Define glaucoma.
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A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss
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Why isn’t elevated IOP mentioned above?
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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
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In addition to being the strongest risk factor for glaucoma, IOP has another quality that renders it unique—what is it?
Define glaucoma.  
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Why isn’t elevated IOP mentioned above?  
Elevated IOP is a strong risk factor for glaucoma, but it need not be present—IOP can be normal, or even low.

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

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

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*That’s why glaucoma management concerns nothing but IOP-lowering maneuvers!*
Define glaucoma.
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Elevated IOP is a strong risk factor for glaucoma, but it need not be present—IOP can be normal, or even low

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

Speaking of IOP... Let's drill down on the factors that determine it
Fill in the IOP equation below.

\[
IOP = \text{Aqueous Formation Rate (µL/min)} + \text{Episcleral Venous Pressure (EVP)}
\]
Fill in the IOP equation below.

\[ IOP = \frac{\text{Aqueous Formation Rate (} \mu\text{L/min)} }{\text{Outflow Facility (} \mu\text{L/min/mmHg)}} + \text{Episceral Venous Pressure (mmHg)} \]
Fill in the IOP equation below. What is its eponymous name?

The equation

\[ IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episceral Venous Pressure (mmHg)} \]
Fill in the IOP equation below. What is its eponymous name? The **Goldmann equation**

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IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}
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The Goldmann equation

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

Note how the \(\mu L/\text{min}\) cancel, leaving IOP in \(mmHg\)
Fill in the IOP equation below. What is its eponymous name?

The Goldmann equation

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

Episcleral venous pressure (EVP) normally measures about # to # mmHg (ie, the same as central venous pressure) in an upright pt.
Fill in the IOP equation below. What is its eponymous name?

The Goldmann equation

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

Episcleral venous pressure (EVP) normally measures about 8-12 mmHg (ie, the same as central venous pressure) in an upright pt.
Fill in the IOP equation below. What is its eponymous name?

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

No question—proceed when ready
Fill in the IOP equation below. What is its eponymous name?
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Fill in the IOP equation below. What is its eponymous name? The Goldmann equation

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IOP = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}
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So to lower IOP, one must:
- decrease aqueous formation
- increase outflow facility
- decrease episcleral venous pressure
- and/or dehydrate the vitreous with a hyperosmotic agent

Three maneuvers implied by the Goldmann equation:
- the numerator
- the denominator
- the other thing
Fill in the IOP equation below. What is its eponymous name? The \textbf{Goldmann equation}

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So to lower IOP, one must:

\[ \begin{align*}
&\text{---decrease aqueous formation, and/or} \\
&\text{---increase outflow facility, and/or} \\
&\text{---decrease episcleral venous pressure}
\end{align*} \]
Fill in the IOP equation below. What is its eponymous name?

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So to lower IOP, one must:

- decrease aqueous formation, **and/or**
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- decrease episcleral venous pressure

Three maneuvers implied by the Goldmann equation

- one word
- three words

Important maneuver **not** implied by the Goldmann equation

... **and/or** with a **agent**
Fill in the IOP equation below. **What is its eponymous name?**

**The Goldmann equation**

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IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}
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So to lower IOP, one must:

1. decrease aqueous formation, *and/or*
2. increase outflow facility, *and/or*
3. decrease episcleral venous pressure

*Three maneuvers implied by the Goldmann equation*

*Important maneuver not implied by the Goldmann equation* ...

... *and/or dehydrate the vitreous* with a *hyperosmotic* agent
Which classes of meds decrease aqueous formation?
-- β blockers
-- CAIs
-- α agonists

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What are the two types of outflow?
- Trabecular meshwork
- Uveoscleral
Fill in the IOP equation below. What is its eponymous name? The Goldmann equation

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Obviously, aqueous-humor dynamics play a central role in glaucoma. Let’s delve into its production…

Which classes of meds decrease aqueous formation?

--β blockers
--CAIs
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What are the two types of outflow?

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Glaucoma Overview

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

The Goldmann equation
Fill in the IOP equation below. What is its eponymous name?
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\[ \text{IOP} = \text{Aqueous Formation Rate (\(\mu\text{L/min}\))} + \text{Outflow Facility (\(\mu\text{L/min/mmHg}\)) + Episceral Venous Pressure (mmHg)} \]

What is the rate of aqueous formation?
Fill in the IOP equation below. What is its eponymous name? The Goldmann equation

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

What is the rate of aqueous formation?
2-3 µL/min
Fill in the IOP equation below. What is its eponymous name? The **Goldmann equation**

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

What is the aqueous volume of the anterior chamber?
Fill in the IOP equation below. What is its eponymous name?
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*What is the rate of aqueous formation?*
2-3 µL/min

*What is the aqueous volume of the anterior chamber?*
200-300 µL
Fill in the IOP equation below. **What is its eponymous name?**

The **Goldmann equation**

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

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

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

So then, **what percent of AC volume is ‘turned over’ every minute?**
Fill in the IOP equation below. What is its eponymous name? The Goldmann equation

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About 1%
Fill in the IOP equation below. What is its eponymous name? The Goldmann equation

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Roughly 100 minutes
So to lower IOP, one must:

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Speaking of aqueous formation…What specific tissue makes aqueous?

The nonpigmented epithelium of the pars plicata portion of the ciliary body

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

The Goldmann equation
To lower IOP, one must:

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The IOP equation is:

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

The eponymous name for this equation is the **Goldmann equation**.

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**What specific tissue makes aqueous?**

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Ciliary body: One perspective, two questions
Ciliary body: One perspective, two questions

This part is the…Pars plicata

This part is the…Pars plana
Ciliary body: Another perspective
Ciliary body: Another
Glaucoma Overview

Ciliary body: Another
Now let’s look at the CB epithelium. Low power photomicrograph.
Now let’s look at the CB epithelium. *Higher.*
*Speaking of aqueous formation... What specific tissue makes aqueous?*

The *nonpigmented* epithelium of the pars plicata portion of the ciliary body.

*What is implied by the fact that aqueous is made by the 'nonpigmented' epithelium?*

The presence of a *pigmented* epithelium.

---

Now let’s look at the CB epithelium. **High.**
Q

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

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

**Neuroectoderm**

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

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Neuroectoderm

What other portion of the eye derives from neuroectoderm?
From what embryonic tissue do the two epithelia of the CB derive?

**Neuroectoderm**

What other portion of the eye derives from neuroectoderm?

The retina (ie, the neurosensory retina + RPE)

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

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

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How are the two epithelial layers of the CB oriented with respect to one another?

Speaking of aqueous formation...What specific tissue makes aqueous?  
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**Glaucoma Overview**

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The same way--apex-to-apex

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Which CB epithelial layer is pigmented--the inner, or the outer?

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Which CB epithelial layer is pigmented--the inner, or the outer?
The outer
From what embryonic tissue do the two epithelia of the CB derive?

**Neuroectoderm**

What other portion of the eye derives from neuroectoderm?

**The retina (ie, the neurosensory retina + RPE)**

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

**Apex-to-apex**

How are the two epithelial layers of the CB oriented with respect to one another?

**The same way--apex-to-apex**

Which CB epithelial layer is pigmented--the inner, or the outer?

**The outer**

Which portion of the retina is contiguous with the pigmented layer of the CB epithelium?

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

The **nonpigmented** epithelium of the pars plicata portion of the ciliary body.

What is implied by the fact that aqueous is made by the 'nonpigmented' epithelium?

The presence of a **pigmented** epithelium.
Glaucoma Overview

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

What other portion of the eye derives from neuroectoderm?
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How are the neurosensory retinal and RPE cells oriented with respect to one another?
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Which CB epithelial layer is pigmented--the inner, or the outer?
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Which portion of the retina is contiguous with the pigmented layer of the CB epithelium?
The RPE, ie, the outer
Embryology of the optic vesicle as it invaginates. What you want to take note of is:

-- The pigmented layer of the CB epi derives from the same structure as the RPE (which is, as its name indicates, also pigmented), and
-- the nonpigmented CB epi derives from the same structure that gives rise to the neurosensory retina.

In other words, what you already know about eye anatomy can help you understand and remember eye embryology. (For more, see the Embryology made simply ridiculous slide-set.)

Note: This vesicle...

...not this one!
Embryology of the optic vesicle as it invaginates. What you want to take note of is:

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

The same way--apex-to-apex.

Which CB epithelial layer is pigmented--the inner, or the outer? The outer.

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

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Silly rabbit—mistakes are for residents! Remember, for us eye dentists the terms *inner* and *outer* are in relation to the globe itself; ie, inner means ‘closer to, or of, the inner aspect of the globe.’

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*Which CB epithelial layer is pigmented--the inner, or the outer?*

That’s what I said--the outer!

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Glaucoma Overview

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Silly rabbit—mistakes are for residents! Remember, for us eye dentists the terms *inner* and *outer* are in relation to the **globe itself**; ie, inner means ‘closer to, or of, the inner aspect of the globe.’ Because the nonpigmented layer faces the vitreous cavity, it is the ‘inner’ layer of the two.

The same way--apex-to-apex.

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

Which portion of the retina is contiguous with the pigmented layer of the CB epithelium?
The RPE, ie the outer.
So to lower IOP, one must:
--decrease aqueous formation,
and/or
--increase outflow facility,
and/or
--decrease episcleral venous pressure
...and/or dehydrate the vitreous with a hyperosmotic agent

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

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

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

What are the two types of outflow?
--Trabecular meshwork
--Uveoscleral

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

So to lower IOP, one must:
--decrease aqueous formation, and/or
--increase outflow facility, and/or
--decrease episcleral venous pressure
Based on the two-name eponym principle: $P = \frac{F}{A}$

(P is for Pressure)
Based on the *Imbert-Fick* principle: \[ P = \frac{F}{A} \]
Based on the **Imbert-Fick principle**: \( P = \frac{F}{A} \)

- Pressure inside a sphere equals force needed to flatten its surface divided by the area of flattening.

- \( F \) stands for...
- \( A \) stands for...

**Principle in words**: ...
Based on the *Imbert-Fick principle*: \[ P = \frac{F}{A} \]

- Pressure inside a sphere equals the force needed to flatten its surface divided by the area of flattening.
Based on the **Imbert-Fick principle**: $P = \frac{F}{A}$

- Pressure inside a sphere equals force needed to flatten its surface divided by the area of flattening.
- Assumes surface is two words, and (cornea is neither, obviously).
Based on the **Imbert-Fick principle**: \( P = \frac{F}{A} \)

- Pressure inside a sphere equals **force needed to flatten its surface** divided by the **area of flattening**
- Assumes surface is **infinitely thin**, and **dry** (cornea is neither, obviously)
Based on the [Imbert-Fick principle](#): \( P = \frac{F}{A} \)

- Pressure inside a sphere equals the [force needed to flatten its surface](#) divided by the [area of flattening](#).
- Assumes surface is [infinitely thin](#) and [dry](#) (cornea is neither, obviously).
- K thickness → [resists applanation](#) → increase vs decrease IOP reading.
Based on the **Imbert-Fick principle**: \( P = \frac{F}{A} \)

- Pressure inside a sphere equals **force needed to flatten its surface** divided by the **area of flattening**
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For the Imbert-Fick principle to hold, the **only** force resisting applanation should be the pressure within the sphere. However, real objects such as the cornea have **intrinsic** resistance to deformation owing to their physical nature, ie, because they’re made of ‘stuff.’
Based on the *Imbert-Fick* principle: \( P = \frac{F}{A} \)

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

*No question—proceed when ready*
Based on the *Imbert-Fick principle*: \( P = \frac{F}{A} \)

- Pressure inside a sphere equals *force needed to flatten its surface* divided by the *area of flattening*.
- Assumes surface is *infinitely thin*, and *dry* (cornea is neither, obviously).
  - \( K \) thickness \( \rightarrow \) resists applanation \( \rightarrow \) *increases* IOP reading
  - Tear film \( \rightarrow \) capillary attraction \( \rightarrow \) IOP reading
Based on the *Imbert-Fick* principle: \( P = \frac{F}{A} \)

- Pressure inside a sphere equals force needed to flatten its surface divided by the area of flattening.
- Assumes surface is infinitely thin, and dry (cornea is neither, obviously).
  - K thickness → resists applanation → *increases* IOP reading.
  - Tear film → capillary attraction → *decreases* IOP reading.
Based on the *Imbert-Fick* principle: \( P = \frac{F}{A} \)

- Pressure inside a sphere equals force needed to flatten its surface divided by the area of flattening.
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- \( K \) thickness → resists applanation → *increases* IOP reading
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*On the other hand:* The first ocular structure encountered by the applanator tip is the tear film. When contact with the tear film is made, a fluid bridge forms between the cornea and the tip. Surface tension of the water in this fluid bridge produces *capillary attraction*, which exerts a slight ‘pull’ on the applanator tip, drawing it toward the cornea.
Based on the **Imbert-Fick principle**: \( P = \frac{F}{A} \)

- Pressure inside a sphere equals force needed to flatten its surface divided by the area of flattening.
- Assumes surface is **infinitely thin**, and **dry** (cornea is neither, obviously).
- K thickness ➞ resists applanation ➞ *increases* IOP reading
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*On the other hand:* The first ocular structure encountered by the applanator tip is the tear film. When contact with the tear film is made, a fluid bridge forms between the cornea and the tip. Surface tension of the water in this fluid bridge produces *capillary attraction*, which exerts a slight ‘pull’ on the applanator tip, drawing it toward the cornea. Because this force is drawing the applanator tip forward, it is causing the pressure reading to be falsely **low**.

No question—proceed when ready
Based on the *Imbert-Fick* principle: \[ P = \frac{F}{A} \]

- Pressure inside a sphere equals force needed to flatten its surface divided by the area of flattening.
- Assumes surface is infinitely thin, and dry (cornea is neither, obviously).

- \( K \) thickness → resists applanation → *increases* IOP reading
- Tear film → capillary attraction → *decreases* IOP reading

*To be useful, an applanator-type device has to account for these factors.* Fortunately, the brilliant Dr. Goldmann was (mostly) up to the challenge…
Based on the *Imbert-Fick* principle: \[ P = \frac{F}{A} \]

- Pressure inside a sphere equals force needed to flatten its surface divided by the area of flattening.
- Assumes surface is infinitely thin, and dry (cornea is neither, obviously).
- K thickness → resists applanation → increases IOP reading.
- Tear film → capillary attraction → decreases IOP reading.
- Dr. Goldmann realized if the diameter of the circle applanated by the device is \[ #.## \text{ mm} \], capillary attraction and corneal thickness would cancel each other out (assuming CCT is \[ # \text{ µm} \]).

*(CCT = Central corneal thickness)*
Based on the *Imbert-Fick* principle: \( P = \frac{F}{A} \)

- Pressure inside a sphere equals force needed to flatten its surface divided by the area of flattening.
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- Dr. Goldmann realized if the diameter of the circle applanated by the device is 3.06 mm, capillary attraction and corneal thickness would cancel each other out (assuming CCT is 520 µm).

\( (CCT = \text{Central corneal thickness}) \)
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- Dr. Goldmann realized if the diameter of the circle applanated by the device is 3.06 mm, capillary attraction and corneal thickness would cancel each other out (assuming CCT is 520 µm).
- Goldmann believed CCT was ~520, with little variation.

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

No question—proceed when ready.
Based on the **Imbert-Fick principle**: \( P = \frac{F}{A} \)

- Pressure inside a sphere equals force needed to flatten its surface divided by the area of flattening.
- Assumes surface is infinitely thin, and dry (cornea is neither, obviously).
- \( K \) thickness \( \rightarrow \) resists applanation \( \rightarrow \) increases IOP reading.
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Dr. Goldmann realized if the diameter of the circle applanated by the device is **3.06 mm**, capillary attraction and corneal thickness would cancel each other out (assuming CCT is **520 \( \mu \text{m} \)**).

- Goldmann believed CCT was \( \approx 520 \), with little variation.
- When the mires line up, the diameter of the applanated area is **3.06 mm**.

No question—proceed when ready.
Define glaucoma. A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss.

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

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

We mentioned previously that glaucoma presents with “progressive ONH damage.” Let’s drill down on the structure of the ONH.
The optic nerves are composed of what?
The optic nerves are composed of what?
The axons of retinal ganglion cells
The optic nerves are composed of what?
The axons of retinal ganglion cells

How many fibers (axons) comprise an optic nerve?
The optic nerves are composed of what?
The axons of retinal ganglion cells

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

Per the…
Glaucoma book: 1.2-1.5M
Neuro book: 1-1.2M
Fundamentals book: “more than a million”
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?
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Do they synapse in the region of the optic nerve head?
No
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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?
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Most? Where will the others synapse, and what are they responsible for?
The optic nerves are composed of what?
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Most? Where will the others synapse, and what are they responsible for?
Most of the others are involved in the three words
The optic nerves are composed of what?
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Most? Where will the others synapse, and what are they responsible for?
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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.
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There’s that word again—’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?
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Anatomically speaking, the optic nerve is considered to have four portions. What are they?
The optic nerves are composed of what?
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Anatomically speaking, the optic nerve is considered to have four portions. What are they?
The intraocular, intraorbital, intracanalicular, and intracranial
Glaucoma Overview

Optic nerve portions (don’t memorize the lengths)
The optic nerves are composed of what?
The axons of retinal ganglion cells

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Glaucoma Overview

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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)

Anatomically speaking, the optic nerve is considered to have four portions. What are they?
The intraocular, intraorbital, intracanalicular, and intracranial

The intraocular portion is also considered to have four portions. What are they?
*What is the blood supply for each?*

<table>
<thead>
<tr>
<th>Portion</th>
<th>Blood supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>NFL portion</td>
<td>Central retinal artery (CRA)</td>
</tr>
<tr>
<td>Pre-laminar</td>
<td>Short posterior ciliary arteries</td>
</tr>
<tr>
<td>Laminar</td>
<td>Arterial circle of Zinn &amp; Haller</td>
</tr>
<tr>
<td>Retrolaminar</td>
<td>Centrifugal CRA branches, centripetal pial branches</td>
</tr>
</tbody>
</table>

A
ONH: Blood supply
For reasons that have yet to be fully elucidated, glaucomatous optic neuropathy tends to damage the superior and inferior poles of the ONH preferentially and early. This leads to thinning at the poles (focal thinning is often referred to as a ‘notch.’)
Because of this tendency, ophthalmologists focus on the *vertical cup-disc ratio* (VCDR) when assessing a pt’s glaucoma status.

For reasons that have yet to be fully elucidated, *glaucomatous optic neuropathy tends to damage the superior and inferior poles of the ONH preferentially and early*. This leads to thinning at the poles (focal thinning is often referred to as a ‘notch.’)
Note that the VCDR can be misleading in this regard, as it can be quite pronounced in some normal eyes (especially those with a large disc).
Note that the VCDR can be misleading in this regard, as it can be quite pronounced in some normal eyes (especially those with a large disc).

Thus, in determining the glaucomatous-ness of an ONH, don’t just rely on the VCDR--make sure you also *inspect and critically evaluate the status of the neuroretinal rim.*
The nonglaucomatous neuroretinal rim tends to follow what’s known as the **ISNT rule**: In decreasing order, the rim is thickest at its Inferior, Superior, Nasal, and Temporal portions. If an ONH’s rim adheres to this rule, it **ISNT** glaucomatous.
The nonglaucomatous neuroretinal rim tends to follow what’s known as the **ISNT rule**: In decreasing order, the rim is thickest at its **I**nferior, **S**uperior, **N**asal, and **T**emporal portions. If an ONH’s rim adheres to this rule, it **ISNT** glaucomatous.

*Note: Not all glaucoma docs find the ISNT rule to be helpful—YMMV. Ask!*
Now consider the ONH and retina in cross section. Note that the RNFL and ONH are both organized in a specific fashion:

No question—proceed when ready
Now consider the ONH and retina in cross section. Note that the RNFL and ONH are both organized in a specific fashion:

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

No question—proceed when ready
Now consider the ONH and retina in cross section. Note that the RNFL and ONH are both organized in a specific fashion:

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

--The ONH is stacked \textit{horizontally}, with its peripheral-most fibers being those originating in the far retina, and its innermost fibers originating in the peripapillary region.
Now let’s look at the topography of the retinal nerve fiber layer, and how that topography relates to the structure of the ONH.

No question—proceed when ready
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First, take note of the horizontal raphé. Fibers do not cross this anatomic boundary—those superior to it join the superior ONH, and those inferior to it, the inferior ONH.
Now let’s look at the topography of the retinal nerve fiber layer, and how that topography relates to the structure of the ONH.

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

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

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

No question—proceed when ready
Because there are so many fibers at the superior and inferior poles, the normal ONH rim tends to be thicker at these sites. (This accounts for the relative proportions of the rim segments as captured by the ISNT rule described previously.)
Note also that a vertical meridian can be described in the retina as well. Unlike the horizontal raphé (which is physically instantiated in the anatomy of the retina), this vertical meridian is purely functional—it cannot be identified via histological examination of the retina.
Note also that a *vertical meridian* can be described in the retina as well. Unlike the horizontal raphé (which is physically instantiated in the anatomy of the retina), this vertical meridian is purely functional—it cannot be identified via histological examination of the retina. Instead, it is identified via *visual field testing*. Fixation divides the VF into nasal and temporal fields, with the photoreceptors (PRs) responsible for the temporal VF being nasal to the vertical meridian, and those responsible for the nasal VF located temporal to it.
Note also that a *vertical meridian* can be described in the retina as well. Unlike the horizontal raphé (which is physically instantiated in the anatomy of the retina), this vertical meridian is purely functional—it cannot be identified via histological examination of the retina. Instead, it is identified via visual field testing. Fixation divides the VF into nasal and temporal fields, with the **If not within the retina, where is the anatomic location for the vertical meridian found in the visual fields?**
Glaucoma Overview

**Fovea centralis (fixation)**

- PRs are temporal to fovea, so VF is nasal to fixation
- PRs are nasal to fovea, so VF is temporal to fixation

Note also that a *vertical meridian* can be described in the retina as well. Unlike the horizontal raphé (which is physically instantiated in the anatomy of the retina), this vertical meridian is purely functional—*it cannot be identified via histological examination of the retina.* Instead, it is identified via visual field testing. Fixation divides the VF into nasal and temporal fields, with the

*If not within the retina, where is the anatomic location for the vertical meridian found in the visual fields?*

The optic chiasm. Recall that it is there that the visual field is divided vertically.
Glaucoma Overview

Note also that a *vertical meridian* can be described in the retina as well. Unlike the horizontal raphé (which is physically instantiated in the anatomy of the retina), this vertical meridian is purely functional—it cannot be identified via histological examination of the retina. Instead, it is identified via *visual field testing*. Fixation divides the VF into nasal and temporal fields, with the photoreceptors (PRs) responsible for the temporal VF being nasal to the vertical meridian, and those responsible for the nasal VF located temporal to it. Finally, note that fixation also divides the VF into superior and inferior VFs. The corresponding portions of the retina related topographically to the horizontal raphé.

*No question—proceed when ready*
Putting it all together: The VF can be divided into four quadrants. Together, retinal topography and ONH structure dictate that each quadrants corresponds with a particular anatomic location on the ONH. This relationship is important to understand as it allows the clinician to determine whether VF changes correlate with structural changes in the ONH as detected via DFE and/or imaging technology.
Here is a representation of the VF for each eye. Which is OD, and which OS?
Here is a representation of the VF for each eye. Which is OD, and which OS? Remember, VFs are **not** drawn as if the pt is looking at you; they’re drawn as if **you** are the pt!
Measured in degrees from fixation, how far does the normal VF extend superiorly, inferiorly, nasally and temporally?
(Don’t get too fixated on these specific numbers--different sources will give slightly different values.)
Measured in degrees from fixation, how far does the normal VF extend superiorly, inferiorly, nasally and temporally?
(Don’t get too fixated on these specific numbers--different sources will give slightly different values.)
Measured in degrees from fixation, how much of the VF is assessed via the automated perimetry machines found in most ophthalmology practices?
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.
How far in degrees from fixation is the blind spot?
How far in degrees from fixation is the blind spot?
About 15 (again, don’t get too hung up on that specific number.)
For reasons that have yet to be fully elucidated, glaucoma initially ‘prefers’ to damage the superior and inferior poles of the ONH. This leads to thinning at the poles (focal thinning is referred to as a ‘notch.’)
Glaucoma Overview

For reasons that have yet to be fully elucidated, glaucoma initially ‘prefers’ to damage the superior and inferior poles of the ONH. This leads to thinning at the poles (focal thinning is referred to as a ‘notch.’) Specifically, glaucoma tends initially to affect fibers that originate on the temporal side of the vertical meridian.
For reasons that have yet to be fully elucidated, glaucoma initially ‘prefers’ to damage the superior and inferior poles of the ONH. This leads to thinning at the poles (focal thinning is referred to as a ‘notch.’) Specifically, glaucoma tends initially to affect fibers that originate on the temporal side of the vertical meridian. The result of this is that glaucomatous VF defects appear in and extend from the nasal visual field.
Define glaucoma. A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss.

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

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

It was noted initially that glaucoma presents with “characteristic VF loss.” That’s what we’re getting at here. Let’s take a detailed look at the way glaucomatous VF defects appear and progress.
Note: The following set of VFs are from a pt who suffered severe, progressive VF loss in a manner classic for glaucomatous optic neuropathy. I am not personally familiar with this case, and thus cannot provide context regarding the clinical circumstances that resulted in such profound, unchecked VF loss.

No question—proceed when ready
The first location at which glaucomatous VF manifests is near the nasal limit of a 24-2 field, sitting on (or ‘hanging’ just below) the horizontal midline. This pattern of loss is called a nasal step.
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*No question—proceed when ready*
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...is associated with *this* location on the retina, meaning that the affected nerve fibers originated there...and entered the ONH peripherally.

No question—proceed when ready.
If left untreated, the nasal step will gradually enlarge.

No question—proceed when ready
If left untreated, the nasal step will gradually enlarge.

Note the area of origin for affected fibers has grown.
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.
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*. 
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 the area of origin for affected fibers now extends all the way to the ONH itself.

No question—proceed when ready
As glaucoma damage progresses, further loss of nerve fibers joining at that portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an *arcuate*.

Note also that an early *inferior* nasal step is now present.
If left unchecked, an arcuate will expand into the surrounding portion of the VF.

No question—proceed when ready
Once an arcuate has expanded sufficiently, it becomes an **defect**. The superior visual field is now all but gone.
Glaucoma Overview

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

- Early superior nasal step
- Superior nasal step
- Superior arcuate
- Early inferior nasal step
- Advanced arcuate
- Altitudinal defect
- Inferior arcuate
- Early inferior nasal step
- Inferior nasal step
- Altitudinal defect

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

A note on the ‘Glaucoma Continuum’

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

No question—proceed when ready
In this regard, a word on the notion of ‘early’ glaucoma. We previously described the above VF defect as an ‘early’ nasal step.
In this regard, a word on the notion of ‘early’ glaucoma. We previously described the above VF defect as an ‘early’ nasal step. But take note of the point along the glaucoma continuum at which such a VF defect occurs—clearly, it doesn’t qualify as ‘early’ disease with respect to the continuum. Don’t mistake early VF changes for early disease!

No question—proceed when ready
Finally, let’s look briefly at how one should think through the new glaucoma case sitting in your exam chair.
The first thought you should have when encountering a pt you suspect has glaucoma is…
The first thought you should have when encountering a pt you suspect has glaucoma is…

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

Glaucoma

Open-angle  Closed- or narrow-angle

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

What is the status of the angle?

How does one determine the status of the angle?
The first thought you should have when encountering a pt you suspect has glaucoma is…

What is the status of the angle?

How does one determine the status of the angle?

**Gonioscopy.** Don’t assume your glaucoma pt has open angles—**prove** it by gonioing them!
Once you have determined a pt has open-angle glaucoma, the next ‘first thought’ is to ask...
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?*
Untreated IOP consistently above 22 mmHg

Untreated IOP consistently below 22 mmHg

Normal-tension glaucoma (NTG)
Untreated IOP consistently above 22 mmHg

Untreated IOP consistently below 22 mmHg

(Note that this distinction is somewhat controversial, as some glaucomalogists contend NTG is not a separate condition.)
Once you have determined a pt has high-pressure open-angle glaucoma, the next ‘first thought’ is to ask…
Once you have determined a pt has high-pressure open-angle glaucoma, the next ‘first thought’ is to ask…

*Is it primary open-angle glaucoma (POAG), or secondary OAG?*
How prevalent is POAG in the US?
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Very. It affects about 2% of the over-40 population.
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Where does POAG rank worldwide as a cause of blindness?
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*Where does POAG rank worldwide as a cause of blindness?*
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*Is there a racial predilection?*
How prevalent is POAG in the US?
Very. It affects about 2% of the over-40 population.

Where does POAG rank worldwide as a cause of blindness?
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Is there a racial predilection?
Yes, individuals of [ ] and [ ] heritage are at a 4x greater risk than are [ ] (and their relative risk of going blind is even higher than that).
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Is age a risk factor?
How prevalent is POAG in the US?
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Where does POAG rank worldwide as a cause of blindness?
It is second only to cataract.

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

Is age a risk factor?
Yes, POAG rates increase dramatically with age.
How prevalent is POAG in the US?
Very. It affects about 2% of the over-40 population.

Where does POAG rank worldwide as a cause of blindness?
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Is age a risk factor?
Yes, POAG rates increase dramatically with age.

What is the #1 risk factor?
How prevalent is POAG in the US?
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What is the #1 risk factor?
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How prevalent is POAG in the US?

Very. It affects about 2% of the over-40 population.

Where does POAG rank worldwide as a cause of blindness?

It is second only to cataract.

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

What is the #1 risk factor?
Elevated IOP.

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

While not listed in the section on risk factors, the Glaucoma book alludes to two other variables as being well-established as significant risk factors for POAG. What are they?
- Central corneal thickness (CCT)
- Ocular perfusion pressure (OPP)
Glaucoma Overview

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

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

--?
--?

It is second only to elevated IOP.

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

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

What is the #1 risk factor?
Elevated IOP
How prevalent is POAG in the US? 

Very. It affects about 2% of the over-40 population.

Where does POAG rank worldwide as a cause of blindness? 

It is second only to cataract.

Is there a racial predilection? 

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

Is age a risk factor? 

Yes, POAG rates increase dramatically with age.

What is the #1 risk factor? 

Elevated IOP.
It's critical to note that POAG is a diagnosis of exclusion—
It's critical to note that POAG is a diagnosis of exclusion—it can only be made once secondary causes of OAG have been ruled out!
Glaucoma Overview

↑ IOP OAG

Primary

Secondary

PXS

Pigmentary

Tumor-Induced
- Phacolytic
- Phacoantigenic
- Lens particle

Lens-Induced
- Posner-Schlossman
- Fuchs heterochromic iridocyclitis

Inflammation-Induced
- Steroids
- Mydriatics

Drug-Induced

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

↑ EVS

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

(Most of these conditions are addressed in detail in other slide-sets—see the Table of Contents)
Once you have determined a pt has angle-closure glaucoma, the next ‘first thought’ is to ask…
Glaucoma Overview

Angle-Closure Glaucoma

Primary  ?  Secondary

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

is it primary or secondary?
Is there a racial predilection regarding the risk of PACG?
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Yes, individuals of Inuit heritage have the highest known risk of PACG.
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Is there a racial predilection regarding the risk of PACG?
Yes, individuals of Inuit heritage have the highest known risk of PACG--their relative risk has been estimated to be as high as 40x that of whites.

What about people of Asian descent?
Is there a racial predilection regarding the risk of PACG?
Yes, individuals of Inuit heritage have the highest known risk of PACG--their relative risk has been estimated to be as high as 40x that of whites.

What about people of Asian descent?
Their relative risk is somewhere between that of the Inuit and whites.
Is there a racial predilection regarding the risk of PACG?
Yes, individuals of Inuit heritage have the highest known risk of PACG--their relative risk has been estimated to be as high as 40x that of whites.

What about people of Asian descent?
Their relative risk is somewhere between that of the Inuit and whites.

Is age a risk factor?
Is there a racial predilection regarding the risk of PACG?
Yes, individuals of Inuit heritage have the highest known risk of PACG--their relative risk has been estimated to be as high as 40x that of whites.

What about people of Asian descent?
Their relative risk is somewhere between that of the Inuit and whites.

Is age a risk factor?
Yes, the incidence increases with age.
Angle-Closure Glaucoma

Is there a racial predilection regarding the risk of PACG?
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What about people of Asian descent?
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Is age a risk factor?
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What about people of Asian descent?
Their relative risk is somewhere between that of the Inuit and whites

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

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

What about people of Asian descent?
Their relative risk is somewhere between that of the Inuit and whites.

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

Is gender a risk factor?
Yes, are at higher risk.
Is there a racial predilection regarding the risk of PACG?
Yes, individuals of Inuit heritage have the highest known risk of PACG--their relative risk has been estimated to be as high as 40x that of whites.

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

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

Is gender a risk factor?
Yes, women are at higher risk
Is there a racial predilection regarding the risk of PACG?
Yes, individuals of Inuit heritage have the highest known risk of PACG--their relative risk has been estimated to be as high as 40x that of whites.

What about people of Asian descent?
Their relative risk is somewhere between that of the Inuit and whites

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

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

Is refraction a risk factor?
**Is there a racial predilection regarding the risk of PACG?**
Yes, individuals of Inuit heritage have the highest known risk of PACG--their relative risk has been estimated to be as high as 40x that of whites.

**What about people of Asian descent?**
Their relative risk is somewhere between that of the Inuit and whites

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

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

**Is refraction a risk factor?**
Yes; PACG is more likely to occur in...
Is there a racial predilection regarding the risk of PACG?
Yes, individuals of Inuit heritage have the highest known risk of PACG--their relative risk has been estimated to be as high as 40x that of whites.

What about people of Asian descent?
Their relative risk is somewhere between that of the Inuit and whites

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

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

Is refraction a risk factor?
Yes; PACG is more likely to occur in hyperopes
Angle-Closure Glaucoma

Primary

Secondary

What are the four subtypes of PACG?
What are the four subtypes of PACG?
The first thought you should have when encountering a pt you suspect has secondary angle-closure glaucoma is…
The first thought you should have when encountering a pt you suspect has secondary angle-closure glaucoma is…

*is it with or without pupillary block*
The first thought you should have when encountering a pt you suspect has secondary angle-closure glaucoma is… 

_is it with or without pupillary block_

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