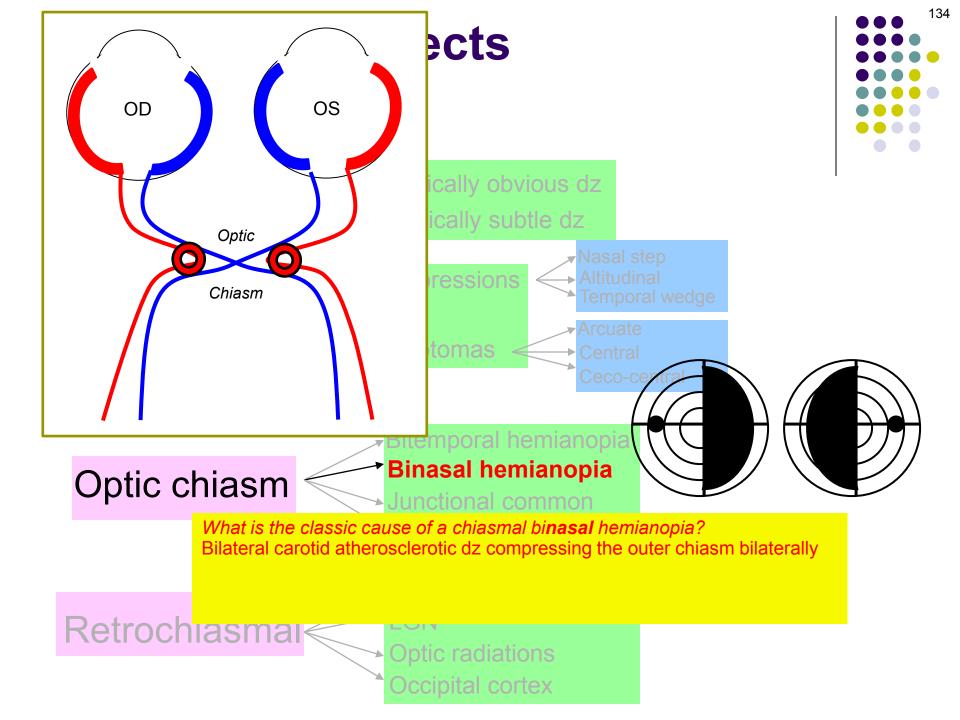




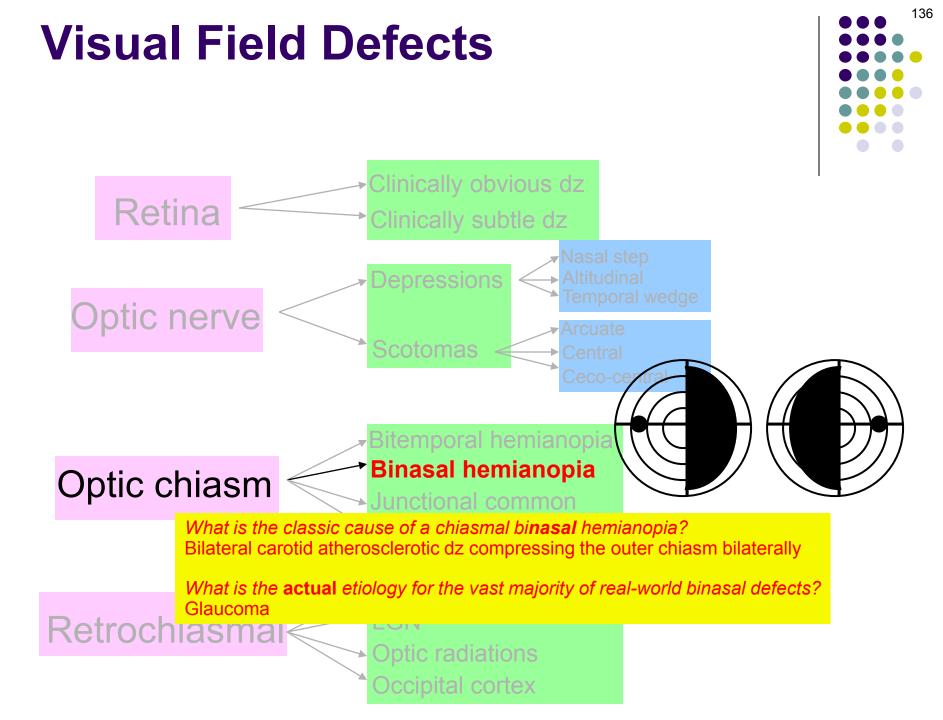


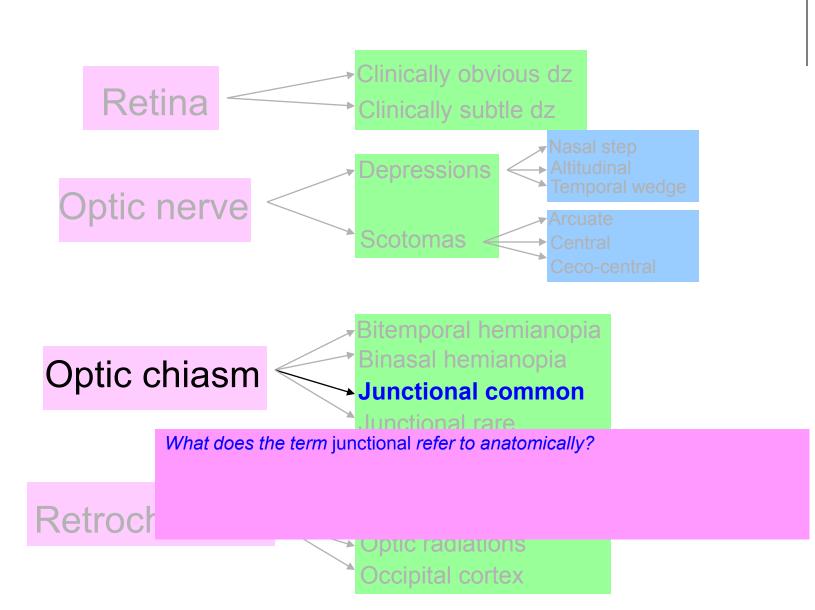


133 Visual Field Defects Clinically obvious dz Retina Clinically subtle dz **Optic nerve** - Arcuate Scotomas Central Bitemporal hemianop **Binasal hemianopia Optic chiasm** Junctional common What is the classic cause of a chiasmal binasal hemianopia? Retrochiasmai **Optic radiations** Occipital cortex

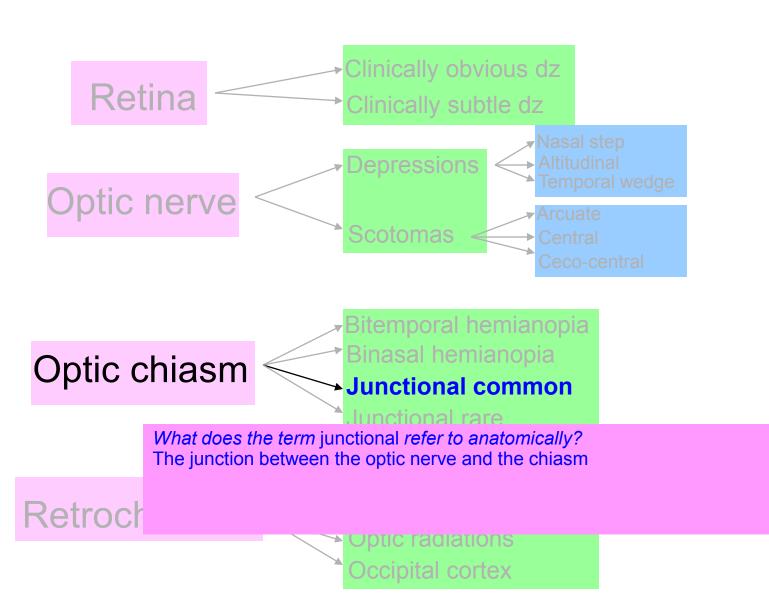


135 Visual Field Defects Clinically obvious dz Retina Clinically subtle dz **Optic nerve** - Arcuate Scotomas Central **Binasal hemianopia Optic chiasm** Junctional common What is the classic cause of a chiasmal binasal hemianopia? Bilateral carotid atherosclerotic dz compressing the outer chiasm bilaterally What is the **actual** etiology for the vast majority of real-world binasal defects? Retrochiasmai **Optic radiations** Occipital cortex

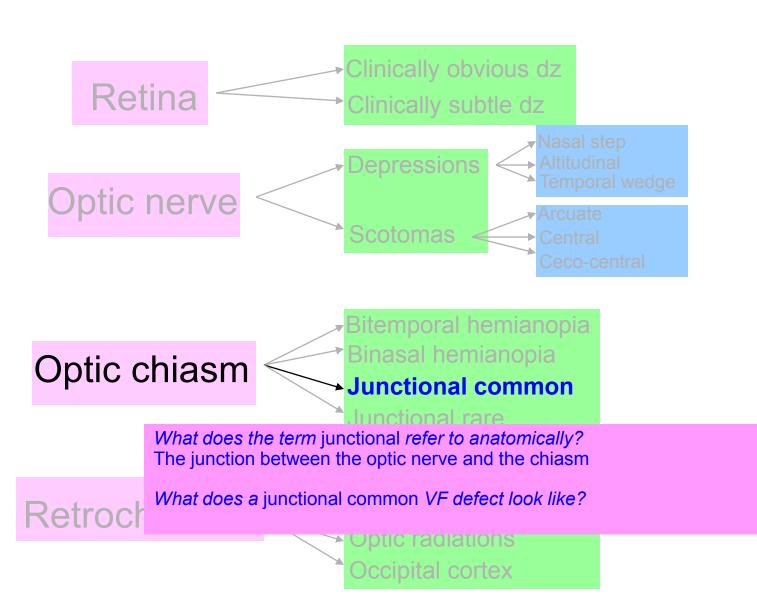




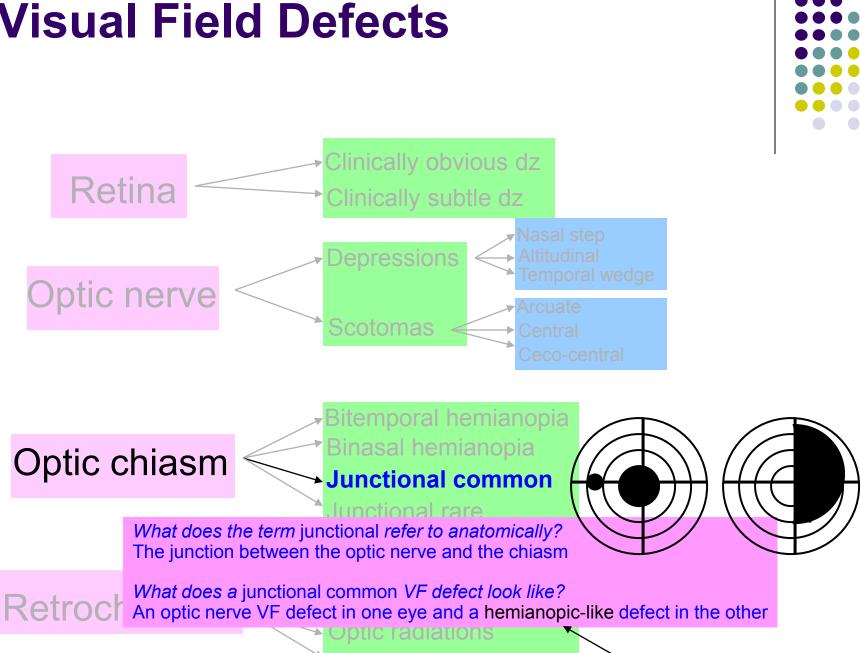








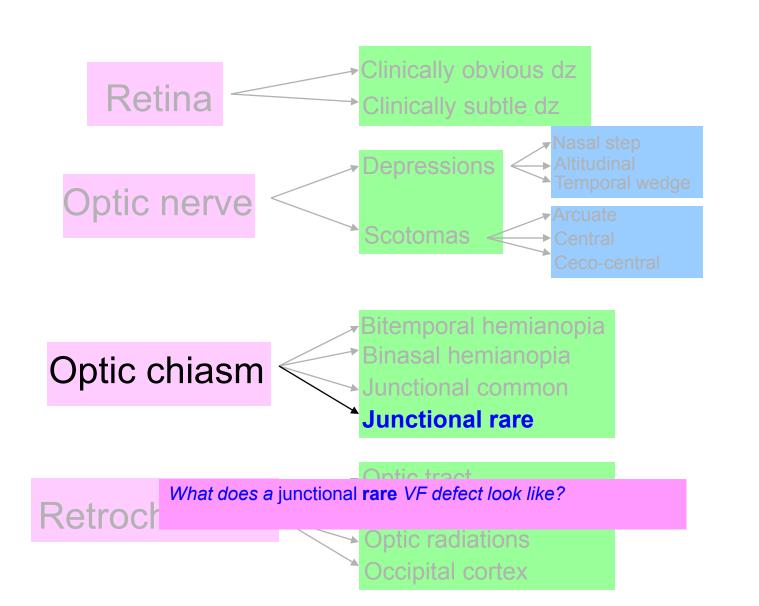




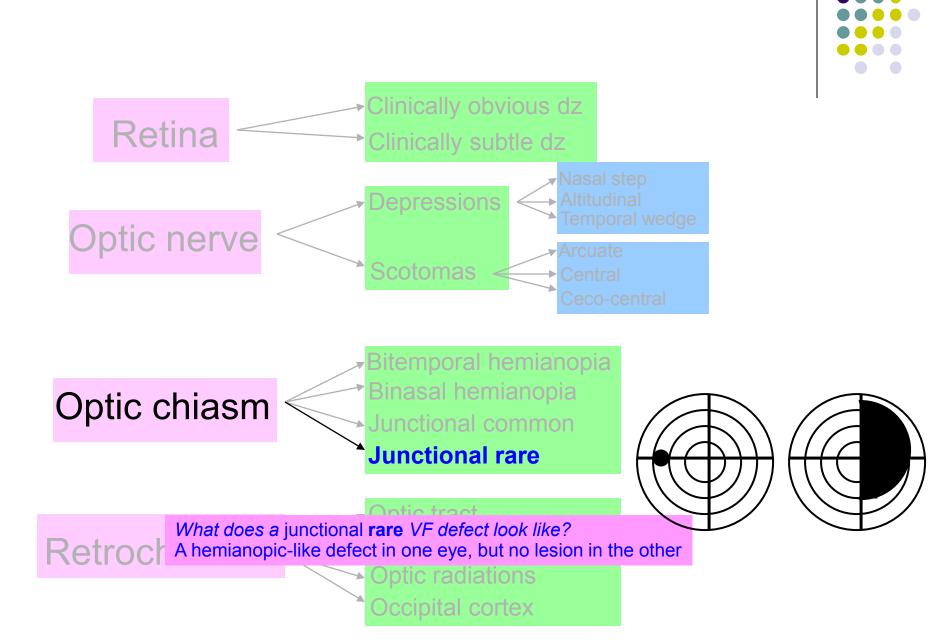
Occipital cortex

i.e., it respects the vertical meridian

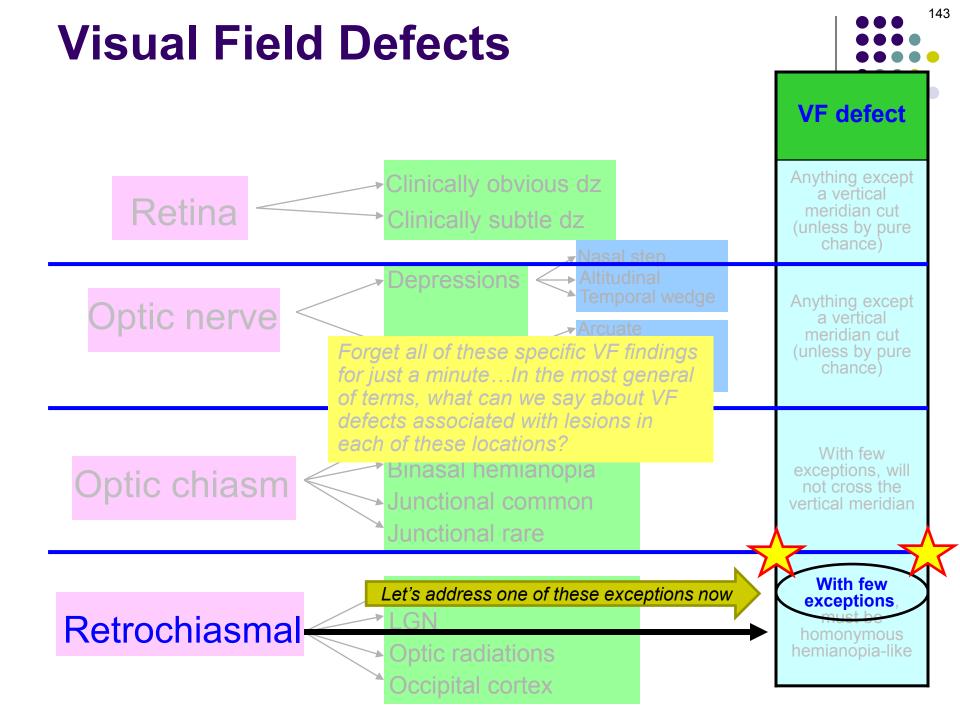
140

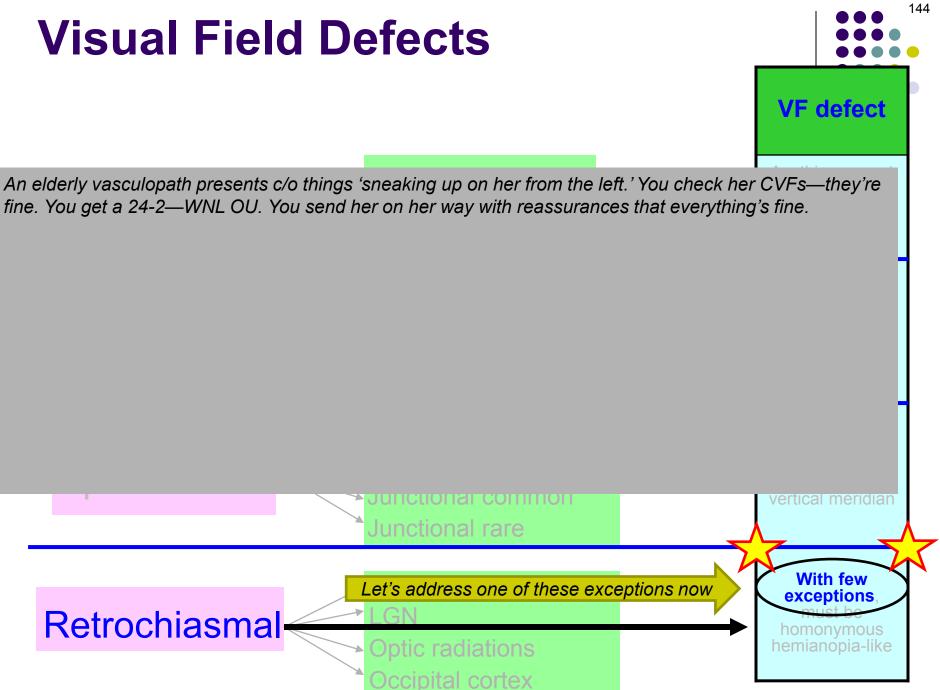






142



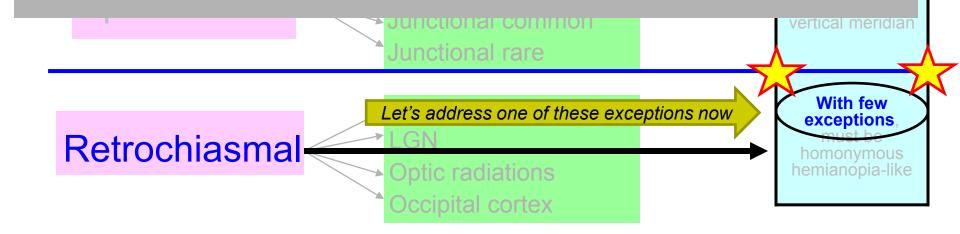


An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Junctional common Junctional rare With few Let's address one of these exceptions now exceptions Retrochiasmalhomonymous hemianopia-like

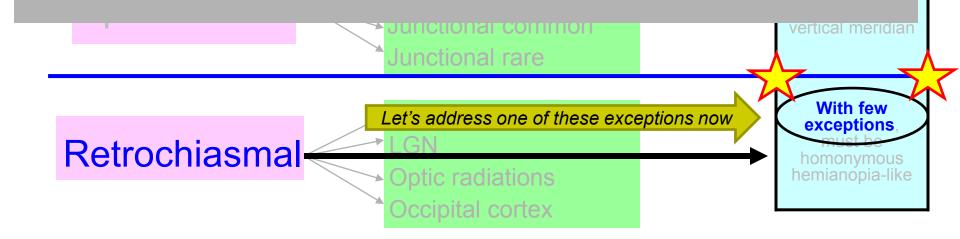
Optic radiations Occipital cortex

An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who...

146

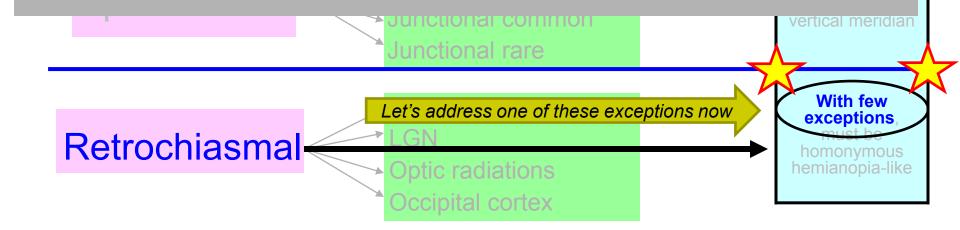


An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who... Sends you a consult note detailing both her VF loss **and** the location of the CNS changes (dx: ischemic CVA) that produced it. What did you miss?

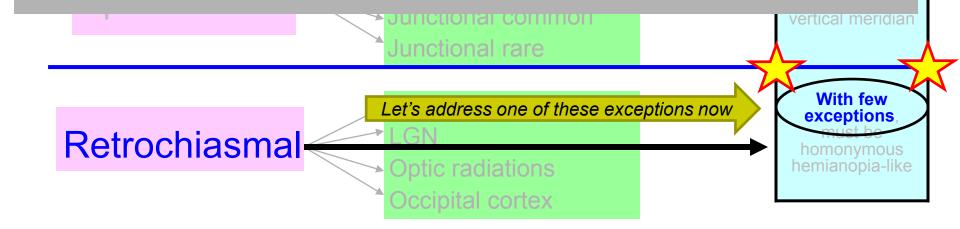


An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who... Sends you a consult note detailing both her VF loss **and** the location of the CNS changes (dx: ischemic CVA) that produced it. What did you miss? You missed a classic case of loss of the two words

148



An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who... Sends you a consult note detailing both her VF loss **and** the location of the CNS changes (dx: ischemic CVA) that produced it. What did you miss? You missed a classic case of loss of the temporal crescent.

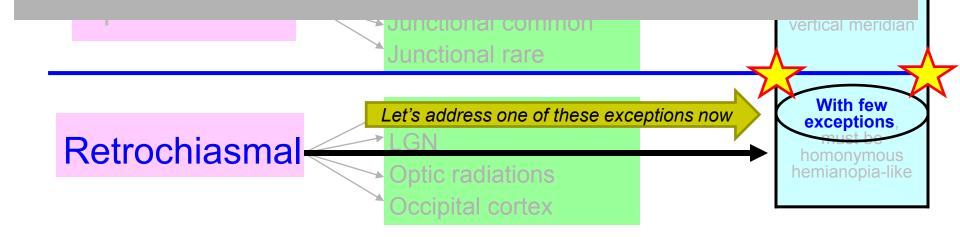


An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who... Sends you a consult note detailing both her VF loss **and** the location of the CNS changes (dx: ischemic CVA) that produced it. What did you miss? You missed a classic case of loss of the temporal crescent. The temporal visual field of the eye ipsilateral

150

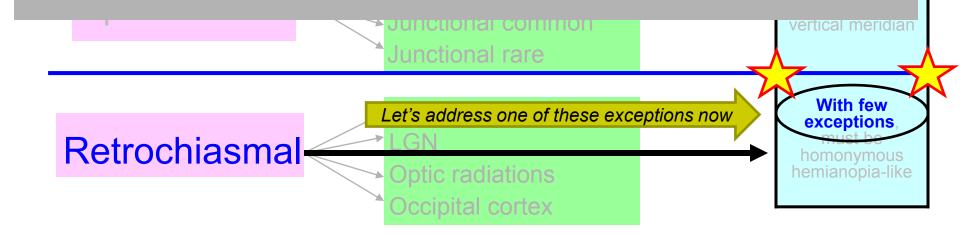
VF defect

to the field in question extends much farther (r deg) than does the contribution from the fellow eye (deg)



An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who... Sends you a consult note detailing both her VF loss **and** the location of the CNS changes (dx: ischemic CVA) that produced it. What did you miss?

You missed a classic case of loss of the temporal crescent. The temporal visual field of the eye ipsilateral to the field in question extends much farther (~ 100°) than does the contribution from the fellow eye (~ 60°).

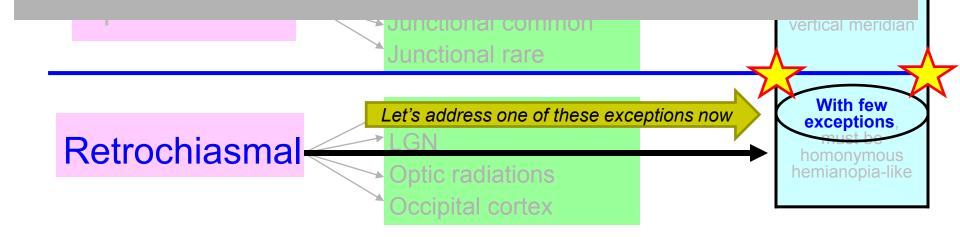


An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who... Sends you a consult note detailing both her VF loss **and** the location of the CNS changes (dx: ischemic CVA) that produced it. What did you miss? You missed a classic case of loss of the temporal crescent. The temporal visual field of the eye ipsilateral

152

VF defect

You missed a classic case of loss of the temporal crescent. The temporal visual field of the eye ipsilateral to the field in question extends much farther ($\sim 100^{\circ}$) than does the contribution from the fellow eye ($\sim 60^{\circ}$). Thus, there is a roughly 40° crescent in the ipsilateral eye that has no corresponding contribution from the fellow eye—it is hemianopia-like, but **not** homonymous.



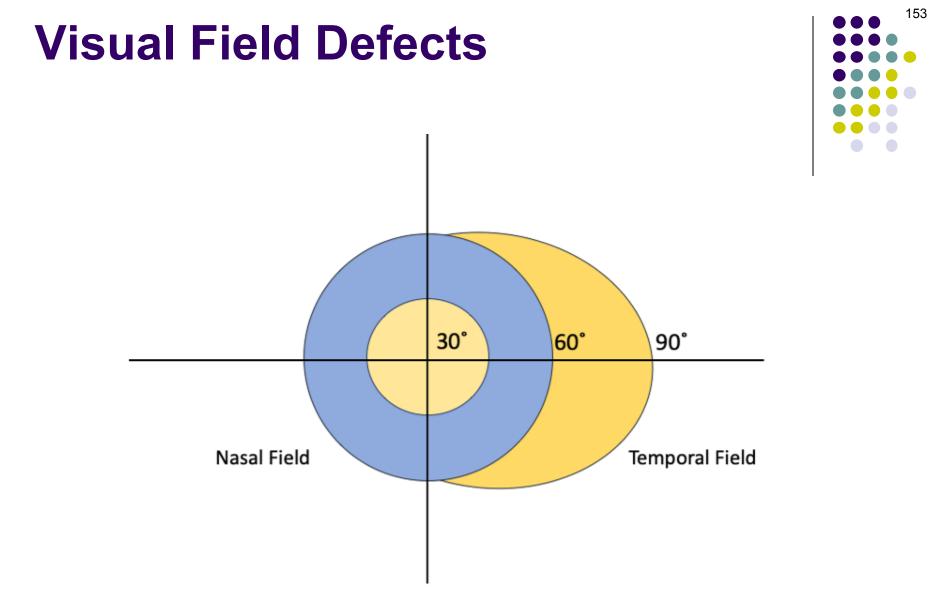


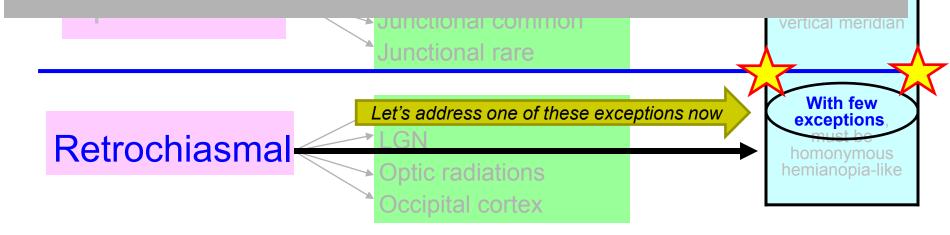
Diagram of the nasal VF (60 degrees) and temporal VF (90-100 degrees). The temporal 60-90° region is the temporal crescent.

An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who... Sends you a consult note detailing both her VF loss **and** the location of the CNS changes (dx: ischemic CVA) that produced it. What did you miss? You missed a classic case of loss of the temporal crescent. The temporal visual field of the eye ipsilateral to the field in question extends much farther (~ 100°) than does the contribution from the fellow eye (~ 60°).

154

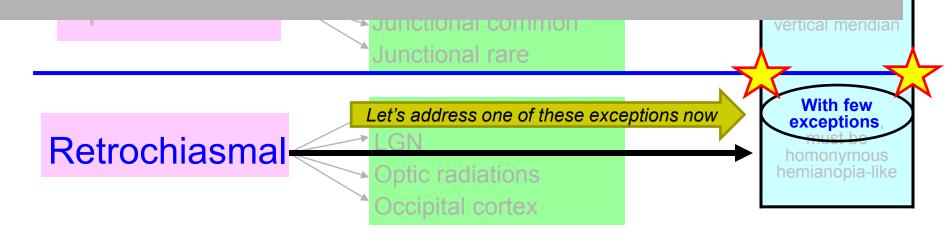
VF defect

Thus, there is a roughly 40° crescent in the ipsilateral eye that has no corresponding contribution from the fellow eye—it is hemianopia-like, but **not** homonymous. No commercially-available automated VF analyzer is capable of detecting a temporal crescent; the only technology that can is perimetry.



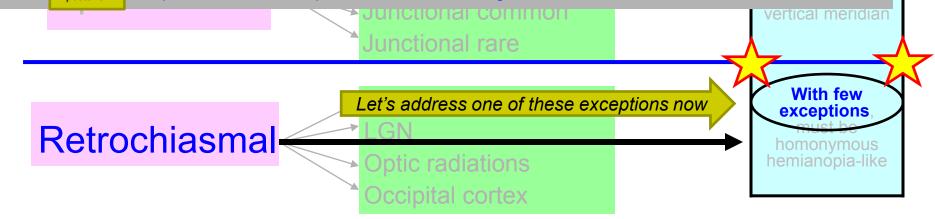
An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who... Sends you a consult note detailing both her VF loss **and** the location of the CNS changes (dx: ischemic CVA) that produced it. What did you miss?

You missed a classic case of loss of the temporal crescent. The temporal visual field of the eye ipsilateral to the field in question extends much farther (~ 100°) than does the contribution from the fellow eye (~ 60°). Thus, there is a roughly 40° crescent in the ipsilateral eye that has no corresponding contribution from the fellow eye—it is hemianopia-like, but **not** homonymous. No commercially-available automated VF analyzer is capable of detecting a temporal crescent; the only technology that can is Goldmann perimetry.



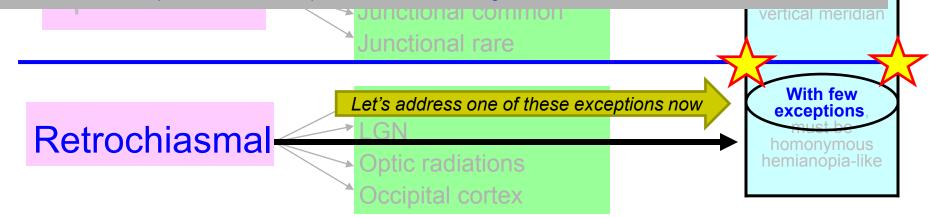
An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who... Sends you a consult note detailing both her VF loss **and** the location of the CNS changes (dx: ischemic CVA) that produced it. What did you miss?

You missed a classic case of loss of the temporal crescent. The temporal visual field of the eye ipsilateral to the field in question extends much farther (~ 100°) than does the contribution from the fellow eye (~ 60°). Thus, there is a roughly 40° crescent in the ipsilateral eye that has no corresponding contribution from the fellow eye—it is hemianopia-like, but **not** homonymous. No commercially-available automated VF analyzer is capable of detecting a temporal crescent; the only technology that can is Goldmann perimetry. Lesions of the anterior vs occipital cortex are responsible for this finding.

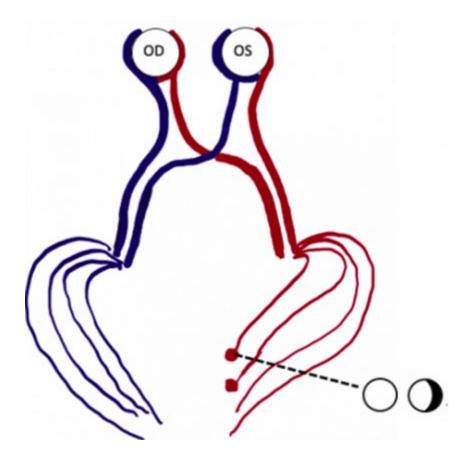


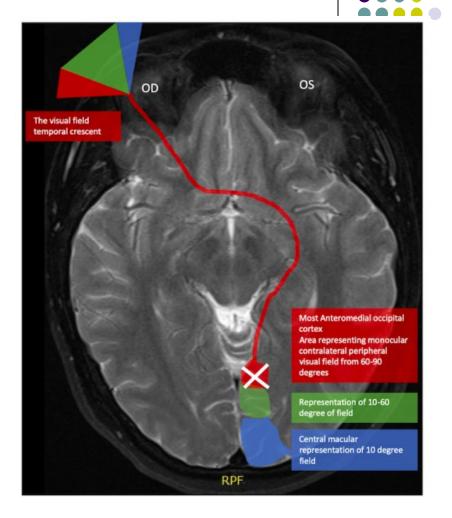
An elderly vasculopath presents c/o things 'sneaking up on her from the left.' You check her CVFs—they're fine. You get a 24-2—WNL OU. You send her on her way with reassurances that everything's fine. Two weeks later she's on your schedule again with the same complaint. Just to be sure you repeat the VF, but with the rarely-used 30-2 protocol—again, WNL. Reassure, discharge. Back the next week. Sigh. This visit, for the first time in your career, you order the never-used 60-2 protocol—again, nothing. YOU'RE FINE, YOU CRAZY OLD BAT! you yell at her. (Not really.) To placate her, you refer her to a neuro-oph, who... Sends you a consult note detailing both her VF loss **and** the location of the CNS changes (dx: ischemic CVA) that produced it. What did you miss?

You missed a classic case of loss of the temporal crescent. The temporal visual field of the eye ipsilateral to the field in question extends much farther (~ 100°) than does the contribution from the fellow eye (~ 60°). Thus, there is a roughly 40° crescent in the ipsilateral eye that has no corresponding contribution from the fellow eye—it is hemianopia-like, but **not** homonymous. No commercially-available automated VF analyzer is capable of detecting a temporal crescent; the only technology that can is Goldmann perimetry. Lesions of the anterior occipital cortex are responsible for this finding.



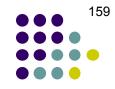
157





Images showcasing the location of a lesion producing Temporal Crescent Syndrome





- Which of the following is *not* associated with bitemporal visual-field loss?
 - Sectoral RP
 - Glaucoma
 - Fuchs coloboma
 - Chiasmal lesion
 - Toxic/hereditary/nutritional optic neuropathy





- Which of the following is *not* associated with bitemporal visual-field loss?
 - Sectoral RP
 - Glaucoma
 - Fuchs coloboma
 - Chiasmal lesion
 - Toxic/hereditary/nutritional optic neuropathy
- Glaucoma. Hemianopic (= respects the vertical midline) bitemporal VF loss is associated exclusively with *lesions compressing the chiasm*, specifically the mid-vs lateral chiasm.



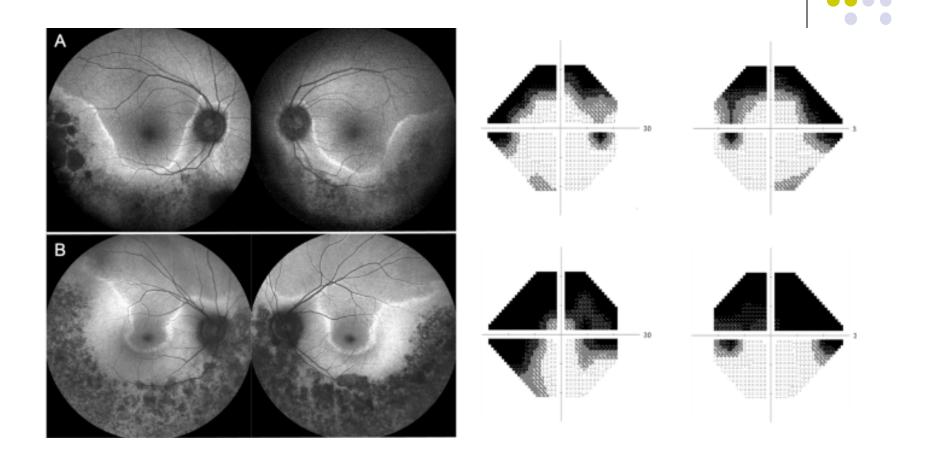


- Which of the following is *not* associated with bitemporal visual-field loss?
 - Sectoral RP
 - Glaucoma
 - Fuchs coloboma
 - Chiasmal lesion
 - Toxic/hereditary/nutritional optic neuropathy
- Glaucoma. Hemianopic (= respects the vertical midline) bitemporal VF loss is associated exclusively with *lesions compressing the chiasm*, specifically the mid-chiasm.





- Which of the following is *not* associated with bitemporal visual-field loss?
 - Sectoral RP
 - Glaucoma
 - Fuchs coloboma
 - Chiasmal lesion
 - Toxic/hereditary/nutritional optic neuropathy
- *Glaucoma*. Hemianopic (= respects the vertical midline) bitemporal VF loss is associated exclusively with *lesions compressing the chiasm*, specifically the mid-chiasm. Other causes of bitemporal loss do not respect the midline (except by happenstance). *Sectoral RP* is symmetric bilaterally, and thus can affect the temporal VF bilaterally.



Sectoral RP

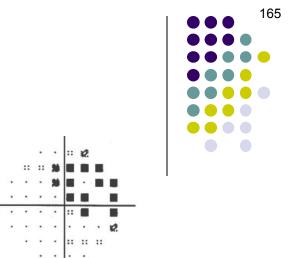
Images from JP Marques et al, *EYS*-Associated Sector Retinitis Pigmentosa. *Graefe's Archives for Clinical and Experimental Ophthalmology*, 2022 Apr;260(4):1405-1413. With kind permission of the first author.

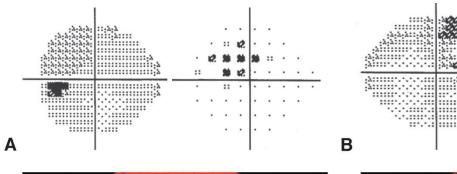
163



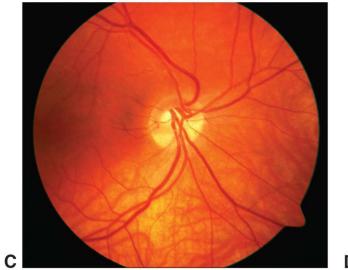


- Which of the following is *not* associated with bitemporal visual-field loss?
 - Sectoral RP
 - Glaucoma
 - Fuchs coloboma
 - Chiasmal lesion
 - Toxic/hereditary/nutritional optic neuropathy
- Glaucoma. Hemianopic (= respects the vertical midline) bitemporal VF loss is associated exclusively with *lesions compressing the chiasm*, specifically the mid-chiasm. Other causes of bitemporal loss do not respect the midline (except by happenstance). Sectoral RP is symmetric bilaterally, and thus can affect the temporal VF bilaterally. *Fuchs coloboma* (aka *tilted disc syndrome*) is associated with bitemporal loss that resolves with proper correction.







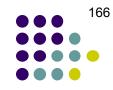


Tilted disc: Superior bitemporal VF defects

.....

·····





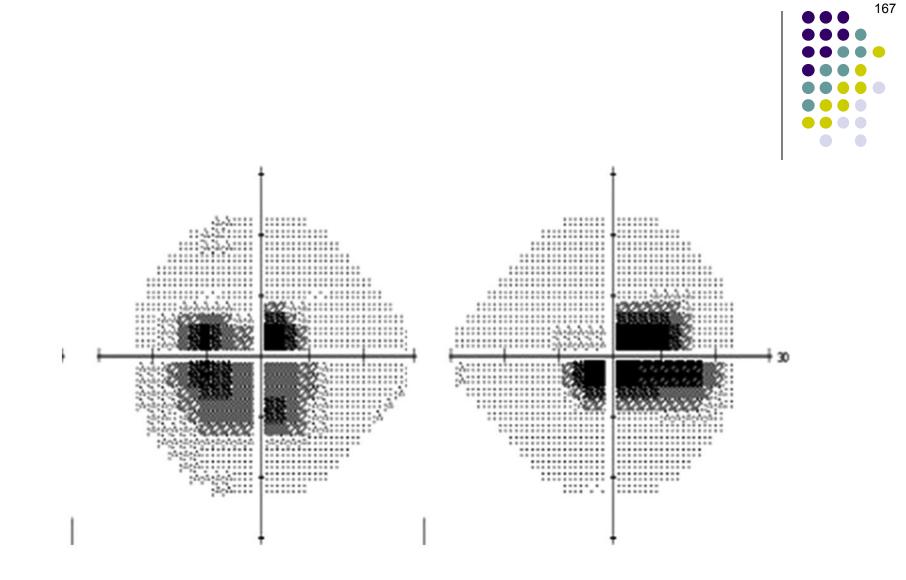
• Which of the following is *not* associated with bitemporal visual-field loss?

- Sectoral RP
- Glaucoma
- Fuchs coloboma
- Chiasmal lesion

• Toxic/hereditary/nutritional optic neuropathy

• *Glaucoma*. Hemianopic (= respects the vertical midline) bitemporal VF loss is associated exclusively with *lesions compressing the chiasm*, specifically the mid-chiasm. Other causes of bitemporal loss do not respect the midline (except by happenstance). *Sectoral RP* is symmetric bilaterally, and thus can affect the temporal VF bilaterally. *Fuchs coloboma* (aka *tilted disc syndrome*) is associated with bitemporal loss that resolves with proper correction.

Toxic/hereditary/nutritional optic neuropathy is associated with bilateral cecocentral VF loss, which can mimic bitemporal loss.



Visual field defects characteristic of toxic and metabolic optic neuropathies





- Which of the following is *not* associated with bitemporal visual-field loss?
 - Sectoral RP
 - Glaucoma
 - Fuchs coloboma
 - Chiasmal lesion
 - Toxic/hereditary/nutritional optic neuropathy
- *Glaucoma*. Hemianopic (= respects the vertical midline) bitemporal VF loss is associated exclusively with *lesions compressing the chiasm*, specifically the mid-chiasm. Other causes of bitemporal loss do not respect the midline (except by happenstance). Sectoral RP is symmetric bilaterally, and thus can affect the temporal VF bilaterally. Fuchs coloboma (aka tilted disc syndrome) is associated with bitemporal loss that resolves with proper correction. Toxic/hereditary/nutritional optic neuropathy is associated with bilateral cecocentral VF loss, which can mimic bitemporal loss. Glaucoma almost always affects the nasal VF long before the temporal field is involved—if anything, glaucoma is far more likely to cause bi**nasal** VF loss (although this is a very rare occurrence).





- Which of the following is *not* associated with bitemporal visual-field loss?
 - Sectoral RP
 - Glaucoma

Fuchs coloboma (aka *tilted disc syndrome*) is associated with bitemporal loss that resolves with proper correction.





- Which of the following is not associated with bitemporal visual-field loss?
 - Sectoral RP
 - Glaucoma

It's actually pretty straightforward.

The area including and adjacent to the inferior pole of a tilted disc is staphylomatous. This means the 'axial length' of the photoreceptors within this region is greater than that of the rest of the posterior pole. Because of this extra axial length, the correction used during VF testing (which is based on the refraction of the non-staphylomatous fovea) is not myopic enough for the inferior peripapillary region.

Fuchs coloboma (aka tilted disc syndrome) is associated with

bitemporal loss that resolves with proper correction.





- Which of the following is not associated with bitemporal visual-field loss?
 - Sectoral RP
 - Glaucoma

It's actually pretty straightforward.

The area including and adjacent to the inferior pole of a tilted disc is staphylomatous. This means the 'axial length' of the photoreceptors within this region is greater than that of the rest of the posterior pole. Because of this extra axial length, the correction used during VF testing (which is based on the refraction of the non-staphylomatous fovea) is not myopic enough for the inferior peripapillary region. Because this region is out of focus, it will manifest a *refractive scotoma* on the test. And because the retina involved in this scotoma is **inferonasal** to the fovea, it follows that the resulting VF defect will be **superotemporal** to fixation.

Fuchs coloboma (aka tilted disc syndrome) is associated with

bitemporal loss that resolves with proper correction.





- Which of the following is not associated with bitemporal visual-field loss?
 - Sectoral RP
 - Glaucoma

It's actually pretty straightforward.

The area including and adjacent to the inferior pole of a tilted disc is staphylomatous. This means the 'axial length' of the photoreceptors within this region is greater than that of the rest of the posterior pole. Because of this extra axial length, the correction used during VF testing (which is based on the refraction of the non-staphylomatous fovea) is not myopic enough for the inferior peripapillary region. Because this region is out of focus, it will manifest a *refractive scotoma* on the test. And because the retina involved in this scotoma is **inferonasal** to the fovea, it follows that the resulting VF defect will be **superotemporal** to fixation. And, as Fuchs coloboma is virtually always bilateral, these superotemporal VF defects are present *bilaterally*.

Fuchs coloboma (aka tilted disc syndrome) is associated with

bitemporal loss that resolves with proper correction.