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

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

Why isn’t elevated IOP mentioned above?
Elevated IOP is a strong risk factor for glaucoma, but it need not be present—IOP can be normal, or even low
Open-angle Glaucoma: *Primary*

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

For more on ONH damage in glaucoma, see slide-set G0
Re characteristic VF loss in glaucoma:
The first location at which glaucomatous VF manifests is near the nasal limit of a 24-2 field, sitting on (or ‘hanging’ just below) the horizontal midline. This pattern of loss is called a *nasal step*. 

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

*Early superior 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 *nasal step*. 

This location in the VF...is associated with this location on the retina...and thus, this location on the ONH
If left untreated, the nasal step will gradually enlarge.
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 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 \textit{arcuate}.

\textbf{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.
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.
The inferior step is now an arc, and appears destined to become altitudinal, resulting in blindness.

For more on VF defects in glaucoma, see slide-set G0
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

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?

Open-angle Glaucoma: Primary
The first thought you should have when encountering a pt you suspect has glaucoma is…

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

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

Open-angle Glaucoma: *Primary*  

OAG  

$\uparrow$ IOP  

Normal-tension glaucoma (NTG)  

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

Open-angle Glaucoma: *Primary*

↓IOP

Untreated IOP consistently below 22 mmHg

Normal-tension glaucoma (NTG)

(Note that this distinction is somewhat controversial, as some glaucomalogists contend NTG is not a separate condition.)
Untreated IOP consistently above 22 mmHg

Open-angle Glaucoma: *Primary*

\[ \uparrow \text{IOP} \]

Untreated IOP consistently below 22 mmHg

Normal-tension glaucoma (NTG)

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

Normal-tension glaucoma *is covered in its own slide-set (G21)*
Once you have determined a pt has high-pressure open-angle glaucoma, the next ‘first thought’ is to ask...
Open-angle Glaucoma: Primary

↑ IOP OAG

Primary ? Secondary

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?*
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?*

*What does it mean to say a case of glaucoma is ‘secondary’?*
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?

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

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

What are some of these specific factors?
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?

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

What are some of these specific factors?
Brace yourself…
Open-angle Glaucoma: Primary

↑ IOP OAG

Primary

Secondary

Schwartz syndrome

↑ EVS

Trauma-Related

Drug-Induced

PXS Pigmentary Tumor-Induced Lens-Induced Inflammation-Induced

Phacoantigenic

Phacolytic

Fuchs heterochromic iridocyclitis

Posner-Schlossman

Lens particle

AVM Venous obstruction SVC syndrome C-C fistula

Angle recession Cyclodialysis cleft Hyphema Hemolytic Ghost cell

(All are addressed in detail in other slide-sets—see the Table of Contents.)
Note that primary open-angle glaucoma (POAG) is a diagnosis of exclusion—it can only be made by first determining that the angle is open, and then ruling out the myriad causes of secondary OAG.
So, you see a pt with ONH and VF loss consistent with glaucomatous optic neuropathy. This can appropriately be referred to as ‘glaucoma.’

(No question—proceed when ready)
Glaucoma

So, you see a pt with ONH and VF loss consistent with glaucomatous optic neuropathy. This can appropriately be referred to as ‘glaucoma.’ But before calling it open-angle glaucoma (OAG), you must first gonio the pt and determine affirmatively that the angle is open.

(No question—proceed when ready)
So, you see a pt with ONH and VF loss consistent with glaucomatous optic neuropathy. This can appropriately be referred to as ‘glaucoma.’ But before calling it open-angle glaucoma (OAG), you must first gonio the pt and determine affirmatively that the angle is open. Don’t use the label OAG until you’ve done so!

(No question—proceed when ready)
So, you see a pt with ONH and VF loss consistent with glaucomatous optic neuropathy. This can appropriately be referred to as ‘glaucoma.’

But before calling it open-angle glaucoma (OAG), you must first gonio the pt and determine affirmatively that the angle is open. Don’t use the label OAG until you’ve done so!

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

(No question—proceed when ready)
Primary Open-Angle Glaucoma (POAG)

So, you see a pt with ONH and VF loss consistent with glaucomatous optic neuropathy. This can appropriately be referred to as ‘glaucoma.’ But before calling it open-angle glaucoma (OAG), you must first gonio the pt and determine affirmatively that the angle is open. Don’t use the label OAG until you’ve done so!

Likewise, before calling it primary open angle glaucoma (POAG), you must first consider and rule out the myriad causes of secondary OAG. Don’t use the label POAG until you’ve done so!

(No question—proceed when ready)
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--?
--IOP
--?
--?
--?
--?
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--?
--?
--?
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

- Race
- IOP
- Family history
- Older age
- Myopia
- Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

- Low ocular perfusion pressure (OPP)
- Low cerebrospinal fluid (CSF) pressure
- Low corneal hysteresis

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

Individuals of black and Hispanic heritage are at a 4x greater risk than are whites.

What about the risk of going blind from POAG?

These same folk are at a 4x higher risk of that as well.
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

- Race
- IOP
- Family history
- Older age
- Myopia
- Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

- Low ocular perfusion pressure (OPP)
- Low cerebrospinal fluid (CSF) pressure
- Low corneal hysteresis

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

Individuals of [ ] and [ ] heritage are at a 4x greater risk than are [ ]
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

- Race
- IOP
- Family history
- Older age
- Myopia
- Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
- Low ocular perfusion pressure (OPP)
- Low cerebrospinal fluid (CSF) pressure
- Low corneal hysteresis

With regards to race, who is at higher risk for POAG in the US?
Individuals of black and Hispanic heritage are at a 4x greater risk than are whites.
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

- Race
- IOP
- Family history
- Older age
- Myopia
- Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

- Low ocular perfusion pressure (OPP)
- Low cerebrospinal fluid (CSF) pressure
- Low corneal hysteresis

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

Individuals of black and Hispanic heritage are at a 4x greater risk than are whites

What about the risk of going blind from POAG?

These same folk are at a 4x higher risk of that as well
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

- Race
- IOP
- Family history
- Older age
- Myopia
- Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

- Low ocular perfusion pressure (OPP)
- Low cerebrospinal fluid (CSF) pressure
- Low corneal hysteresis

**Race**

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

Individuals of black and Hispanic heritage are at a 4x greater risk than are whites

*What about the risk of going blind from POAG?*

These same folk are at a 4x higher risk of that as well
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

-- Race
-- Family history
-- Older age
-- Myopia
-- Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

-- Low ocular perfusion pressure (OPP)
-- Low cerebrospinal fluid (CSF) pressure
-- Low corneal hysteresis

In addition to being the strongest risk factor for glaucoma, IOP has another quality that renders it unique—what is it?
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis

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

-- Race
-- Family history
-- Older age
-- Myopia
-- Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

-- Low ocular perfusion pressure (OPP)
-- Low cerebrospinal fluid (CSF) pressure
-- Low corneal hysteresis

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 treatment turns on IOP-lowering maneuvers!
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis

How significant a risk factor for POAG is age?

Older age
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis

How significant a risk factor for POAG is age?
Very. The probability of having POAG, as well as the probability of it progressing, both increase dramatically with increasing age.
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis

**How significant a risk factor for POAG is age?**
Very. The probability of having POAG, as well as the probability of it progressing, both increase dramatically with increasing age.

**For what racial group is age a particularly impactful risk factor?**
AAs. Consider—fully 11% of AAs over the age of 80 have glaucoma!
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

How significant a risk factor for POAG is age?
Very. The probability of having POAG, as well as the probability of it progressing, both increase dramatically with increasing age.

For what racial group is age a particularly impactful risk factor?
AAs. Consider—fully 11% of AAs over the age of 80 have glaucoma!
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
---Race
---IOP
---Family history
---Older age
---Myopia
---Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
---Low ocular perfusion pressure (OPP)
---Low cerebrospinal fluid (CSF) pressure
---Low corneal hysteresis

**How significant a risk factor for POAG is age?**
Very. The probability of having POAG, as well as the probability of it progressing, both increase dramatically with increasing age.

**For what racial group is age a particularly impactful risk factor?**
AAs. Consider—fully 11% of AAs over the age of 80 have glaucoma!
Q

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

--Race
--IOP
--Family history
--Older age
--Myopia

**Thin central corneal thickness (CCT)**

Which glaucoma clinical trial identified CCT as a risk factor for POAG?
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia

**Thin central corneal thickness (CCT)**

*Which glaucoma clinical trial identified CCT as a risk factor for POAG?*
The Ocular Hypertension Treatment Trial (the OHTS)
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia

**Thin central corneal thickness (CCT)**

*Which glaucoma clinical trial identified CCT as a risk factor for POAG?*
The Ocular Hypertension Treatment Trial (the OHTS)

*A thin CCT results in falsely low IOP readings on applanation. Does this IOP effect account for the relationship between CCT and POAG risk?*
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia

**Thin central corneal thickness (CCT)**

Which glaucoma clinical trial identified CCT as a risk factor for POAG?
The Ocular Hypertension Treatment Trial (the OHTS)

A thin CCT results in falsely low IOP readings on applanation. Does this IOP effect account for the relationship between CCT and POAG risk?
No—thin CCT is a risk factor even after accounting for its effect on IOP measurement, ie, it’s an independent risk factor
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia

Thin central corneal thickness (CCT)

Which glaucoma clinical trial identified CCT as a risk factor for POAG?
The Ocular Hypertension Treatment Trial (the OHTS)

A thin CCT results in falsely low IOP readings on applanation. Does this IOP effect account for the relationship between CCT and POAG risk?
No—thin CCT is a risk factor even after accounting for its effect on IOP measurement, ie, it’s an independent risk factor

How might this work physiologically?
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia

**Thin central corneal thickness (CCT)**

Which glaucoma clinical trial identified CCT as a risk factor for POAG?
The Ocular Hypertension Treatment Trial (the OHTS)

A thin CCT results in falsely low IOP readings on applanation. Does this IOP effect account for the relationship between CCT and POAG risk?
No—thin CCT is a risk factor even after accounting for its effect on IOP measurement, ie, it’s an independent risk factor

How might this work physiologically?
No one knows for certain, but it might be that thinness of the CCT reflects structural characteristics of the eyewall that make the ONH vulnerable to glaucomatous damage
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis

Speaking of structural characteristics of the eyewall that make the ONH vulnerable to glaucomatous damage…What does the term hysteresis refer to?
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

--Race

--IOP

--Family history

--Older age

--Myopia

--Thin central corneal thickness (CCT)

**What are the “other” (ditto) risk factors?**

--Low ocular perfusion pressure (OPP)

--Low cerebrospinal fluid (CSF) pressure

**Low corneal hysteresis**

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

It refers to the fact that changes in the physical property of a structure may lag behind changes in the forces that determine it.
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis

Speaking of structural characteristics of the eyewall that make the ONH vulnerable to glaucomatous damage…What does the term hysteresis refer to?
It refers to the fact that changes in the physical property of a structure may lag behind changes in the forces that determine it.

Wiggity what? Can you unpack that with respect to the cornea, please?
Sure—flip to the next slide…
Open-angle Glaucoma: *Primary*

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

The moment at which corneal flattening hits the reference value

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

Now the amount of air pressure is ramping back down. As the pressure drops, the cornea will proceed to round back out to its normal shape (again, not depicted on graph).
Open-angle Glaucoma: *Primary*

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

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

**But that’s not what happens.** Instead, the cornea ‘lags’ behind, and doesn’t achieve the reference level of flattening until the air pressure has dropped past that which was required to produce it while the air pressure was ramping up.
Consider: A column of air is directed at the cornea, and its pressure is ramped up over time. As the pressure increases, it causes the cornea to flatten more and more (not depicted on graph). At some point, corneal flattening reaches a predetermined reference value. The amount of air pressure required to produce this level of flattening is noted.

Now the amount of air pressure is ramping back down. As the pressure drops, the cornea will proceed to round back out to its normal shape (again, not depicted on graph).

If the cornea was perfectly elastic, it would reach the reference level of flattening on the way ‘out’ at the same air-pressure level that produced it on the way ‘in.’

But that’s not what happens. Instead, the cornea ‘lags’ behind, and doesn’t achieve the reference level of flattening until the air pressure has dropped past that which was required to produce it while the air pressure was ramping up.
Open-angle Glaucoma: Primary

The moment at which corneal flattening hits the reference value

The difference between the ‘in’ and ‘out’ pressures needed to produce a given amount of flattening defines the cornea’s hysteresis

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

Now the amount of air pressure is ramping back down. As the pressure drops, the cornea will proceed to round back out to its normal shape (again, not depicted on graph).

If the cornea was perfectly elastic, it would reach the reference level of flattening on the way ‘out’ at the same air-pressure level that produced it on the way ‘in.’

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

OK, but how does low corneal hysteresis increase glaucoma risk?

As with thin CCT, no one knows for certain. But also like thin CCT, the assumption is low hysteresis reflects structural properties of the eyewall that render the ONH vulnerable to glaucomatous damage.
Open-angle Glaucoma: Primary

The moment at which corneal flattening hits the reference value

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

Now the amount of air pressure is ramping back down. As the pressure drops, the cornea will proceed to round back out to its normal shape (again, not depicted on graph).

If the cornea was perfectly elastic, it would reach the reference level of flattening on the way ‘out’ at the same air-pressure level that produced it on the way ‘in.’

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

The difference between the ‘in’ and ‘out’ pressures needed to produce a given amount of flattening defines the cornea’s hysteresis

OK, but how does low corneal hysteresis increase glaucoma risk? As with thin CCT, no one knows for certain.
Open-angle Glaucoma: *Primary*

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

If the cornea was perfectly elastic, it would reach the reference level of flattening on the way ‘out’ at the same air-pressure level that produced it on the way ‘in.’

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

**OK, but how does low corneal hysteresis increase glaucoma risk?** As with thin CCT, no one knows for certain. But also like thin CCT, the assumption is low hysteresis reflects structural properties of the eyewall that render the ONH vulnerable to glaucomatous damage.
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--- Low ocular perfusion pressure (OPP)
--- Low cerebrospinal fluid (CSF) pressure

What is OPP, ie, how is it defined?
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
---Low ocular perfusion pressure (OPP)
---Low cerebrospinal fluid (CSF) pressure

What is OPP, ie, how is it defined?
It is the difference between mean arterial pressure (MAP) and IOP

Open-angle Glaucoma: Primary
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)

What is OPP, ie, how is it defined?
It is the difference between mean arterial pressure (MAP) and IOP

Is low OPP a risk factor for POAG development, or progression?
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

--- **Low ocular perfusion pressure (OPP)**
--- Low cerebrospinal fluid (CSF) pressure
--- Low corneal hysteresis

**What is OPP, ie, how is it defined?**
It is the difference between mean arterial pressure (MAP) and IOP

**Is low OPP a risk factor for POAG development, or progression?**
For both
What are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

- Race
- IOP
- Family history
- Older age
- Myopia
- Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

- Low ocular perfusion pressure (OPP)
- Low cerebrospinal fluid (CSF) pressure
- Low corneal hysteresis

Open-angle Glaucoma: *Primary*

So, if low OPP is a risk factor for progression, it follows that HTN should be protective against POAG. Is this the case?

What is OPP, ie, how is it defined?
It is the difference between mean arterial pressure (MAP) and IOP

Is low OPP a risk factor for POAG development, or progression? For both
So, if low OPP is a risk factor for progression, it follows that HTN should be protective against POAG. Is this the case?
Like a FB status, it’s complicated. While the data are not completely clear, the evidence suggests HTN reduces the risk of POAG for pts younger than 65, but increases the risk in those older than that.

What are the “other” (ditto) risk factors?
-- Low ocular perfusion pressure (OPP)
-- Low cerebrospinal fluid (CSF) pressure

What is OPP, ie, how is it defined?
It is the difference between mean arterial pressure (MAP) and IOP

Is low OPP a risk factor for POAG development, or progression?
For both
Open-angle Glaucoma: *Primary*

**What are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?**

---
- Race
- IOP
- Family history
- Older age
- Myopia
- Thin central corneal thickness (CCT)

**What are the “other” (ditto) risk factors?**

---
- Low ocular perfusion pressure (OPP)
- Low cerebrospinal fluid (CSF) pressure
- Low corneal hysteresis

---

**What is OPP, ie, how is it defined?**

*It is the difference between mean arterial pressure (MAP) and IOP.*

**Is low OPP a risk factor for POAG development, or progression?**

For both

---

So, if low OPP is a risk factor for progression, it follows that HTN should be protective against POAG. Is this the case?

Like a FB status, it’s complicated. While the data are not completely clear, the evidence suggests HTN reduces the risk of POAG for pts younger than 65, but increases the risk in those older than that.
What are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

--Race--IOP--Family history--Older age--Myopia--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

--Low ocular perfusion pressure (OPP)--Low cerebrospinal fluid (CSF) pressure--Low corneal hysteresis

Open-angle Glaucoma: Primary

So, if low OPP is a risk factor for progression, it follows that HTN should be protective against POAG. Is this the case?

Like a FB status, it’s complicated. While the data are not completely clear, the evidence suggests HTN reduces the risk of POAG for pts younger than 65, but increases the risk in those older than that.

That is complicated. How might this work physiologically?

What is OPP, ie, how is it defined?

It is the difference between mean arterial pressure (MAP) and IOP

Is low OPP a risk factor for POAG development, or progression?

For both
So, if low OPP is a risk factor for progression, it follows that HTN should be protective against POAG. Is this the case?
Like a FB status, it’s complicated. While the data are not completely clear, the evidence suggests HTN reduces the risk of POAG for pts younger than 65, but increases the risk in those older than that.

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

What are the “other” (ditto) risk factors?

- **Low ocular perfusion pressure (OPP)**
- Low cerebrospinal fluid (CSF) pressure

What is OPP, ie, how is it defined?
It is the difference between mean arterial pressure (MAP) and IOP.

Is low OPP a risk factor for POAG development, or progression?
For both
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis
--DM?
--HTN?

What with the question marks for DM and HTN? Are they risk factors, or not?

The short answer is—it’s complicated. --Re DM: Controversy exists as to the relationship between DM and POAG. Several well-regarded studies suggest DM is a risk factor. However, several equally well-regarded studies found no association, and one seemed to suggest DM might be associated with a reduced risk of POAG. (Many experts contend this finding was an artifact of the study’s design.)

--Re HTN: Controversy exists here as well. One (large, well-regarded) study found that HTN is associated with a reduced risk of POAG in individuals <65, but an increased risk in older individuals. However, equally-reliable research found that HTN was at least somewhat protective for both older and younger pts alike.
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis
--DM?
--HTN?

What with the question marks for DM and HTN? Are they risk factors, or not?
The short answer is—it’s complicated.

--Re DM: Controversy exists as to the relationship between DM and POAG.
Several well-regarded studies suggest DM is a risk factor.

--Re HTN
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?

- Race
- IOP
- Family history
- Older age
- Myopia
- Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?

- Low ocular perfusion pressure (OPP)
- Low cerebrospinal fluid (CSF) pressure
- Low corneal hysteresis
- DM?
- HTN?

What with the question marks for DM and HTN? Are they risk factors, or not?

The short answer is—it’s complicated.

Re DM: Controversy exists as to the relationship between DM and POAG. Several well-regarded studies suggest DM is a risk factor. However, several equally well-regarded studies found no association, and one seemed to suggest DM might be associated with a reduced risk of POAG. (Many experts contend this finding was an artifact of the study’s design.)

Re HTN
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis
--DM?
--HTN?

What with the question marks for DM and HTN? Are they risk factors, or not? The short answer is—it’s complicated.
--Re DM: Controversy exists as to the relationship between DM and POAG. Several well-regarded studies suggest DM is a risk factor. However, several equally well-regarded studies found no association, and one seemed to suggest DM might be associated with a reduced risk of POAG. (Many experts contend this finding was an artifact of the study’s design.)
--Re HTN:
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis
--DM?
--HTN?

What with the question marks for DM and HTN? Are they risk factors, or not?
The short answer is—it’s complicated.

--Re DM: Controversy exists as to the relationship between DM and POAG. Several well-regarded studies suggest DM is a risk factor. However, several equally well-regarded studies found no association, and one seemed to suggest DM might be associated with a reduced risk of POAG. (Many experts contend this finding was an artifact of the study’s design.)

--Re HTN: Controversy exists here as well. One (large, well-regarded) study found that HTN is associated with a reduced risk of POAG in individuals <65, but an increased risk in older individuals.
Besides IOP, what are the “important” (per the Glaucoma book) risk factors for POAG development and/or progression?
--Race
--IOP
--Family history
--Older age
--Myopia
--Thin central corneal thickness (CCT)

What are the “other” (ditto) risk factors?
--Low ocular perfusion pressure (OPP)
--Low cerebrospinal fluid (CSF) pressure
--Low corneal hysteresis
--DM?
--HTN?

What with the question marks for DM and HTN? Are they risk factors, or not?
The short answer is—it’s complicated.
--Re DM: Controversy exists as to the relationship between DM and POAG. Several well-regarded studies suggest DM is a risk factor. However, several equally well-regarded studies found no association, and one seemed to suggest DM might be associated with a reduced risk of POAG. (Many experts contend this finding was an artifact of the study’s design.)
--Re HTN: Controversy exists here as well. One (large, well-regarded) study found that HTN is associated with a reduced risk of POAG in individuals <65, but an increased risk in older individuals. However, equally-reliable research found that HTN was at least somewhat protective for both older and younger pts alike.
Where does POAG rank worldwide as a cause of blindness?
Where does POAG rank worldwide as a cause of blindness?
It is second only to

Open-angle Glaucoma: *Primary*
Open-angle Glaucoma: *Primary*

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

How prevalent is POAG in the US?
Where does POAG rank worldwide as a cause of blindness?
It is second only to cataract.

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

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

How prevalent is POAG in the US?
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

POAG is called the ‘silent thief of sight.’ Why?
Where does POAG rank worldwide as a cause of blindness?
It is second only to cataract.

How prevalent is POAG in the US?
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

POAG is called the ‘silent thief of sight.’ Why?
Because of its stealthy nature. It is insidious in onset, and progresses very slowly. It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process, often when the pt is already functionally blind from field loss.
Where does POAG rank worldwide as a cause of blindness?
It is second only to cataract.

How prevalent is POAG in the US?
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

POAG is called the ‘silent thief of sight.’ Why?
Because of its stealthy nature. It is insidious in onset, and progresses very slowly. It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process, often when the pt is already functionally blind from field loss. As we ‘speak,’ untold numbers of people are at this moment going blind from POAG—they just don’t know it.
Where does POAG rank worldwide as a cause of blindness?
It is second only to cataract.

How prevalent is POAG in the US?
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

POAG is called the ‘silent thief of sight.’ Why?
Because of its stealthy nature. It is insidious in onset, and progresses very slowly. It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process, often when the pt is already functionally blind from field loss. As we ‘speak,’ untold numbers of people are at this moment going blind from POAG—they just don’t know it.

In general, what is the visual prognosis for POAG?

Open-angle Glaucoma: Primary
Where does POAG rank worldwide as a cause of blindness?
It is second only to cataract.

How prevalent is POAG in the US?
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

POAG is called the ‘silent thief of sight.’ Why?
Because of its stealthy nature. It is insidious in onset, and progresses very slowly. It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process, often when the pt is already functionally blind from field loss. As we ‘speak,’ untold numbers of people are at this moment going blind from POAG—they just don’t know it.

In general, what is the visual prognosis for POAG?
Not too bad—most pts retain useful vision for life.
Where does POAG rank worldwide as a cause of blindness?
It is second only to cataract.

How prevalent is POAG in the US?
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

POAG is called the ‘silent thief of sight.’ Why?
Because of its stealthy nature. It is insidious in onset, and progresses very slowly. It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process, often when the pt is already functionally blind from field loss. As we ‘speak,’ untold numbers of people are at this moment going blind from POAG—they just don’t know it.

In general, what is the visual prognosis for POAG?
Not too bad—most pts retain useful vision for life.

How many don’t? That is, what proportion end up bilaterally blind?
Where does POAG rank worldwide as a cause of blindness?
It is second only to cataract.

How prevalent is POAG in the US?
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

POAG is called the ‘silent thief of sight.’ Why?
Because of its stealthy nature. It is insidious in onset, and progresses very slowly.
It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process,
often when the pt is already functionally blind from field loss. As we ‘speak,’ untold
numbers of people are at this moment going blind from POAG—they just don’t know it.

In general, what is the visual prognosis for POAG?
Not too bad—most pts retain useful vision for life

How many don’t? That is, what proportion end up bilaterally blind?
About 4% or so
Open-angle Glaucoma: Primary

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

How prevalent is POAG in the US?
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

POAG is called the ‘silent thief of sight.’ Why?
Because of its stealthy nature. It is insidious in onset, and progresses very slowly.
It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process,
often when the pt is already functionally blind from field loss. As we ‘speak,’ untold
numbers of people are at this moment going blind from POAG—they just don’t know it.

In general, what is the visual prognosis for POAG?
Not too bad—most pts retain useful vision for life

How many don’t? That is, what proportion end up bilaterally blind?
About 4% or so

What one intervention has been demonstrated (via clinical trial) to reduce the risk of
glaucoma progression?
Open-angle Glaucoma: *Primary*

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

*How prevalent is POAG in the US?*
Very. Almost *2%* of the over-40 US population—*3M+* people—have POAG.

*POAG is called the ‘silent thief of sight.’ Why?*
Because of its stealthy nature. It is insidious in onset, and progresses very slowly. It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process, often when the pt is already functionally blind from field loss. As we ‘speak,’ untold numbers of people are at this moment going blind from POAG—they just don’t know it.

*In general, what is the visual prognosis for POAG?*
Not too bad—most pts retain useful vision for life.

*How many don’t? That is, what proportion end up bilaterally blind?*
About 4% or so.

*What one intervention has been demonstrated (via clinical trial) to reduce the risk of glaucoma progression?*
IOP reduction.
Open-angle Glaucoma: *Primary*

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

*How prevalent is POAG in the US?*
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

*POAG is called the ‘silent thief of sight.’ Why?*
Because of its stealthy nature. It is insidious in onset, and progresses very slowly. It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process, often when the pt is already functionally blind from field loss. As we ‘speak,’ untold numbers of people are at this moment going blind from POAG—they just don’t know it.

*In general, what is the visual prognosis for POAG?*
Not too bad—most pts retain useful vision for life

*How many don’t? That is, what proportion end up bilaterally blind?*
About 4% or so

*What one intervention has been demonstrated (via clinical trial) to reduce the risk of glaucoma progression?*
*IOP reduction*

*By what specific modes of intervention can this IOP reduction be achieved?*
--?
--?
--?
Where does POAG rank worldwide as a cause of blindness?
It is second only to cataract.

How prevalent is POAG in the US?
Very. Almost \( \frac{2\%}{\text{of the over-40 US population}} \) — \( \frac{3M+}{\text{people}} \) — have POAG.

POAG is called the ‘silent thief of sight.’ Why?
Because of its stealthy nature. It is insidious in onset, and progresses very slowly. It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process, often when the pt is already functionally blind from field loss. As we ‘speak,’ untold numbers of people are at this moment going blind from POAG—they just don’t know it.

In general, what is the visual prognosis for POAG?
Not too bad—most pts retain useful vision for life

How many don’t? That is, what proportion end up bilaterally blind?
About 4% or so

What one intervention has been demonstrated (via clinical trial) to reduce the risk of glaucoma progression?
--- IOP reduction

By what specific modes of intervention can this IOP reduction be achieved?
--- Topical meds
--- Laser surgery
--- Incisional surgery
Where does POAG rank worldwide as a cause of blindness?
It is second only to cataract.

How prevalent is POAG in the US?
Very. Almost 2% of the over-40 US population—3M+ people—have POAG.

POAG is called the ‘silent thief of sight.’ Why?
Because of its stealthy nature. It is insidious in onset, and progresses very slowly. It causes no discomfort. Further, visual acuity isn’t affected until late in the dz process, often when the pt is already functionally blind from field loss. As we ‘speak,’ untold numbers of people are at this moment going blind from POAG—they just don’t know it.

In general, what is the visual prognosis for POAG?
Not too bad—most pts retain useful vision for life.

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

What one intervention has been demonstrated (via clinical trial) to reduce the risk of glaucoma progression?
IOP reduction