Ocular Hypotensives: List the common agents

- \( \beta \) blockers
  - Timolol
  - Betaxolol
  - Carteolol
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Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - The ‘big three’ FDA-approved PGA that dominate the American market
  - An FDA-approved PGA, much less well-known than the big three
  - A PGA ‘combo drug’
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
  - (Tafluprost) *(An FDA-approved PGA, much less well-known than the big three)*
  - (Latanaprostene bunod) *(A PGA ‘combo drug’)*
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost
  - Tafluprost (Latanaprostene bunod)

What is the brand name of tafluprost?
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
  - Tafluprost

*What is the brand name of tafluprost?* Zioptan (and that’s all we’ll have to say about it)
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
  - (Tafluprost) agonist

What is the brand name of latanaprostene bunod?
Ocular Hypotensives: List the common agents

- \( \beta \) blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
  - (Tafluprost) agonist
    - (Latanaprostene bunod)

The three FDA-approved PGA that dominate the American market

What is the brand name of latanaprostene bunod?
Vyzulta

(We’ll have more to say about this drug later)
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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- **Prostaglandin analogues**
  - Latanaprost
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- **Nonselective α/β agonist**
  - Epinephrine
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(This drove me nuts when I was a med student—how could the same disease be treated by two different medicines with the exact opposite effect? The first time I read it, I assumed it was a typo.)
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI (carbonic anhydrase inhibitors)**
Ocular Hypotensives: List the common agents

- \(\beta\) blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

- Nonselective \(\alpha/\beta\) agonist
  - Epinephrine
  - Dipivefrin

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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- **Prostaglandin analogues**
  - Latanaprost
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- **Nonselective α/β agonist**
  - Epinephrine
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- **CAI**
  - Dorzolamide
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There is another, less well-known CAI—what is it?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
  - **Methazolamide**

There is another, less well-known CAI—what is it?

Methazolamide
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
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- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
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  - Brinzolamide
  - Acetazolamide
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  - Apraclonidine
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  - Pilocarpine (Pilo for short)
Ocular Hypotensives: List the common agents

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- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil
Ocular Hypotensives: List the common agents

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<thead>
<tr>
<th>Category</th>
<th>Agents</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>β blockers</strong></td>
<td>Timolol, Betaxolol, Carteolol</td>
</tr>
<tr>
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</tr>
<tr>
<td><strong>Nonselective α/β agonist</strong></td>
<td>Epinephrine, Dipivefrin</td>
</tr>
<tr>
<td><strong>CAI</strong></td>
<td>Dorzolamide, Brinzolamide, Acetazolamide</td>
</tr>
<tr>
<td><strong>Selective α agonists</strong></td>
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<tr>
<td><strong>Miotics</strong></td>
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</tr>
<tr>
<td><strong>Rho kinase inhibitor</strong></td>
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</tr>
</tbody>
</table>

*These meds are rarely used anymore (except for pilo in certain situations), so we won’t bother with their brand names.*
### Ocular Hypotensives: List the common agents

<table>
<thead>
<tr>
<th>Category</th>
<th>Agents</th>
<th>Brand Name?</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>β blockers</strong></td>
<td>Timolol</td>
<td>Timoptic</td>
</tr>
<tr>
<td></td>
<td>Betaxolol</td>
<td>xxxxxxx</td>
</tr>
<tr>
<td></td>
<td>Carteolol</td>
<td></td>
</tr>
<tr>
<td><strong>Prostaglandin analogues</strong></td>
<td>Latanaprost</td>
<td>Xalatan</td>
</tr>
<tr>
<td></td>
<td>Travaprost</td>
<td>Travatan</td>
</tr>
<tr>
<td></td>
<td>Bimataprost</td>
<td>Lumigan</td>
</tr>
<tr>
<td><strong>Nonselective α/β agonist</strong></td>
<td>Epinephrine</td>
<td>xxxxxxx</td>
</tr>
<tr>
<td></td>
<td>Dipivefrin</td>
<td>xxxxxxx</td>
</tr>
<tr>
<td><strong>CAI</strong></td>
<td>Dorzolamide</td>
<td>Trusopt</td>
</tr>
<tr>
<td></td>
<td>Brinzolamide</td>
<td>Azopt</td>
</tr>
<tr>
<td></td>
<td>Acetazolamide</td>
<td>Diamox</td>
</tr>
<tr>
<td><strong>Selective α agonists</strong></td>
<td>Apraclonidine</td>
<td>Iopidine</td>
</tr>
<tr>
<td></td>
<td>Brimonidine</td>
<td>Alphagan</td>
</tr>
<tr>
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<td>Pilo</td>
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What is the name of the equation that describes the factors determining IOP?
Ocular Hypotensives: List the common agents

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The Goldmann equation
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What is the Goldmann equation? (Meaning, write it out)
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What is the name of the equation that describes the factors determining IOP?

The Goldmann equation

What is the Goldmann equation? (Meaning, write it out)

\[ \text{IOP} = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP} \]
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**What is the Goldmann equation? (Meaning, write it out)**

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IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
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Note:
1) EVP = write it out
2) In the interest of simplicity, I fudged a little on the denominator—technically, it’s outflow, not outflow rate
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**The Goldmann equation implies three means by which IOP can be lowered. What are they?**

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What is the name of the equation that describes the factors determining IOP?

The Goldmann equation

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The Goldmann equation implies three means by which IOP can be lowered. What are they?

--Decrease the rate of aqueous formation
--Increase the rate of aqueous outflow
--Decrease episcleral venous pressure
What is the name of the equation that describes the factors determining IOP?
The Goldmann equation

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The Goldmann equation implies three means by which IOP can be lowered. What are they?
--Decrease the rate of aqueous formation
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There is another commonly-employed means of decreasing IOP that is not implied by the Goldmann equation. What is it?
The Goldmann equation implies three means by which IOP can be lowered. What are they?
-- Decrease the rate of aqueous formation
-- Increase the rate of aqueous outflow
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Dehydration of the vitreous
Ocular Hypotensives: List the common agents

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  - Timolol
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- **Rho kinase inhibitor**
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What is the name of the equation that describes the factors determining IOP?

**The Goldmann equation**

Where, specifically, is aqueous formed?

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Dehydration of the vitreous
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- **Selective α agonists**
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- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
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---

What is the name of the equation that describes the factors determining IOP?

**The Goldmann equation**

Where, specifically, is aqueous formed?

In the nonpigmented epithelium of the pars plicata portion of the ciliary body

Where, specifically, is aqueous formed?

- In the nonpigmented epithelium of the pars plicata portion of the ciliary body

The Goldmann equation implies three means by which IOP can be lowered. What are they?

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Dehydration of the vitreous

What are the two types/pathways of aqueous outflow?

- Trabecular meshwork (TM)
- Uveoscleral (U/S)

One of these is referred to as conventional outflow; the other, unconventional. Which is which?

- TM = conventional
- U/S = unconventional

One outflow pathway is pressure dependent; the other, pressure independent. Which is which?

- TM = conventional = pressure-dependent
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Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
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  - Acetazolamide
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  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

What is the name of the equation that describes the factors determining IOP?

**The Goldmann equation**

What is the Goldmann equation? (Meaning, write it out)

\[ IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP} \]

Note:
1) EVP = episcleral venous pressure
2) In the interest of simplicity, I fudged a little on the denominator—technically, it's outflow facility, not outflow rate.

The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

There is another commonly-employed means of decreasing IOP that is not implied by the Goldmann equation. What is it?

Dehydration of the vitreous

What are the two types/pathways of aqueous outflow?

- Trabecular meshwork (TM)
- Uveoscleral (U/S)

One of these is referred to as **conventional** outflow; the other, **unconventional**. Which is which?

- TM = conventional
- U/S = unconventional
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By decreasing the rate of aqueous formation

By what mechanism do they reduce aqueous formation?

By inhibiting production of cAMP in the ciliary epithelium

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IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
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By inhibiting production of cAMP in the ciliary epithelium

By how much do they lower IOP?
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Of the three means implied by the Goldmann equation, how do **β blockers** lower IOP? (Note: It could be more than one)

By decreasing the rate of aqueous formation

By what mechanism do they reduce aqueous formation?

By inhibiting production of cAMP in the ciliary epithelium

By how much do they lower IOP?

20-30%
Ocular Hypotensives

- β blockers
  - Timolol
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The Goldmann equation implies three means by which IOP can be lowered. What are they?

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Of the three means implied by the Goldmann equation, how do PGAs lower IOP? (Note: It could be more than one)
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The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
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Of the three means implied by the Goldmann equation, how do PGAs lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow
Ocular Hypotensives

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The Goldmann equation implies three means by which IOP can be lowered. What are they?
--- Decrease the rate of aqueous formation
--- Increase the rate of aqueous outflow
--- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how do PGAs lower IOP? (Note: It could be more than one)
By increasing the rate of aqueous outflow

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?
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  - Timolol
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By increasing the rate of aqueous outflow

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?
Mainly via the U/S pathway
Ocular Hypotensives

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  - Timolol
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By increasing the rate of aqueous outflow

By what mechanism do they increase U/S outflow?
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It is unknown at this time
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**By what mechanism do they increase U/S outflow?**

It is unknown at this time

**By how much do they lower IOP?**
### Ocular Hypotensives

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<tr>
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By how much do they lower IOP?

25-33%
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*By what mechanism do they reduce aqueous formation?*

By inhibiting the enzyme carbonic anhydrase
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By what mechanism do they reduce aqueous formation?
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By how much do they lower IOP?
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By how much do they lower IOP?

15-20%
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  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how do **selective α agonists** lower IOP? (Note: It could be more than one)
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
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Of the three means implied by the Goldmann equation, how do selective α agonists lower IOP? (Note: It could be more than one)

Both meds decrease aqueous formation and increase outflow. Additionally, apraclonidine reduces EVP.

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IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
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Ocular Hypotensives

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By what mechanism do they reduce aqueous formation?
Ocular Hypotensives

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The Goldmann equation implies three means by which IOP can be lowered. What are they?

--- Decrease the rate of aqueous formation
--- Increase the rate of aqueous outflow
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Of the three means implied by the Goldmann equation, how do **selective α agonists** lower IOP? (Note: It could be more than one)

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By what mechanism do they reduce aqueous formation? This is not addressed in the BCSC.
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  - Timolol
  - Betaxolol
  - Carteolol

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Both meds decrease aqueous formation and increase outflow. Additionally, apraclonidine reduces EVP.

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This is not addressed in the BCSC

By what mechanism do they increase aqueous outflow?
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
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Both meds decrease aqueous formation and increase outflow. Additionally, apraclonidine reduces EVP.

By what mechanism do they reduce aqueous formation?
This is not addressed in the BCSC

By what mechanism do they increase aqueous outflow?
--Apraclonidine increases outflow
--Brimonidine increases outflow
Ocular Hypotensives

- \( \beta \) blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
- Nonselective \( \alpha/\beta \) agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
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  - Apraclonidine
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The Goldmann equation implies three means by which IOP can be lowered. What are they?

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-- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how do **selective \( \alpha \) agonists** lower IOP? (Note: It could be more than one)

Both meds decrease aqueous formation and increase outflow. Additionally, apraclonidine reduces EVP.

By what mechanism do they reduce aqueous formation?
This is not addressed in the BCSC

By what mechanism do they increase aqueous outflow?
-- Apraclonidine increases TM outflow
-- Brimonidine increases U/S outflow
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonists**
  - Epinephrine
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  - Dorzolamide
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The Goldmann equation implies three means by which IOP can be lowered. What are they?
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Of the three means implied by the Goldmann equation, how do selective α agonists lower IOP? (Note: It could be more than one)
Both meds decrease aqueous formation and increase outflow. Additionally, apraclonidine reduces EVP.

By what mechanism do they reduce aqueous formation?
This is not addressed in the BCSC

By what mechanism do they increase aqueous outflow?
- **Apraclonidine** increases TM outflow
- **Brimonidine** increases U/S outflow

Mnemonic for remembering their outflow pathways:
- **Apraclonidine**: ‘ATM’
- **Brimonidine**: ‘BUS’
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
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Of the three means implied by the Goldmann equation, how do selective α agonists lower IOP? (Note: It could be more than one)
Both meds decrease aqueous formation and increase outflow. Additionally, apraclonidine reduces EVP.

By what mechanism do they reduce aqueous formation?
This is not addressed in the BCSC

By what mechanism do they increase aqueous outflow?
- Apraclonidine increases TM outflow
- Brimonidine increases U/S outflow

By how much do they lower IOP?
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
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Both meds decrease aqueous formation and increase outflow. Additionally, apraclonidine reduces EVP.

By what mechanism do they reduce aqueous formation?
This is not addressed in the BCSC

By what mechanism do they increase aqueous outflow?

- Apraclonidine increases TM outflow
- Brimonidine increases U/S outflow

By how much do they lower IOP?
20-30%
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
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- Decrease the rate of aqueous formation
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By increasing the rate of aqueous outflow

**By what mechanism do they increase outflow?**
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- **Increase the rate of aqueous outflow**
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Of the three means implied by the Goldmann equation, how do **miotics** lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow

By what mechanism do they increase outflow?

They stimulate contraction of the **longitudinal** portion of the **ciliary** muscle.
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
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  - Latanaprost
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  - Acetazolamide

- **Selective $\alpha$ agonists**
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- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how do **miotics** lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow

By what mechanism do they increase outflow?

They stimulate contraction of the longitudinal portion of the ciliary muscle. These muscle fibers attach to the **scleral spur**.
Ocular Hypotensives

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  - Timolol
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  - Netarsudil

---

**The Goldmann equation implies three means by which IOP can be lowered. What are they?**

- Decrease the rate of aqueous formation
- *Increase the rate of aqueous outflow*
- Decrease episcleral venous pressure

**Of the three means implied by the Goldmann equation, how do miotics lower IOP? (Note: It could be more than one)**

By increasing the rate of aqueous outflow

**By what mechanism do they increase outflow?**

They stimulate contraction of the longitudinal portion of the ciliary muscle. These muscle fibers attach to the scleral spur. Tension on the scleral spur produces tightness in the trabecular meshwork, thereby allowing aqueous to egress more efficiently.
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  - Timolol
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---

**Goldmann Equation**

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IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
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*The Goldmann equation implies three means by which IOP can be lowered. What are they?*

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

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By increasing the rate of aqueous outflow

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The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- **Increase the rate of aqueous outflow**
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how do **miotics** lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow

By what mechanism do they increase outflow?
They stimulate contraction of the longitudinal portion of the ciliary muscle. These muscle fibers attach to the scleral spur. Tension on the scleral spur produces tightness in the trabecular meshwork, thereby allowing aqueous to egress more efficiently.

**tl;dr** They increase outflow through the TM pathway
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

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  - Latanaprost
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-- Decrease the rate of aqueous formation
-- **Increase the rate of aqueous outflow**
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By what mechanism do they increase outflow?
They stimulate contraction of the longitudinal portion of the ciliary muscle. These muscle fibers attach to the scleral spur. Tension on the scleral spur produces tightness in the trabecular meshwork, thereby allowing aqueous to egress more efficiently.

By how much do they lower IOP?
**Ocular Hypotensives**

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  - Timolol
  - Betaxolol
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- **CAI**
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  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - *Pilo*

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*The Goldmann equation implies three means by which IOP can be lowered. What are they?*

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how do **miotics** lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow

By what mechanism do they increase outflow? They stimulate contraction of the longitudinal portion of the ciliary muscle. These muscle fibers attach to the scleral spur. Tension on the scleral spur produces tightness in the trabecular meshwork, thereby allowing aqueous to egress more efficiently.

By how much do they lower IOP? 15-20%
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin
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  - Dorzolamide
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  - Apraclonidine
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  - Pilo
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The Goldmann equation implies three means by which IOP can be lowered. What are they?
--Decrease the rate of aqueous formation
--Increase the rate of aqueous outflow
--Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how do Rho kinase inhibitors lower IOP? (Note: It could be more than one)
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

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  - Apraclonidine
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- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

\[ IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP} \]

The Goldmann equation implies three means by which IOP can be lowered. What are they?

-- Decrease the rate of aqueous formation?
-- Increase the rate of aqueous outflow
-- Decrease episcleral venous pressure?

Of the three means implied by the Goldmann equation, how do Rho kinase inhibitors lower IOP? (Note: It could be more than one)

Primarily by increasing the rate of aqueous outflow (they may also reduce aqueous formation as well as decrease EVP, but these are thought to make minor contributions to their IOP-lowering effect)
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- Selective α agonists
  - Apraclonidine
  - Brimonidine

- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

\[ IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP} \]

The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation?
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure?

Of the three means implied by the Goldmann equation, how do Rho kinase inhibitors lower IOP? (Note: It could be more than one)
Primarily by increasing the rate of aqueous outflow (they may also reduce aqueous formation as well as decrease EVP, but these are thought to make minor contributions).

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?
**Ocular Hypotensives**

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

---

\[
\text{IOP} = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
\]

The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation?
- **Increase the rate of aqueous outflow**
- Decrease episcleral venous pressure?

Of the three means implied by the Goldmann equation, how do **Rho kinase inhibitors** lower IOP? (Note: It could be more than one)

Primarily by increasing the rate of **aqueous outflow** (they may also reduce aqueous formation as well as decrease EVP, but these are thought to make minor contributions).

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?

Mainly via the **TM pathway**
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- Selective α agonists
  - Apraclonidine
  - Brimonidine

- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

\[ \text{IOP} = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP} \]

The Goldmann equation implies three means by which IOP can be lowered. What are they?
-- Decrease the rate of aqueous formation?
-- Increase the rate of aqueous outflow
-- Decrease episcleral venous pressure?

Of the three means implied by the Goldmann equation, how do Rho kinase inhibitors lower IOP? (Note: It could be more than one)
Primarily by increasing the rate of aqueous outflow (they may also reduce aqueous formation as well as decrease EVP, but these are thought to make minor contributions to their IOP-lowering effect)

By what mechanism do they increase TM outflow?
Ocular Hypotensives

- \( \beta \) blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
- Nonselective \( \alpha/\beta \) agonists
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
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  - Apraclonidine
  - Brimonidine
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\[
\text{IOP} = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
\]

The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation?
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure?

Of the three means implied by the Goldmann equation, how do Rho kinase inhibitors lower IOP? (Note: It could be more than one)

Primarily by increasing the rate of aqueous outflow (they may also reduce aqueous formation as well as decrease EVP, but these are thought to make minor contributions to their IOP-lowering effect)

By what mechanism do they increase TM outflow?
By inducing relaxation of cytoskeletal elements found within TM cells
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
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  - Apraclonidine
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  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

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\[
\text{IOP} = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
\]

The Goldmann equation implies three means by which IOP can be lowered. What are they?
--Decrease the rate of aqueous formation?
--Increase the rate of aqueous outflow
--Decrease episcleral venous pressure?

Of the three means implied by the Goldmann equation, how do **Rho kinase inhibitors** lower IOP? (Note: It could be more than one)
Primarily by increasing the rate of aqueous outflow (they may also reduce aqueous formation as well as decrease EVP, but these are thought to make minor contributions to their IOP-lowering effect)

By what mechanism do they increase TM outflow?
By inducing relaxation of cytoskeletal elements found within TM cells

By how much do they lower IOP?
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
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  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

\[
IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
\]

*The Goldmann equation implies three means by which IOP can be lowered. What are they?*

-- Decrease the rate of aqueous formation?
-- **Increase the rate of aqueous outflow**
-- Decrease episcleral venous pressure?

*Of the three means implied by the Goldmann equation, how do Rho kinase inhibitors lower IOP? (Note: It could be more than one)*

Primarily by increasing the rate of aqueous outflow (they may also reduce aqueous formation as well as decrease EVP, but these are thought to make minor contributions to their IOP-lowering effect)

*By what mechanism do they increase TM outflow?*
By inducing relaxation of cytoskeletal elements found within TM cells

*By how much do they lower IOP?*
By my reading of the research, in the 20-25% range
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost
- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

The Goldmann equation implies three means by which IOP can be lowered. What are they?
-- Decrease the rate of aqueous formation
-- Increase the rate of aqueous outflow
-- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

\[
IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
\]
**Ocular Hypotensives**

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost
- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
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  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
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The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow
Ocular Hypotensives

- \( \beta \) blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprostene bunod
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  - Apraclonidine
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- Rho kinase inhibitor
  - Netarsudil

The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow.

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?

\[
\text{IOP} = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
\]
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprostene bunod
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- **CAI**
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  - Apraclonidine
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  - Netarsudil

The Goldmann equation implies three means by which IOP can be lowered. What are they?
-- Decrease the rate of aqueous formation
-- Increase the rate of aqueous outflow
-- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)
By increasing the rate of aqueous outflow

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway? Via both
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

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The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does **latanaprostene bunod** lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?

Via both

How does manage to affect both outflow pathways?
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
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\text{IOP} = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
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The Goldmann equation implies three means by which IOP can be lowered. What are they?
- Decrease the rate of aqueous formation
- **Increase the rate of aqueous outflow**
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does **latanaprostene bunod** lower IOP? (Note: It could be more than one)
By increasing the rate of

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?
Via **both**

How does manage to affect both outflow pathways?
The latanaprostene bunod molecule is cleaved into two moieties: and **nitric oxide**.
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost
- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
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  - Apraclonidine
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\text{IOP} = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
\]

The Goldmann equation implies three means by which IOP can be lowered. What are they?
- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?
Via both

How does manage to affect both outflow pathways?
The latanaprostene bunod molecule is cleaved into two moieties: latanaprost and nitric oxide (NO).
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

\[ \text{IOP} = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP} \]

The Goldmann equation implies three means by which IOP can be lowered. What are they?
-- Decrease the rate of aqueous formation
-- Increase the rate of aqueous outflow
-- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

**Latanaprost**

**Nitric oxide**

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway? Via both

How does manage to affect both outflow pathways?
The latanaprostene bunod molecule is cleaved into two moieties: latanaprost and nitric oxide (NO). In turn, these increase U/S and TM outflow, respectively.
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- Selective α agonists
  - Apraclonidine
  - Brimonidine

- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

\[ IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP} \]

The Goldmann equation implies three means by which IOP can be lowered. What are they?
- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

- By increasing the rate of aqueous outflow

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?
Via both

How does manage to affect both outflow pathways?
The latanaprostene bunod molecule is cleaved into two moieties: latanaprost and nitric oxide (NO). In turn, these increase U/S and TM outflow, respectively.
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

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The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?

Via both

How does manage to affect both outflow pathways?

The latanaprostene bunod molecule is cleaved into two moieties: **latanaprost** and **nitric oxide (NO)**. In turn, these increase **U/S** and **TM** outflow, respectively.

How do the constituent moieties accomplish their effects?

--Latanaprost:
  --Mechanism unknown (as noted previously)

--NO
  By inducing relaxation of cytoskeletal elements found within TM cells

**↑ TM outflow**

**↑ U/S outflow**
The Goldmann equation implies three means by which IOP can be lowered. What are they?

--Decrease the rate of aqueous formation
--Increase the rate of aqueous outflow
--Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?
Via both

How does manage to affect both outflow pathways?
The latanaprostene bunod molecule is cleaved into two moieties: latanaprost and nitric oxide (NO). In turn, these increase U/S and TM outflow, respectively.

How do the constituent moieties accomplish their effects?
--Latanaprost: Mechanism unknown (as noted previously)
--NO
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - **Latanaprostene bunod**
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

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\[
IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
\]

The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

Latanaprost and nitric oxide (NO)

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?

Via **both**

How does manage to affect both outflow pathways?

The latanaprostene bunod molecule is cleaved into two moieties: **latanaprost** and **nitric oxide (NO)**. In turn, these increase **U/S** and **TM** outflow, respectively.

How do the constituent moieties accomplish their effects?

**Latanaprost**: Mechanism unknown (as noted previously)

**NO**: **↑** TM outflow **↑** U/S outflow
Ocular Hypotensives:

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

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The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?

Via both

How does manage to affect both outflow pathways?

The latanaprostene bunod molecule is cleaved into two moieties: latanaprost and nitric oxide (NO). In turn, these increase U/S and TM outflow, respectively.

How do the constituent moieties accomplish their effects?

- **Latanaprost**: Mechanism unknown (as noted previously)
- **NO**: By inducing relaxation of cytoskeletal elements found within TM cells
Ocular Hypotensives:

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

\[ IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP} \]

The Goldmann equation implies three means by which IOP can be lowered. What are they?

-- Decrease the rate of aqueous formation
-- Increase the rate of aqueous outflow
-- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

Latanaprost and nitric oxide increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway? Via both.

“By inducing relaxation of cytoskeletal elements found within TM cells”...Where have I heard that before? (No cheating by looking back)

How do the constituent moieties accomplish their effects?

-- Latanaprost: Mechanism unknown (as noted previously)

-- **NO**: By inducing relaxation of cytoskeletal elements found within TM cells
**Q/A**

**Ocular Hypotensives**

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - **Latanaprostene bunod**
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

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**Goldmann equation**

\[ IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP} \]

The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- **Increase the rate of aqueous outflow**
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does **latanaprostene bunod** lower IOP? (Note: It could be more than one)

- By increasing the rate of aqueous outflow

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?

Via both

"By inducing relaxation of cytoskeletal elements found within TM cells"...Where have I heard that before? (No cheating by looking back)

This phrase was used to characterize the mechanism of action of the Rho kinase inhibitors.

Does this mean NO and RhoKIs have the same mechanism of action?

In one sense yes—they both interfere with the Rho signaling cascade that stiffens cytoskeletal elements. However, the two agents act at very different points in that signaling cascade.
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
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  - Apraclonidine
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- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

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The Goldmann equation implies three means by which IOP can be lowered. What are they?

-- Decrease the rate of aqueous formation
-- Increase the rate of aqueous outflow
-- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?

Via both

"By inducing relaxation of cytoskeletal elements found within TM cells"...Where have I heard that before? (No cheating by looking back)

This phrase was used to characterize the mechanism of action of the Rho kinase inhibitors

How do the constituent moieties accomplish their effects?

-- Latanaprost: Mechanism unknown (as noted previously)
-- NO: By inducing relaxation of cytoskeletal elements found within TM cells

---

\[ IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP} \]
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - **Latanaprostene bunod**
  - Travaprost
  - Bimataprost
  - **Nonselective α/β agonist**
    - Epinephrine
    - Dipivefrin
  - **CAI**
    - Dorzolamide
    - Brinzolamide
    - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

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  - **Miotics**
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---

The Goldmann equation implies three means by which IOP can be lowered. What are they?

- Decrease the rate of aqueous formation
- **Increase the rate of aqueous outflow**
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does latanaprostene bunod lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow

Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?

Via both

“By inducing relaxation of cytoskeletal elements found within TM cells”…Where have I heard that before? (No cheating by looking back)

This phrase was used to characterize the mechanism of action of the Rho kinase inhibitors

Does this mean NO and RhoKIs have the same mechanism of action?

How do the constituent moieties accomplish their effects?

- Latanaprost: Mechanism unknown (as noted previously)
- **NO**: By inducing relaxation of cytoskeletal elements found within TM cells
**Ocular Hypotensives**

- **β blockers**
  - Timolol
  - Betaxolol
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  - Latanaprostene bunod
  - Travaprost
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This phrase was used to characterize the mechanism of action of the Rho kinase inhibitors.

Does this mean NO and RhoKIs have the same mechanism of action?

In one sense yes—they both interfere with the Rho signaling cascade that stiffens cytoskeletal elements. However, the two agents act at very different points in that signaling cascade.
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

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Via both

By how much does latanaprostene bunod lower IOP?

By about 1 mmHg

\[
IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}
\]
Ocular Hypotensives

- $\beta$ blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprostene bunod
  - Travaprost
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  - Nonselective $\alpha/\beta$ agonist
    - Epinephrine
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  - Dorzolamide
  - Brinzolamide
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Does it increase outflow via the conventional (TM) pathway, or the unconventional (U/S) pathway?
Via both

By how much does latanaprostene bunod lower IOP?
That’s not the right question. The right question is, by how much more does it lower IOP compared to latanaprost alone?

By about 1 mmHg

Latanaprost: Mechanism unknown (as noted previously)

NO: By inducing relaxation of cytoskeletal elements found within TM cells
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

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\[
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*The Goldmann equation implies three means by which IOP can be lowered. What are they?*

---

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
- Decrease episcleral venous pressure

Of the three means implied by the Goldmann equation, how does **latanaprostene bunod** lower IOP? (Note: It could be more than one)

### Latanaprost vs. Nitric Oxide

- **Latanaprost**
  - U/S outflow
  - TM outflow
- **Nitric oxide**
  - U/S outflow
  - TM outflow

By how much does latanaprostene bunod lower IOP? That’s not the right question. The right question is, by how much more does it lower IOP compared to latanaprost alone?

OK then, by how much more does it lower IOP compared to latanaprost alone?

- Latanaprost: Mechanism unknown (as noted previously)
- **NO**: By inducing relaxation of cytoskeletal elements found within TM cells
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

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  - Latanaprostene bunod
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By how much does latanaprostene bunod lower IOP? That’s not the right question. The right question is, by how much more does it lower IOP compared to latanaprost alone?

OK then, by how much more does it lower IOP compared to latanaprost alone?

By about 1 mmHg

Latanaprost: Mechanism unknown (as noted previously)

NO: By inducing relaxation of cytoskeletal elements found within TM cells
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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  - Netarsudil

Rank these four commonly-used drug classes in terms of their IOP-lowering efficacy:

1) 
2) 
3) 
4)

(Rank the topical formulations)
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
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1) PGAs
2) Beta blockers
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4) CAIs

Give two reasons the PGAs beat the β blockers:
1) 
2)
Ocular Hypotensives: List the common agents

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Give two reasons the PGAs beat the β blockers:
1) Slightly better IOP reduction on average
2) Better 24° IOP control (β blocker efficacy drops during ‘acxtivity’).
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2) Better 24-hour IOP control (β blocker efficacy drops during sleep)
**Ocular Hypotensives: List the common agents**

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1) Slightly better IOP reduction on average
2) Better 24<sup>o</sup> IOP control (β blocker efficacy drops during sleep)

This is why the second dose should be instilled a number of hours before bedtime!
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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Rank these four commonly-used drug classes in terms of their IOP-lowering efficacy:

1) PGAs
2) **Beta blockers**
3) **Selective α agonists**
4) CAI

OK, but why are the β blockers ranked ahead of the selective α agonists? As I recall, both reduce IOP in the 20-30% range.

It's true, their efficacies are equal—at peak. However, the β blockers produce slightly better IOPs at trough, so they win.
Ocular Hypotensives: List the common agents

- **β blockers**
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Ocular Hypot ensives: List the common agents

Prostaglandin analogues

Latanaprost

Travaprost

Bimataprost

Nonselective α/β agonist

Epinephrine

Dipivefrin

CAI

Dorzolamide

Brinzolamide

Acetazolamide

Selective α agonists

Apraclonidine

Brimonidine

Miotics

Pilo

Rho kinase inhibitor

Netarsudil

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1) PGAs

2) β blockers

3) Selective α agonists

4) CAIs

Rank these four commonly-used drug classes in terms of their IOP-lowering efficacy:

1) PGAs

2) β blockers

3) Selective α agonists

4) CAIs
Ocular Hypotensives: List the common agents.

Some drugs are dispensed as fixed-combination meds. The drugs/classes involved are:

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost

- **Nonselective α/β agonist**
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  - Acetazolamide

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- **Miotics**
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  - Latanaprost
  - Travaprost
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- **Nonselective $\alpha/\beta$ agonist**
  - Epinephrine
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- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective $\alpha$ agonists**
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  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

---

Timolol

Brimonidine

CAI
(as Brinzolamide)  (as Dorzolamide)

Latanaprost

Netarsudil
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide (as Brinzolamide)
  - Brinzolamide (as Dorzolamide)
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

What is the brand name of the Timolol/Dorzolamide combo drop?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
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- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

What is the brand name of the Timolol/Dorzolamide combo drop?

Cosopt

Timolol
**Ocular Hypotensives: List the common agents**

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective $\alpha/\beta$ agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective $\alpha$ agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

What is the brand name of the Brimonidine/Timolol combo drop?
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- Selective α agonists
  - Apraclonidine
  - Brimonidine

- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

What is the brand name of the Brimonidine/Timolol combo drop? Combigan
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - **Brinzolamide**
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - **Brimonidine**

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

What is the brand name of the Brimonidine/Brinzolamide combo drop?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

What is the brand name of the Latanaprost/Netarsudil combo drop?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - **Latanaprost**
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

What is the brand name of the Latanaprost/Netarsudil combo drop? **Rocklatan**
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

Give five advantages combo drugs provide over simply using the same meds as separate drops.

1) 
2) 
3) 
4) 
5)
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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- **Prostaglandin analogues**
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**Give five advantages combo drugs provide over simply using the same meds as separate drops.**
1) Convenience
2) Costs less (usually)
3) By halving the number of drops, the preservative-load the ocular surface must endure is halved as well, thus making irritation less of an issue
4) Improved compliance
5) Eliminates washout (ie, when an impatient pt instills their second drop too soon after the first, thereby washing it out)
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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- **Prostaglandin analogues**
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  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - **Netarsudil**

What is the standard dosing frequency for latanaprost? Daily

What is the preferred/recommended time to take latanaprost? Bedtime

What is the standard dosing frequency for netarsudil? Daily

What is the preferred/recommended time to take netarsudil? Bedtime

It all checks out….
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

What is the standard dosing frequency for latanaprost?
Daily

What is the preferred/recommended time to take latanaprost?
Bedtime

What is the preferred/recommended time to take netarsudil?
Bedtime

It all checks out….
Rocklatan
Netarsudil
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

What is the standard dosing frequency for latanaprost?
Daily

What is the standard dosing frequency for netarsudil?
Daily

It all checks out….
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - **Latanaprost**
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonists**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - **Netarsudil**

**What is the standard dosing frequency for latanaprost?**
Daily

**What is the standard dosing frequency for netarsudil?**
Daily

Latanaprost  Rocklatan  Netarsudil
Q

β blockers
- Timolol
- Betaxolol
- Carteolol

Prostaglandin analogues
- **Latanaprost**
- Travaprost
- Bimataprost

Nonselective α/β agonist
- Epinephrine
- Dipivefrin

CAI
- Dorzolamide
- Brinzolamide
- Acetazolamide

Selective α agonists
- Apraclonidine
- Brimonidine

Miotics
- Pilo

Rho kinase inhibitor
- **Netarsudil**

Ocular Hypotensives: List the common agents

What is the standard dosing frequency for latanaprost?
Daily

What is the standard dosing frequency for netarsudil?
Daily

What is the preferred/recommended time to take latanaprost?
Bedtime

What is the preferred/recommended time to take netarsudil?
Bedtime

It all checks out….

Rocklatan

Latanaprost

Netarsudil
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - **Latanaprost**
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - **Netarsudil**

**What is the standard dosing frequency for latanaprost?**
Daily

**What is the standard dosing frequency for netarsudil?**
Daily

**What is the preferred/recommended time to take latanaprost?**
Bedtime
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - **Latanaprost**
  - Travaprost
  - Bimataprost
- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - **Netarsudil**

**Questions**

- What is the standard dosing frequency for latanaprost? Daily
- What is the standard dosing frequency for netarsudil? Daily
- What is the preferred/recommended time to take latanaprost? Bedtime
- What is the preferred/recommended time to take netarsudil? Bedtime

Latanaprost  Rocklatan  **Netarsudil**
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - **Latanaprost**
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - **Netarsudil**

What is the standard dosing frequency for latanaprost? Daily

What is the standard dosing frequency for netarsudil? Daily

What is the preferred/recommended time to take latanaprost? Bedtime

What is the preferred/recommended time to take netarsudil? Bedtime

Latanaprost  Rocklatan  Netarsudil
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travoprost
  - Bimataprost

- Nonselective α/β agonist
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  - Brinzolamide
  - Acetazolamide

- Selective α agonists
  - Apraclonidine
  - Brimonidine

- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

What is the standard dosing frequency for latanaprost?
Daily

What is the standard dosing frequency for netarsudil?
Daily

What is the preferred/recommended time to take latanaprost?
Bedtime

What is the preferred/recommended time to take netarsudil?
Bedtime

It all checks out….
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
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  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

*Which is the only agent FDA-approved for prophylaxing against post-procedure IOP spikes?*
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
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  - Dorzolamide
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  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

Which is the only agent FDA-approved for prophylaxing against post-procedure IOP spikes?

Iopidine
Ocular Hypotensives: List the common agents

- **$\beta$ blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective $\alpha/\beta$ agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective $\alpha$ agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

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Which is the only agent FDA-approved for prophylaxing against post-procedure IOP spikes? **Iopidine**

Iopidine works well for this indication, with one exception--in those pts already on a particular hypotensive drop for glaucoma. So if a pt is already on the drop in question, don’t bother with the pre-procedure Iopidine, as it’s not going to work. Which drop are we talking about?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - **Brimonidine**
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

Which is the only agent FDA-approved for prophylaxing against post-procedure IOP spikes? **Iopidine**

Iopidine works well for this indication, with one exception--in those pts already on a particular hypotensive drop for glaucoma. So if a pt is already on the drop in question, don’t bother with the pre-procedure Iopidine, as it’s not going to work. Which drop are we talking about? **Brimonidine**
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

Which is the only agent FDA-approved for prophylaxing against post-procedure IOP spikes? **Iopidine**

Iopidine works well for this indication, with one exception--in those pts already on a particular hypotensive drop for glaucoma. **So if a pt is already on the drop in question, don't bother with the post-procedure Iopidine, as it's not going to work.** Which drop are we talking about? **Brimonidine**

So if a pt is on brimonidine, what drop **should** you use to blunt a post-procedure IOP spike?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

Which is the only agent FDA-approved for prophylaxing against post-procedure IOP spikes? **Iopidine**

Iopidine works well for this indication, with one exception--in those pts already on a particular hypotensive drop for glaucoma. **So if a pt is already on the drop in question, don’t bother with the post-procedure Iopidine, as it’s not going to work.** Which drop are we talking about? **Brimonidine**

So if a pt is on brimonidine, what drop **should** you use to blunt a post-procedure IOP spike? **Pilo**
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

Speaking of pilo--besides prophylaxing IOP spikes in pts on brimonidine, in what other situations is it useful?
1) 
2)
**Ocular Hypotensives: List the common agents**

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

Speaking of pilo--besides prophylaxing IOP spikes in pts on brimonidine, in what other situations is it useful?

1) Managing angle closure
2) Deepening the angle in plateau-iris syndrome
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

 Speaking of pilo--besides prophylaxing IOP spikes in pts on brimonidine, in what other situations is it useful?

1) Managing angle closure
2) Deepening the angle in plateau-iris syndrome

What is the feared side effect of pilo?
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

Speaking of pilo--besides prophylaxing IOP spikes in pts on brimonidine, in what other situations is it useful?
1) Managing angle closure
2) Deepening the angle in plateau-iris syndrome

What is the feared side effect of pilo?
Retinal tears
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

Speaking of pilo--besides prophylaxing IOP spikes in pts on brimonidine, in what other situations is it useful?

1) Managing angle closure
2) Deepening the angle in plateau-iris syndrome

What is the feared side effect of pilo?

Retinal tears

Because of its association with retinal tears, what should be done prior to initiation of (non-emergent) pilo therapy?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

### Speaking of pilo—besides prophylaxing IOP spikes in pts on brimonidine, in what other situations is it useful?

1. Managing angle closure
2. Deepening the angle in plateau-iris syndrome

### What is the feared side effect of pilo?

Retinal tears

### Because of its association with retinal tears, what should be done prior to initiation of (non-emergent) pilo therapy?

A careful retina evaluation
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

In the present context, how many subtypes of α receptors are we concerned about?
### Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

In the present context, how many subtypes of α receptors are we concerned about? Two
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

In the present context, how many subtypes of α receptors are we concerned about?
Two

What are these two α receptor subtypes called?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
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- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

In the present context, how many subtypes of α receptors are we concerned about?
Two

What are these two α receptor subtypes called?
They are called α₁ and α₂
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
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  - Dorzolamide
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  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

---

**With respect to the eyes, what does activation of each subtype produce?**

- \( \alpha_1 \):
  - --?
  - --?

- \( \alpha_2 \):
  - --?

**What are these two α receptor subtypes called?**

They are called \( \alpha_1 \) and \( \alpha_2 \)
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonists**
  - Epinephrine
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- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

With respect to the eyes, what does activation of each subtype produce?

**α₁**:
- Vasoconstriction
- Pupil
- Eyelid

**α₂**:
- ?

What are these two α receptor subtypes called?
They are called α₁ and α₂
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

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  - Latanaprost
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  - Acetazolamide

- Selective α agonists
  - Apraclonidine
  - Brimonidine

- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

With respect to the eyes, what does activation of each subtype produce?

α₁:
-- Vasoconstriction
-- Pupil mydriasis
-- Eyelid retraction

α₂:
--?

What are these two α receptor subtypes called? They are called α₁ and α₂
Ocular Hypotensives: List the common agents

- \( \beta \) blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
- Nonselective \( \alpha/\beta \) agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective \( \alpha \) agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

With respect to the eyes, what does activation of each subtype produce?

\( \alpha_1 \):
- Vasoconstriction
- Pupil mydriasis
- Eyelid retraction

\( \alpha_2 \):
- Reduced IOP

What are these two \( \alpha \) receptor subtypes called? They are called \( \alpha_1 \) and \( \alpha_2 \)
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
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  - Brimonidine

- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

---

With respect to the eyes, what does activation of each subtype produce?

- $\alpha_1$:
  - Vasoconstriction
  - Pupil mydriasis
  - Eyelid retraction

- $\alpha_2$:
  - Reduced IOP

What are these two α receptor subtypes called? They are called $\alpha_1$ and $\alpha_2$. 
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

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  - Acetazolamide

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  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

Note: The *Glaucoma* book states that neuroprotection is another ‘possible’ effect of α_2_ stimulation.

What are these two α receptor subtypes called? They are called α_1_ and α_2_.

With respect to the eyes, what does activation of each subtype produce?

α_1_:
- Vasoconstriction
- Pupil mydriasis
- Eyelid retraction

α_2_:
- Reduced IOP
- Neuroprotection?

---

In the present context, how many subtypes of α receptors are we concerned about?

Two

They are called α_1_ and α_2.

What are these two α receptor subtypes called?

They are called α_1_ and α_2.

*Note: The Glaucoma book states that neuroprotection is another ‘possible’ effect of α_2_ stimulation. That said, it doesn’t elaborate on this claim, or explain what is meant by ‘neuroprotection’ (in fact, the term doesn’t even appear in the index)*
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
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- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

Note: The *Glaucoma* book states that neuroprotection is another ‘possible’ effect of α₂ stimulation. That said, it doesn’t elaborate on this claim, or explain what is meant by ‘neuroprotection’ (in fact, the term doesn’t even appear in the index).

What are these two α receptor subtypes called? They are called α₁ and α₂.
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

In the present context, how many subtypes of α receptors are we concerned about? Two

What are these two α receptor subtypes called? They are called α₁ and α₂

What does it mean to say the selective α agonists are selective? What are they ‘selecting’?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
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- **CAI**
  - Dorzolamide
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**In the present context, how many subtypes of α receptors are we concerned about?**

Two

**What are these two α receptor subtypes called?**

They are called α₁ and α₂

**What does it mean to say the selective α agonists are selective? What are they ‘selecting’?**

It means they preferentially stimulate α₂ receptors more than α₁
In the present context, how many subtypes of $\alpha$ receptors are we concerned about?

Two

What are these two $\alpha$ receptor subtypes called?

They are called $\alpha_1$ and $\alpha_2$

What does it mean to say the selective $\alpha$ agonists are selective? What are they ‘selecting’?

It means they preferentially stimulate $\alpha_2$ receptors more than $\alpha_1$

One agent is significantly more $\alpha_2$-selective than the other (it is often described as a ‘highly selective $\alpha$ agonist’). Which is it?
In the present context, how many subtypes of $\alpha$ receptors are we concerned about?

Two

What are these two $\alpha$ receptor subtypes called?

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One agent is significantly more $\alpha_2$-selective than the other (it is often described as a ‘highly selective $\alpha$ agonist’). Which is it?

Brimonidine
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
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- **Selective α agonists**
  - Apraclonidine
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- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

*Which agent is notoriously allergenic?*
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
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- **Nonselective α/β agonist**
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*Which agent is notoriously allergenic?*

Iopidine
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

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**Q**

Which agent is notoriously allergenic?

**Iopidine**

**How notorious is it, ie, what proportion of pts develop topical sensitivity?**

Almost half!
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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Which agent is notoriously **allergenic**?

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There are two classic manifestations of iopidine sensitivity—what are they?

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- Contact dermatitis of the lid and periorbital skin
- Follicular conjunctivitis

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When you encounter a follicular conjunctivitis, three things (ie, causes) should come to mind. One is reaction to a ‘toxin’ such as iopidine. What are the other two?
--Toxin
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--class infection

--specific bug infection
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- Viral infection
- Chlamydia infection
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**Questions**

How notorious is it, ie, what proportion of pts develop topical sensitivity? Almost half!

Which agent is notoriously **allergenic**?

**Iopidine**

As if a high likelihood of topical sensitivity wasn’t enough, iopidine has another drawback that also renders it inappropriate for long-term IOP control. What is this second dealbreaker?

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  - Apraclonidine
  - Brimonidine
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Which agent is notoriously allergenic?

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How notorious is it, ie, what proportion of pts develop topical sensitivity?
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As if a high likelihood of topical sensitivity wasn’t enough, iopidine has another drawback that also renders it inappropriate for long-term IOP control. What is this second dealbreaker?
A high propensity for the development of tachyphylaxis

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- Miotics
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Almost half!

Which agent is notoriously allergenic?
Iopidine

As if a high likelihood of topical sensitivity wasn’t enough, iopidine has another drawback that also renders it inappropriate for long-term IOP control. What is this second dealbreaker?
A high propensity for the development of tachyphylaxis

What is tachyphylaxis?
The tendency of a drug to lose effectiveness over time

When you encounter a follicular conjunctivitis, three things (ie, causes) should come to mind. One is reaction to a ‘toxin’ such as iopidine. What are the other two?
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  - Epinephrine
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  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

How notorious is it, ie, what proportion of pts develop topical sensitivity? Almost half!

Which agent is notoriously allergenic?

Iopidine

As if a high likelihood of topical sensitivity wasn’t enough, iopidine has another drawback that also renders it inappropriate for long-term IOP control. What is this second dealbreaker? A high propensity for the development of tachyphylaxis.

What is tachyphylaxis?
The tendency of a drug to lose effectiveness over time.

When you encounter a follicular conjunctivitis, three things (ie, causes) should come to mind. One is reaction to a ‘toxin’ such as iopidine. What are the other two?

- Toxin
- Viral infection
- Chlamydia infection
As mentioned above, iopidine is not in common usage as a long-term IOP med. Of the meds that are commonly used long-term, which is most notoriously allergenic?
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

As mentioned above, iopidine is not in common usage as a long-term IOP med. Of the meds that are commonly used long-term, which is most notoriously allergenic? **Brimonidine**
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - **Brimonidine**

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

As mentioned above, iopidine is not in common usage as a long-term IOP med. Of the meds that are commonly used long-term, which is most notoriously **allergenic**?

**Brimonidine**

Are the manifestations of brimonidine sensitivity the same as those to iopidine?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - **Brimonidine**

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

As mentioned above, iopidine is not in common usage as a long-term IOP med. Of the meds that are commonly used long-term, which is most notoriously allergenic?

**Brimonidine**

Are the manifestations of brimonidine sensitivity the same as those to iopidine?

Yes

Iopidine sensitivity:
- Contact dermatitis of the lid and periorbital skin
- Follicular conjunctivitis
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - **Brimonidíne**
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

As mentioned above, iopidine is not in common usage as a long-term IOP med. Of the meds that are commonly used long-term, which is most notoriously allergenic?

**Brimonidíne**

Almost half of iopidine pts develop sensitivity to it. In this regard, how notorious is brimonidíne?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

As mentioned above, iopidine is not in common usage as a long-term IOP med. Of the meds that are commonly used long-term, which is most notoriously allergenic? **Brimonidine**

Almost half of iopidine pts develop sensitivity to it. In this regard, how notorious is brimonidine? Much less so, although still significant—between 10 and 15%
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

As mentioned above, iopidine is not in common usage as a long-term IOP med. Of the meds that are commonly used long-term, which is most notoriously allergenic?

**Brimonidine**

Almost half of iopidine pts develop sensitivity to it. In this regard, how notorious is brimonidine? Much less so, although still significant—between 10 and 15%.

*If a pt is known to be allergic to iopidine, is it a given that s/he will be allergic to brimonidine?*
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

As mentioned above, iopidine is not in common usage as a long-term IOP med. Of the meds that are commonly used long-term, which is most notoriously allergenic?

**Brimonidine**

Almost half of iopidine pts develop sensitivity to it. In this regard, how notorious is brimonidine? Much less so, although still significant—between 10 and 15%. If a pt is known to be allergic to iopidine, is it a given that s/he will be allergic to brimonidine? Surprisingly no—the cross-sensitivity between these meds is minimal.
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
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- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

*Which of these is available PO?*
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide

- **Acetazolamide** (and methazolamide)

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

Which of these is available PO?
**Acetazolamide** and **methazolamide**
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
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  - **Acetazolamide**

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

Which of these is available PO? **Acetazolamide and methazolamide**

What are the common systemic side effects of PO CAIs?

- ?
- ?
- ?
- ?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
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  - Brimonidine
- **Miotics**
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- **Rho kinase inhibitor**
  - Netarsudil

**Which of these is available PO?**
Acetazolamide and methazolamide

**What are the common systemic side effects of PO CAIs?**

-- Malaise/fatigue/depression
-- Paresthesias
-- Hematologic issues:
  -- Aplastic anemia
  -- Thrombocytopenia
-- Bitter ('metallic') taste
-- Nephrolithiasis
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
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  - Dorzolamide
  - Brinzolamide
  - **Acetazolamide**

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- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
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--Bitter (‘metallic’) taste
--Nephrolithiasis

How do the parasthesias typically manifest?
As tingling of fingers, toes, and perioral area
**Q/A**

**Ocular Hypotensives: List the common agents**

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - **Acetazolamide**
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

---

**Which of these is available PO?**

Acetazolamide and methazolamide

**What are the common systemic side effects of PO CAIs?**

-- Malaise/fatigue/depression  
-- Paresthesias

**How do the paresthesias typically manifest?**

As tingling of bodypart and bodypart area

-- Bitter (‘metallic’) taste
-- Nephrolithiasis

**Hematologic issues:**

-- Aplastic anemia
-- Thrombocytopenia

**Other side effects:**

-- Malaise/fatigue/depression
Ocular Hypotensives: List the common agents

- $\beta$ blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
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- Nonselective $\alpha/\beta$ agonist
  - Epinephrine
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- CAI
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Which of these is available PO? Acetazolamide and methazolamide

What are the common systemic side effects of PO CAIs?
- Malaise/fatigue/depression
- **Paresthesias**
- Bitter (‘metallic’) taste
- Nephrolithiasis

How do the parasthesias typically manifest?
As tingling of fingers, toes and perioral area
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
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- **CAI**
  - **Dorzolamide**
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  - Acetazolamide

- **Selective α agonists**
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- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
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---

Which of these is available PO?

Acetazolamide and methazolamide

What are the common systemic side effects of PO CAIs?

--Malaise/fatigue/depression?
--Paresthesias?
--Hematologic issues:
  --Aplastic anemia?
  --Thrombocytopenia?
--Bitter (‘metallic’) taste?
--Nephrolithiasis?

---

Which of these is associated with topical CAIs?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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- **Prostaglandin analogues**
  - Latanaprost
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Acetazolamide and methazolamide

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- Malaise/fatigue/depression
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  - Aplastic anemia
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**Acetazolamide** and **methazolamide**

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-- Malaise/fatigue/depression
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Which of these is associated with topical CAIs?

Topical dorzolamide is notorious for a particular adverse effect—what is it?

Topical
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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**Which of these is available PO?**
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- Malaise/fatigue/depression
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- Bitter ('metallic') taste
- Nephrolithiasis

**Which of these is associated with **topical** CAIs?**
- **Topical dorzolamide is notorious for a particular adverse effect—what is it?**
  - It stings

**Why does it sting?**
The solution has to be somewhat acidic to keep the medicine in solution.
**Ocular Hypotensives: List the common agents**

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- Nephrolithiasis

**Which of these is associated with topical CAIs?**
- Topical dorzolamide is notorious for a particular adverse effect.
  - **stings**
  - If a pt balks at making their eye sting 3x/d, what can you do to ease their suffering (other than d/c’ing it)?
    - Why does it sting?
    - The vehicle has to be somewhat acidic to keep the medicine in solution
  - **topical**

**If a pt balks at making their eye sting 3x/d, what can you do to ease their suffering (other than d/c’ing it)?**

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-- Malaise/fatigue/depression
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  - Aplastic anemia
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-- Nephrolithiasis

Which of these is associated with **topical** CAIs?

Topical dorzolamide is notorious for a particular adverse effect—what is it?

If a pt balks at making their eye sting 3x/d, what can you do to ease their suffering (other than d/c’ing it)?

Dose it bid (it is nearly as efficacious bid as it is tid)

Why does it sting?
The vehicle has to be somewhat acidic to keep the medicine in solution

Which of these is associated with **topical** CAIs?
Ocular Hypotensives: List the common agents

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Which two drugs lowers episcleral venous pressure (EVP)?
Ocular Hypotensives: List the common agents

- **β blockers**
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  - Netarsudil

---

Which two drugs lowers episcleral venous pressure (EVP)?

- Iopidine and netarsudil (maybe)
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
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Are beta blockers known to cause significant ocular side effects?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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- **Rho kinase inhibitor**
  - Netarsudil

Are beta blockers known to cause significant ocular side effects?
No; beta blocker side effects of concern are systemic, not ocular
Ocular Hypotensives: List the common agents

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- **Rho kinase inhibitor**
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---

Are beta blockers known to cause significant ocular side effects?
No; beta blocker side effects of concern are **systemic**, not ocular

What systemic side effects are of particular concern?
1) 
2)
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

---

Are beta blockers known to cause significant ocular side effects?
No; beta blocker side effects of concern are **systemic**, not ocular

What systemic side effects are of particular concern?
1) Cardiac arrhythmias (so avoid in pts with cardiac conduction issues, eg, heart block)
2) Bronchospasm (so avoid in pts with lung dz, especially COPD and asthma)
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- **β blockers**
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*In population-based studies, which prostaglandin analogue is the most efficacious?*
Ocular Hypotensives: List the common agents

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*In population-based studies, which prostaglandin analogue is the most efficacious? They are all of very similar efficacy*
Ocular Hypotensives: List the common agents

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- Rho kinase inhibitor
  - Netarsudil

In population-based studies, which prostaglandin analogue is the most efficacious?

They are all of very similar efficacy

In population-based studies, which prostaglandin analogue has the best tolerability?
Ocular Hypotensives: List the common agents

- \( \beta \) blockers
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So, does this mean they are all therapeutically equal?

- Apraclonidine
- Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
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So, does this mean they are all therapeutically equal?
No! The fact that aggregated data fail to find differences in efficacy/tolerability does not mean such differences do not exist for individual pts. Thus, if you have a pt who either does not respond to, or is intolerant of, one PGA, you should not give up on the class entirely; rather, consider switching to a different PGA.

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- Brimonidine
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1)  
2)  
3)  
4)  
5)
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1) Eyelash growth
2) Conjunctival hyperemia
3) Darkening of irides
4) Cystoid macular edema (CME)
5) PG-associated periorbitopathy (PAP)
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1) **Eyelash growth**

What’s the $2 term for eyelash growth?

- Hypertrichosis

Other deleterious eyelash changes may occur—what, specifically?

- Trichiasis and distichiasis
Ocular Hypotensives: List the common agents

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  - Bimataprost

- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- Selective α agonist
  - Apraclonidine
  - Brimonidine

- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1) Eyelash growth
2) Conjunctival hyperemia
3) Darkening of irides
4) Cystoid macular edema (CME)
5) PG-associated periorbitopathy (PAP)

What’s the $2 term for eyelash growth? ‘Hypertrichosis’

Other deleterious eyelash changes may occur—what, specifically?

Trichiasis and distichiasis

What’s the difference between trichiasis and distichiasis?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
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- **Miotics**
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- **Rho kinase inhibitor**
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

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Other deleterious eyelash changes may occur—what, specifically?

Trichiasis and distichiasis

What’s the difference between trichiasis and distichiasis?

Trichiasis refers to lashes directed against the ocular surface that originate from their normal anatomic location on the lid margin.
Ocular Hypotensives: List the common agents

Trichiasis
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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  - Latanaprost
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  - Epinephrine
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- **CAI**
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Trichiasis refers to lashes directed against the ocular surface that originate from their normal anatomic location on the lid margin.

In contrast, distichiasis refers to lashes abutting the surface that are growing from an abnormal location (specifically, the orifices of the... two words...).
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1. **Eyelash growth**
2. Conjunctival hyperemia
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Other deleterious eyelash changes may occur—what, specifically?
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Ocular Hypotensives: List the common agents

Distichiasis: Lashes arising from MG orifices
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
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  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
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  - Brimonidine
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  - Pilo
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What's the $? term for eyelash growth?

Hypertrichosis

Is the side effect of eyelash growth universally unwelcomed?

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Is the side effect of eyelash growth universally unwelcomed?
Not by a long shot—some individuals welcome and seek out eyelash growth as a cosmetically desirable outcome.

What’s the $2 term for eyelash growth?
Hypertrichosis

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What’s the $? term for eyelash growth?

- Hypertrichosis

Is the side effect of eyelash growth universally unwelcomed?

Not by a long shot—some individuals welcome and seek out eyelash growth as a cosmetically desirable outcome. In fact, a dilute formulation of is sold under the brand-name as an FDA-approved eyelash growth promoter.

What’s the difference between trichiasis and distichiasis?

Trichiasis refers to lashes directed against the ocular surface that originate from their normal anatomic location on the lid margin.

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Ocular Hypotensives: List the common agents

- \( \beta \) blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

- Nonselective \( \alpha/\beta \) agonists
  - Epinephrine
  - Dipivefrin

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- Selective \( \alpha \) agonists
  - Apraclonidine
  - Brimonidine

- Miotics
  - Pilo

- Rho kinase inhibitor
  - Netarsudil

Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1) Eyelash growth
2) Conjunctival hyperemia
3) Darkening of irides
4) Cystoid macular edema (CME)
5) PG-associated periorbitopathy (PAP)

What’s the $2 term for eyelash growth?

Hypertrichosis

Is the side effect of eyelash growth universally unwelcomed?
Not by a long shot—some individuals welcome and seek out eyelash growth as a cosmetically desirable outcome. In fact, a dilute formulation of bimatoprost is sold under the brand-name Latisse as an FDA-approved eyelash growth promoter.

What’s the difference between trichiasis and distichiasis?
Trichiasis refers to lashes directed against the ocular surface that originate from their normal anatomic location on the lid margin.
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Ocular Hypotensives: List the common agents

Latisse
Ocular Hypotensives: List the common agents

Latisse

(Note the active ingredient)
Ocular Hypotensives: List the common agents

- Beta blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
- Nonselective alpha-beta agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective alpha agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

Prostaglandin analogues have a number of notable side effects. Identify 5 of them:
1) Eyelash growth
2) Conjunctival hyperemia
3) Darkening of irides
4) Cystoid macular edema (CME)

Do PGAs cause acute hyperemia, chronic hyperemia, or both?

Eyelash growth, conjunctival hyperemia, darkening of irides, cystoid macular edema (CME).
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- **Nonselective α/β agonists**
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  - Dipivefrin

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  - Brinzolamide
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1) Eyelash growth
2) **Conjunctival hyperemia**
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4) Cystoid macular edema (CME)

Do PGAs cause acute hyperemia, chronic hyperemia, or both? Both
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- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
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1) Eyelash growth
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3) Darkening of irides
4) Cystoid macular edema (CME)

Do PGAs cause acute hyperemia, chronic hyperemia, or both?
Both

How can you minimize the cosmetic impact of acute hyperemia?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
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- **Nonselective α/β agonists**
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2) **Conjunctival hyperemia**
3) Darkening of irides
4) Cystoid macular edema (CME)
5) PG-associated periorbitopathy (PAP)

Do PGAs cause acute hyperemia, chronic hyperemia, or both?
- Both

How can you minimize the cosmetic impact of acute hyperemia?
- By having the pt use their PGA at bedtime, when cosmesis is not an issue
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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- **Prostaglandin analogues**
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  - Timolol
  - Betaxolol
  - Carteolol

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3) **Darkening of irides**
4) Cystoid macular edema (CME)
5) PG-associated periorbitopathy (PAP)

What proportion of pts will experience darkening of their irides after 5 years of PGA use?

- Netarsudil
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1) Eyelash growth
2) Conjunctival hyperemia
3) Darkening of irides
4) Cystoid macular edema (CME)

What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a fraction, with some facing a much higher risk (to be explained)
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

Prostaglandin analogues have a number of notable side effects. Identify 5 of them:
1) Eyelash growth
2) Conjunctival hyperemia
3) **Darkening of irides**
4) Cystoid macular edema (CME)
5) PG-associated periorbitopathy (PAP)

What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained)

- Nonselective α/β agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

In addition to the iris, another structure of ophthalmic concern may darken as a result of PGA use—what is it? The periocular skin
Ocular Hypotensives: List the common agents

Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1) Eyelash growth
2) Conjunctival hyperemia
3) Darkening of irides
4) Cystoid macular edema (CME)
5) PG-associated periorbitopathy (PAP)

What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained).

Will the iris return to its baseline coloration upon discontinuation of the drop?

Now for the 'higher risk' issue: Of the myriad colors the human iris can assume, the BCSC emphasizes two that are particularly likely to darken in response to PGA use.

What are they?

Green-brown and yellow-brown (aka hazel)

In addition to the iris, another structure of ophthalmic concern may darken as a result of PGA use—what is it?

The periocular skin
### Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

*Prostaglandin analogues have a number of notable side effects. Identify 5 of them:*

1) Eyelash growth
2) Conjunctival hyperemia
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  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

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**What proportion of pts will experience darkening of their irides after 5 years of PGA use?**

Overall, as many as a third, with some facing a much higher risk (to be explained)

**Will the iris return to its baseline coloration upon discontinuation of the drop?**

It will **not**
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
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- Prostaglandin analogues
  - Latanaprost
  - Travaprost
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What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained)

Will the iris return to its baseline coloration upon discontinuation of the drop? It will not

Now for the ‘higher risk’ issue: Of the myriad colors the human iris can assume, the BCSC emphasizes two that are particularly likely to darken in response to PGA use.

What are they?

- Netarsudil
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained)

Will the iris return to its baseline coloration upon discontinuation of the drop?
It will not

Now for the ‘higher risk’ issue: Of the myriad colors the human iris can assume, the BCSC emphasizes **two** that are particularly likely to darken in response to PGA use.

What are they?

Green-brown and yellow-brown (aka hazel)
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
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  - Travaprost
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:
1) Eyelash growth
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5) Prostaglandin-associated periorbitopathy (PAP)

What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained)

Will the iris return to its baseline coloration upon discontinuation of the drop? It will **not**

Now for the ‘higher risk’ issue: Of the myriad colors the human iris can assume, the BCSC emphasizes **two** that are particularly likely to darken in response to PGA use.

What are they? **Green-brown** and **yellow-brown** (aka **hazel**)

*(Full disclosure: Being significantly red-green colorweak myself, I deferred to my wife to select the font color best representing ‘hazel.’ So if you disagree, take it up with her.)*
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
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  - Latanaprost
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What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained)

Will the iris return to its baseline coloration upon discontinuation of the drop?

How likely are green-brown and hazel irides to darken?

Green-brown and yellow-brown (aka hazel)
Ocular Hypotensives: List the common agents

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5) PG-associated periorbitopathy (PAP)

What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained).

Will the iris return to its baseline coloration upon discontinuation of the drop?

How likely are green-brown and hazel irides to darken? Very—% will do so after 5 years.

In addition to the iris, another structure of ophthalmic concern may darken as a result of PGA use—what is it? The periocular skin

How likely are green-brown and hazel irides to darken? Very—% will do so after 5 years.

What about blue eyes—are they at high risk as well? Not by comparison—just under 10% will darken.

Now for the 'higher risk' issue: Of the myriad colors the human iris can assume, the BCSC emphasizes two that are particularly likely to darken in response to PGA use.

What are they? Green-brown and yellow-brown (aka hazel)
Ocular Hypotensives: List the common agents

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  - Timolol
  - Betaxolol
  - Carteolol
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  - Latanaprost
  - Travaprost
  - Bimataprost
- Nonselective $\alpha/\beta$ agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
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  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1) Eyelash growth
2) Conjunctival hyperemia
3) Darkening of irides
4) Cystoid macular edema (CME)
5) PG-associated periorbitopathy (PAP)

What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained)

Will the iris return to its baseline coloration upon discontinuation of the drop?

How likely are green-brown and hazel irides to darken? Very—80%+ will do so after 5 years

In addition to the iris, another structure of ophthalmic concern may darken as a result of PGA use—what is it?

The periocular skin

How likely are green-brown and hazel irides to darken? Very—80%+ will do so after 5 years

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Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

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What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained)

Will the iris return to its baseline coloration upon discontinuation of the drop?

How likely are green-brown and hazel irides to darken?
Very—80%+ will do so after 5 years

What about blue eyes—are they at high risk as well?

Green-brown and yellow-brown (aka hazel)

Netarsudil
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost

- Prostaglandin analogues have a number of notable side effects. Identify 5 of them:
  1) Eyelash growth
  2) Conjunctival hyperemia
  3) Darkening of irides
  4) Cystoid macular edema (CME)
  5) PG-associated periorbitopathy (PAP)

- What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained)

- Will the iris return to its baseline coloration upon discontinuation of the drop?

- How likely are green-brown and hazel irides to darken?
  - Very—80%+ will do so after 5 years

- What about blue eyes—are they at high risk as well?
  - Not by comparison—just under 10% will darken

- Green-brown and yellow-brown (aka hazel)
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travoprost
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1) Eyelash growth
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Now for the ‘higher risk’ issue: Of the myriad colors the human iris can assume, the BCSC emphasizes **two** that are particularly likely to darken in response to PGA use.

What are they? **Green-brown** and **yellow-brown** (aka **hazel**)

In addition to the iris, another structure of ophthalmic concern may darken as a result of PGA use—what is it?

- Netarsudil
Ocular Hypotensives: List the common agents

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In one word (ending with –tic), what sort of process is responsible for the darkening of the irises and/or periocular skin?

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What cell is responsible for melanocytic processes?

Ocular Hypotensives: List the common agents
What cell is responsible for melanocytic processes? 
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Let’s consider the embryology of melanocytes. From which primordial cell do they derive?
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Let’s consider the embryology of melanocytes. From which primordial cell do they derive? Neural crest cells (NCCs)
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Neural crest cells (NCCs)

Briefly, what’s the backstory on neural crest cells?
**What cell is responsible for melanocytic processes?**
Hurr durr, Imma guess **melanocytes**?

*Let’s consider the embryology of melanocytes. From which primordial cell do they derive?*  
Neural crest cells (NCCs)

*Briefly, what’s the backstory on neural crest cells?*  
NCCs are a subtype of **embryo cell type** cells.
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Neural crest cells (NCCs)

*Briefly, what’s the backstory on neural crest cells?*

NCCs are a subtype of neuroectodermal cells. In embryogenesis, some of the neuroectodermal cells located along the dorsal aspect of the early neural tube are induced to transition into NCCs.
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Briefly, what’s the backstory on neural crest cells?
NCCs are a subtype of neuroectodermal cells. Early in embryogenesis, some of the neuroectodermal cells located along the dorsal aspect of the neural tube are induced to transition into NCCs. NCCs then migrate widely across the embryo, and upon arriving at their destination they proliferate and differentiate into specialized tissues and cells, including melanocytes.
Ocular Hypotensives: List the common agents

Neural crest cells…
Neural crest cells...and their derivatives

Ocular Hypotensives: List the common agents
Next let’s consider the function of surface melanocytes. What do they do?
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What is the name of the membrane-bound structure in which melanin is contained?
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What is the name of the membrane-bound structure in which melanin is contained? A melanosome
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Do melanocytes hang onto their melanosomes?
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What is the name of the membrane-bound structure in which melanin is contained? A melanosome.

Do melanocytes hang onto their melanosomes? No—once packaged in melanosomes, melanin is transferred to neighboring cells (e.g., skin melanocytes transfer their melanin to nearby keratinocytes).
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Melanocyte and its keratinocytes

Ocular Hypotensives: List the common agents
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Some people have darker skin than others. (Thanks, Captain Obvious.) Is it the case that darker-completed individuals have more melanocytes?
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Some people have darker skin than others. (Thanks, Captain Obvious.) Is it the case that darker-complected individuals have more melanocytes? No, the number of melanocytes does not vary with degree of pigmentation. People with darker complexion have more melanin in their keratinocytes.
Ocular Hypotensives: List the common agents

- \( \beta \) blockers
  - Timolol
  - Betaxolol
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  - Latanaprost
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

1. Eyelash growth
2. Conjunctival hyperemia
3. Darkening of irides
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5. PG-associated periorbitopathy (PAP)

What proportion of pts will experience darkening of their irides after 5 years of PGA use? Overall, as many as a third, with some facing a much higher risk (to be explained)

Will the iris return to its baseline coloration upon discontinuation of the drops?
No.

Now for the ‘higher risk’ issue: Of the myriad colors the human iris can assume, the BCSC emphasizes two that are particularly likely to darken in response to PGA use.

What are they?
Green-brown and yellow-brown (aka hazel)

In addition to the iris, another structure of ophthalmic concern may darken as a result of PGA use—what is it?
The periocular skin

In one word (ending with ‘-tic’), what sort of process is responsible for the darkening of the irises and/or periocular skin?
It is a melanocytic process

What specific aspect of the melanocytic process is responsible?
Before we answer, let’s sidebar to review this process…

Now that the melanocytic process sidebar is complete, let’s answer this question:
What specific aspect of the melanocytic process is responsible for the darkening of the iris and periocular skin secondary to PGA use?

Increased numbers of melanosomes within melanocytes.

What’s not the cause, and that’s melanocyte proliferation Which doesn’t occur).
Ocular Hypotensives: List the common agents

- **β blockers**
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It will not

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- Netarsudil
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- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin

- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

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- **Miotics**
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Now for the higher risk issue. Of the myriad colors the human iris can assume, the BCSC emphasizes two that are particularly likely to darken in response to PGA use. What are they? Green-brown and yellow-brown (aka hazel).

In addition to the iris, another structure of ophthalmic concern may darken as a result of PGA use—what is it? The periocular skin.

In one word (ending with -tic), what sort of process is responsible for the darkening of the irises and/or periocular skin? It is a melanocytic process.

What specific aspect of the melanocytic process is responsible? Before we answer, let’s sidebar to review this process…

Now that the melanocytic process sidebar is complete, let’s answer this question: What specific aspect of the melanocytic process is responsible for the darkening of the iris and periocular skin secondary to PGA use? Increased numbers of melanosomes within melanocytes. Worth emphasizing what’s **not** the cause, and that’s melanocyte proliferation (which doesn’t occur).
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Increased numbers of melanosomes within melanocytes, worth emphasizing what’s not the cause, and that’s melanocyte proliferation (which doesn’t occur).

Does all this melanocytic mischief put PGA users at an increased risk of developing melanoma of the iris and/or periocular skin? No it doesn’t, and this shouldn’t come as a surprise given that melanocytic proliferation is not a component of the darkening response.
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Which are more likely to get PGA-associated CME: phakic, or pseudophakic eyes?
Ocular Hypotensives: List the common agents

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Which are more likely to get PGA-associated CME: phakic, or pseudophakic eyes? Pseudophakic

Does aphakic status also convey an increased risk of CME? Indeed it does
Ocular Hypotensives: List the common agents

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  - Betaxolol
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  - Epinephrine
  - Dipivefrin
- **CAIs**
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

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3) Darkening of irides
4) **Cystoid macular edema (CME)**
5) PG-associated periorbitopathy (PAP)

Which are more likely to get PGA-associated CME: phakic, or pseudophakic eyes?
- Pseudophakic

What renders a pseudophakic eye even more likely to PGA-associated CME?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
  - Latanaprost
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- An open posterior capsule (s/p either intra-op rupture, or YAG)

Does aphakic status also convey an increased risk of CME?

Indeed it does.
Ocular Hypotensives: List the common agents

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A constellation of orbital/periorbital changes 2ndry to...
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--?
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--Enopththalmos
--Deepening of the lower vs upper-lid sulcus
--?
--?
--?
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- Deepening of the upper-lid sulcus
- Ptosis of the upper lid
- ?
- ?
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- Ptosis of the upper lid
- Inferior [two words]
- ?
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--Inferior scleral show
--A 'one word' orbit
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**What are the classic/typical manifestations of PAP?**
--Enophthalmos
--Deepening of the upper-lid sulcus
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--A ‘tight’ orbit
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Is PAP reversible with cessation of PGA therapy?
A

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Is PAP reversible with cessation of PGA therapy?
As of this writing, this remains unsettled
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Note that of the five side effects identified, four are related to **cosmesis**. This implies that caution should be exercised in long-term use of PGAs in one group of pts (other than supermodels). Which pts are these?

Those with **unilateral glaucoma**
A

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Those with unilateral glaucoma
Unilateral hypertrichosis following latanoprost use OS only
The right (A) and left (B) eyes of a patient on unilateral treatment with a topical prostaglandin analogue for the left eye. Left-sided periorbital skin hyperpigmentation, hypertrichosis, deepening of the superior eyelid sulcus, and loss of periorbital fat are evident.
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A small number (~1%) of PGA pts will experience an idiosyncratic reaction significant enough to warrant discontinuation. What is that reaction?
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  - **Brimonidine**

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Another commonly-used med on the list is notorious for causing a granulomatous anterior uveitis. Which one? **Brimonidine**
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**What corneal condition is a strong contraindication to PGA use?**

- HSV keratitis

Are we talking about active dz only, or does this apply also to a history of HSV keratitis?

Both

Why the contraindication?

PGA use has been associated with prolongation and/or recurrence of HSV keratitis
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  - Bimataprost

- **Nonselective α/β agonist**
  - Epinephrine
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- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

**What corneal condition is a strong contraindication to PGA use?**

HSV keratitis

**Are we talking about active dz only, or does this apply also to a history of HSV keratitis?**
Ocular Hypotensives: List the common agents

- $\beta$ blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

- Nonselective $\alpha/\beta$ agonist
  - Epinephrine
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What corneal condition is a strong contraindication to PGA use?

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Why the contraindication?
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What corneal condition is a strong contraindication to PGA use?
HSV keratitis

Are we talking about active dz only, or does this apply also to a history of HSV keratitis?
Both

Why the contraindication?
PGA use has been associated with prolongation and/or recurrence of HSV keratitis
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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*These three are pro-drugs; i.e., they become activated via cleavage by corneal esterases:*
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Which class must be used cautiously in patients who take MAOIs and/or tricyclics?
(Monoamine oxidase inhibitors)
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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Topical CAIs are relatively contraindicated in Fuchs dystrophy pts. Why?
Ocular Hypotensives: List the common agents

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- **Topical CAIs are relatively contraindicated in Fuchs dystrophy pts. Why?**
  - Because they may cause/exacerbate corneal edema.
Ocular Hypotensives: List the common agents

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**Topical CAIs are relatively contraindicated in Fuchs dystrophy pts. Why?**
Because they may cause/exacerbate corneal edema

**What is the mechanism for CAI-induced corneal edema?**

Recall that endothelial cells make use of carbonic anhydrase in performing their pump function to maintain K deturgescence. In addition to inhibiting aqueous formation, topical CAIs inhibit K endothelial pump function. If endothelial pump function is already tenuous (as it is in Fuchs), the addition of a CAI could lead to the occurrence or worsening of edema.
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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What is the ‘nonresponder’ rate for the β blockers, ie, what percent of pts will not manifest a meaningful decrease in IOP?

β blockers
- Timolol
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Rho kinase inhibitor
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What is the ‘nonresponder’ rate for the β blockers, ie, what percent of pts will not manifest a meaningful decrease in IOP? 10-20
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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What is the ‘nonresponder’ rate for the β blockers, ie, what percent of pts will not manifest a meaningful decrease in IOP? 10-20

What is a well-known cause of nonresponding that should probably keep you from trying a β blocker in the first place? If the pt is on a systemic β blocker (eg, for HTN). In such pts, a topical β blocker is unlikely to move IOP much.
**Ocular Hypotensives: List the common agents**

- **β blockers**
  - Timolol
  - Betaxolol
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**What is a well-known cause of nonresponding that should probably keep you from trying a β blocker in the first place?**
If the pt is on a **systemic** β blocker (eg, for HTN). In such pts, a topical β blocker may not move IOP much.
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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With respect to pregnancy, and under the former system of classifying drugs:

(No question yet—proceed when ready)
β blockers
- Timolol
- Betaxolol
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Prostaglandin analogues
- Latanaprost
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Nonselective α/β agonist
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Miotics
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Rho kinase inhibitor
- Netarsudil

With respect to pregnancy, and under the former system of classifying drugs:
Which are Class A?
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
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With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A?

None of them
Ocular Hypotensives: List the common agents

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With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A?
None of them

Which are Class B?
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**With respect to pregnancy, and under the former system of classifying drugs:**

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Which are Class B?
Brimonidine. (The rest are all Class C.)
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OK then, how should glaucoma be managed during pregnancy?
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What treatment options fall under ‘none’?

-uspend all treatment during pregnancy—just monitor the pt, and resume tx after delivery

OK then, how should glaucoma be managed during pregnancy? With as few meds as possible (preferably **none**).
What treatment options fall under ‘none’?
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--Suspend all treatment during pregnancy--just monitor the pt, and resume tx after delivery 
--If suspending tx seems imprudent, consider SLT 

With respect to pregnancy, and under the former system of classifying drugs: 
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**If meds are to be used, which is the best option?**
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OK then, how should glaucoma be managed during pregnancy? With as few meds as possible (preferably none)

If meds are to be used, which is the best option?
Most experts would probably recommend timolol, but at the 0.25% strength rather than the usual 0.5%.
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You probably know that the cap color for T.5 is...

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With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A?
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Which are Class B?
Brimonidine. (The rest are all Class C.)

You probably know that the cap color for T₅ is... yellow

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With respect to pregnancy, and under the former system of classifying drugs:

- **Which are Class A?**
  - None of them

- **Which are Class B?**
  - Brimonidine. (The rest are all Class C.)

You probably know that the cap color for T.5 is... **yellow**

But do you know the cap color for T.25?

- Apraclonidine
- Brimonidine

OK then, how should glaucoma be managed during pregnancy?

With as few meds as possible (preferably **none**)

If meds are to be used, which is the best option?

Most experts would probably recommend timolol, but at the **0.25%** strength rather than the usual **0.5%**.
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol 0.25
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A?
None of them

Which are Class B?
Brimonidine. (The rest are all Class C.)

With as few meds as possible (preferably none)

If meds are to be used, which is the best option?

Most experts would probably recommend timolol, but at the 0.25% strength rather than the usual 0.5%.

But do you know the cap color for T.25? **Light blue**

You probably know that the cap color for T.5 is... **yellow**
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol 0.25
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With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A? None of them

Which are Class B? Brimonidine. (The rest are all Class C.)

OK then, how should glaucoma be managed during pregnancy? With as few meds as possible (preferably none)

Won't the 0.25 strength be only half as effective as the 0.5?

Most experts would probably recommend timolol, but at the 0.25% strength rather than the usual 0.5%
Ocular Hypotensives: List the common agents

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With respect to pregnancy, and under the former system of classifying drugs:

- Which are Class A?
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- Which are Class B?
  - Brimonidine. (The rest are all Class C.)

OK then, how should glaucoma be managed during pregnancy?

With as few meds as possible (preferably none)

Won't the 0.25 strength be only half as effective as the 0.5?

Far from it. In fact, in many pts, it works just as well

Most experts would probably recommend timolol, but at the 0.25% strength rather than the usual 0.5%
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With respect to pregnancy, and under the former system of classifying drugs:

- **Which are Class A?**
  - None of them

- **Which are Class B?**
  - Brimonidine. (The rest are all Class C.)

What about nursing mothers—should T₂₅ be used for them as well?

- **With as few meds as possible (preferably none)**
- **Won’t the 0.25 strength be only half as effective as the 0.5?**
  - Far from it. In fact, in many pts, it works just as well
- **Which is the best option?**
  - Most experts would probably recommend timolol, but at the 0.25% strength rather than the usual 0.5%
Ocular Hypotensives: List the common agents

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With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A?
None of them

Which are Class B?
Brimonidine. (The rest are all Class C.)

What about nursing mothers—should T_{0.25} be used for them as well?
No, because β blocker metabolites get concentrated in breast milk

Won’t the 0.25 strength be only half as effective as the 0.5?
Far from it. In fact, in many pts, it works just as well

Most experts would probably recommend timolol, but at the 0.25% strength rather than the usual 0.5%
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**Why not use a PGA in pregnant women?**

With respect to pregnancy, and under the former system of classifying drugs:

- Which are Class A? None of them
- Which are Class B? Brimonidine. (The rest are all Class C.)

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Why not use a PGA in pregnant women?

Reach way back to your Ob/Gyn rotation and recall that prostaglandins are involved in inducing labor. Given this, it should not be surprising to learn that one shouldn’t give a pregnant woman a prostaglandin analogue.
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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Why not use a PGA in pregnant women?
Reach way back to your Ob/Gyn rotation and recall that prostaglandins are involved in **two words**.

Which are Class B?
Brimonidine. (The rest are all Class C.)
Q/A

Ocular Hypotensives: List the common agents

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  - Timolol
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  - Latanaprost
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Brimonidine. (The rest are all Class C.)
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Brimonidine. (The rest are all Class C.)
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Why not use a CAI?

Which are Class A?
None of them

Which are Class B?
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Ocular Hypotensives: List the common agents

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CAIs have been shown to be teratogenic in mice. For this reason, the *Glaucoma* book states flatly that “oral CAIs should not be used by women in their childbearing years.”
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**What does IIH stand for in this context?**

Idiopathic intracranial hypertension.
Ocular Hypotensives: List the common agents

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(That said, the Neuro-Oph book considers oral CAIs to be **first-line tx** for IIH, a condition most commonly found in women in their childbearing years. Caveat emptor.)

As for **topical** CAIs in pregnancy, the Glaucoma book doesn’t address them directly.