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It is the only risk factor that is modifiable in a manner proven to influence the risk of glaucoma progression.
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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!
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?
The first thought you should have when encountering a pt you suspect has angle-closure glaucoma is…
The first thought you should have when encountering a pt you suspect has angle-closure glaucoma is…

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

What about people of Asian descent?
Their relative risk is somewhere between that of the Inuit and whites.
Is 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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Is age a risk factor?
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Is refraction a risk factor?
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Is gender a risk factor?
Yes, women are at higher risk.

Is refraction a risk factor?
Yes; PACG is more likely to occur in...
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What are the four subtypes of PACG?
Angled Closure Glaucoma

Primary
- Acute
- Subacute
- Chronic
- Plateau Iris

Secondary

What are the four subtypes of PACG?
Angle-Closure Glaucoma

Primary
- Acute
- Subacute
- Chronic
- Plateau Iris

Secondary

In what fundamental way do these three...
Angle-Closure Glaucoma

Primary
- Acute
- Subacute
- Chronic
- Plateau Iris

Secondary

In what fundamental way do these three... differ from this one?
Angle Closure Glaucoma

Primary
- Acute
- Subacute
- Chronic
- Plateau Iris

Secondary

These share a common mechanism: Pupillary block
Angle Closure Glaucoma

Angle-Closure Glaucoma

Primary
- Acute
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These share a common mechanism: **Pupillary block**
Angle Closure Glaucoma

Angle-Closure Glaucoma

**Primary**
- Acute
- Subacute
- Chronic
- **Plateau Iris**

**Secondary**

These share a common mechanism: **Pupillary block**

In plateau iris, angle closure is due primarily to anterior displacement of the ciliary processes (although some cases of plateau iris demonstrate a pupillary block component as well)
The first thought you should have when encountering a pt you suspect has secondary angle-closure glaucoma is…
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?*
Another way to think about the etiology of secondary angle closure glaucoma is…
Another way to think about the etiology of secondary angle closure glaucoma is…
To consider whether the peripheral iris is being ‘pushed’ forward from behind, or being ‘pulled’ forward from the front.
Angle Closure Glaucoma

Primary
- Acute
- Subacute
- Chronic
- Plateau Iris

Secondary
- w/ Pupillary Block: 'Push'
- w/o Pupillary Block: 'Pull'

Q
Angle Closure Glaucoma

Angle-Closure Glaucoma

Primary
- Acute
- Subacute
- Chronic
- Plateau Iris

Secondary
- w/ Pupillary Block: Push
- w/o Pupillary Block: Pull
  - Lens-Induced
  - Aphakic/Pseudophakic
  - RD
Angle Closure Glaucoma

Primary
- Acute
- Subacute
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Secondary
w/ Pupillary Block: 'Push'
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w/o Pupillary Block: 'Pull'
- ?
- ?
- ?
- ?
- ?
- ?
- ?
- ?
- ?
- ?
A

Angle Closure Glaucoma

Angle-Closure Glaucoma

Primary

- Acute
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Secondary

w/ Pupillary Block:

‘Push’

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w/o Pupillary Block:

‘Pull’

- Neovascular
- ICE
- Inflammatory
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In this context, what does the acronym NVG stand for?

Neovascular glaucoma.

What two-word phrase (not 'diabetic retinopathy') describes the cause of most cases of NVG?

'Retinal ischemia'.

How can ischemia of retinal cells induce neovascularization of the iris (NVI) and/or angle (NVA)?

The ischemic retinal cells release the signaling molecule vascular endothelial growth factor (VEGF), a potent inducer of new blood vessel formation.

What are the three most common causes of NV-inducing ischemia?

-- Diabetic retinopathy
-- CRVO
-- Ocular ischemic syndrome (OIS; which involves ischemia of non-retinal structures as well)

Where on the iris does NVI typically appear first, and what does it look like?

It usually appears as small 'tufts' of vessels at the pupillary margin. As it develops further, it grows in a meandering fashion toward the angle (normal iris vessels typically grow in a rather direct radial fashion).

How does NVA cause angle closure?

The new blood vessels don't travel alone, rather, they are accompanied by fibroblasts and similar cells. These fellow-travelers have contractile properties, and thus can 'pull' the iris across the angle, thereby closing it.
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‘Ischemic’ and ‘non-ischemic’

Which sort is implicated in the development of NVG?

Seriously?

What is the typical timeframe for development of NVG after CRVO?

It usually occurs about 3 month’s after. Because of this, NVG after CRVO is often called ‘100-day glaucoma’ (or, ‘90-day glaucoma’).
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‘Pull’ Neovascular

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Can NVG develop after a CRAO?

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Is NVG more, or less likely to develop after CRAO compared to CRVO?

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CRVO
Ocular is structures

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But surely a CRAO causes more retinal ischemia than does a CRVO. Given this, why isn’t NVG more common after CRAO?

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Because the retina is too ischemic after CRAO. That is, CRAO-induced ischemia is so profound that retinal cells die prior to being able to produce and release VEGF. Contrast this with CRVO, in which enough blood flow is maintained to allow the dying retinal cells time to ‘cry for help.’

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If NVG develops after CRAO, what is the typical timeframe?

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If NVG develops after CRAO, what is the typical timeframe?
It usually occurs about 1 month after. Because of this, NVG after CRAO is often called ‘30-day glaucoma.’

What is the typical timeframe for development of NVG after CRVO?
It usually occurs about 3 month’s after. Because of this, NVG after CRVO is often called ‘100-day glaucoma’ (or, ‘90-day glaucoma’)

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There’s an important difference in the clinical presentation of NVG in DBR and CRVO vs the presentation of ‘NVG’ in OIS. What is it?

Angle closure in DBR and CRVO inevitably produces a dramatic spike in IOP. However, angle closure in OIS frequently is not accompanied by a high IOP.

Why doesn’t the IOP spike during angle closure in OIS?
In a word—hypoperfusion. That is, the same lack of blood flow that resulted in ocular ischemia leads to ciliary-body shutdown, such that very little aqueous is made.

What is the most common cause of OIS?
Carotid occlusive disease.

If an eye has a zipped-up angle secondary to NVA from OIS, what can happen to IOP after successful CEA re-establishes blood flow to the ciliary body?
IOP often spikes dramatically. The patient’s ophthalmologist must be prepared for this development in OIS pts who undergo CEA!
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Angle-Closure Glaucoma

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Plateau Iris w/ Pupillary Block: w/o Pupillary Block:
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The ischemic retinal cells release vascular endothelial growth factor (VEGF), a potent inducer of new blood vessel formation.

What are the three most common causes of NV-inducing ischemia?
--Diabetic retinopathy
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Angle closure in DBR and CRVO inevitably produces a dramatic spike in IOP. However, angle closure in OIS frequently is not accompanied by a high IOP.

Why doesn’t the IOP spike during angle closure in OIS?
In a word—hypoperfusion. That is, the same lack of blood flow that resulted in ocular ischemia leads to ciliary-body shutdown, such that very little aqueous is made.

What is the most common cause of OIS?
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If an eye has a zipped-up angle secondary to NVA from OIS, what can happen to IOP after successful CEA re-establishes blood flow to the ciliary body?
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It grows in a meandering fashion toward the angle (normal iris vessels typically grow in a rather direct radial fashion).

How does NVA cause angle closure?
The new blood vessels don’t travel alone, rather, they are accompanied by fibroblasts and similar cells. These fellow-travelers have contractile properties, and thus can ‘pull’ the iris across the angle, thereby closing it.
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Is NVA always preceded by NVI?

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Iridocorneal endothelial syndrome

In a nutshell, what is ICE?

A nonhereditary condition in which corneal endothelial cells maldifferentiates. The resulting cohort of abnormal cells migrate across the angle and onto the iris, laying down a membrane (histologically similar to Descemet’s) as it goes. These abnormal, migrating endothelial cells and their associated membrane account for all of the signs/symptoms found in ICE.

What are the classic signs of ICE syndrome?

--Iris changes--Corneal changes--Peripheral anterior synechiae (PAS)--Elevated IOP

Who is the typical patient?

A young-to-middle-aged adult female

What three sorts of complaints will she have?

--Changes in the eye’s appearance--Ocular pain --Decreased VA

What ‘pertinent negative’ will be elicited when taking a history?

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Angle Closure Glaucoma

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w/o Pupillary Block:

‘Pull’

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Neovascular

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Inflammatory

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Angle Closure Glaucoma

Angle-Closure Glaucoma

SecondaryPrimary

Acute

Chronic

Lens-Induced

Aphakic/Pseudophakic

RD

Neovascular

ICE

Aqueous misdirection

ROP/PHPV

Subacute

Plateau Iris

w/o Pupillary Block: w/o Pupillary Block:

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Is a normal pupil located in the dead-center of the iris?
No, it is slightly nasal of dead center.

What is the formal term for displacement of the pupil from its normal location?
Corectopia

Q

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What is the formal term for the presence of an extra pupil or pupils?
Polycoria

So, polycoria is a feature of ICE?
No. True polycoria requires that each extra pupil have associated dilator and miosis musculature. In ICE, no such musculature is present; rather, the extra ‘pupils’ are the result of local trauma by the ICE membrane tearing the iris stroma. (As an aside, true polycoria is a phenomenally rare condition.)

If ICE pts don’t have true polycoria, what do they have in this regard?
Pseudopolycoria

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The pupil may be out of round or displaced, and it may have one or more extra openings.

What is the formal term for the presence of an extra pupil or pupils?
Polycoria

Is a normal pupil located in the dead-center of the iris?
No, it is slightly nasal of dead center

What is the formal term for displacement of the pupil from its normal location?
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### Angle-Closure Glaucoma

**In this context, what does ICE stand for?**
- Iridocorneal endothelial syndrome

**In a nutshell,**
- A nonhereditary condition in which corneal endothelial cells maldifferentiate.
- The resulting cohort of abnormal cells migrate across the angle and onto the iris, laying down a membrane (histologically similar to Descemet's) as it goes.
- These abnormal, migrating endothelial cells and their associated membrane account for all of the signs/symptoms found in ICE.

**What are the classic signs of ICE syndrome?**
- Iris changes
- Corneal changes
- Peripheral anterior synechiae (PAS)
- Elevated IOP

**Who is the typical patient?**
- A young-to-middle-aged adult female

**What three sorts of complaints will she have?**
- Changes in the eye's appearance
- Ocular pain
- Decreased VA

**What 'pertinent negative' will be elicited when taking a history?**
- She will deny any family history of similar eye findings

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So, polycoria is a feature of ICE?

Angle-Closure Glaucoma

‘Pull’
--Neovascular
--ICE
--Inflammatory
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--ROP/PHPV
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--2° to retinal issues
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So, polycoria is a feature of ICE?
No. True polycoria requires that each extra pupil have associated dilator and miosis musculature. In ICE, no such musculature is present; rather, the extra 'pupils' are the result of local trauma by the ICE membrane tearing the iris stroma. (As an aside, true polycoria is a phenomenally rare condition.)

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If ICE pts don’t have true polycoria, what do they have in this regard?
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If ICE pts don't have true polycoria, what do they have in this regard?
Pseudopolycoria

Is a normal pupil located in the dead-center of the iris?
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What is the formal term for displacement of the pupil from its normal location?
Corectopia

extra openings

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‘Pull’

Neovascular

ICE

Inflammatory

Nanophthalmos

ROP/PHPV

Aqueous misdirection

Epithelial downgrowth

2° to retinal issues
Angle Closure Glaucoma

What sort of corneal changes will be present?

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Pull

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Neovascular

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What sort of corneal changes will be present?
The pt may note that their cornea appears hazy or milky as a result of corneal edema.
On slit lamp exam, endothelial changes will be noted.

What are the classic signs of ICE syndrome?
--Iris changes
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What is the classic term describing the SL appearance of the abnormal endothelium?
'Hammered silver'

Hammered silver? I thought that was the classic term for the appearance of the endothelium in Fuchs dystrophy.
No, the term for that is 'beaten bronze'.
Angle Closure Glaucoma

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Q
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Q/A

Angle Closure Glaucoma

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Two words are used to describe the appearance of the PAS in ICE. What are they?

'Push' and 'Pull'

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Two words are used to describe the appearance of the PAS in ICE. What are they? ‘Broad’ and ‘high’

Why is this considered ‘high’?
That the PAS extend beyond Schwalbe’s line (SL)

Why do neo-related PAS end at SL?
Because neo can’t grow over ‘normal’ endothelium

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