What is the definition of hyphema?
What is the definition of hyphema?
The presence of RBCs in the anterior chamber
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

*With respect to its extent, there are two types of hyphema—what are they?*
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema
Hyphema

Layered hyphema
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema

What is the most common cause of hyphema?
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema.
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema.

What is the most common cause of hyphema?
Trauma
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema

What is the most common cause of hyphema?
Trauma

What type of trauma is most commonly implicated?
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema

What is the most common cause of hyphema?
Trauma

What type of trauma is most commonly implicated?
Blunt
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema

What is the most common cause of hyphema?
Trauma

What is the mechanism by which blunt trauma causes hyphema?

---

Blunt
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema

What is the most common cause of hyphema?
Trauma

What is the mechanism by which blunt trauma causes hyphema?
The globe is incompressible in the sense that its volume cannot be reduced. (It's like a balloon—squeeze it in one place, it has to expand in another.) So when blunt force to the globe compresses its anterior-posterior dimension, the globe compensates by expanding in the equatorial plane—the eye gets momentarily shorter and fatter.
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema

What is the most common cause of hyphema?
Trauma

What is the mechanism by which blunt trauma causes hyphema?
The globe is incompressible in the sense that its volume cannot be reduced. (It's like a balloon—squeeze it in one place, it has to expand in another.) So when blunt force to the globe compresses its anterior-posterior dimension, the globe compensates by expanding in the equatorial plane—the eye gets momentarily shorter and fatter. In turn, equatorial expansion stretches the iris, including its major (and other) arterial circle. Tears of these vessels or their branches are the common source of blood in traumatic hyphema.
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema

What is the most common cause of hyphema?
Trauma

What type of trauma is most commonly implicated?
Blunt

Who is at risk for traumatic hyphema?
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema

What is the most common cause of hyphema?
Trauma

What type of trauma is most commonly implicated?
Blunt

Who is at risk for traumatic hyphema?
Young men
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema

What is the most common cause of hyphema?
Trauma

What type of trauma is most commonly implicated?
Blunt

Who is at risk for traumatic hyphema?
Young men

Why are young men at risk?
What is the definition of hyphema?
The presence of RBCs in the anterior chamber

With respect to its extent, there are two types of hyphema—what are they?
--When the extent of the hyphema is limited to a few RBCs circulating in the AC, it is called a microhyphema
--When the hyphema is extensive enough to form a clot in the AC, it is called a layered hyphema

What is the most common cause of hyphema?
Trauma

What type of trauma is most commonly implicated?
Blunt

Who is at risk for traumatic hyphema?
Young men

Why are young men at risk?
Because they’re dumb, and do things that lead to getting hit in the eye
What is the pre-eminent goal in managing hyphema?
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed
What is the pre-eminent goal in managing hyphema? *Avoid a re-bleed*

*Is re-bleeding a common event in hyphema?*
Q/A

What is the pre-eminent goal in managing hyphema? **Avoid a re-bleed**

*Is re-bleeding a common event in hyphema?*
Not really; only somewhere around % of cases re-bleed
What is the pre-eminent goal in managing hyphema? **Avoid a re-bleed**

*Is re-bleeding a common event in hyphema?*
Not really; only somewhere around 5% of cases re-bleed

Re-bleed rate per:
-- *Glaucma* book: 5-10%
-- *Cornea* book: <5%
What is the pre-eminent goal in managing hyphema? **Avoid a re-bleed**

*Is re-bleeding a common event in hyphema?*
Not really; only somewhere around 5% of cases re-bleed

*Relative to the initial bleed, when is a re-bleed likely to occur?*
What is the pre-eminent goal in managing hyphema? **Avoid a re-bleed**

*Is re-bleeding a common event in hyphema?*
Not really; only somewhere around 5% of cases re-bleed

*Relative to the initial bleed, when is a re-bleed likely to occur?*
3-7 days post-event
What is the pre-eminent goal in managing hyphema? **Avoid a re-bleed**

Is re-bleeding a common event in hyphema?
Not really; only somewhere around 5% of cases re-bleed

Relative to the initial bleed, when is a re-bleed likely to occur?

3-7 days post-event

Why then? What happens 3-7 days after the initial bleed?
What is the pre-eminent goal in managing hyphema? **Avoid a re-bleed**

Is re-bleeding a common event in hyphema? Not really; only somewhere around 5% of cases re-bleed.

Relative to the initial bleed, when is a re-bleed likely to occur? **3-7 days post-event**

Why then? *What happens 3-7 days after the initial bleed?*
This is when the original clot is going through the process of lysis/retraction.
What is the pre-eminent goal in managing hyphema? **Avoid a re-bleed**

*Is re-bleeding a common event in hyphema?*
Not really; only somewhere around 5% of cases re-bleed

*Relative to the initial bleed, when is a re-bleed likely to occur?*
3-7 days post-event

*What’s the big deal about re-bleeding, ie, why is avoiding it so important?*
A

What is the pre-eminent goal in managing hyphema? **Avoid a re-bleed**

*Is re-bleeding a common event in hyphema?*
Not really; only somewhere around 5% of cases re-bleed

*Relative to the initial bleed, when is a re-bleed likely to occur?*
3-7 days post-event

*What’s the big deal about re-bleeding, ie, why is avoiding it so important?*
Because the risk of long-term complications goes up significantly if re-bleeding occurs
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed
What goal is a close second?
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP
What is the pre- eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP

What is the mechanism by which hyphema leads to elevated IOP?
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP

What is the mechanism by which hyphema leads to elevated IOP?
The TM becomes clogged with RBCs, along with the usual inflammatory cells and material. (Not to mention, the inciting trauma may have damaged the angle.)
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? **Control IOP**

*What is the mechanism by which hyphema leads to elevated IOP?*
The TM becomes clogged with RBCs, along with the usual inflammatory cells and material. (Not to mention, the inciting trauma may have damaged the angle.)

*What event modestly increases the risk of significant IOP elevation?*
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? **Control IOP**

What is the mechanism by which hyphema leads to elevated IOP?
The TM becomes clogged with RBCs, along with the usual inflammatory cells and material. (Not to mention, the inciting trauma may have damaged the angle.)

What event modestly increases the risk of significant IOP elevation?
A large hyphema (ie, there is a modest correlation between hyphema size and the incidence of IOP elevation)
What is the pre- eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? **Control IOP**

*What is the mechanism by which hyphema leads to elevated IOP?*
The TM becomes clogged with RBCs, along with the usual inflammatory cells and material. (Not to mention, the inciting trauma may have damaged the angle.)

*What event modestly increases the risk of significant IOP elevation?*
A large hyphema (ie, there is a modest correlation between hyphema size and the incidence of IOP elevation)

*What event **greatly** increases the risk of significant IOP elevation?*
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? **Control IOP**

*What is the mechanism by which hyphema leads to elevated IOP?*
The TM becomes clogged with RBCs, along with the usual inflammatory cells and material. (Not to mention, the inciting trauma may have damaged the angle.)

*What event modestly increases the risk of significant IOP elevation?*
A large hyphema (i.e., there is a modest correlation between hyphema size and the incidence of IOP elevation)

*What event *greatly* increases the risk of significant IOP elevation?*
A re-bleed
What is the pre-eminent goal in managing hyphema? *Avoid a re-bleed*

What goal is a close second? *Control IOP*

Third?
• What is the pre-eminent goal in managing hyphema? Avoid a re-bleed
• What goal is a close second? Control IOP
• Third? Control inflammation
What is the pre-eminently goal in managing hyphema? Avoid a re-bleed
What goal is a close second? Control IOP
Third? Control inflammation

Why is inflammation control important?
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP

Third? Control inflammation

Why is inflammation control important?
To reduce the risk of synechiae formation (and to improve pt comfort of course).
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed
What goal is a close second? Control IOP
Third? Control inflammation
There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?
What is the pre- eminent goal in managing hyphema? Avoid a re-bleed
What goal is a close second? Control IOP
Third? Control inflammation
There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? Prevent corneal bloodstaining
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP

Third? Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? Prevent corneal bloodstaining

Briefly, how does a hyphema lead to corneal bloodstaining?
Briefly, how does a hyphema lead to corneal bloodstaining?
RBCs in the AC release Hgb, which enters the corneal stroma and gets absorbed by keratocytes.

Q/A

- What is the pre-eminent goal in managing hyphema? Avoid a re-bleed.
- What goal is a close second? Control IOP.
- Third? Control inflammation.

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? **Prevent corneal bloodstaining**
Briefly, how does a hyphema lead to corneal bloodstaining?

RBCs in the AC release Hgb, which enters the corneal stroma and gets absorbed by keratocytes.

What is the pre-eminent goal in managing hyphema? Avoid a re-bleed.
What goal is a close second? Control IOP.
Third? Control inflammation.
There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? Prevent corneal bloodstaining.

Prevent corneal bloodstaining

Briefly, how does a hyphema lead to corneal bloodstaining?
RBCs in the AC release Hgb, which enters the corneal stroma and gets absorbed by keratocytes.
Hyphema

Corneal bloodstaining
What is the pre- eminent goal in managing hyphema? Avoid a re-bleed
What goal is a close second? Control IOP
Third? Control inflammation
There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? **Prevent corneal bloodstaining**

*Briefly, how does a hyphema lead to corneal bloodstaining?*
RBCs in the AC release Hgb, which enters the corneal stroma and gets absorbed by keratocytes

*What complication—common in hyphema—increases the risk of bloodstaining?*
Briefly, how does a hyphema lead to corneal bloodstaining?
RBCs in the AC release Hgb, which enters the corneal stroma and gets absorbed by keratocytes

What complication—common in hyphema—increases the risk of bloodstaining?
Increased IOP

What is the pre-eminent goal in managing hyphema? Avoid a re-bleed
What goal is a close second? Control IOP
Third? Control inflammation
There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? Prevent corneal bloodstaining

Briefly, how does a hyphema lead to corneal bloodstaining?
RBCs in the AC release Hgb, which enters the corneal stroma and gets absorbed by keratocytes

What complication—common in hyphema—increases the risk of bloodstaining?
Increased IOP
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP

Third? Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? Prevent corneal bloodstaining

Briefly, how does a hyphema lead to corneal bloodstaining?
RBCs in the AC release Hgb, which enters the corneal stroma and gets absorbed by keratocytes

What complication—common in hyphema—increases the risk of bloodstaining?
Increased IOP

Why is corneal bloodstaining such a concern in young children?
Briefly, how does a hyphema lead to corneal bloodstaining?
RBCs in the AC release Hgb, which enters the corneal stroma and gets absorbed by keratocytes.

What complication—common in hyphema—increases the risk of bloodstaining?
Increased IOP

Why is corneal bloodstaining such a concern in young children?
Because it is potentially amblyogenic

What is the pre- eminent goal in managing hyphema? Avoid a re-bleed
What goal is a close second? Control IOP
Third? Control inflammation
There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? Prevent corneal bloodstaining
Briefly, how does a hyphema lead to corneal bloodstaining?
RBCs in the AC release Hgb, which enters the corneal stroma and gets absorbed by keratocytes.

What complication—common in hyphema—increases the risk of bloodstaining?
Increased IOP

Why is corneal bloodstaining such a concern in young children?
Because it is potentially amblyogenic

Hyphema

Q

What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP

Third? Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?
Prevent corneal bloodstaining

Is the prognosis for corneal bloodstaining good, or bad?
Both. It spontaneously and completely clears (yay!) over a course of months to years (boo!)
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP

Third? Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? **Prevent corneal bloodstaining**

**What is the pre-eminent goal in managing hyphema? Avoid a re-bleed**

**What goal is a close second? Control IOP**

**Third? Control inflammation**

**There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? Prevent corneal bloodstaining**

**Is the prognosis for corneal bloodstaining good, or bad?** Both. It spontaneously and completely clears (yay!) over a course of months to years (boo!)

**What complication—common in hyphema—increases the risk of bloodstaining?** Increased IOP

**Why is corneal bloodstaining such a concern in young children?** Because it is potentially amblyogenic
What is the pre-eminent goal in managing hyphema?

Avoid a re-bleed

Control IOP

Control inflammation

Prevent corneal bloodstaining

**tl;dr The treatment goals in managing hyphema**

No question—proceed when ready
What is the pre-eminent goal in managing hyphema?

- Avoid a re-bleed

What goal is a close second?

- Control IOP

Third?

- Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?

- Prevent corneal bloodstaining

**tl;dr** The treatment goals in managing hyphema

Speaking of managing hyphema…What steps should be taken to minimize the risk of re-bleeding?

- Shield the eye around the clock
- Elevate the head around the clock
- Strict bedrest
- Avoidance of anticoagulants
- See the pt until the hyphema resolves
What is the pre-eminent goal in managing hyphema?
- Avoid a re-bleed

What goal is a close second?
- Control IOP

What goal is third?
- Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed.
- Prevent corneal bloodstaining

---

**Speaking of managing hyphema…What steps should be taken to minimize the risk of re-bleeding?**

-- Shield the eye around the clock
-- Elevate the head around the clock
-- Strict bedrest
-- Avoidance of anticoagulants
-- See the pt daily until the hyphema resolves

**tl;dr**
The treatment goals in managing hyphema
**What is the pre-eminent goal in managing hyphema?**

- Avoid a re-bleed

**What goal is a close second?**

- Control IOP

**What goal is third?**

- Control inflammation

There is another goal, in young children, that is arguably as important as avoiding re-bleed. What is it?

- Prevent corneal bloodstaining

---

**Speaking of managing hyphema… What steps should be taken to minimize the risk of re-bleeding?**

- Shield the eye around the clock
- Elevate the head around the clock
- **Strict bedrest**
- Avoidance of anticoagulants
- See the pt daily until the hyphema resolves

---

**Does this mean the pt should be hospitalized?**

If necessary, yes (as is often the case with children)

---

**tl;dr The treatment goals in managing hyphema**
What is the pre-eminent goal in managing hyphema?
- Avoid a re-bleed

What goal is a close second?
- Control IOP

Third?
- Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?
- Prevent corneal bloodstaining

Speaking of managing hyphema... What steps should be taken to minimize the risk of re-bleeding?
- Shield the eye around the clock
- Elevate the head around the clock
- **Strict bedrest**
- Avoidance of anticoagulants
- See the pt daily until the hyphema resolves

*tl;dr The treatment goals in managing hyphema*

- **Avoid a re-bleed**
- Control IOP
- Control inflammation
- Prevent corneal bloodstaining

*Does this mean the pt should be hospitalized?*
If necessary, yes (as is often the case with children).
What is the pre-eminent goal in managing hyphema?
- Avoid a re-bleed

What goal is a close second?
- Control IOP

Third?
- Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?
- Prevent corneal bloodstaining

Speaking of managing hyphema… What steps should be taken to minimize the risk of re-bleeding?
-- Shield the eye around the clock
-- Elevate the head around the clock
-- **Strict bedrest**
-- Avoidance of anticoagulants
-- See the pt daily until the hyphema resolves

Does this mean the pt should be hospitalized?
If necessary, yes (as is often the case with children)

**tl;dr** The treatment goals in managing hyphema
**Avoid a re-bleed**

**Hyphema**

Speaking of managing hyphema…What steps should be taken to minimize the risk of re-bleeding?

-- Shield the eye around the clock
-- Elevate the head around the clock
-- Strict bedrest
-- **Avoidance of anticoagulants**
-- See the pt daily until the hyphema resolves

How might a pt inadvertently anti-coagulate him/herself?

*tl;dr The treatment goals in managing hyphema*
What is the pre-eminent goal in managing hyphema? 

Avoid a re-bleed

What goal is a close second? 

Control IOP

Third? 

Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? 

Prevent corneal bloodstaining

Speaking of managing hyphema…What steps should be taken to minimize the risk of re-bleeding?

-- Shield the eye around the clock
-- Elevate the head around the clock
-- Strict bedrest
-- Avoidance of anticoagulants
-- See the pt daily until the hyphema resolves

How might a pt inadvertently anti-coagulate him/herself?
By taking aspirin for pain

tl;dr The treatment goals in managing hyphema
What is the pre-eminent goal in managing hyphema?
- Avoid a re-bleed

What goal is a close second?
- Control IOP

Third?
- Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?
- Prevent corneal bloodstaining

**tl;dr** The treatment goals in managing hyphema

Speaking of managing hyphema... What steps should be taken to minimize the risk of re-bleeding?

- Shield the eye around the clock
- Elevate the head around the clock
- Strict bedrest
- **Avoidance of anticoagulants**
- See the pt. daily until the hyphema resolves

How might a pt inadvertently anti-coagulate him/herself?
By taking aspirin for pain

What should you tell the pt in this regard?

- To avoid aspirin (and other NSAIDs, just to be safe)
What is the pre-eminent goal in managing hyphema?

- Avoid a re-bleed

What goal is a close second?

- Control IOP

Third?

- Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?

- Prevent corneal bloodstaining

**tl;dr** The treatment goals in managing hyphema

Speaking of managing hyphema…What steps should be taken to minimize the risk of re-bleeding?

- Shield the eye around the clock
- Elevate the head around the clock
- Strict bedrest
- **Avoidance of anticoagulants**
- See the pt daily until the hyphema resolves

How might a pt inadvertently anti-coagulate him/herself? By taking aspirin for pain

What should you tell the pt in this regard? To avoid aspirin (and other NSAIDs, just to be safe)
Avoid a re-bleed

Speaking of managing hyphema…What steps should be taken to minimize the risk of re-bleeding?
-- Shield the eye around the clock

What is aminocaproic acid, and how does it relate to avoiding re-bleed in hyphema management?
Avoid a re-bleed

Speaking of managing hyphema… What steps should be taken to minimize the risk of re-bleeding?
-- Shield the eye around the clock

What is aminocaproic acid, and how does it relate to avoiding re-bleed in hyphema management?
It is a systemic med that acts as a ‘clot stabilizer’ by enhancing hemostasis during the process of clot fibrinolysis.
What is the pre-eminent goal in managing hyphema?

Avoid a re-bleed

What goal is a close second?
Control IOP

Third?
Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?
Prevent corneal bloodstaining

tl;dr
The treatment goals in managing hyphema

Speaking of managing hyphema... What steps should be taken to minimize the risk of re-bleeding?

- Shield the eye around the clock
- Elevate the head around the clock
- Strict bedrest
- Avoidance of anticoagulants
- See the pt daily until the hyphema resolves

Hyphema

How might a pt inadvertently anti-coagulate him/herself?
By taking aspirin for pain

What should you tell the pt in this regard?
To avoid aspirin (and other NSAIDs, just to be safe)

What is aminocaproic acid, and how does it relate to avoiding re-bleed in hyphema management?

It is a systemic med that acts as a ‘clot stabilizer’ by enhancing hemostasis during the process of clot fibrinolysis

What is the rationale behind its use in managing hyphema?

- The evidence regarding its ability to reduce re-bleed risk is equivocal
- It has myriad unwelcome side effects that limit its acceptance
- There is some evidence that the risk of re-bleed goes up when it is stopped
Avoid a re-bleed

Speaking of managing hyphema…What steps should be taken to minimize the risk of re-bleeding?
-- Shield the eye around the clock

What is aminocaproic acid, and how does it relate to avoiding re-bleed in hyphema management?
It is a systemic med that acts as a ‘clot stabilizer’ by enhancing hemostasis during the process of clot fibrinolysis

What is the rationale behind its use in managing hyphema?
By enhancing hemostasis, it may reduce the risk of a re-bleed
What is the pre-eminent goal in managing hyphema?

Avoid a re-bleed

What goal is a close second?

Control IOP

Third?

Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?

Prevent corneal bloodstaining

Speaking of managing hyphema... What steps should be taken to minimize the risk of re-bleeding?

-- Shield the eye around the clock
-- Elevate the head around the clock
-- Strict bedrest
-- Avoidance of anticoagulants
-- See the pt daily until the hyphema resolves

What is aminocaproic acid, and how does it relate to avoiding re-bleed in hyphema management?

It is a systemic med that acts as a ‘clot stabilizer’ by enhancing hemostasis during the process of clot fibrinolysis

What is the rationale behind its use in managing hyphema?

By enhancing hemostasis, it may reduce the risk of a re-bleed

Aminocaproic acid is not often used in hyphema management—why?

Three reasons:

-- The evidence regarding its ability to reduce re-bleed risk is equivocal
-- It has myriad unwelcome side effects that limit its acceptance
-- There is some evidence that the risk of re-bleed goes up when it is stopped
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP

Third? Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? Prevent corneal bloodstaining

Speaking of managing hyphema... What steps should be taken to minimize the risk of re-bleeding?

-- Shield the eye around the clock

-- Elevate the head around the clock

-- Strict bedrest

-- Avoidance of anticoagulants

-- See the pt daily until the hyphema resolves

What is aminocaproic acid, and how does it relate to avoiding re-bleed in hyphema management?
It is a systemic med that acts as a ‘clot stabilizer’ by enhancing hemostasis during the process of clot fibrinolysis

What is the rationale behind its use in managing hyphema?
By enhancing hemostasis, it may reduce the risk of a re-bleed

Aminocaproic acid is not often used in hyphema management—why?
Three reasons:

--

--

--
Avoid a re-bleed

Speaking of managing hyphema... What steps should be taken to minimize the risk of re-bleeding?
-- Shield the eye around the clock

What is aminocaproic acid, and how does it relate to avoiding re-bleed in hyphema management?
It is a systemic med that acts as a ‘clot stabilizer’ by enhancing hemostasis during the process of clot fibrinolysis.

What is the rationale behind its use in managing hyphema?
By enhancing hemostasis, it may reduce the risk of a re-bleed.

Aminocaproic acid is not often used in hyphema management—why?
Three reasons:
-- The evidence regarding its ability to reduce re-bleed risk is equivocal
-- It has myriad unwelcome side effects that limit its acceptance
-- There is some evidence that the risk of re-bleed goes up when it is stopped.
Avoid a re-bleed

Speaking of managing hyphema… What steps should be taken to minimize the risk of re-bleeding?
-- Shield the eye around the clock

What is aminocaproic acid, and how does it relate to avoiding re-bleed in hyphema management?
It is a systemic med that acts as a ‘clot stabilizer’ by enhancing hemostasis during the process of clot fibrinolysis

What is the rationale behind its use in managing hyphema?
By enhancing hemostasis, it may reduce the risk of a re-bleed

Aminocaproic acid is not often used in hyphema management—why?
Three reasons:
-- The evidence regarding its ability to reduce re-bleed is equivocal
-- It has myriad unwelcome side effects
-- There is some evidence that the risk of re-bleed goes up when it is stopped

What are some of the myriad side effects?
--
--
--(There are many others)
Aminocaproic acid is not often used in hyphema management—why?
Three reasons:
--The evidence regarding its ability to reduce the risk of re-bleeding is equivocal
--It has myriad unwelcome side effects
--There is some evidence that the risk of re-bleeding may increase when it is stopped

What are some of the myriad side effects?
--GI upset
--Hypotension (to the point of syncope)
--Confusion
--(There are many others)
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP

Third? Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? Prevent corneal bloodstaining

**tl;dr**

The treatment goals in managing hyphema

--

**Which two classes of meds are first-line in managing IOP in hyphema?**

---

β blockers  
α2 agonists  
Apraclonidine (iopidine)  
Brimonidine  
Carbonic anhydrase inhibitors (CAIs)  
Mannitol  
Glycerol
The treatment goals in managing hyphema are:

1. **Avoid a re-bleed**
2. **Control IOP**
3. **Control inflammation**

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? **Prevent corneal bloodstaining**

**Which two classes of meds are first-line in managing IOP in hyphema?**

---

- **Aqueous suppressants**
  - β-blockers
  - α₂ agonists
    - Apraclonidine (iopidine)
    - Brimonidine
  - Carbonic anhydrase inhibitors (CAIs)

- **Hyperosmotic agents**
  - Mannitol
  - Glycerol
What is the pre-eminent goal in managing hyphema? Avoid a re-bleed

What goal is a close second? Control IOP

Third? Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it? Prevent corneal bloodstaining

**tl;dr**

The treatment goals in managing hyphema are:

- Avoid a re-bleed
- Control IOP
- Control inflammation
- Prevent corneal bloodstaining

Which two classes of meds are first-line in managing IOP in hyphema?
Which three drop classes are aqueous suppressants?

--- Aqueous suppressants?
----
----

--- Hyperosmotic agents

--- Apraclonidine (iopidine)
--- Brimonidine
--- Carbonic anhydrase inhibitors (CAIs)
--- Mannitol
--- Glycerol
What is the pre-eminent goal in managing hyphema?

Avoid a re-bleed

What goal is a close second?

Control IOP

Third?

Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?

Prevent corneal bloodstaining

The treatment goals in managing hyphema

Which two classes of meds are first-line in managing IOP in hyphema?

Which three drop classes are aqueous suppressants?

--Aqueous suppressants?

----β blockers

----α2 agonists

----Carbonic anhydrase inhibitors (CAIs)

--Hyperosmotic agents
What is the pre-eminent goal in managing hyphema?
Avoid a re-bleed

What goal is a close second?
Control IOP

Third?
Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?
Prevent corneal bloodstaining

The treatment goals in managing hyphema

Avoid a re-bleed
Control IOP
Control inflammation
Prevent corneal bloodstaining

---

Which two classes of meds are first-line in managing IOP in hyphema?

Which three drop classes are aqueous suppressants?

Which two meds are $\alpha_2$ agonists?

---Aqueous suppressants
----β blockers
----$\alpha_2$ agonists?

-----

-----

-----

-----

-----

---Carbonic anhydrase inhibitors (CAIs)
--Hyperosmotic agents
What is the pre-eminent goal in managing hyphema?
- Avoid a re-bleed

What goal is a close second?
- Control IOP

Third?
- Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?
- Prevent corneal bloodstaining
Avoid a re-bleed

Control IOP

Control inflammation

What is the pre-eminent goal in managing hyphema?
Avoid a re-bleed

What goal is a close second?
Control IOP

Third?
Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?
Prevent corneal bloodstaining

Which two classes of meds are first-line in managing IOP in hyphema?

Which three drop classes are aqueous suppressants?

Which two meds are α₂ agonists?

Which two agents are hyperosmotics?

---Aqueous suppressants
----β blockers
----α₂ agonists
------Apraclonidine (iopidine)
------Brimonidine
------Carbonic anhydrase inhibitors (CAIs)

---Hyperosmotic agents?
The treatment goals in managing hyphema are:

1. **Avoid a re-bleed**
2. **Control IOP**
3. **Control inflammation**
4. Prevent corneal bloodstaining

**Which two classes of meds are first-line in managing IOP in hyphema?**

**Which three drop classes are aqueous suppressants?**

**Which two meds are α₂ agonists?**

**Which two agents are hyperosmotics?**

--- *Aqueous suppressants*

- β blockers
- α₂ agonists

--- *Apraclonidine (iopidine)*

--- *Brimonidine*

--- Carbonic anhydrase inhibitors (CAIs)

--- *Hyperosmotic agents?*

- Mannitol
- Glycerol
The treatment goals in managing hyphema are:

- Avoid a re-bleed
- Control IOP
- Control inflammation

Another goal that is arguably as important as avoiding re-bleed in young children is preventing corneal bloodstaining.

---

Which two classes of meds are first-line in managing IOP in hyphema?
Which three drop classes are aqueous suppressants?
Which two meds are α₂ agonists?
Which two agents are hyperosmotics?

---Aqueous suppressants
-----β blockers
-----α₂ agonists
------Apraclonidine (iopidine)
------Brimonidine
----Carbonic anhydrase inhibitors (CAIs)
--Hyperosmotic agents
----Mannitol
----Glycerol

If IOP can't be controlled medically, what surgical procedure should be considered?

AC washout (we'll have more to say about this procedure later in the slide-set)
Hyphema

The treatment goals in managing hyphema:

Avoid a re-bleed
Control IOP
Control inflammation

Which two classes of meds are first-line in managing IOP in hyphema?
Which three drop classes are aqueous suppressants?
Which two meds are $\alpha_2$ agonists?
Which two agents are hyperosmotics?
---Aqueous suppressants
-----Apraclonidine (iopidine)
-----Brimonidine
-----Carbonic anhydrase inhibitors (CAIs)
---Hyperosmotic agents
-----Mannitol
-----Glycerol

If IOP can’t be controlled medically, what surgical procedure should be considered?
AC washout (we’ll have more to say about this procedure later in the slide-set)
What is the pre-eminent goal in managing hyphema?

- Avoid a re-bleed

What goal is a close second?

- Control IOP

Third?

- Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?

- Prevent corneal bloodstaining

**tl;dr** The treatment goals in managing hyphema

How is intraocular inflammation controlled?

The usual way—with topical steroids and cycloplegics.

Note: Some clinicians forego steroids and cycloplegics in young children, contending that the potential benefit is outweighed by the risk incurred in rasslin’ a struggling child to get the drops into her eye.
What is the pre-eminent goal in managing hyphema?

 Avoid a re-bleed

 What goal is a close second?

 Control IOP

 Third?

 Control inflammation

 There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?

 Prevent corneal bloodstaining

 tl;dr The treatment goals in managing hyphema

 How is intraocular inflammation controlled?

 The usual way—with topical steroids and cycloplegics

 Note: Some clinicians forego steroids and cycloplegics in young children, contending that the potential benefit is outweighed by the risk incurred in rasslin' a struggling child to get the drops into her eye.
Avoid a re-bleed

Control IOP

Control inflammation

How is intraocular inflammation controlled? The usual way—with topical steroids and cycloplegics

Note: Some clinicians forego steroids and cycloplegics in young children, contending that the potential benefit is outweighed by the risk incurred in rasslin’ a struggling child to get the drops into her eye.

tl;dr The treatment goals in managing hyphema
What is the pre-eminent goal in managing hyphema?

- Avoid a re-bleed
- Control IOP
- Control inflammation

Another goal is arguably as important as avoiding re-bleed: Prevent corneal bloodstaining.

**tl;dr** The treatment goals in managing hyphema are:

- Avoid a re-bleed
- Control IOP
- Control inflammation
- Prevent corneal bloodstaining

**What steps can be taken to reduce the risk of corneal bloodstaining?**

- Don’t let IOP get out of hand
- Be vigilant clinically in assessing for evidence of bloodstaining, and have a low threshold for intervening surgically if necessary to prevent it from becoming visually significant
What is the pre-eminent goal in managing hyphema?

- Avoid a re-bleed

What goal is a close second?

- Control IOP

Third?

- Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?

- Prevent corneal bloodstaining

---

What steps can be taken to reduce the risk of corneal bloodstaining?

- Don’t let IOP get out of hand
- Be vigilant clinically in assessing for evidence of bloodstaining, and have a low threshold for intervening surgically if necessary to prevent it from becoming visually significant

**tl;dr** The treatment goals in managing hyphema:
Avoid a re-bleed
Control IOP
Control inflammation

Prevent corneal bloodstaining

What steps can be taken to reduce the risk of corneal bloodstaining?
--Don’t let IOP get out of hand
--Be vigilant clinically in assessing for evidence of bloodstaining, and have a low threshold for intervening surgically if necessary to prevent it from becoming visually significant

What surgical procedure are we talking about here?

tl;dr The treatment of Hyphema
What is the pre-eminent goal in managing hyphema?
- Avoid a re-bleed

What goal is a close second?
- Control IOP

Third?
- Control inflammation

There is another goal that, in young children, is arguably as important as avoiding re-bleed. What is it?
- Prevent corneal bloodstaining


tl;dr The treatment of hyphema is focused on:
- Avoiding re-bleeding
- Controlling IOP
- Controlling inflammation

What steps can be taken to reduce the risk of corneal bloodstaining?
- Don’t let IOP get out of hand
- Be vigilant clinically in assessing for evidence of bloodstaining, and have a low threshold for intervening surgically if necessary to prevent it from becoming visually significant.

What surgical procedure are we talking about here?
Again, AC washout. Speaking of...

Hyphema: AC washout

- Goal is to remove from AC, not the two different words.
- **Hyphema: AC washout**
  - Goal is to remove circulating RBCs from AC, not the entire clot.
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot.
- Criteria triggering AC washout:
  - Any sign of
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot
- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot.
- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x amount of time
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot.
- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days
**Q**

- **Hyphema: AC washout**
  - Goal is to remove **circulating RBCs from AC, not the entire clot**
  - Criteria triggering AC washout:
    - Any sign of **corneal bloodstaining**
    - **Total hyphema x 5 days**

What complication is likely to occur if a total hyphema is present for more than 5 days?
Hyphema: AC washout

- Goal is to remove **circulating RBCs** from AC, not the **entire clot**
- Criteria triggering AC washout:
  - Any sign of **corneal bloodstaining**
  - **Total hyphema x 5 days**

**What complication is likely to occur if a total hyphema is present for more than 5 days?**
Peripheral anterior synechiae
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot
- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days

If a total hyphema consists of dark red-black blood, by name is it known?
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot

- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days

If a total hyphema consists of dark red-black blood, by name is it known? An 8-ball hyphema
Hyphema

8-ball hyphema
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot
- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days
  - Hyphema >50% x amount of time
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot
- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days
  - Hyphema >50% x 8 days
**Hyphema: AC washout**

- **Goal**: To remove circulating RBCs from AC, not the entire clot.
- **Criteria triggering AC washout:**
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days
  - Hyphema >50% x 8 days

**Question:**

What complication is likely to occur if the IOP is >25 for longer than 5 days?
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot
- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days
  - Hyphema >50% x 8 days

What complication is likely to occur if the IOP is >25 for longer than 5 days? Corneal bloodstaining
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot
- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days
  - Hyphema >50% x 8 days
  - Average IOP greater than:
    - 60 x amount of time
**Hyphema: AC washout**

- Goal is to remove *circulating RBCs* from AC, not the *entire clot*

- Criteria triggering AC washout:
  - Any sign of *corneal bloodstaining*
  - Total hyphema *x 5 days*
  - Hyphema >50% *x 8 days*
  - Average IOP greater than:
    - 60 *x 2 days*
**Hyphema: AC washout**

- Goal is to remove **circulating RBCs** from AC, not the **entire clot**

- Criteria triggering AC washout:
  - Any sign of **corneal bloodstaining**
  - Total hyphema x **5 days**
  - Hyphema >50% x **8 days**
  - Average IOP greater than:
    - 60 x **2 days**, or
    - 35 x **amount of time**
**Hyphema: AC washout**

- Goal is to remove circulating RBCs from AC, not the entire clot.

Criteria triggering AC washout:

- Any sign of corneal bloodstaining
- Total hyphema x 5 days
- Hyphema >50% x 8 days
- Average IOP greater than:
  - 60 x 2 days, or
  - 35 x 7 days
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot

Criteria triggering AC washout:
- Any sign of corneal bloodstaining
- Total hyphema x 5 days
- Hyphema >50% x 8 days
- Average IOP greater than:
  - 60 x 2 days, or
  - 35 x 7 days

What devastating complication is likely to occur if IOP is >60 for a couple of days, or >35 for seven days?
**Hyphema: AC washout**

- Goal is to remove circulating RBCs from AC, not the entire clot

- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days
  - Hyphema >50% x 8 days
  - Average IOP greater than:
    - $60 \times 2\text{ days}$, or
    - $35 \times 7\text{ days}$

*What devastating complication is likely to occur if IOP is >60 for a couple of days, or >35 for seven days?* Optic atrophy
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot

Criteria triggering AC washout:
- Any sign of corneal bloodstaining
- Total hyphema x 5 days
- Hyphema >50% x 8 days
- Average IOP greater than:
  - 60 x 2 days, or
  - 35 x 7 days, or
  - 25 x amount of time
Hyphema: AC washout

- Goal is to remove *circulating RBCs* from AC, not the *entire clot*

- Criteria triggering AC washout:
  - Any sign of *corneal bloodstaining*
  - Total hyphema x 5 days
  - Hyphema >50% x 8 days
  - Average IOP greater than:
    - 60 x 2 days, or
    - 35 x 7 days, or
    - 25 x 5 days
What systemic condition must be checked for in hyphema pts at risk for it?
What systemic condition **must** be checked for in hyphema pts at risk for it?
Sickle-cell anemia

**Hyphema**
What systemic condition **must** be checked for in hyphema pts at risk for it? Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell? A hemoglobinopathy
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell? A hemoglobinopathy

What is the underlying problem?
What systemic condition **must** be checked for in hyphema pts at risk for it? Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell? A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O$_2$ tension). This results in the characteristic ‘sickling’ of affected RBCs.
Sickle cell: RBC sickling
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell? A hemoglobinopathy

What is the underlying problem? An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--
--
--
--
What systemic condition **must** be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O\textsubscript{2} tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS
--SC
--S-Thal
--SA
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell? A hemoglobinopathy

What is the underlying problem? An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low $O_2$ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
- SS
- SC
- S-Thal
- SA

What is the key difference between SS, SC and S-Thal vs SA disease?
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell? A hemoglobinopathy

What is the underlying problem? An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?

- SS
- SC
- S-Thal
- SA

What is the key difference between SS, SC and S-Thal vs SA disease? The first three manifest as clinically apparent dz, whereas SA is an asymptomatic (under most conditions) carrier state--aka ‘sickle trait’
What systemic condition **must** be checked for in hyphema pts at risk for it?

Sickle-cell anemia

**Broadly speaking, what sort of disease is sickle-cell?**

A hemoglobinopathy

**What is the underlying problem?**

An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

**What are the four common genotypes of sickle-cell disease?**

--SS
--SC
--S-Thal
--SA

**In America, people of which two ethnic identities are at greatest risk?**

--African-American: 1 in 500
--Hispanic-American: 1 in 36,000
**What systemic condition must be checked for in hyphema pts at risk for it?**

Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?

A hemoglobinopathy

What is the underlying problem?

An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?

--SS
--SC
--S-Thal
--SA
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS
--SC
--S-Thal
--SA

In America, people of which two ethnic identities are at greatest risk?
--African-American: 1 in ?
--Hispanic-American: 1 in ?

What is the sickle-cell dz birthrate for these groups?
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

In America, people of which two ethnic identities are at greatest risk?
--African-American: 1 in 500
--Hispanic-American: 1 in 36,000

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS
--SC
--S-Thal
--SA
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell? A hemoglobinopathy

What is the underlying problem? An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease? --SS --SC --S-Thal --SA

In America, people of which two ethnic identities are at greatest risk?--African-American: 1 in 500--Hispanic-American: 1 in 36,000

What percent of African-Americans test positive for sickle trait?
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (e.g., low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS
--SC
--S-Thal
--SA

What percent of African-Americans test positive for sickle trait?
A whopping 8% (1 in 12)!
What systemic condition must be checked for in hyphema pts at risk for it?  
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?  
A hemoglobinopathy

What is the underlying problem?  
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?  
--SS  
--SC  
--S-Thal  
--SA

Why must sickle-status be assessed in at-risk hyphema pts?  
Because being sickle-positive:  
1)  
2)
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS
--SC
--S-Thal
--SA

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
- SS?
- SC?
- S-Thal?
- SA?

Which is/are associated with increased risk and a need for modified management?

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications
2) impacts how a hyphema should be managed
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
- SS!
- SC!
- S-Thal!
- SA!

Which is/are associated with increased risk and a need for modified management?
All of them

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications
2) impacts how a hyphema should be managed
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS!
--SC!
--S-Thal!
--SA!

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed

What two complications are sickle pts at greater risk of developing?
Elevated IOP and optic atrophy
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS!
--SC!
--S-Thal!
--SA!

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications, and
2) impacts how a hyphema should be managed

What two complications are sickle pts at greater risk of developing?
Elevated IOP and optic atrophy
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell? A hemoglobinopathy

What is the underlying problem? An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O2 tension). This results in the characteristic 'sickling' of affected RBCs.

What are the four common genotypes of sickle-cell disease? --SS! --SC! --S-Thal! --SA!

Why must sickle-status be assessed in at-risk hyphema pts? Because being sickle-positive:
1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed

What two complications are sickle pts at greater risk of developing? Elevated IOP and optic atrophy

Why do sickle pts have an increased risk of developing elevated IOP? Because of the physical characteristics of their RBCs. Normal RBCs are very flexible and change shape easily; thus, they are able to slip easily through the TM. In contrast, sickled RBCs are stiff, and therefore cannot readily change conformation to fit through the TM. Thus, sickled RBCs will 'pile up' at the TM, effectively occluding it and thereby causing IOP to spike.
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell? A hemoglobinopathy

What is the underlying problem? An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O2 tension). This results in the characteristic 'sickling' of affected RBCs.

What are the four common genotypes of sickle-cell disease? --SS! --SC! --S-Thal! --SA!

Why must sickle-status be assessed in at-risk hyphema pts? Because being sickle-positive:

1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed

Why do sickle pts have an increased risk of developing elevated IOP? Because of the physical characteristics of their RBCs. Normal RBCs are very flexible and change shape easily; thus, they are able to slip easily through the TM. In contrast, sickled RBCs are stiff, and therefore cannot readily change conformation to fit through the TM. Thus, sickled RBCs will 'pile up' at the TM, effectively occluding it and thereby causing IOP to spike.
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O2 tension). This results in the characteristic 'sickling' of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS!
--SC!
--S-Thal!
--SA!

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications;
   and
2) impacts how a hyphema should be managed

What two complications are sickle pts at greater risk of developing?
Elevated IOP and optic atrophy

Why do sickle pts have an increased risk of developing elevated IOP?
Because of the physical characteristics of their RBCs. Normal RBCs are very flexible and change shape easily; thus, they are able to slip easily through the TM. In contrast, sickled RBCs are stiff, and therefore cannot readily change conformation to fit through the TM. Thus, sickled RBCs will 'pile up' at the TM, effectively occluding it and thereby causing IOP to spike.
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O2 tension). This results in the characteristic 'sickling' of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS!
--SC!
--S-Thal!
--SA!

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed

What two complications are sickle pts at greater risk of developing?
Elevated IOP and optic atrophy

Why do sickle pts have an increased risk of developing elevated IOP?
Because of the physical characteristics of their RBCs. Normal RBCs are very flexible and change shape easily; thus, they are able to slip easily through the TM. In contrast, sickled RBCs are stiff, and therefore cannot readily change conformation to fit through the TM. Thus, sickled RBCs will ‘pile up’ at the TM, effectively occluding it and thereby causing IOP to spike.

Why are the optic nerves in sickle pts at greater risk of atrophying?
As is the case throughout their bodies, the baseline status of the microcirculation of the optic nerve in sicklers is marginal—even under the best of circumstances, their optic nerves are just getting by. Thus, their optic nerves are vulnerable to damage stemming from anything that compromises their already compromised circulation, one of which is elevated IOP. Because of this, the optic nerve in sickle pts will sustain more damage at lower IOPs for shorter durations than will the optic nerves of non-sickle pts.
What systemic condition must be checked for in hyphema pts at risk for it?  
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?  
A hemoglobinopathy

What is the underlying problem?  
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O2 tension). This results in the characteristic 'sickling' of affected RBCs.

What are the four common genotypes of sickle-cell disease?  
--SS!  
--SC!  
--S-Thal!  
--SA!

Why must sickle-status be assessed in at-risk hyphema pts?  
Because being sickle-positive:  
1) places a hyphema pt at higher risk for complications; and  
2) impacts how a hyphema should be managed

What two complications are sickle pts at greater risk of developing?  
Elevated IOP and optic atrophy

Why do sickle pts have an increased risk of developing elevated IOP?  
Because of the physical characteristics of their RBCs. Normal RBCs are very flexible and change shape easily; thus, they are able to slip easily through the TM. In contrast, sickled RBCs are stiff, and therefore cannot readily change conformation to fit through the TM. Thus, sickled RBCs will ‘pile up’ at the TM, effectively occluding it and thereby causing IOP to spike.

Why are the optic nerves in sickle pts at greater risk of atrophying?  
As is the case throughout their bodies, the baseline status of the microcirculation of the optic nerve in sicklers is marginal—even under the best of circumstances, their optic nerves are just getting by. Thus, their optic nerves are vulnerable to damage stemming from anything that compromises their already compromised circulation, one of which is elevated IOP.
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
A hemoglobinopathy

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O2 tension). This results in the characteristic 'sickling' of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS!
--SC!
--S-Thal!
--SA!

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed

What two complications are sickle pts at greater risk of developing?
Elevated IOP
optic atrophy

Why do sickle pts have an increased risk of developing elevated IOP?
Because of the physical characteristics of their RBCs. Normal RBCs are very flexible and change shape easily; thus, they are able to slip easily through the TM. In contrast, sickled RBCs are stiff, and therefore cannot readily change conformation to fit through the TM. Thus, sickled RBCs will 'pile up' at the TM, effectively occluding it and thereby causing IOP to spike.

Why are the optic nerves in sickle pts at greater risk of atrophying?
As is the case throughout their bodies, the baseline status of the microcirculation of the optic nerve in sicklers is marginal—even under the best of circumstances, their optic nerves are just getting by. Thus, their optic nerves are vulnerable to damage stemming from anything that compromises their already compromised circulation, one of which is elevated IOP. Because of this, the optic nerve in sickle pts will sustain more damage at lower IOPs for shorter durations than will the optic nerves of non-sickle pts.
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia.

How does being sickle+ impact hyphema management?

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS!
--SC!
--S-Thal!
--SA!

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

How does being sickle+ impact hyphema management?
By changing:
--The agents employed in medical management, and
--The threshold for advancing to surgical management

What is the underlying problem?
An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O₂ tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS!
--SC!
--S-Thal!
--SA!

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed
What systemic condition must be checked for in hyphema pts at risk for it?
- Sickle-cell anemia

Broadly speaking, what sort of disease is sickle-cell?
- A hemoglobinopathy

What is the underlying problem?
- An amino-acid substitution in the hemoglobin beta-chain leads to its malfolding under certain metabolic conditions (eg, low O2 tension). This results in the characteristic ‘sickling’ of affected RBCs.

What are the four common genotypes of sickle-cell disease?
- SS
- SC
- S-Thal
- SA

Why must sickle-status be assessed in at-risk hyphema pts?
- Because being sickle-positive:
  1) places a hyphema pt at higher risk for complications; and
  2) impacts how a hyphema should be managed

How does being sickle+ impact hyphema management?
- By changing:
  - The agents employed in medical management
  - The threshold for advancing to surgical management

Which two classes of meds are first-line in managing IOP in hyphema?

These are the agents commonly employed in managing IOP in hyphema. Which ones should be avoided in sicklers?
- Aqueous suppressants
  - β blockers?
  - α₂ agonists
  - Apraclonidine (iopidine)?
  - Brimonidine?
- Carbonic anhydrase inhibitors (CAIs)?
- Hyperosmotic agents?
  - Mannitol?
  - Glycerol?

1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

How does being sickle+ impact hyphema management?
By changing:
- The agents employed in medical management
- The threshold for advancing to surgical management

Which two classes of meds are first-line in managing IOP in hyphema?
- Aqueous suppressants
  - β blockers
  - α₂ agonists
  - Apraclonidine (iopidine)!
  - Brimonidine
  ----Carbonic anhydrase inhibitors (CAIs)!
- Hyperosmotic agents?
  ----Mannitol!
  ----Glycerol!

These are the agents commonly employed in managing IOP in hyphema.
Which ones should be avoided in sicklers?
Remember: These are the agents not to use in sicklers!

1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

How does being sickle+ impact hyphema management?
By changing:
--The agents employed in medical management; and
--The threshold for advancing to surgical management

Which two classes of meds are first-line in managing IOP in hyphema?

These are the agents commonly employed in managing IOP in hyphema. Which ones should be avoided in sicklers?

---Aqueous suppressants
-----β blockers
-----α₂ agonists
-----Apraclonidine (iopidine)
-----Brimonidine
-----Carbonic anhydrase inhibitors
-----Hyperosmotic agents?
-----Mannitol
-----Glycerol

Why must apraclonidine be avoided in sicklers?

1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed
What systemic condition **must** be checked for in hyphema pts at risk for it?
- Sickle-cell anemia

How does being sickle+ impact hyphema management?
- By changing:
  - The agents employed in medical management, and
  - The threshold for advancing to surgical management

Why must sickle-status be assessed in at-risk hyphema pts?
- Because being sickle-positive:
  1) places a hyphema pt at higher risk for complications; and
  2) impacts how a hyphema should be managed

Which two classes of meds are first-line in managing IOP in hyphema? **These are the agents commonly employed in managing IOP in hyphema.**
- Aqueous suppressants
  - β blockers
  - α₂ agonists
  - Apraclonidine (iopidine)
  - Brimonidine
  - Carbonic anhydrase inhibitors
- Hyperosmotic agents?
  - Mannitol
  - Glycerol

Why must apraclonidine be avoided in sicklers?
- It has too much receptor class stimulatory effect

Which three drop classes are aqueous suppressants?
- β blockers
- α₂ agonists
- Apraclonidine (iopidine)
- Brimonidine
- Carbonic anhydrase inhibitors

Which two agents are hyperosmotic?
- Mannitol
- Glycerol

These are the agents commonly employed in managing IOP in hyphema.

Which ones should be avoided in sicklers?
- Apraclonidine (iopidine)
**What systemic condition must be checked for in hyphema pts at risk for it?**

Sickle-cell anemia

**How does being sickle+ impact hyphema management?**

By changing:

- The agents employed in medical management, and
- The threshold for advancing to surgical management

**Which two classes of meds are first-line in managing IOP in hyphema?**

- Aqueous suppressants
  - β blockers
  - α₂ agonists
- **Apraclonidine (iopidine)**
- Brimonidine
- Carbonic anhydrase inhibitors
- Hyperosmotic agents?
  - Mannitol
  - Glycerol

**Why must apraclonidine be avoided in sicklers?**

It has too much \( \alpha_1 \) stimulatory effect

**Which three drop classes are aqueous suppressants?**

- Beta blockers
- Alpha₂ agonists
- Apraclonidine (Iopidine)
- Brimonidine
- Carbonic anhydrase inhibitors

**Which two agents are hyperosmotic?**

- Mannitol
- Glycerol

These are the agents commonly employed in managing IOP in hyphema.

**Which ones should be avoided in sicklers?**

1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

How does being sickle+ impact hyphema management?
By changing:
- The agents employed in medical management; and
- The threshold for advancing to surgical management.

Which two classes of meds are first-line in managing IOP in hyphema?

These are the agents commonly employed in managing IOP in hyphema. Which ones should be avoided in sicklers?

- Aqueous suppressants
  - β blockers
  - α₂ agonists
  - Apraclonidine (iopidine)
  - Brimonidine
- Carbonic anhydrase inhibitors
- Hyperosmotic agents?
  - Mannitol
  - Glycerol

1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed

Why must apraclonidine be avoided in sicklers?
It has too much α₁ stimulatory effect

Which α₁ stimulation effect is so deleterious that it precludes the use of apraclonidine in sicklers?
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

How does being sickle+ impact hyphema management? By changing:
- The agents employed in medical management, and
- The threshold for advancing to surgical management

Which two classes of meds are first-line in managing IOP in hyphema?

These are the agents commonly employed in managing IOP in hyphema. Which ones should be avoided in sicklers?

- Aqueous suppressants
  - β blockers
  - α₂ agonists
  - **Apraclonidine (iopidine)**
  - Brimonidine
  - **Carbonic anhydrase inhibitors**
  - Hyperosmotic agents?
    - Mannitol
    - Glycerol

Why must apraclonidine be avoided in sicklers? It has too much α₁ stimulatory effect

Which α₁ stimulation effect is so deleterious that it precludes the use of apraclonidine in sicklers? Vasoconstriction of the anterior segment vasculature

1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

How does being sickle+ impact hyphema management? By changing:
- The agents employed in medical management, and
- The threshold for advancing to surgical management.

Which two classes of meds are first-line in managing IOP in hyphema? These are the agents commonly employed in managing IOP in hyphema. Which ones should be avoided in sicklers?

- Aqueous suppressants
  ---- β blockers
  ---- α₂ agonists
  ----- Apraclonidine (iopidine)
  ----- Brimonidine
  ------ Carbonic anhydrase inhibitors (CAIs)
- Hyperosmotic agents?
  ---- Mannitol
  ---- Glycerol

1) places a hyphema pt at higher risk for;
2) impacts how a hyphema should be managed.

Why should topical CAIs be avoided in sicklers?

Because they will acidify the AC, and an acidic environment promotes RBC sickling.

There’s an additional reason systemic CAIs should be avoided—what is it?

Because of their diuretic effect, they may lead to hemoconcentration and thus circulatory compromise (especially if the pt is already dehydrated).
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

How does being sickle+ impact hyphema management? By changing:

- The agents employed in medical management
- The threshold for advancing to surgical management

Which two classes of meds are first-line in managing IOP in hyphema?

1) Aqueous suppressants
   1. β blockers
   2. α₂ agonists
   3. Apraclonidine (iopidine)
   4. Brimonidine
2) Carbonic anhydrase inhibitors (CAIs)

Why should topical CAIs be avoided in sicklers? Because they will acidify the AC, and an acidic environment promotes RBC sickling

Why should topical CAIs be avoided in sicklers? Because of their diuretic effect, they may lead to hemoconcentration and thus circulatory compromise (especially if the pt is already dehydrated)

Which three drop classes are aqueous suppressants?

- Apraclonidine (iopidine)
- Brimonidine
- Carbonic anhydrase inhibitors (CAIs)

Which two meds are α₂ agonists?

- Apraclonidine (iopidine)
- Brimonidine

Which two agents are hyperosmotic?

- Mannitol
- Glycerol

These are the agents commonly employed in managing IOP in hyphema. Which ones should be avoided in sicklers?

1) Aqueous suppressants
2) Carbonic anhydrase inhibitors (CAIs)
3) Mannitol
4) Glycerol
What systemic condition must be checked for in hyphema pts at risk for it?
Sickle-cell anemia

How does being sickle+ impact hyphema management?

By changing:
- The agents employed in medical management, and
- The threshold for advancing to surgical management

Which two classes of meds are first-line in managing IOP in hyphema?

- Aqueous suppressants
  - β blockers
  - α2 agonists
  - Apraclonidine (iopidine)
  - Brimonidine
- Carbonic anhydrase inhibitors (CAIs)

Which three drop classes are aqueous suppressants?

- β blockers
- α2 agonists
- Carbonic anhydrase inhibitors (CAIs)
- Apraclonidine (iopidine)
- Brimonidine

Which two meds are α2 agonists?

- Apraclonidine (iopidine)
- Brimonidine

Which two agents are hyperosmotic?

- Mannitol
- Glycerol

Why should topical CAIs be avoided in sicklers?
Because they will acidify the AC, and an acidic environment promotes RBC sickling

There’s an additional reason systemic CAIs should be avoided—what is it?
Because of their diuretic effect, they may lead to hemoconcentration and thus circulatory compromise (especially if the pt is already dehydrated)

1) places a hyphema pt at higher risk for complications
2) impacts how a hyphema should be managed
**What systemic condition must be checked for in hyphema pts at risk for it?**

Sickle-cell anemia

**How does being sickle+ impact hyphema management?**

By changing:
- The agents employed in medical management,
- The threshold for advancing to surgical management

**Which two classes of meds are first-line in managing IOP in hyphema?**

Aqueous suppressants

- β blockers
- α₂ agonists
- Apraclonidine (iopidine)
- Brimonidine
- Carbonic anhydrase inhibitors (CAIs)

**Why should topical CAIs be avoided in sicklers?**

Because they will acidify the AC, and an acidic environment promotes RBC sickling.

**There’s an additional reason systemic CAIs should be avoided—what is it?**

Because of their diuretic effect, they may lead to hemoconcentration and thus circulatory compromise (especially if the pt is already dehydrated).
What systemic condition must be checked for in hyphema pts at risk for it?
- Sickle-cell anemia

How does being sickle+ impact hyphema management?
By changing:
- The agents employed in medical management
- The threshold for advancing to surgical management

Why must sickle-status be assessed in at-risk hyphema pts?
Because being sickle-positive:
1) places a hyphema pt at higher risk for complications;
2) impacts how a hyphema should be managed

Which two classes of meds are first-line in managing IOP in hyphema?

These are the agents commonly employed in managing IOP in hyphema. Which ones should be avoided in sicklers?

- Aqueous suppressants
  - β blockers
  - α<sub>2</sub> agonists
  - Apraclonidine (iopidine)
  - Brimonidine
  - Carbonic anhydrase inhibitors (CAIs)
- Hyperosmotic agents?
  - Mannitol
  - Glycerol

Why should the hyperosmotics be avoided?
1) places a hyphema pt at higher risk for complications, and
2) impacts how a hyphema should be managed
**What systemic condition must be checked for in hyphema pts at risk for it?**

- Sickle-cell anemia

**How does being sickle+ impact hyphema management?**

- By changing:
  - The agents employed in medical management, and
  - The threshold for advancing to surgical management

**Which two classes of meds are first-line in managing IOP in hyphema?**

- Aqueous suppressants
  - β blockers
  - α2 agonists
  - Apraclonidine (iopidine)
  - Brimonidine
  - Carbonic anhydrase inhibitors (CAIs)
- Hyperosmotic agents?
  - Mannitol
  - Glycerol

**Why should the hyperosmotics be avoided?**

For the same reason as systemic CAIs--they may lead to hemoconcentration and thus circulatory compromise

1) places a hyphema pt at higher risk for complications, and
2) impacts how a hyphema should be managed
What systemic condition must be checked for in hyphema pts at risk for it? Sickle-cell anemia

How does being sickle+ impact hyphema management? By changing:
- The agents employed in medical management, and —The threshold for advancing to *surgical* management

What is the underlying problem? An amino-acid substitution in the hemoglobin beta chain leads to its malfolding under certain metabolic conditions (eg, low O2 tension). This results in the characteristic 'sickling' of affected RBCs.

What are the four common genotypes of sickle-cell disease?
--SS!
--SC!
--S-Thal!
--SA!

Why must sickle-status be assessed in at-risk hyphema pts? Because being sickle-positive:
1) places a hyphema pt at higher risk for complications; and
2) impacts how a hyphema should be managed

(No question—proceed when ready)
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot

Criteria triggering AC washout:
- Any sign of corneal bloodstaining
- Total hyphema x 5 days
- Hyphema >50% x 8 days
- Average IOP greater than:
  - 60 x 2 days, or
  - 35 x 7 days, or
  - 25 x 5 days

If sickle-positive: Average IOP greater than:
- 25 x amount of time
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot

- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days
  - Hyphema >50% x 8 days
  - Average IOP greater than:
    - 60 x 2 days, or
    - 35 x 7 days, or
    - 25 x 5 days

If sickle-positive: Average IOP greater than:
  - 25 x 1 day
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot.

Criteria triggering AC washout:
- Any sign of corneal bloodstaining
- Total hyphema x 5 days
- Hyphema >50% x 8 days
- Average IOP greater than:
  - 60 x 2 days, or
  - 35 x 7 days, or
  - 25 x 5 days

If sickle-positive: Average IOP greater than:
- 25 x 1 day, or
- Repeated spikes >30 for amount of time
**Hyphema: AC washout**

- Goal is to remove circulating RBCs from AC, not the entire clot.

Criteria triggering AC washout:
- Any sign of corneal bloodstaining
- Total hyphema x 5 days
- Hyphema >50% x 8 days
- Average IOP greater than:
  - 60 x 2 days, or
  - 35 x 7 days, or
  - 25 x 5 days

**If sickle-positive:** Average IOP greater than:
- 25 x 1 day, or
- Repeated spikes >30 for 2 days
Hyphema: AC washout

- Goal is to remove circulating RBCs from AC, not the entire clot
- Criteria triggering AC washout:
  - Any sign of corneal bloodstaining
  - Total hyphema x 5 days
  - Hyphema >50% x 8 days
  - Average IOP greater than:
    - 60 x 2 days, or
    - 35 x 7 days, or
    - 25 x 5 days

If sickle-positive: Average IOP greater than:
- 25 x 1 day, or
- Repeated spikes >30 for 2 days

Note the management dilemma in sickle-cell—IOP control is absolutely essential because of their pre-existing optic-nerve vulnerability, but sickle-status takes most medical IOP-lowering agents out of your hands
Next, let’s turn our attention to the subject of spontaneous hyphema
When considering the DDx for spontaneous hyphema, we need to think in terms of two groups. What are they?
When considering the DDx for spontaneous hyphema, we need to think in terms of two groups. What are they?
What are the three most common causes of spontaneous hyphema in adults?
What are the three most common causes of spontaneous hyphema in adults?
What are the three most common causes of spontaneous hyphema in adults?

Yo Dr Flynn, you listed neovascularization three times. This a mistake, or what?
What are the three most common causes of spontaneous hyphema in adults?

Yo Dr Flynn, you listed neovascularization three times. This a mistake, or what? It's 'or what,' actually. The point being made here is that neovascularization of the anterior segment is the final common pathway for the conditions that commonly produce spontaneous hyphema in adults.
Yo Dr Flynn, you listed neovascularization three times. This a mistake, or what? It’s ‘or what,’ actually. The point being made here is that neovascularization of the anterior segment is the final common pathway for the conditions that commonly produce spontaneous hyphema in adults.

*OK, then, what are the three sorts of common conditions that produce the neo that produces spontaneous hyphema in adults?* -- -- --
Yo Dr Flynn, you listed neovascularization three times. This a mistake, or what? It's 'or what,' actually. The point being made here is that neovascularization of the anterior segment is the final common pathway for the conditions that commonly produce spontaneous hyphema in adults.

OK, then, what are the three sorts of common conditions that produce the neo that produces spontaneous hyphema in adults?
--Ischemia
--Neoplasm
--Inflammation
Speaking of common pathways in hyphema... Let's work backwards through the process that leads to free blood in the AC.

What event sets up the eye to get a hyphema?
Speaking of common pathways in hyphema... Let's work backwards through the process that leads to free blood in the AC.

*What event sets up the eye to get a hyphema?*

Neovascularization of the anterior segment
Speaking of common pathways in hyphema...Let's work backwards through the process that leads to free blood in the AC.

**What event sets up the eye to get a hyphema?**
Neovascularization of the anterior segment

**What incites the development of anterior segment neo?**
Spontaneous Hyphema

Speaking of common pathways in hyphema... Let's work backwards through the process that leads to free blood in the AC.

What event sets up the eye to get a hyphema?
Neovascularization of the anterior segment

What incites the development of anterior segment neo?
The presence of VEGF and other vascular growth factors
Spontaneous Hyphema

Speaking of common pathways in hyphema... Let's work backwards through the process that leads to free blood in the AC.

What event sets up the eye to get a hyphema?
Neovascularization of the anterior segment

What incites the development of anterior segment neo?
The presence of VEGF and other vascular growth factors

What event leads to the release of VEGF, etc?

Adults
- Ischemia
- Neoplasm
- Inflammation

Children
- Release of VEGF, other vascular growth factors
- A.S. Neo
- HYPHEMA

Q
Speckled Spontaneous Hyphema

**Adults**
- Ischemia
- Neoplasm
- Inflammation

**Children**
- Hypoxia
- Release of VEGF, other vascular growth factors
- A.S. Neo
- HYPHEMA

Speaking of common pathways in hyphema... Let's work backwards through the process that leads to free blood in the AC.

*What event sets up the eye to get a hyphema?*
Neovascularization of the anterior segment

*What incites the development of anterior segment neo?*
The presence of VEGF and other vascular growth factors

*What event leads to the release of VEGF, etc?*
Hypoxia
Broadly speaking, ischemia of one of two ‘structures’ (note the ‘scare quotes’) is responsible for producing anterior segment neo. What are these structures?
Spontaneous *Hyphema*

**Adults**

- Ischemia
- Neoplasm
- Inflammation

**Children**

- Hypoxia
- Release of VEGF, other vascular growth factors
- A.S. Neo
- HYPHEMA

Broadly speaking, ischemia of one of two ‘structures’ (note the ‘scare quotes’) is responsible for producing anterior segment neo. What are these structures? The Retina, and the Entire Eye
Broadly speaking, ischemia of one of two ‘structures’ (note the ‘scare quotes’) is responsible for producing anterior segment neo. What are these structures?

The Retina and the Entire Eye

What retinal ischemic events are notorious for producing anterior segment neo?
Broadly speaking, ischemia of one of two ‘structures’ (note the ‘scare quotes’) is responsible for producing anterior segment neo.
What are these structures?
The Retina, and the Entire Eye

What retinal ischemic events are notorious for producing anterior segment neo?
Proliferative diabetic retinopathy (PDR), and retinal vein occlusions—either central (CRVO) or branch (BRVO)
Broadly speaking, ischemia of one of two ‘structures’ (note the ‘scare quotes’) is responsible for producing anterior segment neo. What are these structures? The Retina, and the Entire Eye.

What is the name of the condition in which the entire eye is ischemic?
Spontaneous Hyphema

Adults
- Ischemia
- Neoplasm
- Inflammation

Children
- Hypoxia
- Release of VEGF, other vascular growth factors
- A.S. Neo
- HYPHEMA

Broadly speaking, ischemia of one of two ‘structures’ (note the ‘scare quotes’) is responsible for producing anterior segment neo. What are these structures?

The Retina, and the

Entire Eye

What is the name of the condition in which the entire eye is ischemic?

Ocular ischemic syndrome (OIS)
Broadly speaking, ischemia of one of two ‘structures’ (note the ‘scare quotes’) is responsible for producing anterior segment neo.

What are these structures?
The Retina, and the Entire Eye

What is the name of the condition in which the entire eye is ischemic?
Ocular ischemic syndrome (OIS)

In OIS, where in the vascular tree is the occlusion found?
Broadly speaking, ischemia of one of two ‘structures’ (note the ‘scare quotes’) is responsible for producing anterior segment neo. What are these structures?
The Retina, and the Entire Eye

What is the name of the condition in which the entire eye is ischemic?
Ocular ischemic syndrome (OIS)

In OIS, where in the vascular tree is the occlusion found?
The ipsilateral carotid
OK, I get how ischemia leads to hypoxia, but how does a neoplasm?
OK, I get how ischemia leads to hypoxia, but how does a neoplasm? Some neoplasms grow so rapidly that they end up outgrowing their blood supply. Tumor cells located at the fringe of the available blood supply may be hypoxic, which leads to their production and release of pro-vascular growth factors resulting in neo, and hyphema.
OK, I get how ischemia leads to hypoxia, but how does a neoplasm? Some neoplasms grow so rapidly that they end up outgrowing their blood supply. Tumor cells located at the fringe of the available blood supply may be hypoxic, which leads to their production and release of pro-vascular growth factors resulting in neo, and hyphema.

Which neoplasm is perhaps best known to be associated with hyphema in adults?
OK, I get how ischemia leads to hypoxia, but how does a neoplasm? Some neoplasms grow so rapidly that they end up outgrowing their blood supply. Tumor cells located at the fringe of the available blood supply may be hypoxic, which leads to their production and release of pro-vascular growth factors resulting in neo, and hyphema.

Which neoplasm is perhaps best known to be associated with hyphema in adults?
Uveal melanoma
How does inflammation lead to neo, and then hyphema?
How does inflammation lead to neo, and then hyphema?
This has yet to be fully elucidated, and the BCSC books do not address the ‘mechanism’ question
**Spontaneous Hyphema**

### Adults
- Ischemia
- Neoplasm
- Inflammation

### Children

Hypoxia → Release of VEGF, other vascular growth factors → A.S. Neo → HYPHEMA

**How does inflammation lead to neo, and then hyphema?**
This has yet to be fully elucidated, and the BCSC books do not address the ‘mechanism’ question

**Which forms of uveitis are well-known to be associated with hyphema in adults?**

--

--
How does inflammation lead to neo, and then hyphema?
This has yet to be fully elucidated, and the BCSC books do not address the ‘mechanism’ question

Which forms of uveitis are well-known to be associated with hyphema in adults?
--Herpetic uveitides
--Fuch heterochromic iridocyclitis (FHI)
Spontaneous Hyphema

How does inflammation lead to neo, and then hyphema? This has yet to be fully elucidated, and the BCSC books do not address the ‘mechanism’ question.

Which forms of uveitis are well-known to be associated with hyphema in adults?
- Herpetic uveitides
- Fuch heterochromic iridocyclitis (FHI)

Which herpetic uveitides are especially notorious for hyphema?
Spontaneous Hyphema

How does inflammation lead to neo, and then hyphema? This has yet to be fully elucidated, and the BCSC books do not address the ‘mechanism’ question.

Which forms of uveitis are well-known to be associated with hyphema in adults?

- Herpetic uveitides
- Fuch heterochromic iridocyclitis (FHI)

Which herpetic uveitides are especially notorious for hyphema?
- VZV
- HSV
Spontaneous Hyphema

How does inflammation lead to neo, and then hyphema? This has yet to be fully elucidated, and the BCSC books do not address the ‘mechanism’ question.

Which forms of uveitis are well-known to be associated with hyphema in adults?

--Herpetic uveitides
--Fuch heterochromic iridocyclitis (FHI)

Which herpetic uveitides are especially notorious for hyphema?

--VZV: Iris atrophy is…?
--HSV: Iris atrophy is…?

VZV and HSV both produce iris atrophy. In what key way do they differ in this regard?

1. Ischemia
2. Neoplasm
3. Inflammation
**Spontaneous Hyphema**

### Adults
- Ischemia
- Neoplasm
- Inflammation

### Children
- Hypoxia
- Release of VEGF, other vascular growth factors
- A.S. Neo
- HYPHEMA

**How does inflammation lead to neo, and then hyphema?**
This has yet to be fully elucidated, and the BCSC books do not address the ‘mechanism’ question.

**Which forms of uveitis are well-known to be associated with hyphema in adults?**
- Herpetic uveitides
- Fuch heterochromic iridocyclitis (FHI)

**Which herpetic uveitides are especially notorious for hyphema?**
- VZV: Iris atrophy is... **sectoral**
- HSV: Iris atrophy is... **diffuse**

VZV and HSV both produce iris atrophy. In what key way do they differ in this regard?
In VZV, the atrophy is **sectoral**, whereas in HSV it is **diffuse**.
Who is the typical FHI pt?

---

**Fuch heterochromic iridocyclitis (FHI)**

---

Who is the typical FHI pt?

A middle-aged adult

Is there a gender predilection?

No

What exam findings comprise the 'classic triad' of FHI? (Hyphema is not one of them.)

Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain't it.)

It develops in about 25-50% of cases

What is the etiology of FHI?

It is uncertain at this time. Four infectious entities have been suggested:

--Toxoplasmosis
--HSV
--CMV
--Rubella

As of now, the preponderance of the evidence points to rubella, but it remains unproven.
Who is the typical FHI pt?
A middle-aged adult

--Fuch heterochromic iridocyclitis (FHI)
Q: Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
Spontaneous Hyphema

Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

--Herpetic uveitides

--Fuch heterochromic iridocyclitis (FHI)
Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)

--Herpetic uveitides
--Fuch heterochromic iridocyclitis (FHI)

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)

It develops in about 25-50% of cases

What is the etiology of FHI?
It is uncertain at this time. Four infectious entities have been suggested:
--Toxoplasmosis
--HSV
--CMV
--Rubella

As of now, the preponderance of the evidence points to rubella, but it remains unproven.
Spontaneous Hyphema

Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP
Spontaneous Hyphema

FHI: Heterochromia iridis, cataract, and stellate KP
Spontaneous Hyphema

Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
- **Heterochromia iridis**
- cataract
- stellate KP

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

--Herpetic uveitides
--Fuch heterochromic iridocyclitis (FHI)
Spontaneous Hyphema

Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the 'classic triad' of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

Is the affected eye the darker eye, or the lighter eye?
The lighter (most of the time; see U13 for details)

--Herpetic uveitides
--Fuch heterochromic iridocyclitis (FHI)
Spontaneous Hyphema
Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)

--Herpetic uveitides
--Fuch heterochromic iridocyclitis (FHI)
Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)
Glaucoma—it develops in about 25-50% of cases

--Fuch heterochromic iridocyclitis (FHI)
Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)
Glaucoma—it develops in about 25-50% of cases

What is the etiology of FHI?

--Herpetic uveitides
--Toxoplasmosis
--HSV
--CMV
--Rubella

As of now, the preponderance of the evidence points to rubella, but it remains unproven.
Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)
Glaucoma—it develops in about 25-50% of cases

What is the etiology of FHI?
It is uncertain at this time. Four infectious entities have been suggested:
--?
--?
--?
--?
Spontaneous Hyphema

Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)
Glaucoma—it develops in about 25-50% of cases

What is the etiology of FHI?
It is uncertain at this time. Four infectious entities have been suggested:
--Toxoplasmosis
--HSV
--CMV
--Rubella

--Herpetic uveitides

--Fuch heterochromic iridocyclitis (FHI)
Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)
Glaucoma—it develops in about 25-50% of cases

What is the etiology of FHI?
It is uncertain at this time. Four infectious entities have been suggested:
--Toxoplasmosis?
--HSV?
--CMV?
--Rubella?
As of now, the preponderance of the evidence points to Rubella, but it remains unproven.
How does inflammation lead to neo, and then hyphema? This has yet to be fully elucidated, and the BCSC books do not address the 'mechanism' question.

Which forms of uveitis are well-known to be associated with hyphema in adults?

- Herpetic uveitides
- Fuch heterochromic iridocyclitis (FHI)

Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)
**Glaucoma**—it develops in about 25-50% of cases

What is the etiology of FHI?
It is uncertain at this time. Four infectious entities have been suggested:

--Toxoplasmosis
--HSV
--CMV
--Rubella!
As of now, the preponderance of the evidence points to rubella, but it remains unproven.
Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)
It develops in about 25-50% of cases

How well does FHI respond to steroid therapy?
Poorly--AC cell is notoriously difficult to eradicate in FHI (as is the anterior vitreous cell which frequently occurs)

If/when the inflammation fails to respond to topical steroids, should more aggressive therapies be pursued?
Generally no. In fact, most pts require no anti-inflammatory tx of any sort (including steroids). Instead, the pt should be monitored for the development of glaucoma and cataract.
Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)
It develops in about 25-50% of cases

How well does FHI respond to steroid therapy?
Poorly--AC cell is notoriously difficult to eradicate in FHI (as is the anterior vitreous cell which frequently occurs)

Fuch heterochromic iridocyclitis (FHI)
Spontaneous Hyphema

Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)
It develops in about 25-50% of cases

How well does FHI respond to steroid therapy?
Poorly--AC cell is notoriously difficult to eradicate in FHI (as is the anterior vitreous cell which frequently occurs)

If/when the inflammation fails to respond to topical steroids, should more aggressive therapies be pursued?
Generally no. In fact, most pts require no anti-inflammatory tx of any sort (including steroids). Instead, the pt should be monitored for the development of glaucoma and cataract

--Herpetic uveitides

--Fuch heterochromic iridocyclitis (FHI)
Spontaneous Hyphema

Who is the typical FHI pt?
A middle-aged adult

Is there a gender predilection?
No

What exam findings comprise the ‘classic triad’ of FHI? (Hyphema is not one of them.)
Heterochromia iridis, cataract, and stellate KP

What other ophthalmic issue is common in FHI? (Again, hyphema ain’t it.)
It develops in about 25-50% of cases

How well does FHI respond to steroid therapy?
Poorly--AC cell is notoriously difficult to eradicate in FHI (as is the anterior vitreous cell which frequently occurs)

If/when the inflammation fails to respond to topical steroids, should more aggressive therapies be pursued?
Generally no. In fact, most pts require no anti-inflammatory tx of any sort (including steroids). Instead, the pt should be monitored for the development of glaucoma and cataract.

---Fuch heterochromic iridocyclitis (FHI)
What three conditions are most commonly associated with spontaneous hyphema in children?
What three conditions are most commonly associated with spontaneous hyphema in children?
Spontaneous Hyphema

Adults
- Ischemia
- Neoplasm
- Inflammation

Children
- JXG
- Rh

What does JXG stand for?

Juvenile xanthogranuloma
It is a nonneoplastic histiocytic proliferation
The majority before age 1 year, and almost all by age 2
As skin papules
Unilaterally
What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
Nonneoplastic histiocytic proliferation

At what age does JXG present?
The majority before age 1 year, and almost all by age 2

How does JXG usually present? (Hint: It's not ophthalmic)
As skin papules

When iris JXG nodules are present, is it uni-, or bilaterally?
Unilaterally
What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
It is a nonneoplastic histiocytic proliferation

Children

JXG

Leukemia

Inflammation

Neoplasm

Ischemia

Adults
What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
It is a **nonneoplastic histiocytic proliferation**
Spontaneous Hyphema

Adults
- Ischemia
- Neoplasm
- Inflammation

Children
- JXG

What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
It is a nonneoplastic histiocytic proliferation

At what age does JXG present?
The majority before age 1 year, and almost all by age 2

As skin papules
When iris JXG nodules are present, is it uni-, or bilaterally?
Unilaterally
What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
It is a **nonneoplastic histiocytic proliferation**

At what age does JXG present?
The majority before age 1 year, and almost all by age 2
What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
It is a nonneoplastic histiocytic proliferation

At what age does JXG present?
The majority before age 1 year, and almost all by age 2

How does JXG usually present? (Hint: It’s not ophthalmic)
What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
It is a nonneoplastic histiocytic proliferation

At what age does JXG present?
The majority before age 1 year, and almost all by age 2

How does JXG usually present? (Hint: It’s not ophthalmic)
As skin papules
JXG: Skin papules. The orangish color is classic
What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
It is a nonneoplastic histiocytic proliferation

At what age does JXG present?
The majority before age 1 year, and almost all by age 2

How does JXG usually present? (Hint: It’s not ophthalmic)
As skin papules

When iris JXG nodules are present, is it uni-, or bilaterally?
What does JXG stand for? Juvenile xanthogranuloma
In three words, what sort of condition is it? It is a nonneoplastic histiocytic proliferation
At what age does JXG present? The majority before age 1 year, and almost all by age 2
How does JXG usually present? (Hint: It’s not ophthalmic) As skin papules
When iris JXG nodules are present, is it uni-, or bilaterally? Unilaterally
JXG: Iris lesion
JXG: Spontaneous hyphema
What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
Nonneoplastic histiocytic proliferation

At what age does JXG present?
The majority before age 1 year, and almost all by age 2

How does JXG usually present? (Hint: It’s not ophthalmic)
As skin papules

When iris JXG nodules are present, is it uni-, or bilaterally?
Unilaterally

In addition to spontaneous hyphema, in what three ways are JXG iris nodules clinically significant?
- They are in the DDx as a ‘masquerade syndrome’ in pediatric uveitis
- If enough are present, they will cause heterochromia iridis
- They can result in severe glaucoma

Should they be removed surgically?
Only if the glaucoma is uncontrollable

What is the natural history of the disease?
JXG is self-limited, usually resolving by age 5 years
What does JXG stand for?
Juvenile xanthogranuloma
In three words, what sort of condition is it?
Nonneoplastic histiocytic proliferation
At what age does JXG present?
The majority before age 1 year, and almost all by age 2
How does JXG usually present? (Hint: It’s not ophthalmic)
As skin papules
When iris JXG nodules are present, is it uni-, or bilaterally?
Unilaterally
In addition to spontaneous hyphema, in what three ways are JXG iris nodules clinically significant?
--They are in the DDx as a ‘masquerade syndrome’ in peds uveitis
--If enough are present, they will cause heterochromia iridis
--They can result in severe glaucoma
Should they be removed surgically?
Only if the glaucoma is uncontrollable
What is the natural history of the disease?
JXG is self-limited, usually resolving by age 5 years
Spontaneous Hyphema

Adults
- Ischemia
- Neoplasm
- Inflammation

Children
- JXG
- Rb

What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
Nonneoplastic histiocytic proliferation

At what age does JXG present?
The majority before age 1 year, and almost all by age 2

How does JXG usually present? (Hint: It’s not ophthalmic)
As skin papules

When iris JXG nodules are present, is it uni-, or bilaterally?
Unilaterally

In addition to spontaneous hyphema, in what three ways are JXG iris nodules clinically significant?
-- They are in the DDx as a ‘masquerade syndrome’ in peds uveitis
-- If enough are present, they will cause heterochromia iridis
-- They can result in severe glaucoma

Should they be removed surgically?
Only if the glaucoma is uncontrollable

What is the natural history of the disease?
JXG is self-limited, usually resolving by age 5 years
What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
Nonneoplastic histiocytic proliferation

At what age does JXG present?
The majority before age 1 year, and almost all by age 2

How does JXG usually present? (Hint: It's not ophthalmic)
As skin papules

When iris JXG nodules are present, is it uni-, or bilaterally?
Unilaterally

In addition to spontaneous hyphema, in what three ways are JXG iris nodules clinically significant?
--They are in the DDx as a ‘masquerade syndrome’ in peds uveitis
--If enough are present, they will cause heterochromia iridis
--They can result in severe glaucoma

Should they be removed surgically?
Only if the glaucoma is uncontrollable

What is the natural history of the disease?
JXG is self-limited, usually resolving by age 5 years
In addition to spontaneous hyphema, in what three ways are JXG iris nodules clinically significant?
--They are in the DDx as a ‘masquerade syndrome’ in peds uveitis
--If enough are present, they will cause heterochromia iridis
--They can result in severe glaucoma

Should they be removed surgically?
Only if the glaucoma is uncontrollable

What is the natural history of the disease?
What does JXG stand for?

Juvenile xanthogranuloma

In three words, what sort of condition is it?
Nonneoplastic histiocytic proliferation

At what age does JXG present?
The majority before age 1 year, and almost all by age 2

How does JXG usually present? (Hint: It's not ophthalmic)
As skin papules

When iris JXG nodules are present, is it uni-, or bilaterally?
Unilaterally

In addition to spontaneous hyphema, in what three ways are JXG iris nodules clinically significant?

--They are in the DDx as a ‘masquerade syndrome’ in peds uveitis
--If enough are present, they will cause heterochromia iridis
--They can result in severe glaucoma

Should they be removed surgically?
Only if the glaucoma is uncontrollable

What is the natural history of the disease?
JXG is self-limited, usually resolving by age 5 years
What does JXG stand for?
Juvenile xanthogranuloma

In three words, what sort of condition is it?
Nonneoplastic histiocytic proliferation

At what age does JXG present?
The majority before age 1 year, and almost all by age 2

How does JXG usually present? (Hint: It's not ophthalmic)
As skin papules

When iris JXG nodules are present, is it uni-, or bilaterally?
Unilaterally

In addition to spontaneous hyphema, in what three ways are JXG iris nodules clinically significant?
--They are in the DDx as a ‘masquerade syndrome’ in pediatric uveitis
--If enough are present, they will cause heterochromia iridis
--They can result in severe glaucoma

Should they be removed surgically?
Only if the glaucoma is uncontrollable

What is the natural history of the disease?
JXG is self-limited, usually resolving by age 5 years
Spontaneous Hyphema

Adults
- Ischemia
- Neoplasm
- Inflammation

Children
- JXG
- Rb
- Leukemia

What does Rb stand for in this context?
What does Rb stand for in this context?
Retinoblastoma
What does Rb stand for in this context?
Retinoblastoma

The BCSC doesn’t have too much to say about hyphema in Rb, save the following two points:
--It is [common vs uncommon]
Spontaneous Hyphema

Adults
- Ischemia
- Neoplasm
- Inflammation

Children
- JXG
- Rb
- Leukemia

What does Rb stand for in this context?
Retinoblastoma

The BCSC doesn’t have too much to say about hyphema in Rb, save the following two points:
--It is uncommon
What does Rb stand for in this context?
Retinoblastoma

The BCSC doesn’t have too much to say about hyphema in Rb, save the following two points:
--It is uncommon
--It is more likely to occur in children than 5 years of age
What does Rb stand for in this context?
Retinoblastoma

The BCSC doesn’t have too much to say about hyphema in Rb, save the following two points:
--It is uncommon
--It is more likely to occur in children < than 5 years of age
Spontaneous Hyphema

Adults
- Ischemia
- Neoplasm
- Inflammation

Children
- JXG
- Rb
- Leukemia

What does Rb stand for in this context?
Retinoblastoma

The BCSC doesn’t have too much to say about hyphema in Rb, save the following two points:
--It is uncommon
--It is more likely to occur in children < than 5 years of age

For more on Rb, see slide-set R2
Where does leukemia rank among childhood malignancies in terms of incidence?
Where does leukemia rank among childhood malignancies in terms of incidence? It is #1 by a mile.
Where does leukemia rank among childhood malignancies in terms of incidence? It is #1 by a mile.

Re its histology: Which form of leukemia is most common in children?
Where does leukemia rank among childhood malignancies in terms of incidence? It is #1 by a mile

Re its histology: Which form of leukemia is most common in children? Acute lymphocytic (ALL)
Where does leukemia rank among childhood malignancies in terms of incidence?
It is #1 by a mile

Re its histology: Which form of leukemia is most common in children?
Acute lymphocytic (ALL)

What other anterior-chamber condition is a well-known manifestation of leukemia?
Where does leukemia rank among childhood malignancies in terms of incidence?
It is #1 by a mile

Re its histology: Which form of leukemia is most common in children?
Acute lymphocytic (ALL)

What other anterior-chamber condition is a well-known manifestation of leukemia?
Pseudohypopyon
Where does leukemia rank among childhood malignancies in terms of incidence? It is #1 by a mile.

Re its histology: Which form of leukemia is most common in children? Acute lymphocytic (ALL)

What other anterior-chamber condition is a well-known manifestation of leukemia? Pseudohypopyon

For more on ophthalmic manifestations of pediatric leukemia, see slide-set P20