Before you begin: This is a big topic, and big topics beget big slide-sets. There’s a natural break around slide 247; I placed a *break time!* slide at that location.
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

Secondary Angle Closure Glaucoma
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 go about determining the status of the angle?

By performing [gonioscopy] on the pt
The first thought you should have when encountering a pt you suspect has glaucoma is...

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How does one go about determining the status of the angle?
By performing gonioscopy on the pt
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What does it mean to say the angle is closed?
The first thought you should have when encountering a pt you suspect has glaucoma is… What is the status of the angle?

What does it mean to say the angle is closed?
It means the peripheral iris is in contact with the trabecular meshwork (TM).
The first thought you should have when encountering a pt you suspect has glaucoma is…

*What is the status of the angle?*

**What does it mean to say the angle is closed?**
It means the peripheral iris is in contact with the trabecular meshwork (TM)

*This contact comes in two basic flavors—what are they?*

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

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

*Closed- or narrow-angle*

Open-angle

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This contact comes in two basic flavors—what are they?

--The iris can **appose** the TM, ie, touch it without adhering to it

--The iris can be **syneched** to the TM, ie, adhered to it
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*I don’t know if *syneched* is actually a word, but you catch my drift*
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- Closed- or narrow-angle

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Secondary Angle Closure Glaucoma

How do you go about determining whether the iris-angle touch is appositional, or synechial?
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**Closed- or narrow-angle**

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**Glaucoma**

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Via dynamic (aka compression, aka indentation) gonioscopy
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Secondary Angle Closure Glaucoma

How do you go about determining whether the iris-angle touch is appositional, or synechial? Via dynamic (aka compression, aka indentation) gonioscopy.

How is dynamic gonioscopy performed?

During gonioscopy, the examiner manipulates the lens to gently compress the central cornea, in the process displacing aqueous peripherally, toward the angle. If the iris-angle contact is appositional, the influx of displaced aqueous will separate them. But at locations where the iris is syneched to the angle, the aqueous influx will have no effect on the iris-angle contact.
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A Glaucoma

**Closed- or narrow-angle**

**Open-angle**

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*“Syneched” is not a word, but it is commonly used in ophthalmology.
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Does it matter what sort of gonio lens is used?

It does indeed. The lens of choice is a Posner, Zeiss or Sussman. These applanate the central cornea, pushing aqueous peripherally and thereby opening (or not) the angle. In contrast, the flange on a Goldmann-style goniolens compresses the peripheral cornea, and thus is less efficient for displacing aqueous into the angle.
The first thought you should have when encountering a pt you suspect has glaucoma is...

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Indentation with Zeiss four-mirror lens causes deepening of the anterior chamber, which opens areas of appositional angle closure or exposes synechiae.
Secondary Angle Closure Glaucoma

Zeiss four-mirror view of iris bombé in an elderly hyperopic patient. The trabecular meshwork is not visualized.
Secondary Angle Closure Glaucoma

Zeiss four-mirror view of iris bombé in an elderly hyperopic patient. The trabecular meshwork is not visualized.

Same patient when a Zeiss lens is used to indent the cornea. The trabecular meshwork is visible.
Secondary Angle Closure Glaucoma

Eye in angle closure. No TM is visible.
Secondary Angle Closure Glaucoma

Eye in angle closure. No TM is visible.

With indentation gonioscopy parts of the TM are visualized (small arrow), but here is a broad peripheral anterior synechia (large arrow) precluding visualization of the remainder of the TM.
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?
Q

Secondary Angle Closure Glaucoma

Glaucoma

Closed- or narrow-angle

Primary

Secondary

What differentiates primary from secondary angle-closure glaucoma?
What differentiates primary from secondary angle-closure glaucoma?
In secondary, a specific pathological cause of angle closure can be identified, whereas no such cause is present in primary dz
Primary angle-closure glaucoma is discussed in detail in its own slide-set; see the Table of Contents.
Secondary Angle Closure Glaucoma

Two basic mechanisms of 2ndry angle closure

? ?
Secondary Angle Closure Glaucoma

Two basic mechanisms of 2ndry angle closure

w/ Pupillary Block  w/o Pupillary Block
What does pupillary block refer to, exactly?

Secondary Angle Closure Glaucoma

w/ Pupillary Block  w/o Pupillary Block

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Secondary Angle Closure Glaucoma

1. Resistance to aqueous flow from the PC to the AC

‘Pupillary block’
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Pupillary block leads to the development of a pressure gradient across the iris, which causes the iris to bow forward.
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Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block
1. Resistance to aqueous flow from the PC to the AC

2. The PC>AC pressure gradient causes the iris to bow forward, like a sail in the wind

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Pupillary block leads to the development of a pressure gradient across the iris, which causes the iris to bow forward. If the iris bows far enough, the peripheral iris will come into apposition with and occlude the drainage angle, precipitating acute closure of the angle and a prodigious rise in IOP.
3. Forward movement of the iris leads to apposition of the peripheral iris against the drainage angle, occluding it.

2. The PC>AC pressure gradient causes the iris to bow forward, like a sail in the wind.

1. Resistance to aqueous flow from the PC to the AC.

‘Pupillary block’
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It isn’t. The posterior chamber is the space immediately behind the lens and anterior to the vitreous residues in the vitreous cavity.
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In which pupil position—constricted, mid-dilated or fully dilated—is such contact likely to develop?
The mid-dilated position is the danger zone for the development of pupillary block.

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Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Two types of pupillary-block mechanism
Secondary Angle Closure Glaucoma

- w/ Pupillary Block
  - Two types of pupillary-block mechanism
    - Lens-Induced
    - Aphakic/Pseudophakic
  - w/o Pupillary Block
Secondary Angle Closure Glaucoma

- w/ Pupillary Block
  - Lens-Induced
  - Aphakic/ Pseudophakic
- w/o Pupillary Block
  - ?
  - ?
Secondary Angle Closure Glaucoma

w/ Pupillary Block

- Lens-Induced
  - Phacomorphic
  - Ectopia lentis

- Aphakic/Pseudophakic

w/o Pupillary Block
In a nutshell, what is the pathologic process in phacomorphic ACG?

In phacomorphic ACG, cataractous increase in lens size has two effects that are a setup for the development of pupillary-block angle closure:

1. It alters the anatomic relationship between the anterior lens surface and the pupil margin in a manner that leads to pupillary block and subsequent angle closure;
2. It pushes the peripheral iris forward, narrowing the angle, thereby reducing the magnitude of the PC-AC pressure gradient needed to induce angle closure.
In a nutshell, what is the pathologic process in phacomorphic ACG? Cataractous increase in lens size has two effects that are a setup for the development of pupillary-block ACG:

1) 

2)
In a nutshell, what is the pathologic process in phacomorphic ACG?

Cataractous increase in lens size has two effects that are a setup for the development of pupillary-block ACG:

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A crowded eye with a tight angle and a lens-iris relationship prone to pupillary block… That sounds like a garden-variety primary angle-closure glaucoma (PACG) eye. How do you differentiate between a phacomorphic glaucoma eye and a PACG eye?
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How is phacomorphic ACG managed?

The same way as PACG—LPI ASAP. The offending cataract should be removed once the eye quiets down. (Some ophthos forego the LPI and go straight to CE.)

Should miotics be employed? No, because they will likely only narrow the angle more by allowing the lens to drift anteriorly. Further, their use may make the soon-to-occur CE more difficult.
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Secondary Angle Closure Glaucoma

**w/ Pupillary Block**

**w/o Pupillary Block**

**Phacomorphic**

**Ectopia lentis**

**Lens-Induced**

**Aphakic/Pseudophakic**

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Secondary Angle Closure Glaucoma

- w/ Pupillary Block
  - Lens-Induced
    - Phacomorphic
    - Ectopia lentis
  - Aphakic/Pseudophakic

- w/o Pupillary Block

What is ectopia lentis?

Displacement of the lens from its normal anatomic position

With regard to lens 'displacement'—what do the following terms mean?

-- Subluxated:
The lens is partially displaced, but remains in the 'general area'

-- Luxated:
The lens is dislocated—completely removed from the pupillary aperture. All zonular attachments have been disrupted.

How does ectopia lentis lead to pupillary block and ACG?

By allowing the displaced lens to move into and block the pupil, producing the pressure gradient, with subsequent iris bombé and angle closure.
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

Aphakic/Pseudophakic

Phacomorphic

Ectopia lentis

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Displacement of the lens from its normal anatomic position
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Secondary Angle Closure Glaucoma

Subluxed lens
What is ectopia lentis?
Displacement of the lens from its normal anatomic position

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Lens-Induced Aphakic/Pseudophakic w/ Pupillary Block

Secondary Angle Closure Glaucoma

Aphakic/ Pseudophakic

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Secondary Angle Closure Glaucoma

Aphakic

Lens resting on the retina

b-scan: lens on ONH

Luxated lens
What is ectopia lentis?
Displacement of the lens from its normal anatomic position

With regard to lens ‘displacement’—what do the following terms mean?
--Sublux(at)ed: The lens is partially displaced, but remains in the ‘general area’
--Lux(at)ed: The lens is dislocated—completely removed from the pupillary aperture. All zonular attachments have been disrupted.

How does ectopia lentis lead to pupillary block and ACG?
Secondary Angle Closure Glaucoma

w/ Pupillary Block

Lens-Induced

Phacomorphic

Ectopia lentis

Aphakic/ Pseudophakic

w/o Pupillary Block

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How does ectopia lentis lead to pupillary block and ACG?
By allowing the displaced lens to move into and blocks the pupil, producing the pressure gradient, with subsequent iris bombé and angle closure.
While there are many conditions associated with ectopia lentis…
While there are many conditions associated with ectopia lentis…
While there are many conditions associated with ectopia lentis… The BCSC Glaucoma book singles out only one for discussion as causing pupillary block. Which one?
While there are many conditions associated with ectopia lentis… The BCSC Glaucoma book singles out only one for discussion as causing pupillary block. Which one? Microspherophakia
What is \textit{ectopia lentis}? Displacement of the lens from its normal anatomic position.

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\textit{In a few words, how would you describe the shape of a microspherophakic lens?}
The name says it all: the lens is small (‘micro’) and round (‘sphero’).
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The name says it all: the lens is small ('micro') and round ('sphero')
Microspherophakia. Note the small size, extreme curvature of the lens
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

Aphakic/ Pseudophakic

Phacomorphic

Ectopia lentis

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Secondary Angle Closure Glaucoma

Microspherophakia. With mydriasis, the lens is able to fit through the pupillary aperture
What is ectopia lentis?
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What common slit-lamp observation owes to the lens’ small size?
Typically, the entirety of the lens equator can be seen in the pupillary aperture when the pt is widely dilated.

Pts with microspherophakia are almost always high myopes. Why?
Because the lens is small, it has a short radius of curvature. Further, because it is spherical, it is more curved than is a normal lens. These two factors give the spheric lens vastly more converging power than a normal lens possesses.
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

Phacomorphic

Ectopia lentis

Aphakic/Pseudophakic

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**Secondary Angle Closure Glaucoma**

w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

- Phacomorphic
- **Ectopia lentis**

Aphakic/ Pseudophakic

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Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

Aphakic/ Pseudophakic

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Ectopia lentis

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Typically, the entirety of the lens equator can be seen in the pupillary aperture when the pt is widely dilated.

Pts with microspherophakia are almost always high myopes. Why?

Because the lens is small, it has a short radius of curvature. Further, because it is spherical, it is more curved than is a normal lens. These two factors give the m’spheric lens vastly more converging power than a normal lens possesses.
**Secondary Angle Closure Glaucoma**

- **w/ Pupillary Block**
  - Lens-Induced
    - Phacomorphic
    - Ectopia lentis
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- **w/o Pupillary Block**

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Displacement of the lens from its normal anatomic position

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*With what condition is microspherophakia most frequently associated?*

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Secondary Angle Closure Glaucoma

- **Aphakic/Pseudophakic**
  - w/ Pupillary Block
  - w/o Pupillary Block

- **Lens-Induced**
  - Phacomorphic
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**With what condition is microspherophakia most frequently associated?**
Weill-Marchesani syndrome

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Secondary Angle Closure Glaucoma

Microspherophakia in Weill-Marchesani syndrome
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

Aphakic/ Pseudophakic

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With what condition is microspherophakia most frequently associated?

What common slit-lamp examination owes to the lens’ small size?
Typically, the entirety of the lens equator can be seen in the pupillary aperture

What are the findings in Weill-Marchesani?

Patients with Weill-Marchesani have:

- Short stature
- Short fingers
- Stiff joints

(Think of it as the opposite of Marfan syndrome)
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

Aphakic/ Pseudophakic

Ectopia lentis

Phacomorphic

With respect to lens 'displacement':

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Displacement of the lens from its normal anatomic position.

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Because the lens is small, it has a short radius of curvature. Further, because it is spherical, it is more curved than is a normal lens. These two factors give the microspheric lens vastly more converging power than a normal lens possesses.

In a few words, how would you describe the shape of a microspherophakic lens?

The name says it all: the lens is small ('micro') and round ('sphero').

What common slit-lamp observation owes to the lens' small size?

Typically, the entirety of the lens equator can be seen in the pupillary aperture when the pt is widely dilated.

With what condition is microspherophakia most frequently associated?

Weill-Marchesani syndrome
Weill-Marchesani syndrome: Short stature
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---

In a few words, how would you describe the shape of a microspherophakic lens?
Small and round

With what condition is microspherophakia most frequently associated?
Weill-Marchesani syndrome

What are the findings in Weill-Marchesani?
Patients with Weill-Marchesani have:
...short stature
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How does ectopia lentis lead to pupillary block and ACG?
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Secondary Angle Closure Glaucoma

- Lens-Induced
  - Phacomorphic
  - Ectopia lentis
- Aphakic/Pseudophakic

w/ Pupillary Block  w/o Pupillary Block

What is ectopia lentis?
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In a few words, how would you describe the shape of a microspherophakic lens?
‘Micro’ and ‘sphero’, indicating a small and round lens.

With what condition is microspherophakia most frequently associated?
Weill-Marchesani syndrome

What are the findings in Weill-Marchesani?
Patients with Weill-Marchesani have:
- short stature
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Weill-Marchesani syndrome: Short fingers
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Patients with Weill-Marchesani have:
...short stature
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With what condition is microspherophakia most frequently associated?
Weill-Marchesani syndrome

What common slit-lamp examination owes to the lens’ small size?
With regard to lens ‘displacement’, why is ‘luxation’ more severe. Further, because it is spherical, it is more curved than a normal lens possesses.

Weill-Marchesani syndrome

What is microspherophakia?
The name says it all: the lens is small (‘micro’) and round (‘spherophakic’).
In a few words, how would you describe the shape of a microspherophakic lens?

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With what condition is microspherophakia most frequently associated?
Weill-Marchesani syndrome

What are the findings in Weill-Marchesani?
Patients with Weill-Marchesani have:

- ...short stature
- ...short fingers
- ...stiff joints

Think of it as the opposite of Marfan syndrome.
Secondary Angle Closure Glaucoma

- Lens-Induced w/ Pupillary Block
- Aphakic/ Pseudophakic w/o Pupillary Block
- Ectopia lentis
- Phacomorphic

What is ectopia lentis?
Displacement of the lens from its normal anatomic position

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Typically, the entirety of the lens equator can be seen in the pupillary aperture when the pt is widely dilated

With what condition is microspherophakia most frequently associated?
Weill-Marchesani syndrome

What are the findings in Weill-Marchesani?
Patients with Weill-Marchesani have:
- short stature
- short fingers
- stiff joints

Think of it as the opposite of Marfan syndrome

Tall stature
Long fingers
Lax joints
Secondary Angle Closure Glaucoma

Weill-Marchesani syndrome

Marfan syndrome
**Weill-Marchesani syndrome**

Weill-Marchesani is strongly associated with microspherophakia. With what conditions is microspherophakia **occasionally** associated?

---

By allowing the displaced lens to move into and blocks the pupil, producing the pressure gradient, with subsequent iris bombé and angle closure.
**Secondary Angle Closure Glaucoma**

- **With Pupillary Block**
  - Lens-Induced
    - Phacomorphic
    - Ectopia lentis

- **Without Pupillary Block**
  - Aphakic/ Pseudophakic

**Ectopia lentis**

*Weill-Marchesani syndrome*

*Weill-Marchesani is strongly associated with microspherophakia.*

*With what conditions is microspherophakia **occasionally** associated?*

- Lowe syndrome
- Alport syndrome
- Marfan syndrome
- Peters anomaly
- Congenital rubella

**How does ectopia lentis lead to pupillary block and ACG?**

By allowing the displaced lens to move into and block the pupil, producing the pressure gradient, with subsequent iris bombé and angle closure.
**Secondary Angle Closure Glaucoma**

- **w/ Pupillary Block**
  - **Lens-Induced**
  - **Aphakic/ Pseudophakic**
    - Phacomorphic
    - **Ectopia lentis**

- **w/o Pupillary Block**

---

What is **ectopia lentis**?

Displacement of the lens from its normal anatomic position

With regard to lens 'displacement'—what do the following terms mean?

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In a few words, how would you describe the shape of a microspherophakic lens?

The name says it all: the lens is small (‘micro’) and round (‘sphero’).

What common slit-lamp observation owes to the lens' small size?

Typically, the entirety of the lens equator can be seen in the pupillary aperture when the pt is widely dilated.

Pts with microspherophakia are almost always high myopes. Why?

Because the lens is small, it has a short radius of curvature. Further, because it is spherical, it is more curved than is a normal lens. These two factors give the m'spheric lens vastly more converging power than a normal lens possesses.

With what condition is microspherophakia most frequently associated?

Weill-Marchesani syndrome

Note: The *Glaucoma* book mentions only two syndromic associations for microspherophakia: Weill-Marchesani and Marfan. Further, it implies that microspherophakia occurs at equal rates in the two conditions. However, as the other *BCSC* books make clear, microspherophakia is associated with these other conditions as well. Further, it is far more likely to occur in Weill-Marchesani than in any of these other conditions, including Marfan.

With what conditions is microspherophakia occasionally associated?

-- Lowe syndrome
-- Alport syndrome
-- Marfan syndrome
-- Peters anomaly
-- Congenital rubella

Weill-Marchesani is strongly associated with microspherophakia.

How does **ectopia lentis** lead to pupillary block and ACG?

By allowing the displaced lens to move into and blocks the pupil, producing the pressure gradient, with subsequent iris bombé and angle closure.
Secondary Angle Closure Glaucoma

- **Lens-Induced**
  - Phacomorphic
  - Ectopia lentis

- **Aphakic/Pseudophakic**

**w/ Pupillary Block**

**w/o Pupillary Block**

**In a few words, how would you describe the shape of a microspherophakic lens?**

**With what condition is microspherophakia most frequently associated?**

**How does ectopia lentis lead to pupillary block and ACG?**

By allowing the displaced lens to move into and block the pupil, producing the pressure gradient, with subsequent iris bombé and angle closure.

**How does ectopia lentis lead to pupillary block and ACG?**

**Weill-Marchesani syndrome**

**Ruby LAMP Weill-Marchesani** is a mnemonic for the conditions associated with microspherophakia:

- **Ruby** = Rubella
- **Lowe syndrome**
- **Alport syndrome**
- **Marfan syndrome**
- **Peters anomaly**
- **Congenital rubella**

How does ectopia lentis lead to pupillary block and ACG?

By allowing the displaced lens to move into and block the pupil, producing the pressure gradient, with subsequent iris bombé and angle closure.

**Pts with microspherophakia are almost always high myopes. Why?**

Further, because it is small, it has a short radius of curvature. Further, because it is spherical, it is more curved than is a normal lens. These two factors give the microspherophakic lens vastly more converging power than a normal lens possesses.
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Lentic-Induced

Aphakic/ Pseudophakic

Phacomorphic

Ectopia lentis w/o Pupillary Block

Ectopia lentis w/ Pupillary Block

In a few words, how would you describe the shape of a microspherophakic lens?

With what condition is microspherophakia most frequently associated?

Weill-Marchesani syndrome

In three words (including syndrome), what are Lowe and Alport syndromes?

Lenticonus

How does ectopia lentis lead to pupillary block and ACG?

By allowing the displaced lens to move into and block the pupil, producing the pressure gradient, with subsequent iris bombe and angle closure.

With regard to lens 'displacement'—what do the following terms mean?

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PTs with microspherophakia are almost always high myopes. Why?

Because the lens is small, it has a short radius of curvature. Further, because it is spherical, it is more curved than is a normal lens. These two factors give the lens vastly more converging power than a normal lens possesses.

With what conditions is microspherophakia occasionally associated?

--Lowe syndrome
--Alport syndrome
--Marfan syndrome
--Peters anomaly
--Congenital rubella

In three words (including syndrome), what are Lowe and Alport syndromes?

Familial oculorenal syndromes

What is their classic (nonocular) presenting sign?

Hematuria

Microspherophakia is not the classic lens finding in the oculorenal syndromes (and should not be the first one out of your mouth if pimped about them). What is?

Lenticonus

Weill-Marchesani is strongly associated with microspherophakia.
**Secondary Angle Closure Glaucoma**

- **w/ Pupillary Block**
- **w/o Pupillary Block**

**Lens-Induced**
- **Aphakic/Pseudophakic**
  - **Phacomorphic**
  - **Ectopia lentis**

**With regard to lens 'displacement'—what do the following terms mean?**
- **Sublux(at)ed:** The lens is partially displaced, but remains in the 'general area'.
- **Lux(at)ed:** The lens is dislocated—completely removed from the pupillary aperture. All zonular attachments have been disrupted.

**How does ectopia lentis lead to pupillary block and ACG?**
By allowing the displaced lens to move into and block the pupil, producing the pressure gradient, with subsequent iris bombe and angle closure.

**In a few words, how would you describe the shape of a microspherophakic lens?**
In three words (including syndrome), what are Lowe and Alport syndromes?
- Familial oculorenal syndromes
- Lowes syndrome
- Alport syndrome
- Marfan syndrome
- Peters anomaly
- Congenital rubella

**With what condition is microspherophakia most frequently associated?**
Weill-Marchesani syndrome

**Weill-Marchesani is strongly associated with microspherophakia.**

**With what conditions is microspherophakia occasionally associated?**
- Lowe syndrome
- Alport syndrome
- Marfan syndrome
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**In three words (including syndrome), what are Lowe and Alport syndromes?**
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- **In a few words, how would you describe the shape of a microspherophakic lens?**
  - The name says it all: the lens is small ('micro') and round ('sphero').

**Typically, the entirety of the lens equator can be seen in the pupillary aperture when the pt is widely dilated.**

**Pts with microspherophakia are almost always high myopes. Why?**
Because the lens is small, it has a short radius of curvature. Further, because it is spherical, it is more curved than a normal lens. These two factors give the microspherophakic lens vastly more converging power than a normal lens possesses.

**What common slit-lamp observation owes to the lens' small size?**
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Reminder:

**Secondary Angle Closure Glaucoma**

- **Lens-Induced**
  - Phacomorphic
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- **Aphakic/Pseudophakic**

  **w/ Pupillary Block**

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**In three words (including syndrome), what are Lowe and Alport syndromes?**

Familial oculorenal syndromes

**What is their classic (nonocular) presenting sign?**

Hematuria

**Microspherophakia is not the classic lens finding in the oculorenal syndromes (and should not be the first one out of your mouth if pimped about them). What is?**

Lenticonus
**Secondary Angle Closure Glaucoma**

w/ Pupillary Block

w/o Pupillary Block

**Lens-Induced**

- Phacomorphic
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- **w/ Pupillary Block**
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Secondary Angle Closure Glaucoma

Anterior lenticonus in Alport syndrome
**Secondary Angle Closure Glaucoma**

- **Lens-Induced**
  - Phacomorphic
  - **Ectopia lentis**
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**w/ Pupillary Block**

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Displacement of the lens from its normal anatomic position.

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**What protein is abnormal in Marfan’s?**

**Fibrillin**

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**What three structures/systems manifest abnormalities in Marfan’s?**

-- The eye (duh)
-- The cardiovascular
-- The musculoskeletal
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

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-- The eye (duh)
-- The cardiovascular
-- The musculoskeletal

What cardiovascular abnormalities are common?

-- Dilatation of the aortic root and descending aorta
-- Aortic aneurysms
-- Mitral valve prolapse

Are these abnormalities clinically significant?

Indeed they are—they are responsible for the significantly shortened lifespan of Marfan pts
Secondary Angle Closure Glaucoma

- Lens-Induced w/ Pupillary Block
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Displacement of the lens from its normal anatomic position.

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Secondary Angle Closure Glaucoma

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Indeed they are—they are responsible for the significantly shortened lifespan of Marfan pts.

How significant is the lifespan shortening?

Quite. The life-expectancy of Marfan pts is about half that of the so-called normal population.
**Q/A**

**Secondary Angle Closure Glaucoma**

- **w/ Pupillary Block**
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- Phacomorphic
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**Secondary Angle Closure Glaucoma**

**With Pupillary Block**

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Secondary Angle Closure Glaucoma

Marfan syndrome: Aortic dissection
**Secondary Angle Closure Glaucoma**

- **Lens-Induced**
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Because the lens is small, it has a short radius of curvature. Further, because it is spherical, it is more curved than is a normal lens. These two factors give the m'spheric lens vastly more converging power than a normal lens possesses.

**With what condition is microspherophakia most frequently associated?**

**Weill-Marchesani syndrome**

Weill-Marchesani is strongly associated with microspherophakia.

**With what conditions is microspherophakia occasionally associated?**

- **Lowe syndrome**
- **Alport syndrome**
- **Marfan syndrome**
- **Peters anomaly**
- **Congenital rubella**

**What protein is abnormal in Marfan’s?**

Fibrillin

**What three structures/systems manifest abnormalities in Marfan’s?**

- The eye (duh)
- The cardiovascular
- The musculoskeletal

**What musculoskeletal abnormalities are common?**

- **The musculoskeletal**
  - —
  - —
**Q/A**

**Secondary Angle Closure Glaucoma**

- **Lens-Induced**
  - **Aphakic/Pseudophakic**
  - Phacomorphic
  - Ectopia lentis

- **w/ Pupillary Block**

- **w/o Pupillary Block**

**With regard to lens 'displacement'—what do the following terms mean?**

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**How does ectopia lentis lead to pupillary block and ACG?**

By allowing the displaced lens to move into and block the pupil, producing the pressure gradient, with subsequent iris bombé and angle closure.

**In a few words, how would you describe the shape of a microspherophakic lens?**

The name says it all: the lens is small ('micro') and round ('sphero').

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**What musculoskeletal abnormalities are common?**

- Arachnodactyly
- Hypermobile joints
- Sternum deformities (eg, pectus excavatum)
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

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What proportion of Marfan pts manifest ocular abnormalities?

At least 80%

Other than ectopia lentis and (occasionally) microspherophakia, what two ocular structural abnormalities are often present?

--Corneal shape abnormalities
--Increased axial length
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The cornea tends to be flatter than normal as well as larger than normal

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We said earlier that ‘pupillary block’ involves contact between the pupillary margin and the lens. If there’s no lens, what’s blocking the pupil in aphakic secondary ACG?
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The culprit is the vitreous face.
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We said earlier that ‘pupillary block’ involves contact between the pupillary margin and the lens. If there’s no lens, what’s blocking the pupil in aphakic secondary ACG? The culprit is the vitreous face. If it bulges forward, it can block the pupil just as readily as can the lens.
Aphakic pupillary block. Now, this pt is not aphakic (an AC IOL is present). Nevertheless, the pic beautifully depicts the mechanism of aphakic pupillary block, that being the vitreous face (*line*) occupying the pupillary aperture, thereby impeding the circulation of newly-created aqueous from the PC to the AC.
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How is aphakic secondary ACG managed?
We said earlier that ‘pupillary block’ involves contact between the pupillary margin and the lens. If there’s no lens, what’s blocking the pupil in aphakic secondary ACG? The culprit is the vitreous face. If it bulges forward, it can block the pupil just as readily as can the lens.

*How is aphakic secondary ACG managed?* Pretty much the same as if the culprit was the native lens—pour aqueous suppressants onto the eye (+/- hyperosmotic agents to dehydrate the V), then perform as many LPIs as necessary as soon as possible.
What sort of IOL is commonly implicated in pupillary block secondary ACG?
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The AC sort. Pressure in the posterior chamber pushes the iris against the IOL from behind, preventing aqueous from passing freely through the pupil. This initiates the now-familiar PC>AC pressure gradient→anterior bowing of the peripheral iris→occlusion of the angle.
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OK, so why doesn’t this happen every time an AC IOL is placed?
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Because a peripheral iridotomy is created during the cataract surgery
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OK, so why doesn’t this happen every time an AC IOL is placed?
Because a peripheral iridotomy is created during the cataract surgery.
That feeling when an AC IOL is implanted, but the surgeon forgets to create a PI
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

Lens-Induced

Aphakic/
Pseudophakic

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OK, so why doesn't this happen every time an AC IOL is placed?
Because a peripheral iridotomy is created during the cataract surgery.

Then why does ACG ever develop?
Because on occasion the PI gets blocked, either by an (unabridged word) or the (two words).
Secondary Angle Closure Glaucoma

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Secondary Angle Closure Glaucoma

Pseudophakic secondary ACG. In this case, a too-central PI (@5 o’clock) is occluded by the IOL optic. Note the ballooning iris.
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How is pseudophakic ACG managed?
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Because a peripheral iridotomy is created during the cataract surgery

Then why does ACG ever develop?
Because on occasion the PI gets blocked, either by an IOL haptic or the vitreous face

How is pseudophakic ACG managed?
The usual way—aqueous suppressants and urgent LPI(s)
Secondary Angle Closure Glaucoma

w/ Pupillary Block

- Lens-Induced
  - Phacomorphic
  - Ectopia lentis
- Aphakic/Pseudophakic

w/o Pupillary Block

? ?

Two mechanisms
Secondary Angle Closure Glaucoma

- w/ Pupillary Block
  - Lens-Induced
    - Phacomorphic
    - Ectopia lentis
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- w/o Pupillary Block
  - Two mechanisms
    - ‘Push’
    - ‘Pull’
What do push and pull mean in this context?

--*Push* refers to anterior displacement of the peripheral iris by...

--*Pull* refers to anterior displacement of the peripheral iris by...
**Secondary Angle Closure Glaucoma**

- **w/ Pupillary Block**
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  - ‘Push’
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**What do push and pull mean in this context?**

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**Push** refers to anterior displacement of the peripheral iris by... a space-occupying process occurring immediately behind it; ie, the peripheral iris is being ‘pushed’ into the angle.

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--**Pull** refers to anterior displacement of the peripheral iris by... a contractile process occurring on its anterior surface; ie, the peripheral iris is being ‘pulled’ into the angle.
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Some conditions have the ability to close the angle by both pushing and/or pulling the peripheral iris
Secondary Angle Closure Glaucoma

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    - ?
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    - Both/Either
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- 'Push'
  - Aqueous misdirection
  - ERD/choroidal effusion
  - Retinal surgery
  - Nanophthalmos
  - Drug-induced
    - PFV

- 'Pull'
  - Both/ Either
What is aqueous misdirection syndrome?

Aqueous misdirection

Secondary Angle Closure Glaucoma

Lens-Induced Aphakic/Pseudophakic w/ Pupillary Block w/o Pupillary Block

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ERD/choroidal effusion

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Retinal surgery

Drug-induced

Both/Either

How does it present?

With high IOP and a uniformly shallow AC in the acute post-op period after intraocular surgery

What is the chief risk factor?

Surgery in an eye with tight angles or PAS

By what other names is it known?

--Malignant glaucoma

--Ciliary-block glaucoma

How is aqueous misdirection syndrome managed medically?

With the triad of aggressive aqueous suppression, aggressive cycloplegia and dehydration of the vitreous with hyperosmotic agents

Is there a role for surgery in managing aqueous misdirection?

Yes; resolution often requires surgical or laser disruption of the vitreous face
What is aqueous misdirection syndrome?
A rare condition in which anterior rotation of the ciliary body causes newly-produced aqueous to be (mis)directed toward the vitreous rather than into the posterior, then anterior chambers.
**Secondary Angle Closure Glaucoma**

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A rare condition in which anterior rotation of the ciliary body causes newly-produced aqueous to be (mis)directed toward the vitreous rather than into the posterior, then anterior chambers.

How does it present?
With high IOP and a uniformly shallow AC in the acute post-op period after intraocular surgery.

Is there a role for surgery in managing aqueous misdirection?
Yes, resolution often requires surgical or laser disruption of the vitreous face.
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Take note—the iris does not have a ‘bombé’ configuration as occurs in pupillary-block ACG.

How is aqueous misdirection syndrome managed medically?
With the triad of aggressive aqueous suppression, aggressive cycloplegia, and dehydration of the vitreous with hyperosmotic agents.

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Aqueous misdirection. The iris does not have a bombé configuration.

Aqueous misdirection. Lateral illumination produces shadowing nasally, revealing the extent of AC shallowing. Note the presence of an LPI, ineffective because pupillary block is not present.
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If a post-op pt presents with a flat chamber and **low IOP**, what tops the DDx?

Aqueous misdirection
**Secondary Angle Closure Glaucoma**

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If a post-op pt presents with a flat chamber and **low IOP**, what tops the DDx?
Wound leak

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The Glaucoma book mentions three types of retinal surgery that can lead to secondary angle-closure glaucoma—what are they?
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- Scleral buckling
- PRP
- PPV
Secondary Angle Closure Glaucoma

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What is the typical mechanism of secondary ACG after SB surgery?
The Glaucoma book mentions three types of retinal surgery that can lead to secondary angle-closure glaucoma—what are they?

What is the typical mechanism of secondary ACG after SB surgery?

Elongation of the eye produces shallowing of the peripheral AC, sometimes aggravated by a choroidal effusion rotating the CB forward.

- Scleral buckling
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The Glaucoma book mentions three types of retinal surgery that can lead to secondary angle-closure glaucoma—what are they?

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- Retinal surgery
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The use of a tamponading injectable (eg, air; silicone oil)
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- Scleral buckling
- Scleral buckling PRP
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How does PRP lead to secondary ACG?
It can produce a choroidal effusion that rotates the CB forward.
What does it mean to say an eye is nanophthalmic?

- It means the eye is small—axial length < 20 mm; small/shallow AC, small (possibly micro-) cornea.
- The lens is comparatively large for the otherwise small eye.
- The sclera tends to be abnormally thick, which can impede venous drainage of the eye by compromising flow through the vortex veins.
- All of these factors combine to render nanophthalmic eyes highly susceptible to angle closure.
Secondary Angle Closure Glaucoma

Q/A

w/ Pupillary Block

w/o Pupillary Block

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Secondary Angle Closure Glaucoma

Nanopthalmic eye. Note the thickness of the sclera.
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**Secondary Angle Closure Glaucoma**

**w/ Pupillary Block**

**w/o Pupillary Block**

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**How about filtering surgery?**

As these eyes are highly prone to intraoperative choroidal effusion, it should be avoided if possible.
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Secondary Angle Closure Glaucoma

w/ Pupillary Block

The Glaucoma book addresses only one drug re inducing ACG. Which one?

w/o Pupillary Block

- 'Push'
  - Aqueous misdirection
  - ERD/choroidal effusion
  - Retinal surgery
  - Nanophthalmos

- 'Pull'
  - Drug-induced
  - PFV
  - Both/Either
Secondary Angle Closure Glaucoma

w/ Pupillary Block

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Topiramate

Aqueous misdirection
ERD/choroidal effusion
Retinal surgery
Nanophthalmos

Drug-induced

PFV

Both/ Either

What are the common indications for topiramate use?
-- Migraine prophylaxis
-- Idiopathic intracranial hypertension

What is the mechanism of angle closure?
Ciliochoroidal effusion leads to zonular relaxation, which leads to pronounced anterior movement of the lens-iris diaphragm, which shallows the AC and causes the peripheral iris to appose and close the angle.

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Severe bilateral ocular pain, plus blurry vision
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**Secondary Angle Closure Glaucoma**

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The Glaucoma book mentions two other indications—what are they?
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--Migraine prophylaxis
--Epilepsy
--Depression

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Secondary Angle Closure Glaucoma

w/ Pupillary Block

- Lens-Induced Aphakic/Pseudophakic
- Phacomorphic Ectopia lentis
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The *bilaterality* of topiramate-induced ACG must be stressed. If it ain’t bilateral, it ain’t topiramate-induced!*

Severe *bilateral* ocular pain, plus blurry vision

*On the OKAP and/or Boards, that is
Slit-lamp photograph at presentation, revealing conjunctival chemosis, corneal edema and markedly shallow anterior chamber in right (A) and left eye (B). Insets: Slit-image showing shallow peripheral anterior chamber; depth is marked with line. B-scan ultrasound at presentation showed peripheral choroidal effusions (arrow) in Right (C) and left (D) eyes.
Secondary Angle Closure Glaucoma

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What causes the blurry vision? (Other than corneal edema.)
Myopic shift

Why do these pts get myopic shift?
Forward displacement of the lens increases its effective power (ie, the secondary focal point of a previously emmetropic eye will be pulled forward into the vitreous)

Drug-induced

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Retinal surgery
Nanophthalmos
PFV
Both/ Either

Primary vs. secondary
Secondary Angle Closure Glaucoma

**w/ Pupillary Block**

- Lens-induced Aphakic/Pseudophakic w/ Pupillary Block w/o Pupillary Block
- Secondary Angle Closure Glaucoma
  - Phacomorphic
  - Ectopia lentis
  - 'Push' 'Pull'
  - ERD/choroidal effusion
  - Nanophthalmos
  - Retinal surgery

**w/o Pupillary Block**

- The Glaucoma book addresses only one drug re inducing ACG. Which one? Topiramate

- What are the common indications for topiramate use?
  -- Migraine prophylaxis
  -- Idiopathic intracranial hypertension

- What is the mechanism of angle closure?
  - Ciliochoroidal effusion leads to zonular relaxation, which leads to pronounced anterior movement of the lens-iris diaphragm, which shallows the AC and causes the peripheral iris to appose and close the angle

- What is the classic presentation of topiramate-induced ACG?
  - Severe bilateral ocular pain, plus blurry vision

- What causes the blurry vision? (Other than corneal edema.) Myopic shift

- Why do these pts get myopic shift?
  - Forward displacement of the lens increases its effective power (ie, the secondary focal point of a previously emmetropic eye will be pulled forward into the vitreous)

- Drug-induced
  - Aqueous misdirection
  - ERD/choroidal effusion
  - Retinal surgery
  - Nanophthalmos
  - PFV

- Both/Either
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How is topiramate-induced ACG managed?
Severe bilateral ocular pain, plus blurry vision

How is topiramate-induced ACG managed?
The most important step is stopping the topiramate ASAP. Aqueous suppressants should be used to acutely lower IOP. Finally, aggressive cycloplegia may pull the iris back and lessen or break the angle closure.
## Secondary Angle Closure Glaucoma

### w/ Pupillary Block

- Lens-Induced
- Aphakic/Pseudophakic
- w/ Pupillary Block w/o Pupillary Block
- Phacomorphic Ectopia lentis
- "Push" / "Pull" ERD/choroidal effusion
- Nanophthalmos
- Retinal surgery
- Drug-induced PFV

### w/o Pupillary Block

- Aqueous misdirection
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- Drug-induced
- PFV

w/o Pupillary Block

- Persistent fetal vasculature

Q

What does PFV stand for in this context?

Anterior and posterior

Which form can cause secondary ACG?

The anterior

In general terms, how does anterior PFV manifest?

As a retrolental fibrovascular membrane that contracts over time, in the process shallowing the AC angle

What is the inheritance pattern for PFV?

None (it is sporadic)

Does it present unilaterally, or bilaterally?

It is unilateral in 90% of cases
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Secondary Angle Closure Glaucoma

w/ Pupillary Block

What does PFV stand for in this context? Persistent fetal vasculature "aka..."

By what name was this condition known previously?

w/o Pupillary Block

'Push'

- Aqueous misdirection
- ERD/choroidal effusion
- Retinal surgery
- Nanophthalmos
- Drug-induced

'Pull'

PFV

Both/Either
Secondary Angle Closure Glaucoma

w/ Pupillary Block

What does PFV stand for in this context?
Persistent fetal vasculature aka...PHPV

By what name was this condition known previously?
Persistent hyperplastic primary vitreous (PHPV)

w/o Pupillary Block

‘Push’
- Aqueous misdirection
- ERD/choroidal effusion
- Retinal surgery
- Nanophthalmos
- Drug-induced
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‘Pull’
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Secondary Angle Closure Glaucoma

w/ Pupillary Block

- Lens-Induced Aphakic/Pseudophakic w/ Pupillary Block
- w/o Pupillary Block

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PFV comes in two forms—what are they?

- Aqueous misdirection
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PFV

Both/Either

‘Push’

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Secondary Angle Closure Glaucoma

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Secondary Angle Closure Glaucoma

PFV: Retrolental membrane
Secondary Angle Closure Glaucoma

PFV: Retrolental membrane. Note the ciliary processes (arrow)
Secondary Angle Closure Glaucoma

PFV: Shallow AC

PFV: Retrolental membrane (2); ciliary processes (3); note also the very shallow AC. (4 is pointing to the iris in what amounts to the world’s worst PAS on that side)
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**Secondary Angle Closure Glaucoma**

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Is PFV inevitably a blinding disease? No—early cataract extraction and membranectomy may salvage the eye and useful vision.
Secondary Angle Closure Glaucoma

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‘Push’

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- Drug-induced
- **PFV**

‘Pull’

- Both/ Either

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(This is a good point in the set to take a break)
Secondary Angle Closure Glaucoma

- w/ Pupillary Block
  - Lens-Induced
    - Phacomorphic
    - Ectopia lentis
  - Aphakic/ Pseudophakic

- w/o Pupillary Block
  - ‘Push’
    - Aqueous misdirection
    - ERD/choroidal effusion
    - Retinal surgery
    - Nanophthalmos
    - Drug-induced
    - PFV
      - Both/ Either
  - ‘Pull’
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    - ?
    - ?
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    - ?
Secondary Angle Closure Glaucoma

w/ Pupillary Block

- Lens-Induced
  - Phacomorphic
  - Ectopia lentis
- Aphakic/Pseudophakic

w/o Pupillary Block

- ‘Push’
  - Aqueous misdirection
  - ERD/choroidal effusion
  - Retinal surgery
  - Nanophthalmos
  - Drug-induced
  - PFV
- ‘Pull’
  - NVG
  - ICE
  - Flat AC
  - Epithelial/fibrous ingrowth

Both/Either
Neovascularization of the angle (NVA) leads to angle-closure glaucoma. The NVA vessels don't ride solo; rather, they are accompanied by contractile elements (e.g., fibroblasts). Along with the neo vessels, these elements will establish a network that crosses from the peripheral iris to the peripheral cornea. Once established, contractile elements will contract, and when they do, they pull the iris up against the angle, rendering it closed.

What two-word phrase (not 'diabetic retinopathy'—think more generally) describes the fundamental cause of most cases of NVG?

'Retinal ischemia'

How does retinal ischemia lead to NVI and NVA?

In a desperate attempt to acquire the oxygen they're lacking, the ischemic retinal cells release the signaling molecule VEGF, a potent inducer of new blood vessel formation. This VEGF diffuses from the vitreous cavity into the anterior segment, where it induces the NVI/NVA process.
Neovascularization of what structure causes neovascular glaucoma (NVG)?
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Secondary Angle Closure Glaucoma

NVA
Secondary Angle Closure Glaucoma

Neovascularization of the angle (NVA)

Neovascularization of what structure causes neovascular glaucoma (NVG)?

Neovascularization of the angle (NVA)

Neovascularization of what structure typically precedes and leads to NVA?

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Neovascularization of the iris (NVI)

Where on the iris does NVI typically first appear? What does it look like?

At the pupillary margin. As small 'tufts' of vessels.

As it develops further, how does it grow (ie, direction, and course)?

In a meandering fashion toward the angle (normal iris vessels typically run in a rather direct radial fashion).
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NVI
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Is NVA always the result of NVI reaching the angle?

No, it can arise de novo in the angle itself.
Secondary Angle Closure Glaucoma

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No higher than Schwalbe’s line

Why can’t they go any higher?
Because vessels cannot grow onto normal corneal endothelium
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In a desperate attempt to acquire the oxygen they’re lacking, the ischemic retinal cells release the signaling molecule VEGF, a potent inducer of new blood vessel formation. This VEGF diffuses from the vitreous cavity into the anterior segment, where it induces the NVI/NVA process.

How far up the peripheral cornea do the NVA vessels go?

No higher than Schwalbe’s line

Why can’t they go any higher?

Because vessels cannot grow onto normal corneal endothelium
Neovascularization of what structure causes neovascular glaucoma (NVG)? Neovascularization of the angle (NVA)

How does NVA lead to angle-closure glaucoma?
The NVA vessels don’t ride solo; rather, they are accompanied by contractile elements (eg, fibroblasts). Along with the neo vessels, these elements will establish a network that crosses from the peripheral iris to the peripheral cornea. Once established, contractile elements gonna contract, and when they do, they pull the iris up against the angle, rendering it closed.

(No question—proceed when ready)
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This configuration—iris affixed to the angle—is known by what name?
**Secondary Angle Closure Glaucoma**

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This configuration—iris affixed to the angle—is known by what name? **Peripheral anterior synechia**e (PAS)
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CRVOs are classified into one of two categories. What are these?
Ischemic and nonischemic

Which sort is implicated in the development of NVG?
Seriously?

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

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Much less likely
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--CRAO
--Ocular ischemia of other causes

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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.
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In a desperate attempt to acquire the oxygen they're lacking, the ischemic retinal cells release the signaling molecule VEGF, a potent inducer of new blood vessel formation. This VEGF diffuses from the vitreous cavity into the anterior segment, where it induces the NVI/NVA process.

What are the three most common causes of ischemia that result in the development of NVG?

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

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, resulting in very little aqueous being 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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Neovascularization of the angle (NVA)

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The NVA vessels don't ride solo; rather, they are accompanied by contractile elements (e.g., fibroblasts). Along with the neo vessels, these elements will establish a network that crosses from the peripheral iris to the peripheral cornea. Once established, contractile elements gonna contract, and when they do, they pull the iris up against the angle, rendering it closed.

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Drug-induced
Both/Either
Retinal surgery
Flat AC
Epithelial/fibrous ingrowth
ICE
NVG
Q/A

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What is the treatment of choice for NVG 2ndry to retinal ischemia?

Panretinal photocoagulation (PRP)

What is the goal of PRP, ie, what are we trying to do?
The goal is to kill most of the cells in the peripheral retina

What is the therapeutic rationale? Why kill the peripheral retina?
As stated several times now: DBR renders portions of the retina hypoxic, and hypoxic cells release VEGF, initiating a cascade of deleterious events. OTOH, dead cells do not release VEGF. So by euthanizing the hypoxic retina, the intraocular VEGF burden is reduced, neovascularization is halted, and SVL is avoided.
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Finally: There are a few clinical scenarios in which NVI/NVA develop in the absence of retinal ischemia. One condition in particular is notorious for this—what is it?

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The NVI/NVA in FHI: In what regard is it highly unusual?

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The NVI/NVA in FHI: In what regard is it highly unusual?

It never leads to the development of PAS, and thus doesn’t provoke NVG in the first place. In fact, and when they do, they pull the iris up against the angle, rendering it closed.

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Secondary Angle Closure Glaucoma
In this context, what does ICE stand for?

Iridocorneal endothelial syndrome

In a nutshell, what is ICE?

A sporadic condition in which abnormal corneal endothelial cells lead to a variety of corneal, iris and angle problems.

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 (recall it's sporadic, not inherited).
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w/ Pupillary Block

w/o Pupillary Block

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‘Pull’

NVG

Flat AC

Epithelial/fibrous ingrowth

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What’s abnormal about the endothelial cells in ICE? They behave like epithelial cells, with a strong tendency to migrate. These so-called ‘ICE cells’ will migrate across the angle and onto the iris, laying down a fibrillar membrane as they go. These cells and their associated membrane account for all of the signs and symptoms found in ICE.
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-- Iris changes  
-- Corneal changes  
-- Peripheral anterior synechiae (PAS)  
-- Elevated IOP/glaucoma
  
  -- 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 (recall it’s sporadic, not inherited)
In this context, what does ICE stand for?
Iridocorneal endothelial syndrome

In a nutshell, what is ICE?
A sporadic condition in which abnormal corneal endothelial cells lead to a variety of corneal, iris and angle problems.

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

What sort of iris changes will be present?
-- The pupil may be out-of-round or displaced
-- The iris may be atrophic and ‘torn’
-- Ectropion uveae may be present
-- Iris nodules and/or nevi may be present

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--Iris nodules and/or nevi may be present

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Secondary Angle Closure Glaucoma

ICE: Corectopia (displaced pupil)
Secondary Angle Closure Glaucoma

ICE: Iris atrophy
Secondary Angle Closure Glaucoma

ICE: Iris nodules (note also the ectropion uveae)
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  --Changes in the eye’s appearance
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  --Decreased VA

What sort of corneal changes will be present?

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- Iris changes
- Corneal changes
- Peripheral anterior synechiae (PAS)
- Elevated IOP/glaucoma
  - Changes in the eye’s appearance
  - Ocular pain
  - Decreased VA

What sort of corneal changes will be present?
It may appear hazy or milky as a result of corneal edema
Secondary Angle Closure Glaucoma

ICE: Corneal edema
In this context, what does ICE stand for?
Iridocorneal endothelial syndrome

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What ‘pertinent negative’ will be elicited when taking a history?
She will deny any family history of similar eye findings (recall it’s sporadic, not inherited)

What are the classic terms for describing the slit-lamp appearance of the abnormal endothelium?
'Hammered silver' or 'beaten bronze'
In this context, what does ICE stand for?
Iridocorneal endothelial syndrome

In a nutshell, what is ICE?
A sporadic condition in which abnormal corneal endothelial cells lead to a variety of corneal, iris and angle problems

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What are the classic terms for describing the slit-lamp appearance of the abnormal endothelium?
‘Hammered silver’ or ‘beaten bronze’
Secondary Angle Closure Glaucoma

ICE: ‘Hammered silver’ corneal endothelium
In this context, what does ICE stand for?
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--Decreased VA

What are the classic terms for describing the slit-lamp appearance of the abnormal endothelium?
‘Hammered silver’ or ‘beaten bronze’

Beaten bronze is also used to describe the appearance of the endothelium in what condition?

What ‘pertinent negative’ will be elicited when taking a history?
She will deny any family history of similar eye findings (recall it’s sporadic, not inherited)
**Secondary Angle Closure Glaucoma**

w/ Pupillary Block

w/o Pupillary Block

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

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What are the classic terms for describing the slit-lamp appearance of the abnormal endothelium?
‘Hammered silver’ or ‘beaten bronze’

---

Beaten bronze is also used to describe the appearance of the endothelium in what condition?
Fuchs dystrophy
In this context, what does ICE stand for?
Iridocorneal endothelial syndrome

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

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

What ‘pertinent negative’ will be elicited when taking a history?
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--Iris changes
--Corneal changes
--Peripheral anterior synechiae (PAS)
--Elevated IOP/glaucoma

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

What ‘pertinent negative’ will be elicited when taking a history?
She will deny any family history of similar eye findings (recall it’s sporadic, not inherited)
In this context, what does ICE stand for? Iridocorneal endothelial syndrome

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

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

What does high mean in this context? Taking a history? Findings (recall it’s sporadic, not inherited)
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

In this context, what does ICE stand for?
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- Iris changes
- Corneal changes
- **Peripheral anterior synechiae (PAS)**
- Elevated IOP/glaucoma

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

What does high mean in this context? That the PAS extend above Schwalbe’s line (SL)

What are the signs of ICE? NVG, Flat AC, Epithelial/fibrous ingrowth
Secondary Angle Closure Glaucoma

ICE: Broad and high PAS
Secondary Angle Closure Glaucoma

w/ Pupillary Block

w/o Pupillary Block

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- Corneal changes
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- Elevated IOP/glaucoma

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

But we said earlier that PAS don’t cross SL. What’s the deal?
That the PAS extend above Schwalbe’s line (SL)

What does high mean in this context?
That the PAS extend above Schwalbe’s line (SL)
In this context, what does ICE stand for?
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Two words are used to describe the appearance of the PAS in ICE. What are they? ‘Broad’ and ‘high’

But we said earlier that PAS don’t cross SL. What’s the deal?
What we said was that PAS don’t cross normal endothelium, but the endothelium in ICE is highly abnormal

What does high mean in this context?
That the PAS extend above Schwalbe’s line (SL)

But we said earlier that PAS don’t cross SL. What’s the deal?
That the PAS extend above normal endothelium, but the endothelium in ICE is highly abnormal
How common is glaucoma in ICE?

ICE (Iridocorneal Endothelial Syndrome) is a sporadic condition where abnormal corneal endothelial cells lead to a variety of corneal, iris, and angle problems. The typical patient is a young-to-middle-aged adult female. Three common complaints are:

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

When taking a history, the pertinent negative will be:

She will deny any family history of similar eye findings (recall it’s sporadic, not inherited).

The common signs of ICE syndrome include:

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

Glaucoma develops in about half of ICE cases. Secondary pupillary-block glaucoma can be managed medically in some cases, but many require filtering surgery. SLT (Selective Laser Trabeculoplasty) is ineffective in ICE and should be avoided.
How common is glaucoma in ICE? Quite—it develops in about half of cases.

What ‘pertinent negative’ will be elicited when taking a history? She will deny any family history of similar eye findings (recall it’s sporadic, not inherited).

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

In a nutshell, what is ICE? A sporadic condition in which abnormal corneal endothelial cells lead to a variety of corneal, iris and angle problems.

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

How common is glaucoma in ICE? Quite—it develops in about half of cases.

How does ICE produce secondary pupillary-block glaucoma? As mentioned earlier, ICE cells may cross the angle, leaving a membrane in their wake. This membrane can contract, producing the broad and high PAS discussed previously. Or, the membrane can occlude the angle simply by covering it.

Can the glaucoma be managed medically? In some cases, yes. However, many go on to filtering surgery.

Is SLT a good option? No—it is ineffective in ICE, and should be avoided.
Secondary Angle Closure Glaucoma

How common is glaucoma in ICE?
Quite—it develops in about half of cases

How does ICE produce secondary pupillary-block glaucoma?

--Peripheral anterior synechiae (PAS)
--Elevated IOP/glaucoma

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

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**In a nutshell, what is ICE?**
A sporadic condition in which abnormal corneal endothelial cells lead to a variety of corneal, iris and angle problems.

**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
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Can the glaucoma be managed medically?  
--Peripheral anterior synechiae (PAS)  
--Elevated IOP/glaucoma

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

What ‘pertinent negative’ will be elicited when taking a history?  
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How common is glaucoma in ICE? 
Quite—it develops in about half of cases

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As mentioned earlier, ICE cells may cross the angle, leaving a membrane in their wake. This membrane can contract, producing the broad and high PAS discussed previously. Or, the membrane can occlude the angle simply by covering it.

Can the glaucoma be managed medically? 
In some cases, yes. However, many go on to filtering surgery.

What ‘pertinent negative’ will be elicited when taking a history? 
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Peripheral anterior synechiae (PAS)

Elevated IOP/glaucoma

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Can the glaucoma be managed medically? In some cases, yes. However, many go on to filtering surgery.

Is SLT a good option? No—it has no role in managing ICE, and should be avoided

---Peripheral anterior synechiae (PAS)
---Elevated IOP/glaucoma
---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 (recall it’s sporadic, not inherited)
**Secondary Angle Closure Glaucoma**

**w/ Pupillary Block**

**w/o Pupillary Block**

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

*In a nutshell, what is ICE?*

**The BCSC books recognize three variants of ICE. What are they?**

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

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In a nutshell, what is ICE? The BCSC books recognize three variants of ICE. What are they?
- Iris nevus syndrome, aka Cogan-Reese syndrome
- Chandler syndrome
- Essential iris atrophy

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

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- Handler syndrome
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Take note of the sweet mnemonic!
-- Ocular pain
-- Decreased VA

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The BCSC books recognize three variants of ICE. What are they?
What is the predominant finding for each?
--Iris nevus syndrome, aka Cogan-Reese syndrome:
  --Chandler syndrome
  --Essential iris atrophy

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

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The BCSC books recognize three variants of ICE. What are they? What is the predominant finding for each?
--Iris nevus syndrome, *aka* Cogan-Reese syndrome: Iris nevi/nodules
--Chandler syndrome:
--Essential iris atrophy

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--Ocular pain
--Decreased VA

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- Chandler syndrome: Corneal edema
- Essential iris atrophy:

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-- Ocular pain
-- Decreased VA

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**Secondary Angle Closure Glaucoma**

- **w/ Pupillary Block**
- **w/o Pupillary Block**

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The BCSC books recognize three variants of ICE. What are they? What is the predominant finding for each?
- Iris nevus syndrome, aka Cogan-Reese syndrome: Iris nevi/nodules
- Chandler syndrome: Corneal edema
- Essential iris atrophy: Iris atrophy/tears

**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 (recall it’s sporadic, not inherited)
What clinical scenario typically produces the flat AC that leads to secondary ACG?
What clinical scenario typically produces the flat AC that leads to secondary ACG? 
Wound leak after cataract or filtering surgery
Secondary Angle Closure Glaucoma

w/ Pupillary Block

- Lens-Induced
  - Phacomorphic
  - Ectopia lentis
- Aphakic/Pseudophakic

w/o Pupillary Block

- ‘Push’
  - Aqueous misdirection
  - EDR/choroidal effusion
- ‘Pull’
  - NVG
  - ICE
  - Epithelial/fibrous ingrowth

What clinical scenario typically produces the flat AC that leads to secondary ACG?
Wound leak after cataract or filtering surgery

Will the IOP be high, or low?
What clinical scenario typically produces the flat AC that leads to secondary ACG?
Wound leak after cataract or filtering surgery

Will the IOP be high, or low?
Low
What clinical scenario typically produces the flat AC that leads to secondary ACG?

Wound leak after cataract or filtering surgery

Will the IOP be high, or low?

Low

How does a flat AC lead to ACG?

By allowing PAS to form

Recall that a flat AC + high (or even normal) IOP after CE is suggestive of aqueous misdirection syndrome.
What clinical scenario typically produces the flat AC that leads to secondary ACG?
Wound leak after cataract or filtering surgery

Will the IOP be high, or low?
Low

How does a flat AC lead to ACG?
Secondary Angle Closure Glaucoma

w/ Pupillary Block
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Will the IOP be high, or low?
Low

How does a flat AC lead to ACG?
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**Wound leak** after cataract or filtering surgery

How is a wound leak managed medically?

--

--

--
Q/A

Secondary Angle Closure Glaucoma

w/ Pupillary Block

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- ‘Push’
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- ‘Pull’

What clinical scenario typically produces the flat AC that leads to secondary ACG?

**Wound leak** after cataract or filtering surgery

How is a wound leak managed medically? It’s as simple as ABC(D):

-- A
-- B
-- C
-- D

Flat AC

Epithelial/fibrous ingrowth

NVG

ICE
Secondary Angle Closure Glaucoma

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  - Lens-Induced
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- w/o Pupillary Block
  - ‘Push’
    - Aqueous misdirection
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    - ICE
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What clinical scenario typically produces the flat AC that leads to secondary ACG?
**Wound leak** after cataract or filtering surgery

How is a wound leak managed medically? It’s as simple as ABC(D):

--- Aqueous suppressants
--- Bandage contact lens (BCL), if we’re talking post-CE
--- Cycloplegia
--- Discontinue (or at least Diminish) topical steroids
Q

What clinical scenario typically produces the flat AC that leads to secondary ACG?

**Wound leak** after cataract or filtering surgery

How is a wound leak managed medically?

- **Aqueous suppressants**
  - Bandage contact lens (BCL)
  - Cycloplegia
  - Discontinue (or at least diminish) topical steroids

Hol up—the IOP is already super low. What is the rationale for using aqueous suppressants?
Secondary Angle Closure Glaucoma

w/ Pupillary Block

- Lens-Induced
  - Phacomorphic
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- Aphakic/Pseudophakic

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  - Aqueous misdirection
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- 'Pull'

What clinical scenario typically produces the flat AC that leads to secondary ACG?

Wound leak after cataract or filtering surgery

How is a wound leak managed medically?

Hol up—the IOP is already super low. What is the rationale for using aqueous suppressants?

The idea is to promote closure of the leak by decreasing the flow of aqueous across it

Aqueous suppressants
- Bandage contact lens (BCL)
- Cycloplegia
- Discontinue (or at least Diminish) topical steroids

Flat AC

Epithelial/fibrous ingrowth

NVG

ICE
What clinical scenario typically produces the flat AC that leads to secondary ACG?

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How is a wound leak managed medically?

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**Aqueous suppressants**

-- Bandage contact lens (BCL), if we're talking post-CE
-- Cycloplegia
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Which 3 drug classes are aqueous suppressants?

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--
--
Secondary Angle Closure Glaucoma

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  - Cycloplegia
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How is a wound leak managed medically?

- **Hol up—the IOP is already super low. What is the rationale**

Which 3 drug classes are aqueous suppressants?

- α agonists
- β blockers
- Carbonic anhydrase inhibitors

Flat AC

Epithelial/fibrous ingrowth
What clinical scenario typically produces the flat AC that leads to secondary ACG?

**Wound leak** after cataract or filtering surgery

How is a wound leak managed medically? It’s as simple as ABC(D):

-- Aqueous suppressants
-- Bandage contact lens (BCL) if epithelial/choroidal effusion
-- Discontinue (or at least diminish) topical steroids

What is the purpose of cycloplegia?

To deepen the AC by rotating the ciliary body back.
**Secondary Angle Closure Glaucoma**

- **w/ Pupillary Block**
  - Lens-Induced
    - Phacomorphic
    - Ectopia lentis
  - Aphakic/Pseudophakic
- **w/o Pupillary Block**
  - ‘Push’
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  - ‘Pull’
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    - ICE

**Flat AC**
- Epithelial/fibrous ingrowth

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How is a wound leak managed medically? It’s as simple as ABC(D):
- Aqueous suppressants
- Bandage contact lens (BCL) if needed to prevent ACF
- Cycloplegia
- Discontinue (or at least diminish) topical steroids

What is the purpose of cycloplegia?
To deepen the AC by rotating the ciliary body back
What clinical scenario typically produces the flat AC that leads to secondary ACG?

Wound leak after cataract or filtering surgery

Why stop steroids? Won’t that increase inflammation?

- Discontinue (or at least Diminish) topical steroids

How is a wound leak managed medically?

- Aqueous suppressants
- Bandage contact lens (BCL), if we’re talking post-CE
- Cycloplegia
- Discontinue (or at least Diminish) topical steroids
What clinical scenario typically produces the flat AC that leads to secondary ACG?

**Wound leak** after cataract or filtering surgery

Why stop steroids? Won’t that increase inflammation?

Yes, but it will also promote leak closure by removing steroid-induced inhibition of wound healing

Discontinue (or at least Diminish) topical steroids

How is a wound leak medically managed?

--Aqueous suppressants
--Bandage contact lens (BCL), if we’re talking post-CE
--Cycloplegia
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**Wound leak** after cataract or filtering surgery

How is a **wound leak** managed medically? It's as simple as ABC(D):

- **A**queous suppressants
- **B**andage contact lens (BCL), if we're talking post-CE
- **C**ycloplegia
- **D**iscontinue (or at least **D**iminish) topical steroids

Under what circumstances should a wound leak be managed surgically?

1)  
2)  
3)  
4)
What clinical scenario typically produces the flat AC that leads to secondary ACG?

**Wound leak** after cataract or filtering surgery.

How is a wound leak managed medically?

- Aqueous suppressants
- Bandage contact lens (BCL), if we’re talking post-CE
- Cycloplegia
- Discontinue (or at least diminish) topical steroids

Under what circumstances should a wound leak be managed surgically?

1. No improvement by about 48 hours or so
2. Obvious wound gape
3. IOL-cornea touch
4. Iris prolapse
In a nutshell, what is epithelial/fibrous ingrowth?

Intraocular invasion by epithelial or fibrous tissue via a surgical or traumatic wound.

How do these entities produce secondary ACG?

- If the invading tissue grows over the angle, it can produce PAS, or even destroy the TM.

What do they look like at the slit lamp?

- Epithelial ingrowth: A thin gray sheet
- Fibrous ingrowth: A thick, gray-white, vascular layer

What is the treatment?

Extensive intraocular debridement can be attempted.

What is the prognosis?

Poor.
In a nutshell, what is epithelial/fibrous ingrowth?

Intraocular invasion by epithelial or fibrous tissue via a surgical or traumatic wound.
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If the invading tissue grows over the angle, it can produce PAS, or even destroy the TM
**Secondary Angle Closure Glaucoma**

- **w/ Pupillary Block**
  - Lens-Induced
  - Aphakic/Pseudophakic w/ Pupillary Block w/o Pupillary Block
- **w/o Pupillary Block**
  - Phacomorphic Ectopia lentis
  - 'Push' 'Pull'
  - NVG
  - ICE
  - Epithelial/fibrous ingrowth
  - Flat AC
  - Aqueous misdirection
  - Retinal surgery
  - Drug-induced
  - Both/Either
  - Nanophthalmos
  - PFV
  - ERD/choroidal effusion

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--Fibrous ingrowth
Secondary Angle Closure Glaucoma

Epithelial ingrowth after DSAEK
Secondary Angle Closure Glaucoma

Eye with a grey sheet with scalloped edges extending along the endothelium with overlying mild corneal edema

Anterior segment optical coherence tomography of the eye demonstrating a hyperreflective sheet extending through the surgical incision and spreading across the endothelium

Epithelial ingrowth after cataract surgery
In a nutshell, what is epithelial/fibrous ingrowth?
Intraocular invasion by epithelial or fibrous tissue via a surgical or traumatic wound

What simple procedure can be performed in the clinic to confirm the presence of epithelial ingrowth?
Shoot the suspected sheet of epithelial ingrowth with an argon laser

What reaction to a laser burn would indicate the tissue is in fact epithelial?
The production of a white burn

Epithelial ingrowth: A thin gray sheet
Fibrous ingrowth
In a nutshell, what is epithelial/fibrous ingrowth?
Intraocular invasion by epithelial or fibrous tissue via a surgical or traumatic wound

What simple procedure can be performed in the clinic to confirm the presence of epithelial ingrowth?
Shoot the suspected sheet of epithelial ingrowth with an [type of] laser

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Secondary Angle Closure Glaucoma

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Fibrous ingrowth:

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Drug-induced
Both/Either
Retinal surgery
Nanophthalmos
PFV
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What reaction to a laser burn would indicate the tissue is in fact epithelial?
Production of a white burn

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Shoot the suspected sheet of epithelial ingrowth with an argon laser.

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The production of a white burn color.

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--Fibrous ingrowth
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--Fibrous ingrowth: A thick, gray-white, vascular layer
Secondary Angle Closure Glaucoma

Fibrous ingrowth after cataract surgery
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How do these entities produce secondary ACG? If the invading tissue grows over the angle, it can produce PAS, or even destroy the TM.

What do they look like at the slit lamp?—Epithelial ingrowth: A thin gray sheet—Fibrous ingrowth: A thick, gray-white, vascular layer.

What is the treatment? Extensive intraocular debridement can be attempted.

What is the prognosis? Poor.
In a nutshell, what is epithelial/fibrous ingrowth?
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What is the prognosis?
Poor
Secondary Angle Closure Glaucoma

w/ Pupillary Block
- Lens-Induced
  - Phacomorphic
  - Ectopia lentis
- Aphakic/Pseudophakic

w/o Pupillary Block
- ‘Push’
  - Aqueous misdirection
  - ERD/choroidal effusion
  - Retinal surgery
  - Nanophthalmos
  - Drug-induced
  - PFV
- ‘Pull’
  - NVG
  - ICE
  - Flat AC
  - Epithelial/fibrous ingrowth

Both/Either

Next
Secondary Angle Closure Glaucoma

w/ Pupillary Block
- Lens-Induced
  - Phacomorphic
  - Ectopia lentis
- Aphakic/Pseudophakic

w/o Pupillary Block
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  - Nanophthalmos
  - Drug-induced
  - PFV
- ‘Pull’
  - NVG
  - ICE
  - Flat AC
  - Epithelial/fibrous ingrowth

Both/Either
- Inflammation
- Tumor

Next
Secondary Angle Closure Glaucoma

w/ Pupillary Block

- Lens-Induced
- Aphakic/Pseudophakic

w/o Pupillary Block

- ‘Push’
- ‘Pull’

By what mechanism could inflammation push the angle closed?

- NVG
- ICE
- Flat AC
- Epithelial/fibrous ingrowth

- PFV
- Both/Either

Inflammation

Inflammation

Tumor
Q/A

By what mechanism could inflammation push the angle closed?

A massive three words could push the lens into the pupil.
By what mechanism could inflammation push the angle closed?

A massive exudative RD could push the lens into the pupil.
By what mechanism could inflammation **push** the angle closed?
A massive exudative RD could push the lens into the pupil

How does inflammation **pull** the angle closed?

Inflammation compromises the blood-aqueous barrier, allowing copious amounts of inflammatory proteins and fibrin to accumulate in the AC. These substances can produce posterior synechiae leading to iris bombé and eventually angle closure. Additionally, PAS can form, especially if peripheral iris edema is present.
By what mechanism could inflammation **push** the angle closed? A massive exudative RD could push the lens into the pupil.

How does inflammation **pull** the angle closed? Inflammation compromises the blood-aqueous barrier, allowing copious amounts of inflammatory proteins and fibrin to accumulate in the AC. These substances can produce posterior synechiae leading to iris bombé and eventually angle closure. Additionally, PAS can form, especially if peripheral iris edema is already narrowing the angle.
Inflammatory glaucoma. Note the posterior synechiae as well as PAS.
How would a tumor push the angle closed?

If a tumor is anterior enough (or large enough), it can either directly (via mass effect) or indirectly (via associated exudation) move the lens-iris diaphragm forward, thereby shallowing the angle.

What two general types of tumor are known to do this?

-- Choroidal
-- Retinal
How would a tumor push the angle closed?
If a tumor is anterior enough (or large enough), it can either directly (via mass effect) or indirectly (via associated exudation) move the lens-iris diaphragm forward, thereby shallowing the angle.
Ring melanoma of the ciliary body. Pigmented ciliary body lesion noted on gonioscopy (arrow)

Ultrasound biomicroscopy of a ring melanoma of the ciliary body. (A) Main mass of tumor at 9:00 o'clock. (B) Tumor involving ciliary body at 11:00 o'clock. (C) Tumor extends under the iris at 6:00 o'clock and is associated with a small cyst (arrow). (T, tumor.)
How would a tumor push the angle closed?
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Secondary Angle Closure Glaucoma

w/ Pupillary Block
- Lens-Induced
- Phacomorphic
- Ectopia lentis
- Aphakic/Pseudophakic

w/o Pupillary Block
- ‘Push’
  - Aqueous misdirection
  - ERD/choroidal effusion
- ‘Pull’
  - NVG
  - ICE
  - Flat AC
  - Epithelial/fibrous ingrowth

How would a tumor pull the angle closed?

Both/Either
- Inflammation

Tumor
How would a tumor pull the angle closed?
Via one of two mechanisms:

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How would a tumor pull the angle closed?
Via one of two mechanisms:
--Tumor-induced NVI/NVA leading to NVG
--Tumor necrosis can produce severe inflammation, leading to posterior synechiae and/or PAS
Secondary Angle Closure Glaucoma

w/ Pupillary Block

- Lens-Induced
  - Phacomorphic
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- Aphakic/ Pseudophakic

w/o Pupillary Block

- ‘Push’
  - Aqueous misdirection
  - ERD/choroidal effusion

- ‘Pull’

- NVG
- ICE
- Flat AC
- Epithelial/ fibrous ingrowth

How would a tumor pull the angle closed?
Via one of two mechanisms:
--Tumor-induced NVI/NVA leading to NVG
  Tumor necrosis can produce severe inflammation, leading to PAS

Which tumors are notorious for inducing NVI/NVA?

- Tumor
- Inflammation

Both/ Either
Secondary Angle Closure Glaucoma

How would a tumor pull the angle closed?
Via one of two mechanisms:
-- Tumor-induced NVI/NVA leading to NVG
  Tumor necrosis can produce severe inflammation, leading to posterior synechiae and/or PAS

Which tumors are notorious for inducing NVI/NVA?
-- Retinoblastoma
-- Medulloepithelioma
-- Choroidal melanoma

Both/Either

Tumor

Inflammation

Flat AC

Epithelial/fibrous ingrowth

NVG

ICE

ERD/choroidal effusion
In a nutshell, what sort of tumor is a medulloepithelioma?

- Medulloepithelioma
- Retinoblastoma
- Choroidal melanoma

How would a tumor pull the angle closed?

- Tumor-induced NVI/NVA leading to NVG
- Tumor necrosis can produce severe inflammation, leading to posterior synechiae and/or PAS

Which tumors are notorious for inducing NVI/NVA?

- Retinoblastoma
- Medulloepithelioma
- Choroidal melanoma
In a nutshell, what sort of tumor is a medulloepithelioma? A locally very aggressive tumor of the non-pigmented epithelium of the ciliary body.

Medulloepithelioma

Both/Either

- Retinoblastoma
- Medulloepithelioma
- Choroidal melanoma

Tumor

Inflammation

Retinal surgery

Drug-induced

Nanophthalmos

PFV

ERD/choroidal effusion

Flat AC

Epithelial/fibrous ingrowth

ICE

NVG

Aqueous misdirection

- Tumor-induced NVI/NVA leading to NVG
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Which tumors are notorious for inducing NVI/NVA?

- Retinoblastoma
- Medulloepithelioma
- Choroidal melanoma

During what life-period does medulloepithelioma present?

Childhood

What clinical findings are commonly present?

- ACG (duh)
- Iris mass
- Hyphema
- Sectoral cataract

How is it managed?

Enucleation is usually required

Rare

non- vs pigmented
Secondary Angle Closure Glaucoma

In a nutshell, what sort of tumor is a medulloepithelioma? A locally very aggressive tumor of the nonpigmented epithelium of the ciliary body

- Retinoblastoma
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- Medulloepithelioma
- Choroidal melanoma

How is it managed?

Enucleation is usually required
In a nutshell, what sort of tumor is a medulloepithelioma?
A locally very aggressive tumor of the nonpigmented epithelium of the ciliary body.

By what other name is medulloepithelioma known?

Retinoblastoma

Medulloepithelioma

Choroidal melanoma

Inflammation

Tumor

-- Retinoblastoma

-- Medulloepithelioma

-- Choroidal melanoma

Both/Either

Inflammation

Tumor
Secondary Angle Closure Glaucoma

In a nutshell, what sort of tumor is a **medulloepithelioma**?
A locally very aggressive tumor of the nonpigmented epithelium of the ciliary body.

**By what other name is medulloepithelioma known?**
Diktyoma

**Which tumors are notorious for inducing NVI/NVA?**
- Retinoblastoma
- Medulloepithelioma
- Choroidal melanoma

Inflammation

How would a tumor pull the angle closed?
Via one of two mechanisms:
- Tumor-induced NVI/NVA leading to NVG
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- Retinoblastoma
- Medulloepithelioma
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In summary, medulloepithelioma is a locally very aggressive tumor of the nonpigmented epithelium of the ciliary body. It is rare and typically presents during childhood. Common clinical findings include ACG, iris mass, hyphema, and sectoral cataract. Management usually requires enucleation.
In a nutshell, what sort of tumor is a medulloepithelioma? A **locally very aggressive** tumor of the nonpigmented epithelium of the ciliary body.

How ‘locally aggressive’ is it?

Retinoblastoma

Medulloepithelioma

Medulloepithelioma

Choroidal melanoma

How would a tumor pull the angle closed?

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- Medulloepithelioma
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In a nutshell, what sort of tumor is a medulloepithelioma? A **locally very aggressive** tumor of the nonpigmented epithelium of the ciliary body.

Is it common, or rare?

Rare

During what life-period does medulloepithelioma present?

Childhood

What clinical findings are commonly present?

- ACG (duh)
- Iris mass
- Hyphema
- Sectoral cataract

How is it managed?

Enucleation is usually required

How ‘locally aggressive’ is it?

Aggressive enough to cause death
Secondary Angle Closure Glaucoma

A locally very aggressive tumor of the nonpigmented epithelium of the ciliary body

How ‘locally aggressive’ is it? Aggressive enough to cause death

In a nutshell, what sort of tumor is a medulloepithelioma?

A locally very aggressive tumor of the nonpigmented epithelium of the ciliary body

How ‘locally aggressive’ is it? Aggressive enough to cause death

- Retinoblastoma
- Medulloepithelioma
- Choroidal melanoma

Both/Either

Inflammation

Tumor

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How ‘locally aggressive’ is it? Aggressive enough to cause death
In a nutshell, what sort of tumor is a medulloepithelioma? A locally very aggressive tumor of the nonpigmented epithelium of the ciliary body.

Is it common, or rare?

--Medulloepithelioma
--Retinoblastoma
--Choroidal melanoma

Which tumors are notorious for inducing NVI/NVA?

--Retinoblastoma
--Medulloepithelioma
--Choroidal melanoma

Inflammation

Both/Either

Tumor

Both/Either

Inflammation

Flat AC

Epithelial/fibrous ingrowth

NVI/NVA

NVG

ICE

Aqueous misdirection

ERD/choroidal effusion

How would a tumor pull the angle closed?

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Childhood

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--Iris mass
--Hyphema
--Sectoral cataract

How is it managed?

Enucleation is usually required.
Secondary Angle Closure Glaucoma

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Is it common, or rare?
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How is it managed?
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In a nutshell, what sort of tumor is a medulloepithelioma? A locally very aggressive tumor of the nonpigmented epithelium of the ciliary body.

Is it common, or rare? Rare

During what life-period does medulloepithelioma present? Childhood

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How is it managed? Enucleation is usually required

Which tumors are notorious for inducing NVI/NVA? -- Retinoblastoma -- Medulloepithelioma -- Choroidal melanoma

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Is it common, or rare?
Rare

During what life-period does medulloepithelioma present?
Childhood

How is it managed?
Enucleation is usually required.
Secondary Angle Closure Glaucoma

Medulloepithelioma/diktyoma
In a nutshell, what sort of tumor is a medulloepithelioma? A locally very aggressive tumor of the nonpigmented epithelium of the ciliary body.

Is it common, or rare?
Rare.

During what life-period does medulloepithelioma present?
Childhood.

What clinical findings are commonly present?
-- ACG (duh)
-- Sectoral cataract
**Secondary Angle Closure Glaucoma**

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A locally very aggressive tumor of the nonpigmented epithelium of the ciliary body

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Childhood

**What clinical findings are commonly present?**
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-- Sectoral cataract

**How is it managed?**
Enucleation is usually required

**During what life-period does medulloepithelioma present?**
Childhood

**What clinical findings are commonly present?**
-- ACG (duh)
-- Iris mass
-- Hyphema
-- Sectoral cataract

**How is it managed?**
Enucleation is usually required
(a) 2-year-old girl who presented with translucent white mass (arrow) and NVI. (b) Beige-white medulloepithelioma of the ciliary body.

Medulloepithelioma/diktyoma
Medulloepithelioma/diktyoma: Note the cataract
**Secondary Angle Closure Glaucoma**

**In a nutshell, what sort of tumor is a medulloepithelioma?**
A locally very aggressive tumor of the nonpigmented epithelium of the ciliary body

**Is it common, or rare?**
Rare

**During what life-period does medulloepithelioma present?**
Childhood

**What clinical findings are commonly present?**
--ACG (duh)
--Iris mass
--Hyphema
--Sectoral cataract

**How is it managed?**
--Retinoblastoma
---Medulloepithelioma
--Choroidal melanoma

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---Both/Either
---Inflammation
---NVI/NVA
---NVG
---ICE
---Flat AC
---Epithelial/fibrous ingrowth

**Which tumors are notorious for inducing NVI/NVA?**
--Retinoblastoma
--Medulloepithelioma
--Choroidal melanoma

**How would a tumor pull the angle closed?**
Via one of two mechanisms:
--Tumor-induced NVI/NVA leading to NVG
--Tumor necrosis can produce severe inflammation, leading to posterior synechiae and/or PAS

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Tumor

Glucoma

Ileo Pupillary Block

Push’’

‘‘Pull’’

NVG
ICE
Flat AC
Epithelial/fibrous ingrowth

Retinal surgery
Drug-induced
Both/Either

Nanophthalmos
PFV
ERD/choroidal effusion

How is it managed?
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