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

*Why isn’t elevated IOP mentioned above?*
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.
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?
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
Define glaucoma.
A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss

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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?
It is the only risk factor that is *modifiable* in a manner proven to influence the risk of glaucoma progression

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

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

\[ IOP = \text{Aqueous Formation Rate} + \text{Outflow Facility} + \text{Episcleral Venous Pressure} \]

**Glaucoma Overview**
The Goldmann equation

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

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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)}
\]

Note how the \(\mu\text{L/min}\) cancel, leaving IOP in 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 (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}
\]

Note how the \(\mu\text{L/min}\) cancel, leaving IOP in mmHg

**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. In other words, even if aqueous formation ceased (which would take the first term in the Goldmann equation down to zero), IOP should not fall below EVP; that is, IOP would 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. However, neither of these extrapolations hold up to empirical scrutiny. The point being, *the Goldmann equation is a simplified, idealized model of IOP determination that does not account for all the real-world factors that influence IOP.*
Fill in the IOP equation below. **What is its eponymous name?** **The Goldmann equation**

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

So to lower IOP, one must:

- __and/or__ decrease aqueous formation
- __and/or__ increase outflow facility
- __and/or__ decrease episcleral venous pressure
- __and/or__ dehydrate the vitreous with a hyperosmotic agent

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

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*Three maneuvers implied by the Goldmann equation*
Fill in the IOP equation below. **What is its eponymous name?**

The **Goldmann equation**

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Three maneuvers implied by the Goldmann equation

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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Three maneuvers implied by the Goldmann equation

Important maneuver not implied by the Goldmann equation

...and/or dehydrate the vitreous with a hyperosmotic agent
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The Goldmann equation

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

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

So to lower IOP, one must:
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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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**Glaucoma Overview**

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

**The Goldmann equation**

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

What are the two types of outflow?
- 
- 

---

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

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

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

So to lower IOP, one must:
- decrease aqueous formation, and/or
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...and/or dehydrate the vitreous with a hyperosmotic agent
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

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

The Goldmann equation

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

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What are the two types of outflow?
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Glaucoma Overview
Fill in the IOP equation below. **What is its eponymous name?**

The **Goldmann equation**

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

*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 (\(\mu\text{L/min}\))} + \text{Outflow Facility (\(\mu\text{L/min/mmHg}\))} + \text{Episceral Venous Pressure (mmHg)}
\]

What is the rate of aqueous formation?
2-3 \(\mu\text{L/min}\)
Fill in the IOP equation below. What is its eponymous name? The Goldmann equation

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

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

What is the aqueous volume of the anterior chamber?
Fill in the IOP equation below. *What is its eponymous name?* The **Goldmann equation**

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

*What is the rate of aqueous formation?*
2-3 $\mu$L/min

*What is the aqueous volume of the anterior chamber?*
270 $\mu$L, give or take
Fill in the IOP equation below. What is its eponymous name? The Goldmann equation

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

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

What is the aqueous volume of the anterior chamber?
270 μL, give or take

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

\begin{equation*}
IOP = \text{Aqueous Formation Rate (µL/min)} - \text{Outflow Facility (µL/min/mmHg)} + \text{Episceral Venous Pressure (mmHg)}
\end{equation*}

\textbf{What is the rate of aqueous formation?}
\[2-3 \, \text{µL/min}\]

\textbf{What is the aqueous volume of the anterior chamber?}
\[270 \, \text{µL, give or take}\]

\textbf{So then, what percent of AC volume is ‘turned over’ every minute?}
\[About \, 1\%\]
Fill in the IOP equation below. **What is its eponymous name?**

The **Goldmann equation**

\[
IOP = \frac{Aqueous \, Formation \, Rate \, (\mu L/min)}{Outflow \, Facility \, (\mu L/min/mmHg)} + Episcleral \, Venous \, Pressure \, (mmHg)
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**What is the rate of aqueous formation?**
2-3 \( \mu L/min \)

**What is the aqueous volume of the anterior chamber?**
270 \( \mu L \), give or take

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

**Given this, how long does it take for the aqueous content of the AC to be fully replaced?**
Fill in the IOP equation below. **What is its eponymous name?**

**The Goldmann equation**

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

**What is the rate of aqueous formation?**

2-3 µL/min

**What is the aqueous volume of the anterior chamber?**

270 µL, give or take

So then, **what percent of AC volume is ‘turned over’ every minute?**

About 1%

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

Roughly 100 minutes
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

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

The Goldmann equation

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

Speaking of aqueous formation…What specific tissue makes aqueous?

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

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

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

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

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

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--increase outflow facility, and/or
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Fill in the IOP equation below. What is its eponymous name?
The Goldmann equation

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

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

What are the two types of outflow?
--Trabecular meshwork
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Glaucoma Overview
So to lower IOP, one must:

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

The Goldmann equation

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

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

What are the two types of outflow?

--Trabecular meshwork

--Uveoscleral

A Glaucoma Overview
Glaucoma Overview

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

Ciliary body: Another perspective
Glaucoma Overview

Ciliary body: Another
Ciliary body: Another

Glaucoma Overview
Now let’s look at the CB epithelium. **Low power** photomicrograph.
Glaucoma Overview

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.
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.

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

Neuroectoderm

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

What other portion of the eye derives from neuroectoderm?

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
A

Glaucoma Overview

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.

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

What other portion of the eye derives from neuroectoderm? The retina (i.e., the neurosensory retina + RPE).
What specific tissue makes aqueous?
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What is implied by the fact that aqueous is made by the ‘nonpigmented’ epithelium?
The presence of a pigmented epithelium.

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?
Glaucoma Overview

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?
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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?
Apex-to-apex
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?

**Apex-to-apex**

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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Apex-to-apex

How are the two epithelial layers of the CB oriented with respect to one another?
The same way--apex-to-apex

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The same way--apex-to-apex

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

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

**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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--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.)
Glaucoma Overview

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

Neuroectoderm

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

Q

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

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
Which intraocular structure 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

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

Hol up Dr Flynn. Looking at this photomicrograph, the pigmented epi layer appears to be the inner one. Did you make a mistake?
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
Fill in the IOP equation below. What is its eponymous name? The Goldmann equation

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

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

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

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

Which classes of meds decrease aqueous formation?
- \(\beta\) blockers
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What are the two types of outflow?
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- Uveoscleral

Glaucoma Overview
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 the force needed to flatten its surface divided by the area of flattening.

**Principle in words**

- \( F \) stands for what the…
- \( A \) stands for what the…
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.
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 \(\rightarrow\) resists applanation \(\rightarrow\) increase vs decrease in IOP reading.
Based on the **Imbert-Fick principle**: \( P = \frac{F}{A} \)

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- 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

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, i.e., 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.)
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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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**.
Based on the **Imbert-Fick principle**: \( P = \frac{F}{A} \)

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*To be useful, an applanation-type device has to account for these factors. Fortunately, the brilliant Dr. Goldmann was (mostly) up to the challenge…*
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Dr. Goldmann realized if the area appplanated by the device is \( \#.#\# \) mm\(^2\), capillary attraction and corneal thickness would cancel each other out (assuming CCT is \( \# \mu m \))

\( \text{(CCT = Central corneal thickness)} \)
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- Dr Goldmann realized if the area applanated by the device is **3.06 mm\(^2\)**, capillary attraction and corneal thickness would cancel each other out (assuming CCT is **520**).
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- Dr Goldmann realized if the area applanated by the device is 3.06 mm$^2$, capillary attraction and corneal thickness would cancel each other out (assuming CCT is 520)
  - Goldmann believed CCT was $\sim$520, with little variation.
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(We now know that CCT averages about 550, but that it varies widely among individuals)
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- Goldmann believed CCT was ~520, with little variation.
- When mires line up, planated area = 3.06 mm\(^2\).
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?
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How many fibers (axons) comprise an optic nerve?
The optic nerves are composed of what?
The axons of retinal ganglion cells

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

Per the…
Glaucoma book: 1.2-1.5M
Neuro book: 1-1.2M
Fundamentals book: “more than a million”
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Do they synapse in the region of the optic nerve head?
The optic nerves are composed of what?
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Where will they synapse?
The optic nerves are composed of what?
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Where will they synapse?
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Most? Where will the others synapse, and what are they responsible for?
Q/A

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ONH: Blood supply
For reasons that have yet to be fully elucidated, glaucomatous optic neuropathy tends to damage the superior and inferior poles of the ONH preferentially and early. This leads to thinning at the poles (focal thinning is often referred to as a ‘notch.’)
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).
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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 **I**nferior, **S**uperior, **N**asal, and **T**emporal 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 Inferior, Superior, Nasal, and Temporal portions. If an ONH’s rim adheres to this rule, it is **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:
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--The RNFL is stacked *vertically*, with fibers that originate at points distant from the ONH running at the bottom (ie, closer to the RPE); and
Now consider the ONH and retina in cross section. Note that the RNFL and ONH are both organized in a specific fashion:

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

--The ONH is stacked *horizontally*, with its peripheral-most fibers being those originating in the far retina, and its innermost fibers originating in the peripapillary region.
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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First, take note of the *horizontal raphé*. Fibers do not cross this anatomic boundary—those superior to it join the superior ONH, and those inferior to it, the inferior ONH.
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Next, the papillomacular (PM) bundle—the swath of nerve fibers originating in the foveal region. Note how this bundle takes up the lion’s share of the temporal ONH.
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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.
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.
Glaucoma Overview

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

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

[Diagram showing the nasal step]
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.
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 \textit{nasal step}. 

\textbf{This} location in the VF...is associated with \textbf{this} location on the retina, meaning that the affected nerve fibers originated there...and entered the ONH peripherally.
If left untreated, the nasal step will gradually enlarge.
Glaucoma Overview

'Early superior nasal step'

'Superior nasal step'

Note the area of origin for affected fibers has grown

If left untreated, the nasal step will gradually enlarge.
As glaucoma damage progresses, further loss of nerve fibers joining at that portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an *arcuate*. 
As glaucoma damage progresses, further loss of nerve fibers in the superior portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an **arcuate**.

Note the area of origin for affected fibers now extends all the way to the ONH itself.
As glaucoma damage progresses, further loss of nerve fibers joining at that portion of the ONH will cause the VF defect to arc toward the blind spot. Once the VF loss has connected to the blind spot, the resulting defect is termed an *arcuate*.  

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

If left unchecked, an arcuate will expand into the surrounding portion of the VF.
Once an arcuate has expanded sufficiently, it becomes an altitudinal defect. The superior visual field is now all but gone. The inferior nasal step continues to enlarge.
The inferior step is now an arc, and appears destined to become altitudinal, resulting in blindness.
Glaucoma is a progressive condition, passing from undetectable early disease to asymptomatic-but-detectable (via RNFL imaging) disease to functional (ie, marked by VF loss) disease, the last stage of which is severe vision loss and blindness.
In this regard, a word on the notion of ‘early’ glaucoma. We previously described the above VF defect as an ‘early’ nasal step.
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!
Finally, let’s look briefly at how one should think through the new glaucoma case sitting in your exam chair
Glaucoma Overview

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

*What is the status of the angle?*
How does one determine the status of the angle?
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? 
**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

Normal-tension glaucoma (NTG)

Untreated IOP consistently below # mmHg
Untreated IOP consistently above 22 mmHg

Normal-tension glaucoma (NTG)

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

*Is it primary open-angle glaucoma (POAG), or secondary OAG?*
How prevalent is POAG in the US?
How prevalent is POAG in the US?
Very. It affects about $\%$ of the over-40 population.
How prevalent is POAG in the US?
Very. It affects about 2% of the over-40 population.
Glaucoma Overview

How prevalent is POAG in the US?
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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?
It is second only to...
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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?
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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).
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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?
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.
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What is the #1 risk factor?
How prevalent is POAG in the US?
Very. It affects about 2% of the over-40 population.

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What is the #1 risk factor?
Elevated IOP.
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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?
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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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?
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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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

↑ IOP OAG

Primary

Secondary
Glaucoma Overview

Primary

Secondary

↑ IOP OAG
Glaucoma Overview

↑ IOP OAG

Primary

Secondary

PXS

Pigmentary

Tumor-Induced

Lens-Induced

Inflammation-Induced

Drug-Induced

Trauma-Related

↑ EVS

Schwartz syndrome
Glaucoma Overview

↑ IOP OAG

Primary

Secondary

PXS

Pigmentary

Tumor-Induced

Lens-Induced

Inflammation-Induced

Drug-Induced

Trauma-Related

↑ EVS

Schwartz syndrome

- Phacolytic
- Phacoantigenic
- Lens particle

- Posner-Schlossman
- Fuchs heterochromic iridocyclitis

- Steroids
- Mydriatics

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

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

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

Angle-Closure Glaucoma

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

is it primary or secondary?
Is there a racial predilection regarding the risk of PACG?
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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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.
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?
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 there a racial predilection regarding the risk of PACG?
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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?
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 hyperopes.
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
What are the four subtypes of PACG?
Angle-Closure Glaucoma

Primary
- Acute
- Subacute
- Chronic
- Plateau Iris

Secondary

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*
Angle-Closure Glaucoma

Primary
- Acute
- Subacute
- Chronic
- Plateau Iris

Secondary

? w/ Pupillary Block  w/o 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