Ocular Hypotensives: List the common agents

- $\beta$ blockers

...
A

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

Ocular Hypotensives: List the common agents
Ocular Hypotensives: List the common agents

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

- Prostaglandin analogues
  - An FDA-approved PGA, much less well-known than the big three
  - A PGA ‘combo drug’

The ‘big three’ FDA-approved PGA that dominate the American market
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
  - (Tafluprost)  
  - (Latanaprostene bunod)

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    - What is the brand name of tafluprost?

The three FDA-approved PGA that dominate the American market
Ocular Hypotensives: List the common agents

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

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
  - Tafluprost (*Agonist*)

*The three FDA-approved PGA that dominate the American market

**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

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

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost
  - (Tafuraprost) The three FDA-approved PGA that dominate the American market
  - (Latanaprostene bunod) An FDA-approved AGI

What is the brand name of latanaprostene bunod?
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) – An FDA-approved PGA combo drug

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

- $\beta$ blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
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- Nonselective $\alpha/\beta$ agonist
  - Epinephrine
  - Dipivefrin
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

(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
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  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI** (carbonic anhydrase inhibitors)
  -
  -
  -
Ocular Hypotensives: List the common agents

- $\beta$ blockers
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Ocular Hypotensives: List the common agents

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  - Timolol
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- **Prostaglandin analogues**
  - Latanaprost
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  - Bimataprost

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

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

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

?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - 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**
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- **CAI**
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  - Brinzolamide
  - Acetazolamide
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  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilocarpine (*Pilo* for short)
Ocular Hypotensives: List the common agents

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  - Latanaprost
  - Travaprost
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- **Nonselective α/β agonist**
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- **CAI**
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  - Brinzolamide
  - Acetazolamide

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

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
A

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

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>β blockers</strong></td>
<td>Timolol, Betaxolol, Carteolol</td>
</tr>
<tr>
<td><strong>Prostaglandin analogues</strong></td>
<td>Latanaprost, Travaprost, Bimataprost</td>
</tr>
<tr>
<td><strong>Nonselective α/β agonist</strong></td>
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<tr>
<td><strong>CAI</strong></td>
<td>Dorzolamide, Brinzolamide, Acetazolamide</td>
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<td><strong>Selective α agonists</strong></td>
<td>Apraclonidine, Brimonidine</td>
</tr>
<tr>
<td><strong>Miotics</strong></td>
<td>Pilo</td>
</tr>
<tr>
<td><strong>Rho kinase inhibitor</strong></td>
<td>Netarsudil</td>
</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

- **β blockers**
  - Timolol
  - Betaxolol
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<table>
<thead>
<tr>
<th>Brand Name?</th>
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<tbody>
<tr>
<td>Timoptic</td>
</tr>
<tr>
<td>x</td>
</tr>
<tr>
<td>x</td>
</tr>
<tr>
<td>Xalatan</td>
</tr>
<tr>
<td>Travatan</td>
</tr>
<tr>
<td>Lumigan</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Resudil</td>
</tr>
<tr>
<td>Rhopressa</td>
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23
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What is the name of the equation that describes the factors determining IOP?
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What is the name of the equation that describes the factors determining IOP?
The Goldmann equation
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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
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**What is the name of the equation that describes the factors determining IOP?**

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*Note:*
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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?

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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

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

Ocular Hypotensives: List the common agents

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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)

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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?

Where, specifically, is aqueous formed?

- Decrease the rate of aqueous formation
- Increase the rate of aqueous outflow
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Note:
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Dehydration of the vitreous
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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

The Goldmann equation

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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)

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

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**.

One outflow pathway is **pressure dependent**; the other, **pressure independent**.
Ocular Hypotensives: List the common agents

- β blockers
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- TM = conventional = pressure-dependent
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  - 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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- **Selective α agonists**
  - 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)**

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

**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 = pressure-dependent
- U/S = unconventional = pressure-independent
Ocular Hypotensives: List the common agents

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
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**Q** Of the three means implied by the Goldmann equation, how do **β blockers** 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
- Decrease episcleral venous pressure

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
**Ocular Hypotensives**

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  - 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 **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?
Ocular Hypotensives:

- **β blockers**
  - Timolol
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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

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
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
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-- Decrease the rate of aqueous formation
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-- Decrease episcleral venous pressure

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?
Ocular Hypotensives

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travaprost
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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 **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%

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

- \( \beta \) blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
  - Latanaprost
  - Travoprost
  - Bimataprost
- Nonselective \( \alpha/\beta \) agonists
  - Epinephrine
  - Dipivefrin
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  - Dorzolamide
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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)
Ocular Hypotensive Agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travoprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
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  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
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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 do PGAs lower IOP? (Note: It could be more than one)

By increasing the rate of aqueous outflow
Ocular Hypotensive Agents

- **β blockers**
  - Timolol
  - Betaxolol
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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?
A

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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

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?
Mainly via the U/S pathway
Ocular Hypotensives

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

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- **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

By what mechanism do they increase U/S outflow?
Ocular Hypotensives

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  - Timolol
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- 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

By what mechanism do they increase U/S outflow?

It is unknown at this time
Ocular Hypotensives

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

- **Prostaglandin analogues**
  - Latanaprost
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It is unknown at this time

By how much do they lower IOP?
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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?

25-33%
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
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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 CAIs 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**
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- Dipivefrin

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- Dorzolamide
- Brinzolamide
- Acetazolamide

**Selective α agonists**
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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?

---

1. **Decrease the rate of aqueous formation**
2. **Increase the rate of aqueous outflow**
3. **Decrease episcleral venous pressure**

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

By decreasing the rate of aqueous formation.
Ocular Hypotensives

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

- **Prostaglandin analogues**
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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 CAIs lower IOP? (Note: It could be more than one)

By decreasing the rate of aqueous formation

By what mechanism do they reduce aqueous formation?
Ocular Hypotensives

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

- **Prostaglandin analogues**
  - Latanaprost
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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

Of the three means implied by the Goldmann equation, how do **CAIs** 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 the enzyme carbonic anhydrase
Ocular Hypotensives

- \( \beta \) blockers
  - Timolol
  - Betaxolol
  - Carteolol
- Prostaglandin analogues
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The Goldmann equation implies three means by which IOP can be lowered. What are they?
1. Decrease the rate of aqueous formation
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Of the three means implied by the Goldmann equation, how do CAIs 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 the enzyme carbonic anhydrase

By how much do they lower IOP?
Ocular Hypotensives:

- β blockers
  - Timolol
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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 CAIs 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 the enzyme carbonic anhydrase

By how much do they lower IOP?

15-20%
Ocular Hypotensives

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  - Timolol
  - Betaxolol
  - Carteolol

- **Prostaglandin analogues**
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  - Travaprost
  - Bimataprost

- **Nonselective α/β agonist**
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- **CAI**
  - Dorzolamide
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The Goldmann equation implies three means by which IOP can be lowered. What are they?
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-- 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
  - 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 do **selective α agonists** lower IOP? (Note: It could be more than one)

Both meds decrease aqueous formation and increase outflow. Additionally, apra reduces EVP.
Ocular Hypotensives

- \( \alpha/\beta \) 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

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 (apra)

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.
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

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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 **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?
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

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)

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 book.
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
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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 book

By what mechanism do they increase aqueous outflow?
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

---

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 (apra)

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 book

*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 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 (apra)

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

**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 agonist**
  - Epinephrine
  - Dipivefrin

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

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

- **Miotics**
  - 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 (apra)

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 book

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
  - 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 (apra)

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 book

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
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
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  - Dorzolamide
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  - Pilo
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  - 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 (apra)

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 book

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
  - 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 do **miotics** 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

- **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 do miotics lower IOP? (Note: It could be more than one)*

By increasing the rate of aqueous outflow
Ocular Hypotensives

- **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**
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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?
Ocular Hypotensive 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

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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.
Ocular Hypotensives

- **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
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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 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
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonists**
  - Epinephrine
  - Dipivefrin
- **CAI**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
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  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
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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?
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 **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: List the common agents

Note: The next two meds are too new to appear in the latest (in my possession) edition of the BCSC *Glaucoma* book. Thus, questions about them are unlikely to appear on the OKAP or WQEs—yet.
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

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
- **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)
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

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).

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

---

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).

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

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

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:

- **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

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  - 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
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?
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**
  - 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 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)
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
**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

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  - 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)

By increasing the rate of aqueous outflow

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

In turn, these increase U/S and TM outflow, respectively.

-- 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**
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  - Pilo

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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)

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

- $\beta$ blockers
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - **Latanaprostene bunod**
  - Travaprost
  - Bimataprost
- Nonselective $\alpha/\beta$ agonist
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective $\alpha$ 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)

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?

---

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

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

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

- **Nonselective \( \alpha/\beta \) agonist**
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- **CAI**
  - Dorzolamide
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  - 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)].
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
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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).
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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**Q/A**

**Rate of aqueous formation**
**Rate of aqueous outflow**
**EVP**

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

*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)

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
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- **Miotics**
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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)

**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

- **Non-selective α/β agonist**
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- **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)

---

**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**:  
-- **NO**: indications.
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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  - Acetazolamide
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  - 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 \textit{latanaprostene bunod} lower IOP? (Note: It could be more than one)

Lataprost \hspace{1cm} \text{Nitric oxide}

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

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

How do the constituent moieties accomplish their effects?
--\textit{Latanaprost}: Mechanism unknown (as noted previously)
--\textit{NO}: By inducing relaxation of cytoskeletal elements found within TM cells
Ocular Hypotensives

- β blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprostene bunod
  - Travoprost
  - Bimataprost

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- Selective α/β 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)

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 do the constituent moieties accomplish their effects?

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

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

- Blockers
  - Timolol
  - Betaxolol
  - Carteolol

- Prostaglandin analogues
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

- Non-selective \(\alpha/\beta\) agonist
  - Epinephrine
  - Dipivefrin

- CAIs
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

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

- Miotics
  - Pilo

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  - Netarsudil

\[
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)

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

Both

How do the constituent moieties accomplish their effects?

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

“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
Ocular Hypotensives

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

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

- Prostaglandin analogues
  - Latanaprostene bunod
  - Travaprost
  - Bimataprost

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- Selective \( \alpha \) 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)

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

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

Via both

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

“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?

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

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

- **Prostaglandin analogues**
  - Latanaprostene bunod
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- Nonselective \( \alpha/\beta \) agonist
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  - Brinzolamide
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- Selective \( \alpha \) 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)

- By increasing the rate of aqueous outflow

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

- Via both

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

"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?
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

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

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
- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α/β 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)

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?
By about 1 mmHg

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

- **$\beta$ blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprostene bunod
  - Travoprost
  - Bimataprost
- **Nonselective $\alpha/\beta$ agonist**
  - Epinephrine
  - Dipivefrin
- **CAIs**
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- **Selective $\alpha$ agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
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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

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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

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 how much does latanaprostene bunod lower IOP?
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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?
Ocular Hypotensives

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

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

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

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

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)

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 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
  - 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

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

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

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

- **Prostaglandin analogues**
  - Latanaprost
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- **Selective α agonists**
  - Apraclonidine
  - Brimonidine

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

Rank these four commonly-used drug classes in terms of their IOP-lowering efficacy:
1) PGAs
2) Beta blockers
3) Selective α agonists
4) CAIs
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**
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  - Pilo
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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) CAIs

Give two reasons the PGAs beat the β blockers:
1) 
2)
A/Q

Ocular Hypotensives: List the common agents

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

Rank these four commonly-used drug classes in terms of their IOP-lowering efficacy:
1) **PGAs**
2) **Beta blockers**
3) Selective α agonists
4) **CAIs**

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 'activity')
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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  - Latanaprost
  - Travaprost
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1) **PGAs**
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4) CAIs

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

- **β blockers**
  - Timolol
  - Betaxolol
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  - Latanaprost
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Give two reasons the PGAs beat the β blockers:
1) Slightly better IOP reduction on average
2) Better 24-hour IOP control (β blocker efficacy drops during sleep)

This is why the second dose should be instilled a number of hours before bedtime!
Q

Ocular Hypotensives: List the common agents

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

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Rank these four commonly-used drug classes in terms of their IOP-lowering efficacy:
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2) Beta blockers
3) Selective α agonists
4) CAIs

**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.

By better 24-hour control (β blocker efficacy drops during sleep)
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
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  - Dorzolamide
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  - Acetazolamide
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  - Dorzolamide
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  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
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Rank these four commonly-used drug classes in terms of their IOP-lowering efficacy:

1) PGAs
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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.

Better 24-hour control (β blocker efficacy drops during sleep)
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
  - 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.

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

- **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

Timolol

CAI
  (as Brinzolamide)
  (as Dorzolamide)

Brimonidine

Netarsudil

Latanaprost
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
- **Nonselective α/β agonist**
  - Epinephrine
  - Dipivefrin
  - Brimonidine
- **CAI**
  - Dorzolamide (as Brinzolamide)
  - Brinzolamide (as Dorzolamide)
  - Acetazolamide
- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
  - Latanaprost
- **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**
  - Epinephrine
  - Dipivefrin

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

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

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

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

**Cosopt**

**Timolol**

**Dorzolamide**
Q: 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 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
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
  - 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 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

- **\( \beta \) 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

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

1. 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.
2. Eliminates washout (i.e., when an impatient pt instills their second drop too soon after the first, thereby washing it out).
3. (as Brinzolamide)
4. (as Dorzolamide)

Combigan

Brimonidine

Simbrinza

Cosopt

Timolol
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

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
  - 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**

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**Questions**

1. What is the standard dosing frequency for latanaprost?
   - **Daily**
2. What is the standard dosing frequency for netarsudil?
   - **Daily**
3. What is the preferred/recommended time to take latanaprost?
   - **Bedtime**
4. What is the preferred/recommended time to take netarsudil?
   - **Bedtime**

- Rocklatan
Ocular Hypotensives: List the common agents

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

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

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

- CAI
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide

- Selective \(\alpha\) 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

Latanaprost

Netarsudil
Q

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

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

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- **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….
Ocular Hypotensives: List the common agents

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

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

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- **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
Ocular Hypotensives: List the common agents

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

- **Prostaglandin analogues**
  - **Latanaprost**
  - Travoprost
  - 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 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
  - Bimataprost
- **Nonselective α/β agonist**
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  - Brinzolamide
  - Acetazolamide
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  - 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

Latanaprost

Rocklatan

Netarsudil
Ocular Hypotensives: List the common agents

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

- **Prostaglandin analogues**
  - **Latanaprost**
  - Travaprost
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- **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
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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  - **Latanaprost**
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  - 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**
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- **Selective α agonists**
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  - 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
  - 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?
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
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

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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
  - Travoprost
  - 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? **Lopidine**

**Lopidine** 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 lopidine, 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
  - Travoprost
  - 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?
A

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

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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
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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*
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?
Ocular Hypotensives: List the common agents

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

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
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  - 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 $\alpha_1$ and $\alpha_2$
Ocular Hypotensives: List the common agents

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

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

\[ \alpha_1: \]

- 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

- Prostaglandin analogues
  - Latanaprost
  - Travaprost
  - Bimataprost

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  - Brinzolamide
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- Miotics
  - Pilo

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  - Netarsudil

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

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

- \( \alpha_2 \):
  - Reduced IOP

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

- \( \beta \) 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

**Questions:**

- 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
  - Carteolol

- **Prostaglandin analogues**
  Note: The latest (in my possession) BCSC Glaucoma book states that **neuroprotection** is another ‘possible’ effect of $\alpha_2$ 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 book’s index)

- **Nonselective $\alpha_1/\alpha_2$ agonist**
  - Epinephrine
  - Dipivefrin

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

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

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

In the present context, how many subtypes of $\alpha$ receptors are we concerned about?

Two

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**Ocular Hypotensives: List the common agents**

- **β blockers**
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  - Dorzolamide
  - Brinzolamide
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- **Selective α agonists**
  - Apraclonidine
  - Brimonidine
- **Miotics**
  - Pilo
- **Rho kinase inhibitor**
  - Netarsudil

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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 $\alpha_1$ and $\alpha_2$

What does it mean to say the selective α agonists are selective? What are they ‘selecting’?
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 $\alpha_1$
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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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 $\alpha_1$ and $\alpha_2$

What does it mean to say the selective α 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 α 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?
They are called $\alpha_1$ and $\alpha_2$

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

Which agent is notoriously allergenic?
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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  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

Which agent is notoriously allergenic? 
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

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

Almost half!

Which agent is **notoriously** allergenic?

Iopidine
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
  - Bimataprost
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Which agent is notoriously allergenic?

Iopidine

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

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

--

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Q/A

Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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  - Latanaprost
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  - Dorzolamide
  - 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

**There are two classic manifestations of iopidine sensitivity—what are they?**
- Contact dermatitis of the lid and periorbital skin
- Follicular conjunctivitis
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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  - Latanaprost
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- **Selective α agonists**
  - Apraclonidine
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- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

Which agent is notoriously *allergenic*?

Iopidine

How notorious is it, i.e., what proportion of pts develop topical sensitivity?

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

-- Contact dermatitis of the lid and periorbital skin
-- Follicular conjunctivitis
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
- \( \mbox{Rho kinase inhibitor} \)
  - Netarsudil

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

Which agent is notoriously\( \text{allergenic} \)?

Iopidine

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

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

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
--
Q/A

Ocular Hypotensives: List the common agents

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

- **Prostaglandin analogues**
  - Latanaprost
  - Travaprost
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  - Netarsudil

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

Which agent is notoriously allergenic?
Iopidine

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

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

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

-- Toxin
  -- Class
  -- Specific bug
-- Infection
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 agent is notoriously allergenic?

**Iopidine**

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

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

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

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

Almost half!
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

**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?

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

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

Almost half!
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
  - Betaxolol
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- **Prostaglandin analogues**
  - Latanaprost
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  - Dorzolamide
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- **Selective α agonists**
  - Apraclonidine
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- **Rho kinase inhibitor**
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**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

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
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

---

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? 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

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

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

- **Miotics**
  - Pilo

- **Rho kinase inhibitor**
  - Netarsudil

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**Iodidine sensitivity:**
- Contact dermatitis of the lid and periorbital skin
- Follicular conjunctivitis

**Are the manifestations of brimonidine sensitivity the same as those to iodidine?**

As mentioned above, iodidine 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**?

**Brimonidin**
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

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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

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

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 10–15%.

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
Q

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 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
  - 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
- Hematologic issues:
  - Aplastic anemia
  - Thrombocytopenia
- Bitter ('metallic') taste
- Nephrolithiasis
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 of these is available PO?*
**Acetazolamide** and **methazolamide**

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

- **β blockers**
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  - Latanaprost
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  - 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?**
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- **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
- Bitter (‘metallic’) taste
- Nephrolithiasis

How do the parasthesias typically manifest?

- As tingling of fingers, toes and perioral area
- Thrombocytopenia
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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  - 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**
-- Bitter (‘metallic’) taste
-- Thrombocytopenia
-- Nephrolithiasis

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  - Betaxolol
  - Carteolol
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  - Latanaprost
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  - 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?
- 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
  - 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 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

- \( \beta \) 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

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

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

It stings
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 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?

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

It stings

Why does it sting?
A

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 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?*

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

It stings

*Why does it sting?*

The vehicle has to be somewhat acidic to keep the medicine in solution
Ocular Hypotensives: List the common agents

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

- **Prostaglandin analogues**
  - Latanaprost
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- **CAI**
  - Dorzolamide
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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?
- Malaise/fatigue/depression
- Paresthesias
- Hematologic issues:
  - Aplastic anemia
  - Thrombocytopenia
  - Bitter ('metallic') taste
  - Nephrolithiasis

Which of these is associated with topical CAIs?

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
**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

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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?

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

It **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)?

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

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

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  - Timolol
  - Betaxolol
  - Carteolol
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*Which two drugs lowers episcleral venous pressure (EVP)?*

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

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- **Miotics**
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  - Netarsudil

Are beta blockers known to cause significant ocular side effects?
Ocular Hypotensives: List the common agents

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  - Timolol
  - Betaxolol
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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.
Ocular Hypotensives: List the common agents

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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)
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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) 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)
Ocular Hypotensives: List the common agents

- **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*
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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

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So, does this mean they are all therapeutically equal?

In population-based studies, which prostaglandin analogue is the most efficacious? 
*They are all of very similar efficacy*

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*They are all of very similar tolerability*
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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?

They are all of very similar tolerability

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. As a general rule, at least two PGAs should be tried before you give up on the class.
Ocular Hypotensives: List the common agents

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

1)
2)
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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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Do PGAs cause acute hyperemia, chronic hyperemia, or both?
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Do PGAs cause acute hyperemia, chronic hyperemia, or both? Both

How can you minimize the cosmetic impact of acute hyperemia?
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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

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Which are more likely to darken, light irides or dark irides?
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1) Eyelash growth
2) Conjunctival hyperemia
3) **Darkening of irides**
4) Cystoid macular edema (CME)
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Which are more likely to darken, **light** irides or **dark** irides? Light
Ocular Hypotensives: List the common agents

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Which are more likely to get PGA-associated CME, phakic or pseudophakic eyes?
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Which are more likely to get PGA-associated CME, phakic or pseudophakic eyes?
Pseudophakic
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Which are more likely to get PGA-associated CME, phakic or pseudophakic eyes?

Pseudophakic

What event during cataract surgery will render a pseudophakic eye even more likely to experience PGA-associated CME?

Posterior-capsule rupture

Why is the term cystoid macular edema something of a misnomer for this condition?

Because the condition is usually 'dry' (ie, there is no edema present).
Ocular Hypotensives: List the common agents

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  - Brimonidine
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  - Pilo
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- Miotics
  - Pilocarpine
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- Pseudophakic

What event during cataract surgery will render a pseudophakic eye even more likely to experience PGA-associated CME?
- Posterior-capsule rupture

Why is the term cystoid macular edema something of a misnomer for this condition?
- Because the condition is usually 'dry' (ie, there is no edema present)

For this reason, the condition is sometimes described as cystoid macular degeneration (ie, CMD)
Ocular Hypotensives: List the common agents

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

What is Prostaglandin-Associated Periorbitopathy (PAP)?

Unknown
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- **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 is Prostaglandin-Associated Periorbitopathy (PAP)?
A constellation of orbital/periorbital changes including deepening of the sulcus, atrophy of periorbital fat, enophthalmos, and tight eyelids.

Unknown
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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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 class of pts (other than supermodels). Which pts are these?
A

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Ocular Hypotensives: List the common agents

- **β blockers**
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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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A small number (~1%) of PGA pts will experience an idiosyncratic reaction significant enough to warrant discontinuation. What is that reaction? **Uveitis**
Q

**Ocular Hypotensives: List the common agents**

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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?

**Uveitis**

Another commonly-used med on the list is can cause uveitis, specifically a granulomatous anterior uveitis. Which one?
Ocular Hypotensives: List the common agents

- **β blockers**
  - Timolol
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Another commonly-used med on the list is can cause uveitis, specifically 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.
Ocular Hypotensives: List the common agents

- β blockers
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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

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Topical CAIs are relatively contraindicated in Fuchs dystrophy pts. Why?
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Topical CAIs are relatively contraindicated in Fuchs dystrophy pts. Why?
Because they may cause/exacerbate corneal edema.
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What is the mechanism for CAI-induced corneal edema?
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**Topical CAIs are relatively contraindicated in Fuchs dystrophy pts. Why?**
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**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.
What is the ‘nonresponder’ rate for the \( \beta \) blockers, ie, what percent of pts will not manifest a meaningful decrease in IOP?

If the pt is on a systemic \( \beta \) blocker (eg, for HTN). In such pts, a topical \( \beta \) blocker is unlikely to move IOP much.
What is the ‘nonresponder’ rate for the β blockers, ie, what percent of pts will not manifest a meaningful decrease in IOP?

10-20

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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?

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With respect to pregnancy, and under the former system of classifying drugs: Which are Class A?
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
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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:*

**Which are Class A?**
None of them

**Which are Class B?**
Brimonidine. (The rest are all Class C.)
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None of them

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**OK then, how should glaucoma be managed during pregnancy?**
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**OK then, how should glaucoma be managed during pregnancy?**

With as few meds as possible (preferably **none**).
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- Which are Class A?
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---

What treatment options fall under ‘none’?

- Suspend 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**).
Ocular Hypotensives: List the common agents

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

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

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

- **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.)

**What treatment options fall under ‘none’?**

- Suspend all treatment during pregnancy--just monitor the pt, and resume tx after delivery
- If suspending tx seems imprudent, consider
- If conditions warrant it, consider

- Brinzolamide
- Acetazolamide
- Dorzolamide
- Brinzolamide

OK then, how should glaucoma be managed during pregnancy?

With as few meds as possible (preferably) none
Ocular Hypotensives: List the common agents

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

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

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

- **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.)

**What treatment options fall under 'none'?**
- Suspend all treatment during pregnancy—just monitor the pt, and resume tx after delivery
- If suspending tx seems imprudent, consider SLT
- If conditions warrant it, consider incisional surgery

OK then, how should glaucoma be managed during pregnancy?
With as few meds as possible (preferably **none**).
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

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)

If meds are to be used, which is the best option?
Q/A

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

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)

If meds are to be used, which is the best option?
Most experts would probably recommend timolol, but at the
**%** strength rather than the usual **%**
**Ocular Hypotensives: List the common agents**

- **β blockers**
  - Timolol 0.5/0.25
  - 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

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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**)

**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.)

You probably know that the cap color for T.5 is…

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.)

You probably know that the cap color for T.5 is... yellow

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
- 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.)

You probably know that the cap color for T.5 is… yellow

But do you know the cap color for T.25?
- Apraclonidine
- Brimonidine

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
  - Travoprost
  - Bimataprost

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

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

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.)

But do you know the cap color for T.25? **Light blue**

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.)

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

- **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.)

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%.

Wont 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.
**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.)

What about nursing mothers—should T.25 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**
- Most experts would probably recommend timolol, but at the **0.25% strength** rather than the usual 0.5%

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
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  - Bimataprost

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  - 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.)

What about nursing mothers—should T.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%
Ocular Hypotensives: List the common agents

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  - Timolol
  - Betaxolol
  - Carteolol
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  - Latanaprost
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  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- Selective α agonists
  - Apraclonidine
  - Brimonidine
- Miotics
  - Pilo
- Rho kinase inhibitor
  - Netarsudil

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.)
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

**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.

**Which are Class B?**
Brimonidine. (The rest are all Class C.)
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

**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.

**Which are Class B?**
Brimonidine. (The rest are all Class C.)
**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

**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.

**Why not use a CAI?**

While the BCSC Glaucoma book states flatly that “oral CAIs should not be used by women in their childbearing years,” 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.
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

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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.

**Why not use a CAI?**
CAIs have been shown to be teratogenic in mice. For this reason, the BCSC Glaucoma book states flatly that “oral CAIs should not be used by women in their childbearing years.” 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.