--Intraocular inflammation



-- Corneal endothelial abnormality

--Increased episcleral venous pressure

We are going to look at unilateral elevated IOP in each of these clinical scenarios

--Intraocular inflammation

Generally speaking, do you expect an inflamed eye to be hypertensive, or hypotensive?

--Corr



--Intraocular inflammation

```
Generally speaking, do you expect an inflamed eye to be hypertensive, or hypotensive?
Hypotensive

--Corr
```

--Intraocular inflammation

Generally speaking, do you expect an inflamed eye to be hypertensive, or hypotensive?
Hypotensive

--Corr Why hypotensive?



--Intraocular inflammation

Generally speaking, do you expect an inflamed eye to be hypertensive, or hypotensive?

Hypotensive

--Corr Why hypotensive?

Ciliary-body inflammation leads to decreased aqueous production, with subsequent low IOP.

--Incre



--Intraocular inflammation

Generally speaking, do you expect an inflamed eye to be hypertensive, or hypotensive?

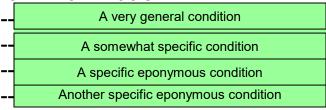
Hypotensive

--Corr Why hypotensive?

Ciliary-body inflammation leads to decreased aqueous production, with subsequent low IOP. That said, certain uveitic entities are notorious for *elevated* IOP, thus rendering it an important clue re the etiology of the inflammation.



--Intraocular inflammation



--Corneal endothelial abnormality

--Increased episcleral venous pressure



--Intraocular inflammation

- --Anterior uveitis
- --Trabeculitis
- --Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- --Corneal endothelial abnormality

--Increased episcleral venous pressure



- -- Anterior uveitis
- -- Trabeculitis
- --Fuchs heterochrom
- --Posner-Schlossmar
- -- Corneal endothelial abnormal

Both of these present with unilateral AC cell and ↑ IOP. How does one differentiate between them?



--Increased episcleral venous pressure

- --Intraocular inflammation
 - -- Anterior uveitis
 - -- Trabeculitis
- -- Corneal endothelial abnormal

Both of these present with unilateral AC cell and ↑ IOP. How does one differentiate between them? The severity of the AC reaction is an important clue.

In order for a 'simple' anterior uveitis to cause ↑ IOP, the inflammatory reaction must be quite severe.

--Increased episcleral venous pressure



- --Intraocular inflammation
 - -- Anterior uveitis
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Both of these present with unilateral AC cell and ↑ IOP. How does one differentiate between them?

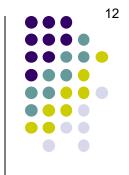
The severity of the AC reaction is an important clue. In order for a 'simple' anterior uveitis to cause ↑ IOP, the inflammatory reaction must be quite severe. In contrast, the cell associated with trabeculitis can be quite mild.



--Increased episcleral venous pressure

--Intraocular inflammation

- --Anterior uveitis
- -- Trabeculitis
- What etiology should come to mind with this?
- --Fuchs hetero
- --Posner-Schlossman
- --Corneal endothelial abnormality
- --Increased episcleral venous pressure



--Intraocular inflammation

- Anterior uveitis
- -- Trabeculitis
- What etiology should come to mind with this? Herpesvirus infection, especially HSV and VZV
- Danie in Calabarra
- -Posner-Schlossman
- --Corneal endothelial abnormality

--Increased episcleral venous pressure



Unilateral † IOP associated with: What four anterior uveitis etiologies are notorious for elevating IOP?

(hints soon forthcoming...)

(Etiologies other than these two, that is)

2)

--Intraocular inflammation

-- Anterior uveitis

3)

--Corneal endothelial abnormality

--Increased episcleral venous pressure

3)

4)



What four anterior uveitis etiologies are notorious for elevating IOP?

- 1) this one is a family of infectious agents2)
- --Intraocular inflammation
 - -- Anterior uveitis
 - -- Trabeculitis
 - -- Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
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3)



What four anterior uveitis etiologies are notorious for elevating IOP?

- 1) Herpesvirus (all forms)2)
- --Intraocular inflammation
 - -- Anterior uveitis
 - -- Trabeculitis
 - -- Fuchs heterochromic iridocyclitis
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- --Corneal endothelial abnormality
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What four anterior uveitis etiologies are notorious for elevating IOP?

- 1) Herpesvirus (all forms)
- --Intraocular inflammation 2) this one is a specific bug 3)

4)

- -- Anterior uveitis
- --Fuchs heterochromic iridocyclitis
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What four anterior uveitis etiologies are notorious for elevating IOP?

- 1) Herpesvirus (all forms)2) Toxoplasmosis
- --Intraocular inflammation
 - -- Anterior uveitis
 - Traheculitie
- 4)

3)

- --Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- --Corneal endothelial abnormality
- --Increased episcleral venous pressure



What four anterior uveitis etiologies are notorious for elevating IOP?

- 1) Herpesvirus (all forms)
- 2) Toxoplasmosis
 - 3) this one is a common noninfectious entity
- Trabeculitis

-- Anterior uveitis

- -- Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- --Corneal endothelial abnormality

--Intraocular inflammation

--Increased episcleral venous pressure



What four anterior uveitis etiologies are notorious for elevating IOP?

- 1) Herpesvirus (all forms)
- --Intraocular inflammation 2) Toxoplasmosis -- Anterior uveitis
 - 3) Sarcoidosis

 - --Fuchs heterochromic iridocyclitis
- --Corneal endothelial abnormality
- --Increased episcleral venous pressure



What four anterior uveitis etiologies are notorious for elevating IOP?

- 1) Herpesvirus (all forms)
- 2) Toxoplasmosis
- 3) Sarcoidosis
- 4) this one is another specific bug
- -- Fuchs heterochromic iridocyclitis
- --Posner-Schlossman

-- Anterior uveitis

--Corneal endothelial abnormality

--Intraocular inflammation

--Increased episcleral venous pressure



What four anterior uveitis etiologies are notorious for elevating IOP?

- 1) Herpesvirus (all forms)
- 2) Toxoplasmosis
 - 3) Sarcoidosis
- -- Trabeculitis

-- Anterior uveitis

--Intraocular inflammation

- 4) Syphilis
- -- Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
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What four anterior uveitis etiologies are notorious for elevating IOP?

- 1) Herpesvirus (all forms)
- 2) Toxoplasmosis
- 3) Sarcoidosis
- 4) Syphilis

Which of these can present with granulomatous-appearing KP?

- -- Anterior uveitis
 - --Trabeculitis

--Intraocular inflammation

- --Fuchs heterochromic iridocyclitis
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--Increased episcleral venous pressure



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- 3) Sarcoidosis
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Which of these can present with granulomatous-appearing KP?

All of them

--Trabeculitis

-- Anterior uveitis

--Intraocular inflammation

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1) Herpesvirus (all forms)

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g IOP?

--Intraocular inflammation

- -- Anterior uveitis
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- --Corneal endothelial abnormality
- --Increased episcleral venous pressure

--Secondary glaucoma

Which can present with stellate KP?

--Intraocular inflammation

- -- Anterior uveitis
- -- Trabeculitis
- 2) Toxoplasmosis 3) Syphilis
- 4) Sarcoidosis

Which can present with stellate KP?

All but sarcoid (and don't forget about FHI)

--Fuchs heterochromic iridocyclitis FHI causes stellate KP too

1) Herpesvirus (all forms)

- --Corneal endothelial abnormality

--Increased episcleral venous pressure

accompanied by increased IOP?



--Intraocular inflammation

- --Anterior uveitis
- -- Trabeculitis
- --Fuchs heterochromic_iridocyclitis
- --Corneal endothelial abnormality
- --Increased episcleral venous pressure



--Intraocular inflammation

- --Anterior uveitis
- -- Trabeculitis

- What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?
- --Topical steroids
- --Cycloplegia
- --Fuchs heterochrom --Anti-infectives if bug is known
- --Posner-Schlossmar -- +/- ocular hypotensives

Corneal endothelial abnormality

--Increased episcleral venous pressure



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What two classes of topical hypotensives should probably be avoided?

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Corneal endothelial abnormali

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- --Miotics
- -- Prostaglandin analogues

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Why should these be avoided? Both are potentially inflammogenic

33

--Intraocular inflammation

- --Anterior uveitis
- -- Trabeculitis
- -- Fuchs heterochrom -- Anti-
- --Posner-Schlossmar -- +/-
- Corneal endothelial abnormality
- --Increased episcleral venous pressur

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In what way might steroid therapy complicate the IOP issue?



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Corneal endothelial abnormality

--Increased episcleral venous pressur

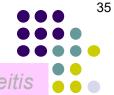
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In what way might steroid therapy complicate the IOP issue? Recall that some pts are **steroid responders**, meaning their IOP increases secondary to steroid therapy. Thus, when a uveitis pt (under treatment) develops increased IOP, it could mean either:

a)

b)



--Intraocular inflammation

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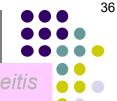
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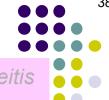
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- **b)** they are a steroid responder, and their steroid should be **decreased** (or even stopped)

So how do you know whether steroids are the solution to, or the cause of, ocular hypertension in uveitis?

--Seco



--Intraocular inflammation

- -- Anterior uveitis
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As a rule, it takes at least 2 weeks before steroid-induced ocular hypertension occurs. Thus, elevated IOP within this time frame indicates inadequate uveitis control, and the steroid dose should be increased.

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Corneal endothelial abnormality

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On the other hand, if IOP spikes after the uveitis is controlled, consideration should be given to tapering the steroid and/or adding an aqueous suppressant to the treatment regimen.

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
 - -- Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- Corneal endothelial abnormality

What exam findings comprise the 'classic triad' of FHI? (Hint: Elevated IOP is not one of them.)



--Intraocular inflammation

- -- Anterior uveitis
- --Trabeculitis
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What exam findings comprise the 'classic triad' of FHI? (Hint: Elevated IOP is not one of them.) Heterochromia iridis, cataract, and stellate KP







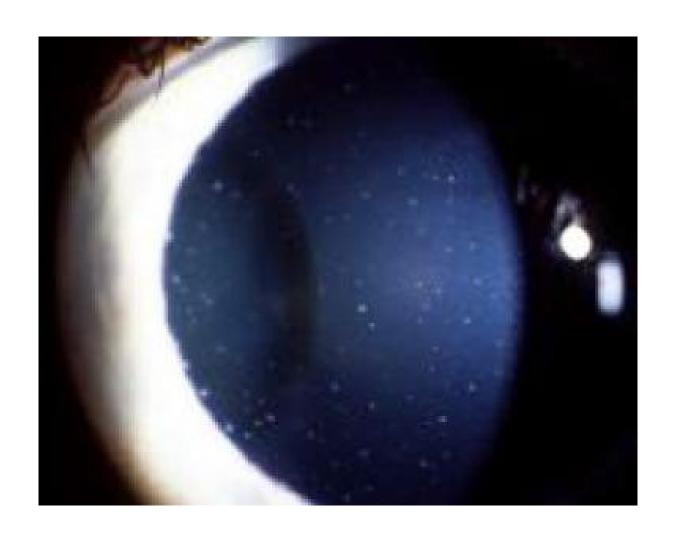
FHI: Heterochromia





FHI: Note the cataract

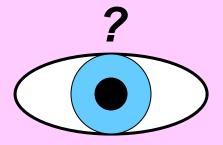


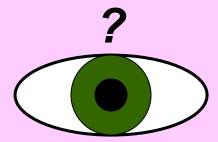


FHI: Stellate KP

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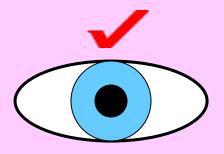
Is the affected eye the **darker** eye or the **lighter** eye?

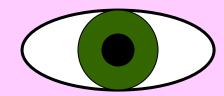


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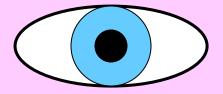
Is the affected eye the **darker** eye or the **lighter** eye? The **lighter** (with one exception)

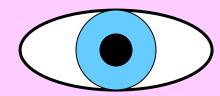


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Is the affected eye the darker eye or the lighter eye?
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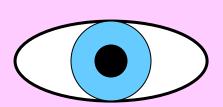
What is the exception; ie, under what circumstances is the darker eye the one with FHI? In individuals with light-blue eyes...

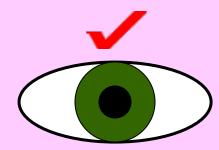


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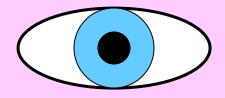
What is the exception; ie, under what circumstances is the darker eye the one with FHI? In individuals with light-blue eyes...the iris atrophy stemming from the FHI process will make visible the darkly-pigmented epithelium of the posterior iris, thus making the eye appear darker

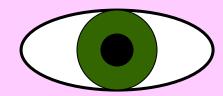


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Heterochromia iridis, cataract, and stellate KP





ls : Th In addition to heterochromia, the atrophic changes give the iris an appearance that has been likened to damage caused by an insect. What is the two-word description for this appearance?

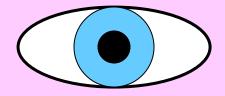
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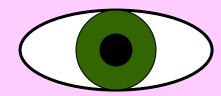


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'Moth eaten'

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FHI: 'Moth eaten' iris. Note the smooth stromal architecture and loss of iris crypts

--Intraocular inflammation

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The KP in FHI have a couple of other notable characteristics—what are they?

__



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The KP in FHI have a couple of other notable characteristics—what are they?

- --They may be interconnected by lines described as 'lacy tendrils'
- --They are diffusely scattered (as opposed to being concentrated in anterior uveitides)

two words

, as is the case in most



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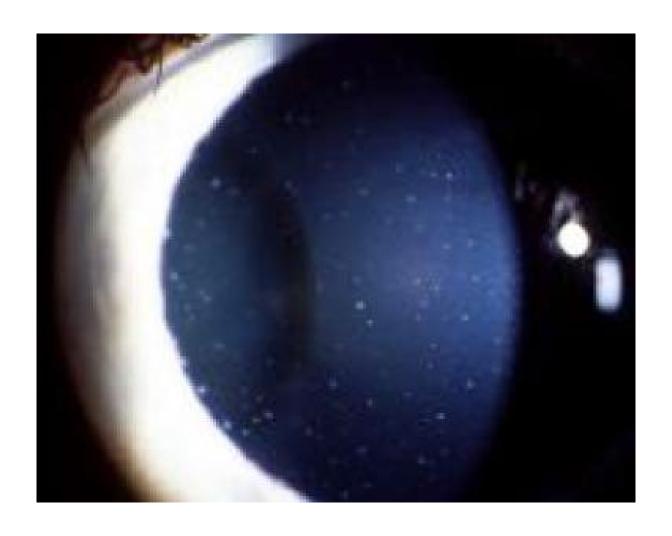
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FHI: Stellate KP. Note the diffuse distribution

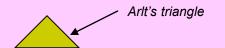
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Where/what is Arlt's triangle?





- --Intraocular inflammation
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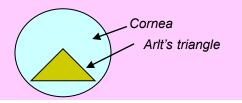
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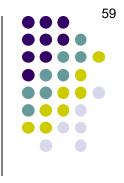
The KP in FHI have a couple of other notable characteristics—what are they?

- --They may be interconnected by lines described as 'lacy tendrils'
- --They are diffusely scattered (as opposed to being concentrated in anterior uveitides)

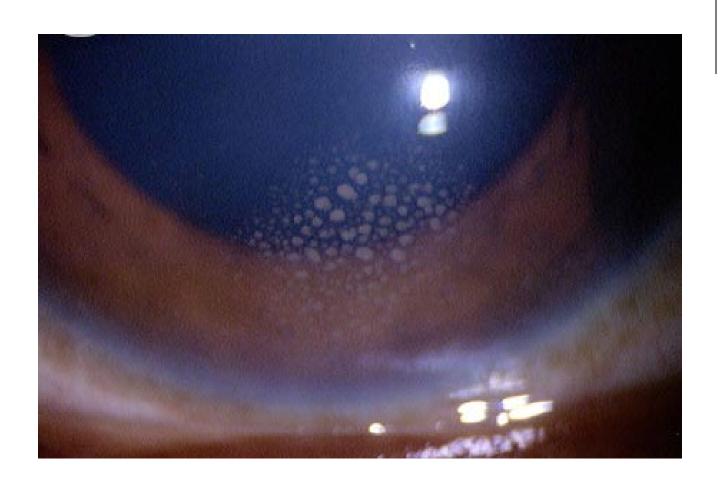
Arlt's triangle as is the case in most

Where/what is Arlt's triangle?
It's an equilateral triangle with its apex at the corneal center and base near the inferior border of the cornea









KP (in sarcoidosis) concentrated in Arlt's triangle

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How common is glaucoma in FHI?



--Intraocular inflammation

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How common is glaucoma in FHI? It develops in about 25-50% of cases



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65

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--Despite the chronic nature of the iridocyclitis in FHI, three words (and an abb.)

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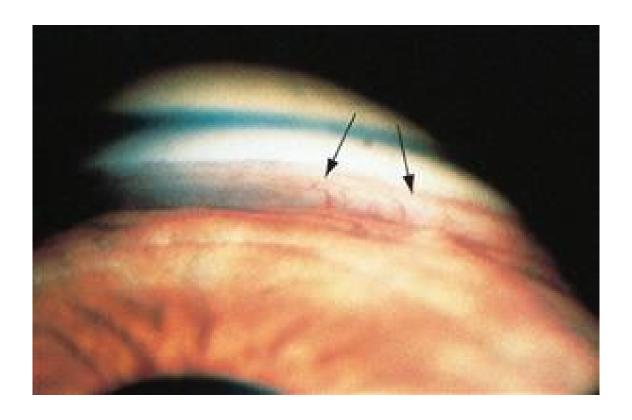


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Fuchs heterochromic iridocyclitis. Fine vessels (arrows) are seen crossing the TM. This NVA is **not** accompanied by a fibrovascular membrane and does not result in PAS formation and subsequent secondary angle closure.

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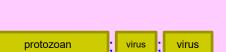
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Is there a gender predilection?

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Is cataract surgery in FHI associated with an increased risk of intraoperative complications?



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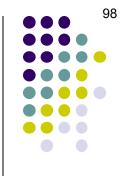


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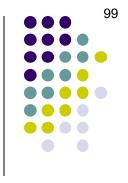
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 An adult age 20-50



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Does the inflammatory component tend to be mild, or severe?

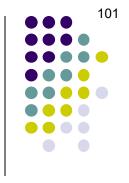


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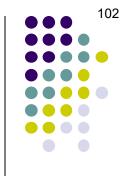
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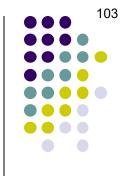
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How severe?



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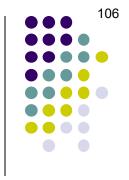
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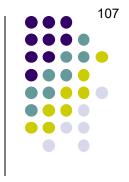
IOP in the 40-60 range is typical



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How long do the crises last?



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How long do the crises last?
Hours to weeks



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How long do the crises last? Hours to weeks

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What is/are the presenting complaint(s)?
Unilateral pain, blurred vision, haloes around lights

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Unilateral pain, blurred vision, haloes around lights

What is the cause of the blurred vision/haloes?



114

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--Secondary

Is the angle open, or closed?
Open

How long do the crises last?
Hours to weeks

Do they recur? Yes What is/are the presenting complaint(s)?

Corneal edema secondary to the high IOP

Unilateral pain, blurred vision, haloes around lights

What is the cause of the blurred vision/haloes?

115

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CMV, a member of the family

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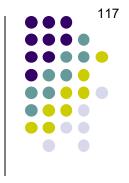
--Secondary Is the angle open, or closed? Open

How long do the crises last?

Hours to weeks

Do they recur?

Yes





What four anterior uveitis etiologies are notorious for elevating IOP?

- 1) Herpesvirus (all forms)
- 2) Toxoplasmosis
- 3) Sarcoidosis
- 4) Syphilis
- --Fuchs heterochromic iridocyclitis
- -- Posner-Schlossman
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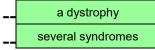
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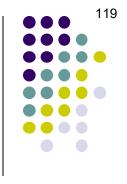
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Increased episcleral venous pressure

--Secondary glaucoma



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 - --PPMD (posterior polymorphous dystrophy)
 - -- ICE (iridocorneal endothelial syndrome)
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In a nutshell, what is abnormal about the endothelial cells in PPMD?

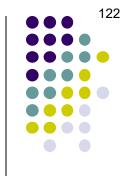
--Seco



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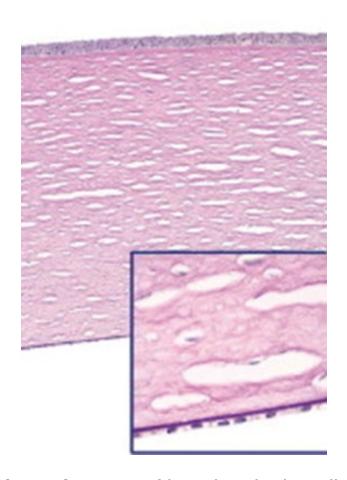
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In a nutshell, what is abnormal about the endothelial cells in PPMD? They 'behave' like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells **proliferate**, form **multiple layers**, and **migrate**—none of which normal endothelial cells do.

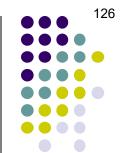


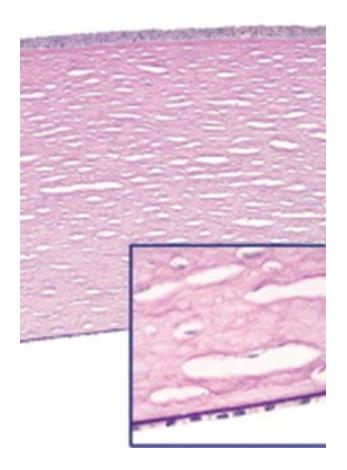




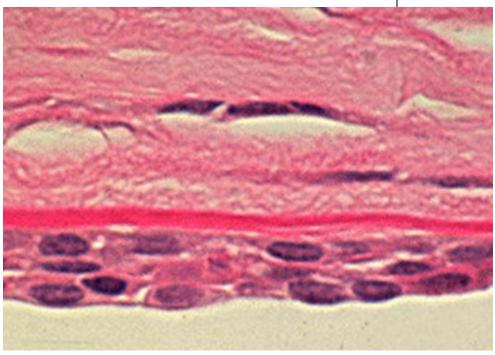


Normal cornea. Note the single-cell-thick nature of the endothelial cells.





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Posterior polymorphous corneal dystrophy. Instead of being lined by cells with the attributes of corneal endothelium, the posterior cornea is covered by cells with **epithelial/fibroblast-like** features.

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PPMD presents with three posterior-K slit-lamp findings. What are they?

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127

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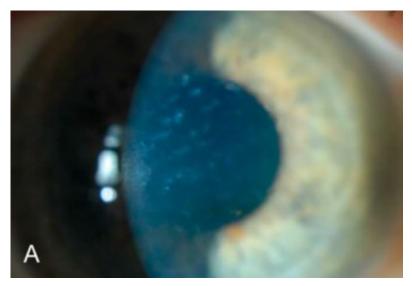
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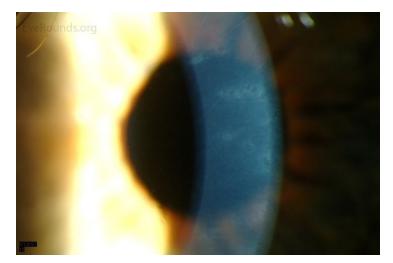
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- --Linear band-shaped opacities
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- --Diffuse opacities

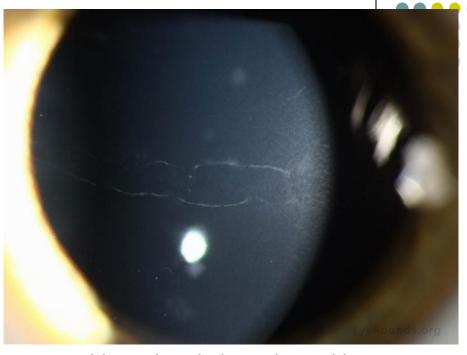




Diffuse opacities



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PPMD

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Because it can be highly asymmetric

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Briefly, what is ICE?



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Briefly, what is ICE?

A progressive, nonfamilial, unilateral disorder characterized by abnormalities of the corneal endothelium and iris, as well as PAS formation



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140

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- --The pt will c/o changes in the eye's appearance, and/or pain, and/or decreased VA
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- --The cornea of the affected eye will have abnormal endothelium, and may be edematous
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- -- Iris nevus syndrome
- --Chandler syndrome
- -- Essential iris atrophy

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The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they?

-- Iris nevus syndrome: ?

--Chandler syndrome: ?

--Essential iris atrophy: ?

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How will a pt with ICE present?

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- --The affected eye will have elevated IOP +/- glaucomatous ONH damage
- --The cornea of the affected eye will have abnormal endothelium, and may be edematous
- --The fellow eye will be essentially normal



al

- --Intraocular inflammation
 - -- Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
 - --ICE

The BCSC books recognize three ICE variants—what are they? All have three findings

in common—what are they?

--Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP

--Chandler syndrome: Abnormal endothelium; PAS; elevated IOP

-- Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

A young-to-middle-aged addit lemale

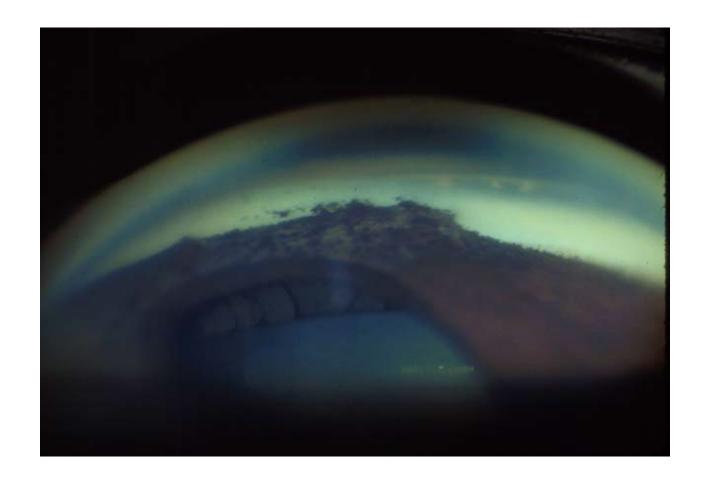
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ICE: PAS

- --Intraocular inflammation
 - -- Anterior uveitis
 - -- Trabeculitis
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- -- Corneal endothelial abnormality

 - --ICF

in common—what are they

-- Iris nevus syndrom : Abnormal endothelium; PAS; elevated IOP

--Chandler syndron(e: Abnormal endothelium; PA); elevated IOP

-- Essential iris atropic: Abnormal endothelium: AS; elevated IOP

A young-to-middle-aged addit lemale

What is the classic metal-related description of the endothelium?

- --The permitty of an angular mental of a appearance, and or decreased VA
- --The affected eye will have elevated IOP +/- glaucomatous ONH damage
- --The cornea of the affected eye will have abnormal endothelium, and may be edematous
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in common—what are they

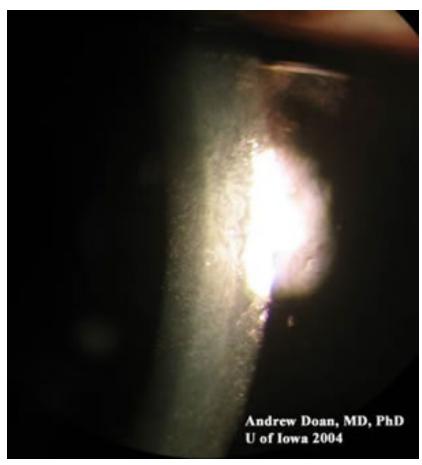
- -- Iris nevus syndrom : Abnormal endothelium; P\S; elevated IOP
- --Chandler syndron(e: Abnormal endothelium; PA\$)
- -- Essential iris atropi .: Abnormal endothelium; AS; elevated IOP

A young-to-middle-aged soull remaie

What is the classic metal-related description of the endothelium? 'Hammered silver'

- Unangeom are eye e appearance, analer pain, and/or decreased VA
- --The affected eye will have elevated IOP +/- glaucomatous ONH damage
- --The cornea of the affected eye will have abnormal endothelium, and may be edematous
- --The fellow eye will be essentially normal





Low mag



150

High mag

ICE: 'Hammered silver' corneal endothelium

- --Intraocular inflammation
 - -- Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICE

Ingresed anicaloral vancus pressure

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they?

ea

- --Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP (not so much)?
- --Chandler syndrome: Abnormal endothelium; PAS; elevated IOP (not so much)?
- -- Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP (not so much)?

A young-to-middle-aged addit lemale

One variant is less likely than the others to manifest elevated IOP—which one?

How will a pt with ICE

- --The pt will c/o changes in the eye's appearance, and/or pain, and/or decreased VA
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- -- Corneal endothelial abnormality
 - --PPMD
 - --ICE

Increased anicalaral vanaua procesure

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they?

ea

- --Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOF
- --Chandler syndrome: Abnormal endothelium; PAS; elevated IOP (not so much)
- -- Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

A young-to-middle-aged addit lemale

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How will a pt with ICE Chandler syndrome

- --The pt will c/o changes in the eye's appearance, and/or pain, and/or decreased VA
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- --Corneal endothelial abnormality
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The BCSC books recognize three ICE variants—what are they? All have three findings

in common—what are they? Each has a predominant finding—what is it?

- --Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; ?
- -- Chandler syndrome: Abnormal endothelium; PAS; elevated IOF
- -- Essential iris atrophy: Abnormal endothelium; PAS; elevated IOF

A young-to-middle-aged addit lemale

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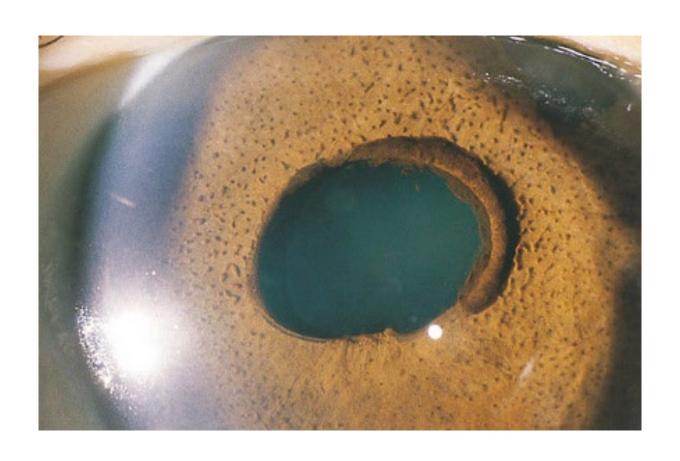
- --Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; iris nevi/nodules

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Iris nevus syndrome

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 - --Posner-Schlossman
- -- Corneal endothelial abnormality
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 - --ICE

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 - A young-to-middle-aged addit remale

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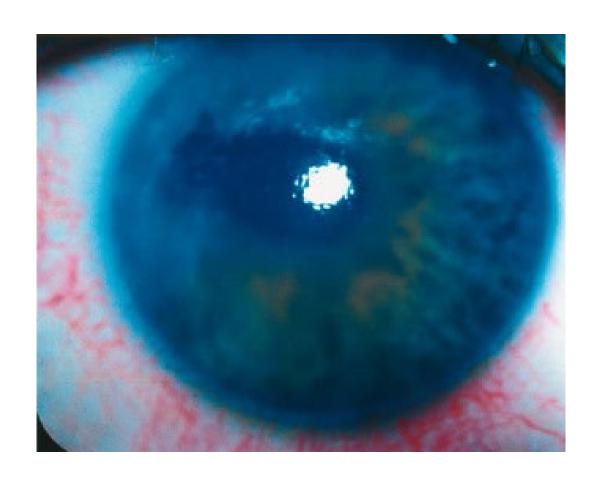
157

- --Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; iris nevi/nodules
- --Chandler syndrome: Abnormal endothelium; PAS; elevated IOP; corneal edema
- --Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP
- A young-to-middle-aged addit lemale

How will a pt with ICE present?

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Chandler syndrome

- --Intraocular inflammation
 - -- Anterior uveitis
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 - --Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
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A young-to-middle-aged addit lemale

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Iria povuja avadroma: Abnormal andathalium: DAS: alayated IOD; iria pavi/a

--Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; iris nevi/nodules

--Chandler syndrome: Abnormal endothelium; PAS; elevated IOP; corneal edema

--Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP; iris atrophy/holes

A young-to-middle-aged addit remaie

How will a pt with ICE present?

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al





Essential iris atrophy

162

```
--Intraocular inflammation
```

- -- Anterior uveitis
- --Trabeculitis
- --Fuchs heterochromic irido
- --Posner-Schlossman

--Corneal endothelial abnormality

- --PPMD
- --ICE
- --Increased episcleral venous pressure

How common is glaucoma in each?

- --ICE:
- --PPMD:

163

- --Intraocular inflammation
 - -- Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic irido
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressure

How common is glaucoma in each?
--ICE: Very common—80 to 100% of cases

--PPMD: Not that common—25% of cases

164

```
--Intraocular inflammation
```

- -- Anterior uveitis
- --Trabeculitis
- --Fuchs heterochromic irido
- --Posner-Schlossman

--Corneal endothelial abnormality

- --PPMD
- --ICE
- --Increased episcleral venous pressure

How common is glaucoma in each?

--ICE: Very common—80 to 100% of cases

--PPMD: Not that common—25% of cases

What is the mechanism for increased IOP?

- --ICE:
- --PPMD:

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic irido
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressure

How common is glaucoma in each?

--ICE: Very common—80 to 100% of cases

--PPMD: Not that common—25% of cases

What is the mechanism for increased IOP?

- --ICE: Angle closure via PAS and/or a membrane
- --PPMD: Unclear; angle can be closed a la ICE,

but some have an open angle



166

```
--Intraocular inflammation
```

- -- Anterior uveitis
- --Trabeculitis
- --Fuchs heterochromic irido
- --Posner-Schlossman

--Corneal endothelial abnormality

- --PPMD
- --ICE
- --Increased episcleral venous pressure

How common is glaucoma in each?

What is the take-home message regarding PPMD/ICEand unilateral elevated IOP?

--PPMD: Unclear; angle can be closed a la ICE, but some have an open angle

- --Intraocular inflammation
 - -- Anterior uveitis
 - -- Trabeculitis
 - --Fuchs heterochromic irido
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressure

How common is glaucoma in each?

- What is the take-home message regarding PPMD/ICEand unilateral elevated IOP?
- It is incumbent upon you to do a thorough slit-lamp examination of the posterior corneal surface in all pts with unilateral elevated IOP!

--PPMD: Unclear; angle can be closed a la ICE but some have an open angle



168

```
--Intraocular inflammation
```

- --Anterior uveitis
- --Trabeculitis
- --Fuchs heterochromic irido
- --Posner-Schlossman
- -- Corneal endothelial abnormality

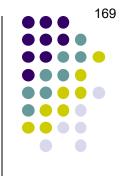
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- What is the take-home message regarding PPMD/ICE
- and unilateral elevated IOP?

For more on PPMD, see slide-set K45 For more ICE, see slide-set K26

but some have an open angle

- --Intraocular inflammation
 - -Anterior uveitis
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- --Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressure



170

- --Intraocular inflammation
 - -- Anterior uveitis
 - --Trabeculitis
 - Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICF
- --Increased episcleral venous pressure

Recall that the Goldmann equation for IOP contains two terms: --The first term...

171

```
--Intraocular inflammation
```

- -- Anterior uveitis
- --Trabeculitis
- --Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
 - --ICF
- --Increased episcleral venous pressure

Recall that the Goldmann equation for IOP contains two terms:

--The first term...quantifies the balance between aqueous formation and aqueous egress

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

172

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressure

Recall that the Goldmann equation for IOP contains two terms:

- --The first term...quantifies the balance between aqueous *formation* and aqueous *egress*
- --The second term in the equation is...

--Secondary glaucoma

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

Second term

173

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICF
- --Increased episcleral venous pressure

Recall that the Goldmann equation for IOP contains two terms:

- --The first term...quantifies the balance between aqueous formation and aqueous egress
- --The second term in the equation is... EVP.

--Secondary glaucoma

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

174

- --Intraocular inflammation
 - -- Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
 - --ICF
- --Increased episcleral venous pressure

Recall that the Goldmann equation for IOP contains two terms:

- --The first term...quantifies the balance between aqueous *formation* and aqueous *egress* --The second term in the equation is...*EVP*.
- Thus, any ocular and/or systemic conditions that lead to a persistent increase in EVP can produce a persistent elevation in IOP and thus glaucoma. And if this condition affects only one eye, it can produce unilateral elevated IOP.
- --Secondary glaucoma



175

```
--Intraocular inflammation
```

- -Anterior uveitis
- --Trabeculitis
- Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
 - --ICF

--Increased episcleral venous pressure

a specific intracranial problem
a general intrathoracic problem
a phakomatosis
a very general ophthalmic problem

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

- --Intraocular inflammation
 - -Anterior uveitis
 - --Trabeculitis
 - -- Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICF
- --Increased episcleral venous pressure
 - --CCF (carotid-cavernous sinus fistula)
 - --SVC syndrome (SVC = superior vena cava)
 - --Sturge-Weber
 - -- Orbital inflammation

--Secondary glaucoma

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





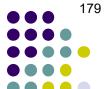
```
--Intraocular infla What is the fundamental anatomical abnormality in CCF?
--Corneal endoth
--Increased episc
          --CCF
--Secondary glauc
```

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



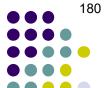
```
--Intraocular infla What is the fundamental anatomical abnormality in CCF?
                   A channel forms that allows direct communication between the carotid artery (or
                   one of its dural branches) and the cavernous sinus (CS)
--Corneal endoth
--Increased episc
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IOP = \frac{\text{Aqueous Formation Rate (}_{\mu\text{L/min}}\text{)} + \frac{\text{Episcleral Venous}}{\text{Pressure (mmHg)}}
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```
--Intraocular infla What is the fundamental anatomical abnormality in CCF?
                   A channel forms that allows direct communication between the carotid artery (or
                   one of its dural branches) and the cavernous sinus (CS)
                   What important effect does this have on the CS?
-- Corneal endoth
--Increased episc
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IOP = \frac{\text{Aqueous Formation Rate (}_{\mu\text{L/min}}\text{)} + \frac{\text{Episcleral Venous}}{\text{Pressure (mmHg)}}
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```
--Intraocular infla What is the fundamental anatomical abnormality in CCF?
                   A channel forms that allows direct communication between the carotid artery (or
                   one of its dural branches) and the cavernous sinus (CS)
                   What important effect does this have on the CS?
                   The normal pressure within the CS is about the same as central venous
-- Corneal endothe
                   pressure (CVP; #6# mmHg)
--Increased episc
          --CCF
--Secondary glauc
```

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





--Intraocular infla What is the fundamental anatomical abnormality in CCF?

A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS)

What important effect does this have on the CS?

-- Corneal endoth

The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg)

--Increased episc

--CCF

--Secondary glauc



--Intraocular infla What is the fundamental anatomical abnormality in CCF?

A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS)

What important effect does this have on the CS?

-- Corneal endothe

The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP; usually at least # to # mmHg).

--Increased episc

--CCF

--Secondary glauc



-- Corneal endothe

--Increased episc

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A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS)

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The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP; usually at least 70-100 mmHg).

--Secondary glauc





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How does a CCF lead to unilateral increased IOP?

--Secondary glauc





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--Increased episc

--CCF

How does a CCF lead to unilateral increased IOP?

When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded.

--Secondary glauc

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





-- Corneal endothe

--Increased episc

--CCF

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When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

--Secondary glauc

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--Increased episc

How does a CCF lead to unilateral increased IOP?

--CCF

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What two classes of mechanism account for the formation of CCF?

--Secondary glauc



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--Increased episc

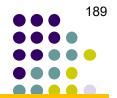
How does a CCF lead to unilateral increased IOP?

--CCF

When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF? --Secondary glauc They can be traumatic or spontaneous

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



--Intraocular infla What is the fundamental anatomical abnormality in CCF?

A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS)

-- Corneal endothe

--Increased episc

--CCF

What important effect does this have on the CS?

The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP; usually at least 70-100 mmHg).

How does a CCF lead to unilateral increased IOP?

When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is ultimately results in

Demographically speaking, who gets:

--Traumatic CCF?

--Spontaneous CCF?

What two classes of mechanism account for the formation of CCE?

--Secondary glauc They can be traumatic or spontaneous



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--Increased episc

--CCF

How does a CCF lead to unilateral increased IOP?

When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is ultimately results in

Demographically speaking, who gets:

-- Traumatic CCF? Young persons

--Spontaneous CCF? Middle-aged to elderly females

What two classes of mechanism account for the formation of CCE?

--Secondary glauc They can be traumatic or spontaneous

191

Unilateral ↑ IOP associated with:



--Intraocular infla What is the fundamental anatomical abnormality in CCF?

A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS)

What important effect does this have on the CS?

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--CCF

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When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCE?

--Secondary glauc They can be traumatic or spontaneous

192

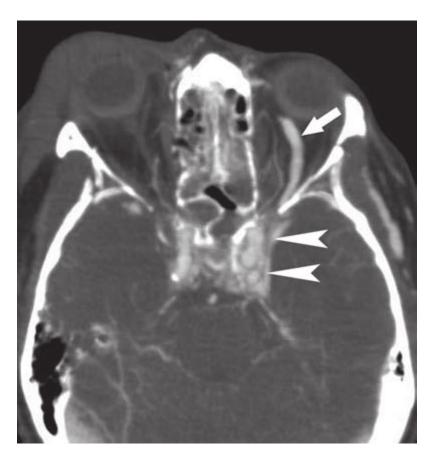
Unilateral ↑ IOP associated with:



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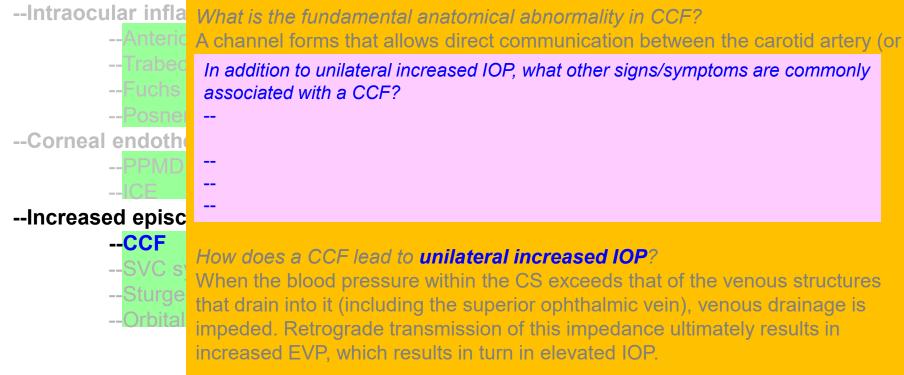




Carotid cavernous fistula in a 20-year-old man s/p motor vehicle collision. CT angiography shows asymmetric enlargement of the left cavernous sinus (arrowheads) and left superior ophthalmic vein (arrow).

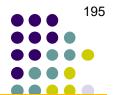
--Secondary glauc They can be traumatic or spontaneous





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

What two classes of mechanism account for the formation of CCE?



-- Corneal endothe

--Increased episc

--CCF

--Intraocular infla What is the fundamental anatomical abnormality in CCF?

A channel forms that allows direct communication between the carotid artery (or

In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?

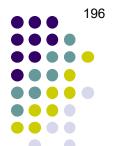
- --A chronically **red eye** (beware of the little old lady with 'chronic conjunctivitis' that never seems to get any better!)
- --Chemosis
- --Proptosis (in high-flow CCF)
- --Tinnitus

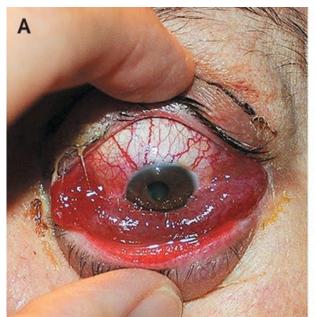
How does a CCF lead to unilateral increased IOP?

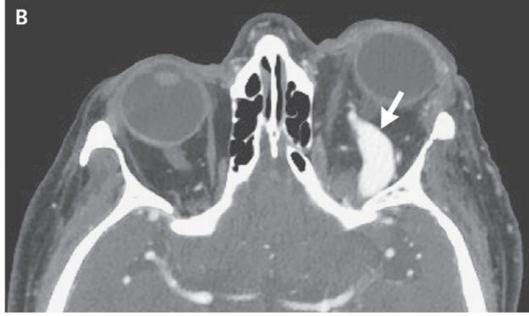
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What two classes of mechanism account for the formation of CCE?

--Secondary glauc They can be traumatic or spontaneous







A 55 year old woman with a history of HTN presented with a 1-day history of periorbital discomfort, inferior **chemosis**, and **conjunctival injection** of the left eye (Panel A). IOP OS was 48. Exam OD was unremarkable. She reported a 2-year history of episodic headache and pulsatile **tinnitus** in the left ear. Contrast-enhanced computed tomography of the orbit showed **proptosis** and a dilated left superior ophthalmic vein (Panel B, arrow), suggesting the presence of a carotid–cavernous sinus fistula.



--Intraocular infla What is the fundamental anatomical abnormality in CCF?

A channel forms that allows direct communication between the carotid artery (or In addition to unilateral increased IOP, what other signs/symptoms are commonly

associated with a CCF?

--A chronically red eye (beware of the little old lady with 'chronic conjunctivitis' that never seems to get any better!)

--Corneal endoth

What slit-lamp finding concerning the appearance of the conj vessels is a tipoff that you're dealing with a CCF?

--Incre

impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCE?

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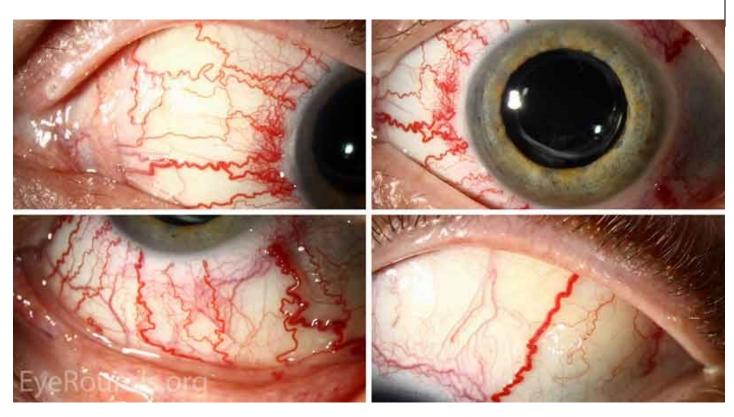
The vessels have a kinked or 'corkscrew' appearance

impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

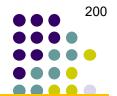
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Corkscrewing of conj vessels 2ndry to CCF



--Intraocular infla What is the fundamental anatomical abnormality in CCF?

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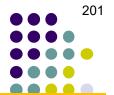
The vessels have a kinked or 'corkscrew' appearance

What causes this corkscrewing?

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What slit-lamp finding concerning the appearance of the conj vessels is a tipoff that you're dealing with a CCF?

The vessels have a kinked or 'corkscrew' appearance

What causes this corkscrewing?

Arterialization of blood flow in the vessels (think about how a garden hose will twist and kink when the water is turned on but is held in by a closed nozzle on the end of the hose)

impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCE?

--Secondary glauc They can be traumatic or spontaneous

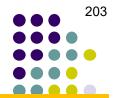


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Aqueous Formation Rate (μL/min)
Outflow Facility (μL/min/mmHg) **Episcleral Venous** Pressure (mmHg)

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-- Corneal endothe

--Chemosis

What sign may be present in a pt who reports the symptom of tinnitus, and should be checked for?

(-Tinnitus)

A bruit

--Increased episc

--CCF

How does a When the blo

How should one check for a bruit in cases of suspected CCF?

that drain into required and superior option of the venil, venious draining impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

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-- Corneal endoth

--Chemosis (-Tinnitus)

What sign may be present in a pt who reports the symptom of tinnitus, and should be checked for?

A bruit

--Increased episc

--CCF

How does a When the blo

How should one check for a bruit in cases of suspected CCF? By auscultating the globe (through closed lids of course), and at the temporal region

that drain into a thousand the superior opinion venil, venese aramag impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCE? --Secondary glauc They can be traumatic or spontaneous

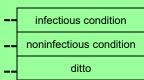
206

```
--Intraocular inflammation
```

- -Anterior uveitis
- --Trabeculitis
- --Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICE

--Increased episcleral venous pressure

- --CCF
- --SVC syndrome
- --Sturge-Weber
- -- Orbital inflammation



--Secondary glaucoma

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

207

```
--Intraocular inflammation
```

- --Anterior uveitis
- -- Trabeculitis
- -- Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICF

--Increased episcleral venous pressure

- --CCF
- --SVC syndrome
- --Sturge-Weber
- -- Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED (Thyroid eye disease)

--Secondary glaucoma

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

208

```
--Intraocular inflammation
```

- -- Anterior uveitis
- --Trabeculitis
- Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICF

--Increased episcleral venous pressure

- --CCF
- --SVC syndrome
- --Sturge-Weber
- --Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED

--Secondary glaucoma

By what two mechanisms do these raise EVP?

1)

2)

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

209

```
--Intraocular inflammation
```

- -- Anterior uveitis
- --Trabeculitis
- --Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
 - --ICF

--Increased episcleral venous pressure

- --CCF
- --SVC syndrome
- --Sturge-Weber
- --Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED

--Secondary glaucoma

By what two mechanisms do these raise EVP?

 Inflammation near the vortex veins causes them to swell, decreasing lumen diameter and thereby impeding drainage and/or

2)



210

```
--Intraocular inflammation
```

- -- Anterior uveitis
- --Trabeculitis
- Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
 - --ICF

--Increased episcleral venous pressure

- --CCF
- --SVC syndrome
- --Sturge-Weber
- --Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED

--Secondary glaucoma

By what two mechanisms do these raise EVP?

- Inflammation near the vortex veins causes them to swell, decreasing lumen diameter and thereby impeding drainage and/or
- 2) Orbital congestion mechanically compresses the vortex veins



211

```
--Intraocular inflammation
```

- --Anterior uveitis
- --Trabeculitis
- --Fuchs heterochromic iridocyclitis
- --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressure
 - --CCF
 - --SVC syndrome
 - --Sturge-Weber
 - --Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED
- --Secondary glaucoma

Before we proceed, a note... It is misleading to have a section labeled 'secondary glaucoma.'



212

```
--Intraocular inflammation
         --Anterior uveitis
-- Corneal endothelial abnormality
--Increased episcleral venous pressure
         --SVC syndrome
         --Sturge-Weber
         --Orbital inflammation
```

--Secondary glaucoma

Before we proceed, a note...It is misleading to have a section labeled 'secondary glaucoma.' Why? Because every etiology above also qualifies as a secondary glaucoma (assuming the elevated IOP results in glaucomatous damage, that is). The term here is used as a catch-all for conditions that didn't fit easily into other categories.

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
 - -- Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressure
 - --CCF
 - --SVC syndrome
 - --Sturge-Weber
 - --Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TEI

--Secondary glaucoma

These are bilateral, but can be so asymmetric as to seem unilateral

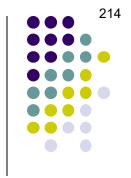
a general sort of condition

a specific condition

another specific condition



- --Intraocular inflammation -- Corneal endothelial abnormality --Increased episcleral venous pressure
- These are bilateral, but can be so asymmetric as to seem unilateral



- --Intraocular inflammation
 - -Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressure
 - --CCF
 - --SVC syndrome
 - --Sturge-Weber
 - --Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED

--Secondary glaucoma

```
These are bilateral, but can be so asymmetric as to seem unilateral
```

```
--Lens-related: Phacolytic, phacomorphic
```

--PXS

--PDS

215

216

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
 - Fuchs heterochromic iridocyclitis
 - ---Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous press
 - --CCF
 - --SVC syndrome
 - --Sturge-Weber
 - --Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED

--Secondary glaucoma

These are bilateral, but can be so asymmetric as to seem unilateral

- --Lens-related: Phacolytic
 - -Lens-related. **Phacolyt** -PXS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)

217

- --Intraocular inflammation
 - --Anterior uveitis
- -- Corneal endothelial abnormality
- --Increased episcleral venous press

--Secondary glaucoma

These are bilateral. but can be so asymmetric as to seem unilateral

- --Lens-related: Phacolytic

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?

- --Elevated IOP (duh)
- --A mature or hypermature cataract

218

```
--Intraocular inflammation
```

- --Anterior uveitis
- --Trabeculitis
- --Fuchs heterochromic iridocyclitis
- --Posner-Schlossman

--Corneal endothelial abnormality

- --PPMD
- --ICE
- --Increased episcleral venous pressi
 - --CCF
 - --SVC syndrome
 - --Sturge-Weber
 - Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED

--Secondary glaucoma

These are bilateral, but can be so asymmetric as to seem unilateral

- --Lens-related: Phacolytic
 - -PXS -PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?

- --Elevated IOP (duh)
- --A mature or hypermature cataract

What is a mature cataract?

219

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
 - Fuchs heterochromic iridocyclitis
 - ---Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressi
 - --CCF
 - --SVC syndrome
 - --Sturge-Weber
 - Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED

--Secondary glaucoma

These are bilateral, but can be so asymmetric as to seem unilateral

- --Lens-related: Phacolytic
 - -PXS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?

- --Elevated IOP (duh)
- --A mature or hypermature cataract

What is a mature cataract?

A cortical cataract that has advanced to the point that the entire lens is opaque





Mature cataract

221

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- --Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressi
 - --CCF
 - --SVC syndrome
 - --Sturge-Weber
 - Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED

--Secondary glaucoma

These are bilateral, but can be so asymmetric as to seem unilateral

- --Lens-related: Phacolytic
 - -PXS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?

- --Elevated IOP (duh)
- --A mature or hypermature cataract

What is a mature cataract?

A cortical cataract that has advanced to the point that the entire lens is opaque

What is a hypermature cataract?

222

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
- -- Corneal endothelial abnormality
- --Increased episcleral venous press

 - --SVC syndrome
 - --Sturge-Weber

--Secondary glaucoma

These are bilateral. but can be so asymmetric as to seem unilateral

- --Lens-related: Phacolytic

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?

- --Elevated IOP (duh)
- --A mature or hypermature cataract

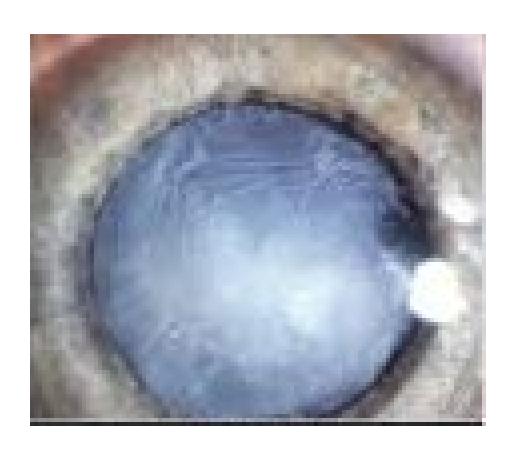
What is a mature cataract?

A cortical cataract that has advanced to the point that the entire lens is opaque

What is a hypermature cataract?

A mature cataract in which the cortical material has partially liquefied





Hypermature cataract

224

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic iridocyclitis
 - --Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous pressi
 - --CCF
 - --SVC syndrome
 - --Sturge-Weber
 - --Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED

--Secondary glaucoma

These are bilateral, but can be so asymmetric as to seem unilateral

- --Lens-related: Phacolytic
 - -PXS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?

- --Elevated IOP (duh)
- --A mature or hypermature cataract

What is a mature cataract?

A cortical cataract that has advanced to the point that the entire lens is opaque

What is a hypermature cataract?

A mature cataract in which the cortical material has partially liquefied

Once the cortical component has liquefied enough to allow the nuclear component to move freely, what is the cataract called?

225

- --Intraocular inflammation
 - --Anterior uveitis
 - --Trabeculitis
 - --Fuchs heterochromic iridocyclitis
 - ---Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
 - --ICE
- --Increased episcleral venous press
 - --CCF
 - --SVC syndrome
 - --Sturge-Weber
 - --Orbital inflammation
 - --cellulitis
 - --pseudotumor
 - --TED

--Secondary glaucoma

These are bilateral, but can be so asymmetric as to seem unilateral

- --Lens-related: Phacolytic
 - -PXS -PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?

- --Elevated IOP (duh)
- --A mature or hypermature cataract

What is a mature cataract?

A cortical cataract that has advanced to the point that the entire lens is opaque

What is a hypermature cataract?

A mature cataract in which the cortical material has partially liquefied

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A morgagnian cataract





Morgagnian cataract

227

- --Intraocular inflammation
 - --Anterior uveitis
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 - --Posner-Schlossman
- -- Corneal endothelial abnormality
 - --PPMD
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--Secondary glaucoma

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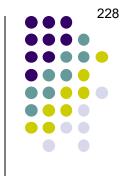
--Lens-related: Phacolytic

-PXS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?

- --Elevated IOP (duh)
- --A mature or hypermature cataract

What is the classic presentation and history?



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--Intraocular inflammation
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- --Anterior uveitis

-- Corneal endothelial abnormality

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--Secondary glaucoma

These are bilateral. but can be so asymmetric as to seem unilateral

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229

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Is the angle open or closed?

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Liquefaction of the cataract leads to lens proteins leaching across the capsule into the AC, where they prompt an inflammatory reaction; the protein + inflammatory cells clog the trabecular meshwork, leading to the IOP rise

236

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What will cytologic evaluation of an aqueous aspirate reveal? (This is another classic feature of phacolytic glaucoma.)

AC, where they prompt an e protein + inflammatory cells cl leading to the IOP rise

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Big fat macrophages loaded down with phagocytized lens proteins

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--Intraocular inflammation
                   Is phacomorphic glaucoma a form of open-angle, or angle-closure glaucoma?
--Corneal endoth
--Increased episo
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238

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Angle closure
In phacolytic glaucoma, cataractous lens proteins in the AC are the culprit. What is
the culprit in phacomorphic glaucoma?
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240

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```

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242
```

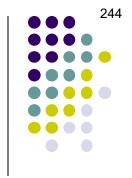
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                    1)
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                     How does cataractous increase in lens size lead to IOP elevation?
                     1) It alters the anatomic relationship between the anterior lens surface and the pupil
                     margin in a manner that leads to pupillary block and subsequent angle closure; and
                     2)
```

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 - Cataractous increase in lens size

How does cataractous increase in lens size lead to IOP elevation?

- 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; and
- 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

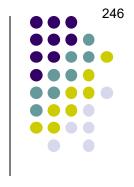
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 - --PXS
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What is the status of the lens capsule in these conditions?

```
--Intraocular inflammation
-- Corneal endothelial abnormality
--Increased episcleral v
                                  sure
                        through
                        intact
                        capsule
--Secondary glaucoma
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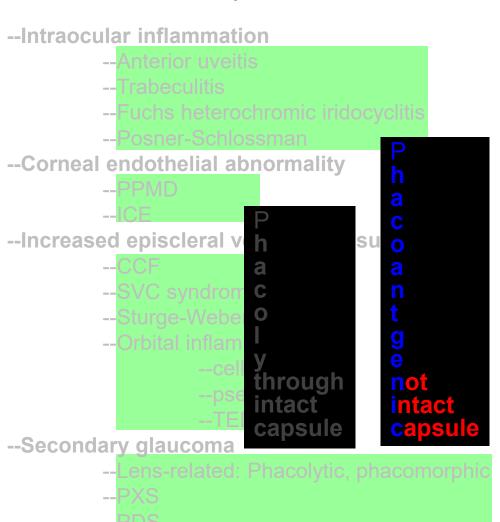
What is the status of the lens capsule in these conditions?
By definition, **intact**

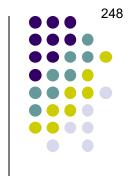
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247
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--Intraocular inflammation
         --Anterior uveitis
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                                   sure
                        through
                        intact
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What lens condition associated with uveitis has, by definition, a capsule that **has** been traumatically or surgically breached?

What is the status of the lens capsule in these conditions?
By definition, intact





What lens condition associated with uveitis has, by definition, a capsule that **has** been traumatically or surgically breached?

Phacoantigenic uveitis

What is the status of the lens capsule in these conditions?
By definition, intact

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- --PXS
- --PDS

PXS and PDS are compared and contrasted in detail in slide-set G4

250

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There are many other causes of secondary glaucoma—see both G13 (2ndry open-angle glaucoma) and G16 (2ndry angle-closure glaucoma)

--TED

- --Secondary glaucoma
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 - --PXS
 - --PDS