Q

Ocular Hypotensives: List the common agents

- *β blockers*
    -
    -
    -

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol

Q

Ocular Hypotensives: List the common agents

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

The 'big three' FDA-approved PGA that dominate the American market

An FDA-approved PGA, much less well-known than the big three

A PGA 'combo drug'

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
  - (Tafluprost)
  - (Latanaprostene bunod)

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  - **(Tafluprost)**
  - (Latanaprostene bunod)

The three FDA-approved PGA that dominate the American market

...ne big three

A PGA combo drug

What is the brand name of tafluprost?

A

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  - Carteolol
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  - Latanaprost
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  - **(Tafluprost)**
  - (Latanaprostene bunod)

The three FDA-approved PGA that dominate the American market

...ne big three

A PGA combo drug

What is the brand name of tafluprost?
Zioptan (and that's all we'll have to say about it)

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
  - (Tafluprost) ← *An FDA approv*
  - (**Latanaprostene bunod**) ← *A*

The three FDA-approved PGA that dominate the American market

What is the brand name of latanaprostene bunod?

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
  - (Tafluprost) ← *An FDA-approv*
  - (**Latanaprostene bunod**) ←

The three FDA-approved PGA that dominate the American market

What is the brand name of latanaprostene bunod?
Vyzulta

(We'll have more to say about this drug later)

Q

Ocular Hypotensives: List the common agents

- *β blockers*
    - Timolol
    - Betaxolol
    - Carteolol
- *Prostaglandin analogues*
    - Latanaprost
    - Travaprost
    - Bimataprost
- *Nonselective α/β agonist*
    -
    -

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
- *Nonselective α/β agonist*
  - Epinephrine
  - Dipivefrin

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- ***β blockers***
  - Timolol
  - Betaxolol
  - Carteolol
- *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.)

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
- *Nonselective α/β agonist*
  - Epinephrine
  - Dipivefrin
- *CAI* (carbonic anhydrase inhibitors)
  -
  -
  -

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

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  - Latanaprost
  - Travaprost
  - Bimataprost
- *Nonselective α/β agonist*
  - Epinephrine
  - Dipivefrin
- *CAI*
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
  - *?*

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

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
  - Methazolamide

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

Methazolamide

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*
  -
  -

A

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  - Carteolol
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  - Travaprost
  - Bimataprost
- *Nonselective α/β agonist*
  - Epinephrine
  - Dipivefrin
- *CAI*
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- *Selective α agonists*
  - Apraclonidine
  - Brimonidine

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  - Bimataprost
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  - Epinephrine
  - Dipivefrin
- *CAI*
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- *Selective α agonists*
  - Apraclonidine
  - Brimonidine
- *Miotics*
  -

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- *Nonselective α/β agonist*
  - Epinephrine
  - Dipivefrin
- *CAI*
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- *Selective α agonists*
  - Apraclonidine
  - Brimonidine
- *Miotics*
  - Pilocarpine (*Pilo* for short)

Q

Ocular Hypotensives: List the common agents

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

A

Ocular Hypotensives: List the common agents

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

Q

Ocular Hypotensives: List the common agents

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

Brand Name?

xxxxx
xxxxx

xxxxx
xxxxx

xxxxx

These meds are rarely used anymore (except for pilo in certain situations), so we won't bother with their brand names

A

Ocular Hypotensives: List the common agents

	Brand Name?
• *β blockers*	
• **Timolol**	**Timoptic**
• Betaxolol	xxxxx
• Carteolol	xxxxx
• *Prostaglandin analogues*	
• **Latanaprost**	**Xalatan**
• **Travaprost**	**Travatan**
• **Bimataprost**	**Lumigan**
• *Nonselective α/β agonist*	
• Epinephrine	xxxxx
• Dipivefrin	xxxxx
• *CAI*	
• **Dorzolamide**	**Trusopt**
• **Brinzolamide**	**Azopt**
• **Acetazolamide**	**Diamox**
• *Selective α agonists*	
• **Apraclonidine**	**Iopidine**
• **Brimonidine**	**Alphagan**
• *Miotics*	
• Pilo	xxxxx
• *Rho kinase inhibitor*	
• **Netarsudil**	**Rhopressa**

Q

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- *CAI*
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- *Selective α agonists*
  - Apraclonidine
  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

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

A

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- *Miotics*
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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

Q

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

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

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

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Note:
1) EVP = write it out
2) In the interest of simplicity, I fudged a little on the denominator—technically, it's outflow one word, not outflow **rate**

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Note:
1) EVP = episcleral venous pressure
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The Goldmann equation implies three means by which IOP can be lowered. What are they?
--
--
--

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

Q

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

Q

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Dehydration of the vitreous

Q

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  - Pilo
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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?

$$\text{IOP} = \frac{\text{Rate of } \textbf{aqueous formation}}{\text{Rate of aqueous outflow}} + \text{EVP}$$

Note:
1) EVP = episcleral venous pressure
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The Goldmann equation implies three means by which IOP can be lowered. What are they?
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- *CAI*
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- *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

Where, specifically, is aqueous formed?
In the nonpigmented epithelium of the pars plicata portion of the ciliary body

$$IOP = \frac{\text{Rate of } \textbf{aqueous formation}}{\text{Rate of aqueous outflow}} + EVP$$

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**

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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)

$$\text{IOP} = \frac{\text{Rate of aqueous formation}}{\text{Rate of } \textbf{aqueous outflow}} + \text{EVP}$$

Note:

What are the two types/pathways of aqueous outflow?
--
--

A

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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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Note:

What are the two types/pathways of aqueous outflow?
--Trabecular meshwork (TM)
--Uveoscleral (U/S)

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
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- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
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What is the name of the equation that describes the factors determining IOP?
The Goldmann equation

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

$$IOP = \frac{\text{Rate of aqueous formation}}{\text{Rate of } \textbf{aqueous outflow}} + EVP$$

Note:

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 =
--U/S =

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
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  - Dipivefrin
- *CAI*
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Q

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--TM = conventional
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One outflow pathway is pressure **dependent***; the other, pressure* ***independent****. Which is which?*
--TM = conventional =
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A

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Q

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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)*

A

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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

Q

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By decreasing the rate of aqueous formation

By what mechanism do they reduce aqueous formation?

A

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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

Q

Ocular Hypotensives

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

By decreasing the rate of aqueous formation

By what mechanism do they reduce aqueous formation?

By inhibiting production of cAMP in the ciliary epithelium

By how much do they lower IOP?

A

Ocular Hypotensives

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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 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%

Q

Ocular Hypotensives

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

A

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--Decrease the rate of aqueous formation

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

By increasing the rate of aqueous outflow

Q

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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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*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**

Q

Ocular Hypotensives

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By increasing the rate of aqueous outflow

By what mechanism do they increase U/S outflow?

A

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

By increasing the rate of aqueous outflow

By what mechanism do they increase U/S outflow?

It is unknown at this time

Q

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By increasing the rate of aqueous outflow

By what mechanism do they increase U/S outflow?

It is unknown at this time

By how much do they lower IOP?

A

Ocular Hypotensives

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

By increasing the rate of aqueous outflow

By what mechanism do they increase U/S outflow?

It is unknown at this time

By how much do they lower IOP?

25-33%

Q

Ocular Hypotensives

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

A

Ocular Hypotensives

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  - **Acetazolamide**
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  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

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

Q

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By what mechanism do they reduce aqueous formation?

A

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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

Q

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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?

A

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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?
15-20%

Q

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- *CAI*
  - Dorzolamide
  - Brinzolamide
  - Acetazolamide
- ***Selective α agonists***
  - **Apraclonidine**
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- *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 **selective α agonists** lower IOP? (Note: It could be more than one)*

Q/A

Ocular Hypotensives

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
- *Nonselective α/β agonist*
  - Epinephrine
  - Dipivefrin
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  - Dorzolamide
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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 vs brimo reduces EVP.

A

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By what mechanism do they reduce aqueous formation?

This is not addressed in the *BCSC*

Q

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By what mechanism do they reduce aqueous formation?

This is not addressed in the *BCSC*

By what mechanism do they increase aqueous outflow?

Q/A

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  - Timolol
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By what mechanism do they increase aqueous outflow?

--Apraclonidine increases outflow

--Brimonidine increases outflow

A

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

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

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

By what mechanism do they reduce aqueous formation?

This is not addressed in the *BCSC*

By what mechanism do they increase aqueous outflow?

--**A**praclonidine increases **TM** outflow
--**B**rimonidine increases **U/S** outflow

Mnemonic for remembering their outflow pathways:
Apraclonidine: 'ATM'
Brimonidine: 'BUS'

Q

Ocular Hypotensives

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By what mechanism do they increase aqueous outflow?

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--Brimonidine increases U/S outflow

By how much do they lower IOP?

A

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By what mechanism do they reduce aqueous formation?

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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%

Q

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By increasing the rate of aqueous outflow

Q

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By what mechanism do they increase outflow?

Q/A

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By increasing the rate of aqueous outflow

By what mechanism do they increase outflow?

They stimulate contraction of the [direction] portion of the [ ] muscle.

Q/A

Ocular Hypotensives

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

By increasing the rate of aqueous outflow

By what mechanism do they increase outflow?

They stimulate contraction of the longitudinal portion of the ciliary muscle.

Q/A

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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 structure (two words) .

Q/A

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Q/A

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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 [two words], thereby allowing aqueous to egress more efficiently.

Q/A

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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.

A

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By what mechanism do they increase outflow?

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

tl;dr They increase outflow through the TM pathway

Q

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By how much do they lower IOP?

A

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

By increasing the rate of aqueous outflow

By what mechanism do they increase outflow?

They stimulate contraction of the longitudinal portion of the ciliary muscle. 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%

Q

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)*

A

Ocular Hypotensives

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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)

Q

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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 … contributi…

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

A

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- *β blockers*
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Primarily by increasing the rate of **aqueous outflow**

(they may also reduce aqueous formation as well as

decrease

contributi

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

Mainly via **the TM pathway**

Q

Ocular Hypotensives

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By what mechanism do they increase TM outflow?

By inducing relaxation of cytoskeletal elements found within TM cells

Q

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- *β blockers*
  - Timolol
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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?

A

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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

Q

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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)*

By increasing the rate of **aqueous outflow**

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

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Via **both**

Q

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Via **both**

How does manage to affect both outflow pathways?

Q/A

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Via **both**

How does manage to affect both outflow pathways?

The latanaprostene bunod molecule is cleaved into two moieties: ______ and ______.

A

Ocular Hypotensives

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  - Betaxolol
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- ***Prostaglandin analogues***
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- *Miotics*
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Latanaprost **Nitric oxide**

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

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By increasing the rate of **aqueous outflow**

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

Via **both**

How does manage to affect both outflow pathways?

The latanaprostene bunod molecule is cleaved into two moieties: **latanaprost** and **nitric oxide (NO)** .

Q/A

Ocular Hypotensives

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- ***Prostaglandin analogues***
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- *Miotics*
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Latanaprost → *U/S vs TM*

Nitric oxide → *U/S vs TM*

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

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

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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)*

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 ____ and ____ outflow, respectively.

A

Ocular Hypotensives

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

Latanaprostene bunod → **Latanaprost** → **↑ U/S outflow**

Latanaprostene bunod → **Nitric oxide** → **↑ TM outflow**

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

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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.

Q

Ocular Hypotensives

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  - Netarsudil

Latanaprost → **↑ U/S outflow**

Nitric oxide → **↑ TM outflow**

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By increasing the rate of **aqueous outflow**

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

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

A

Ocular Hypotensives

- *β blockers*
  - Timolol
  - Betaxolol
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- ***Prostaglandin analogues***
  - *Latanaprostene bunod*
  - Travaprost
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  - Acetazolamide
- *Selective α agonists*
  - Apraclonidine
  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

Latanaprostene bunod → **Latanaprost** → **↑ U/S outflow**

Latanaprostene bunod → **Nitric oxide** → **↑ TM outflow**

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

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

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By increasing the rate of **aqueous outflow**

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

Via **both**

How does manage to affect both outflow pathways?

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

How do the constituent moieties accomplish their effects?

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

--**NO**

Q

Ocular Hypotensives

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

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

Latanaprostene bunod → **Nitric oxide** → **↑ TM outflow**

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

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

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

How do the constituent moieties accomplish their effects?
--**Latanaprost**: Mechanism unknown (as noted previously)
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A

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Latanaprost → **↑ U/S outflow**

Nitric oxide → **↑ TM outflow**

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

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 ...uld be more than one)*

By increasing the rate of **aqueous outflow**

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

Via **both**

How does manage to affect both outflow pathways?

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

How do the constituent moieties accomplish their effects?

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

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

Q

Ocular Hypotensives

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- ***Prostaglandin analogues***
  - *Latanaprostene bunod*
  - Travaprost
  - Bimatoprost
- N... *α/β ag...*
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorz...
  - Brinz...
  - Aceta...
- *Selective...*
  - Apra...
  - 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**

Latanaprost → **↑ U/S outflow**

Nitric oxide → **↑ TM outflow**

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

Via both

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

How do the constituent moieties accomplish their effects?

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

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

Q/A

Ocular Hypotensives

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- ***Prostaglandin analogues***
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  - Bimataprost
- N *α/β ag*
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorz
  - Brinz
  - Aceta
- *Selective*
  - Apra
  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

Latanaprostene bunod → **Latanaprost** → **↑ U/S outflow**

Latanaprostene bunod → **Nitric oxide** → **↑ TM outflow**

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

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

--Decrease the rate of aqueous formation

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--Decrease episcleral venous pressure

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

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Via **both**

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This phrase was used to characterize the mechanism of action of the

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Nitric oxide → **↑ TM outflow**

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

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

How do the constituent moieties accomplish their effects?

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

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

Q

Ocular Hypotensives

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

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

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  - Betaxolol
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  - Dorz...
  - Brinz...
  - Aceta...
- *Selective*...
  - Apra...
  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

Latanaprost → **↑ U/S outflow**

Nitric oxide → **↑ TM outflow**

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By increasing the rate of **aqueous outflow**

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Via **both**

"By inducing relaxation of cytoskeletal elements found within TM cells"…Where have I heard that before? (No cheating by looking back)
This phrase was used to characterize the mechanism of action of the **Rho kinase inhibitors**

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

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Ocular Hypotensives

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- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

Latanaprost → ↑ U/S outflow

Nitric oxide → ↑ TM outflow

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By increasing the rate of **aqueous outflow**

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

Via both

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

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

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

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

How do the constituent moieties accomplish their effects?

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

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

Q

Ocular Hypotensives

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  - Dorzolamide
  - Brinzolamide
  - A...
- *Selec...*
  - Apraclonidine
  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

Latanaprost → **↑ U/S outflow**

Nitric oxide → **↑ TM outflow**

↓ *IOP by ?*

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

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  - Dipivefrin
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  - Dorzolamide
  - Brinzolamide
  - A...
- *Selec...*
  - Apraclonidine
  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

Latanaprost

Nitric oxide

↑ U/S outflow

↑ TM outflow

↓ IOP by ?

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

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

--Decrease the rate of aqueous formation

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

Via **both**

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

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

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*?

Q

Ocular Hypotensives

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- ***Prostaglandin analogues***
  - *Latanaprostene bunod*
  - Travaprost
  - Bimataprost
- N… *α/β ag*…
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - A…
- *Selec*…
  - Apraclonidine
  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

Latanaprost → **↑ U/S outflow**

Nitric oxide → **↑ TM outflow**

↓ IOP ▭ *mmHg vs latanaprost alone*

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

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?

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

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

A

Ocular Hypotensives

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- ***Prostaglandin analogues***
  - *Latanaprostene bunod*
  - Travaprost
  - Bimatoprost
- N *α/β ag*
  - Epinephrine
  - Dipivefrin
- CAI
  - Dorzolamide
  - Brinzolamide
  - A
- *Selec*
  - Apraclonidine
  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

Latanaprost → **↑ U/S outflow**

Nitric oxide → **↑ TM outflow**

↓ IOP 1.2 mmHg vs latanaprost alone

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

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

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

(Rank the topical formulations)

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

1)

2)

3)

4)

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

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

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

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
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  - Apraclonidine
  - Brimonidine
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  - 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 24º IOP control (β blocker efficacy drops during 'acxtivity'

A

Ocular Hypotensives: List the common agents

- ***β blockers***
  - Timolol
  - Betaxolol
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- ***Prostaglandin analogues***
  - Latanaprost
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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 24º 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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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 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
- ***Prostaglandin analogues***
  - Latanaprost
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  - Dipivefrin
- *CAI*
  - Dorzolam
  - 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**

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.

during sleep)

Q/A

Ocular Hypotensives: List the common agents

- ***β* blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- ***Prostaglandin analogues***
  - Latanaprost
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- *Nonselective α/β agonist*
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  - Dipivefrin
- *CAI*
  - Dorzolam...
  - 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

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 one word. However, the β blockers produce slightly better IOPs at diff word, so they win.

2) Better 24-hr IOP control (β blocker efficacy drops during sleep)

A

Ocular Hypotensives: List the common agents

- ***β* blockers**
  - Timolol
  - Betaxolol
  - Carteolol
- ***Prostaglandin analogues***
  - Latanaprost
  - Travaprost
  - Bimataprost
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  - Epinephr
  - Dipivefrin
- *CAI*
  - Dorzolam
  - 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**

during sleep)

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.

Q

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

Class?

Drug? ***Drug?***

Drug? ***Drug?***

A

Ocular Hypotensives: List the common agents

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

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

CAI

(as Brinzolamide) (as Dorzolamide)

Brimonidine **Timolol**

Latanaprost **Netarsudil**

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - **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

(as Brinzolamide)

CAI

(as Dorzolamide)

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

Brimonidine

Timolol

Latanaprost

Netarsudil

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

CAI

(as Brinzolamide)

(as Dorzolamide)

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

Cosopt

Brimonidine

Timolol

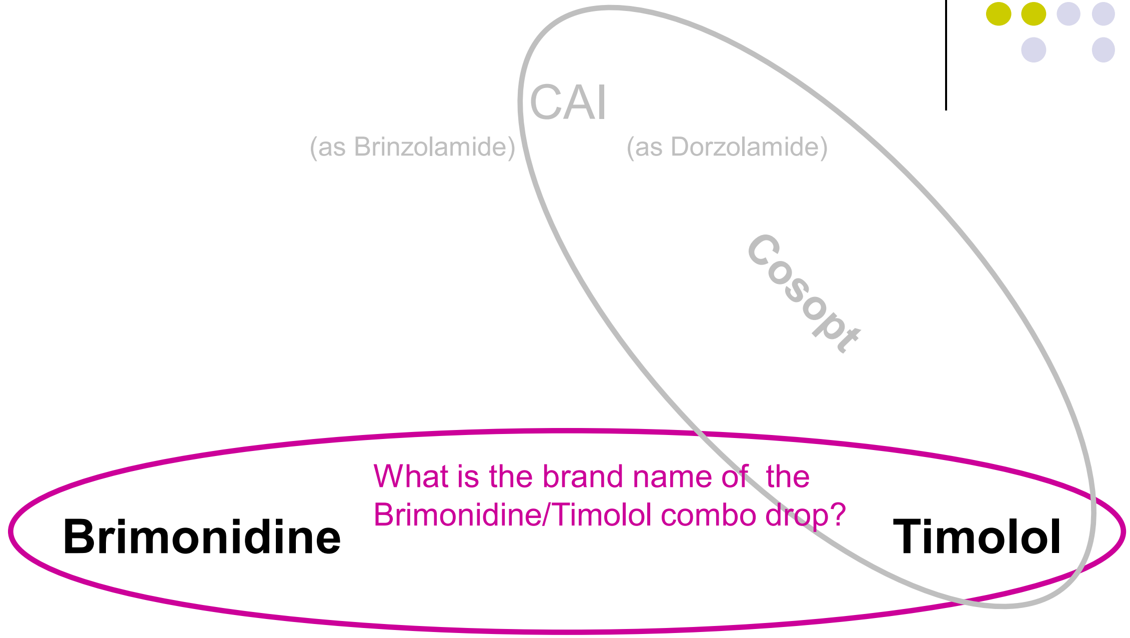
Latanaprost

Netarsudil

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



Latanaprost

Netarsudil

A

Ocular Hypotensives: List the common agents

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

CAI

(as Brinzolamide) (as Dorzolamide)

Cosopt

Brimonidine

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

Combigan

Timolol

Latanaprost

Netarsudil

Q

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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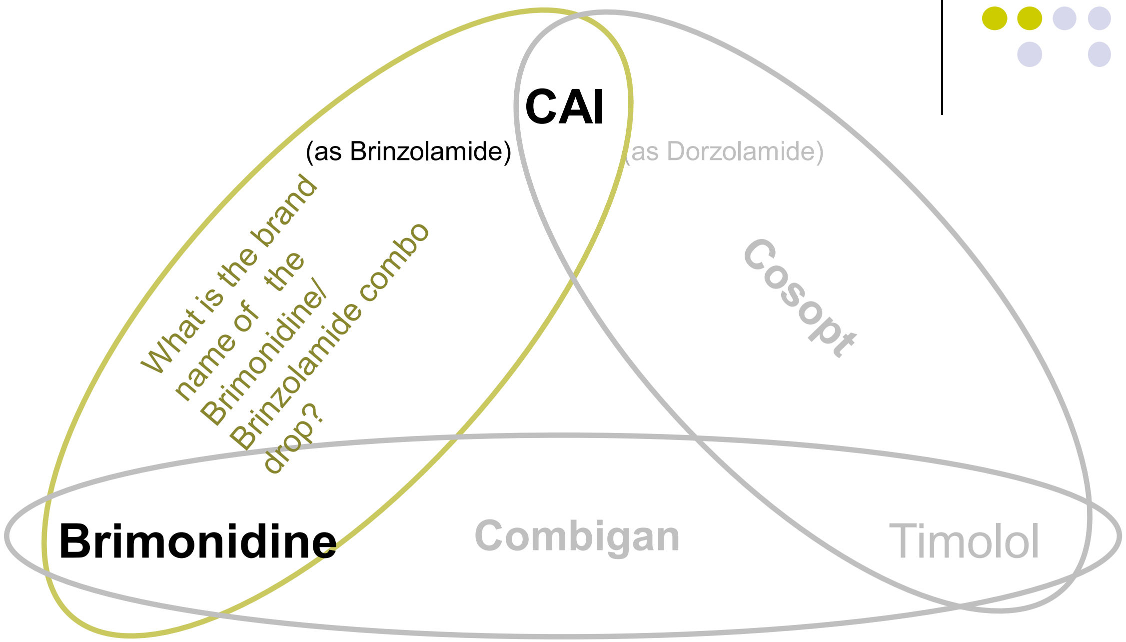

Latanaprost

Netarsudil

A

Ocular Hypotensives: List the common agents

- *β blockers*
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  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
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  - Travaprost
  - Bimataprost
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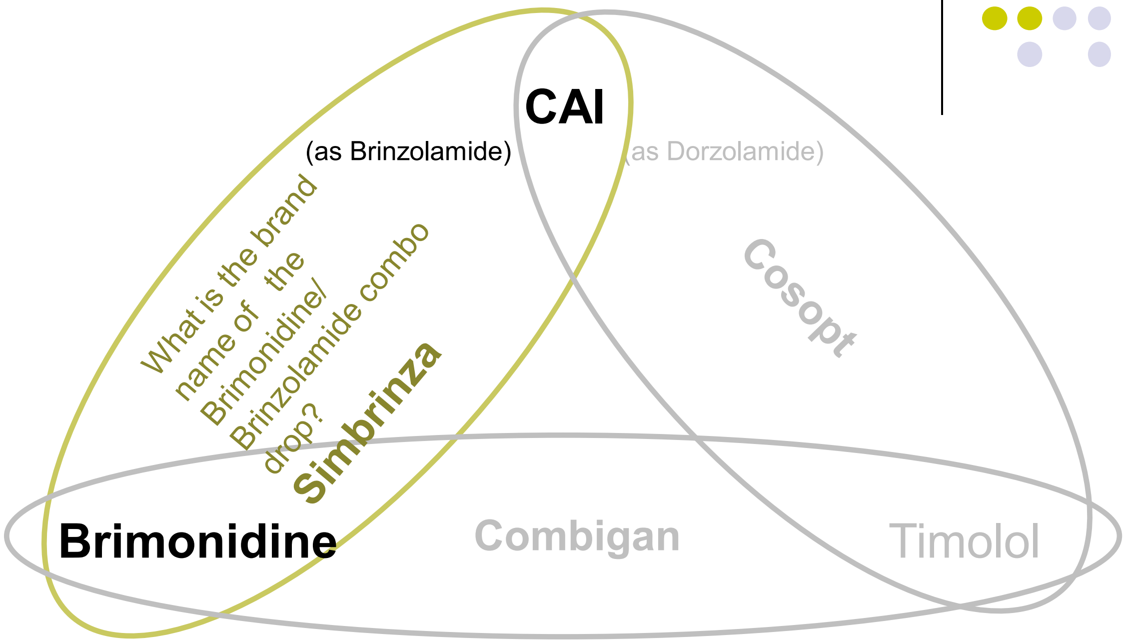

Latanaprost

Netarsudil

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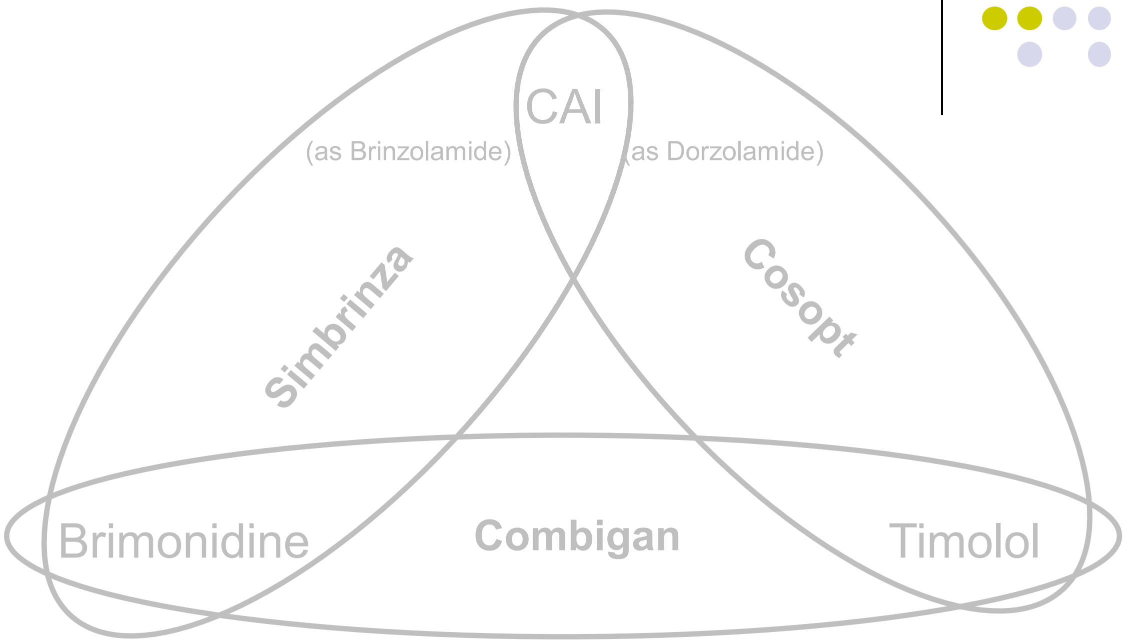

Latanaprost

What is the brand name of the Latanaprost/Netarsudil combo drop?

Netarsudil

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - **Latanaprost**
  - Travaprost
  - Bimataprost
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  - Pilo
- *Rho kinase inhibitor*
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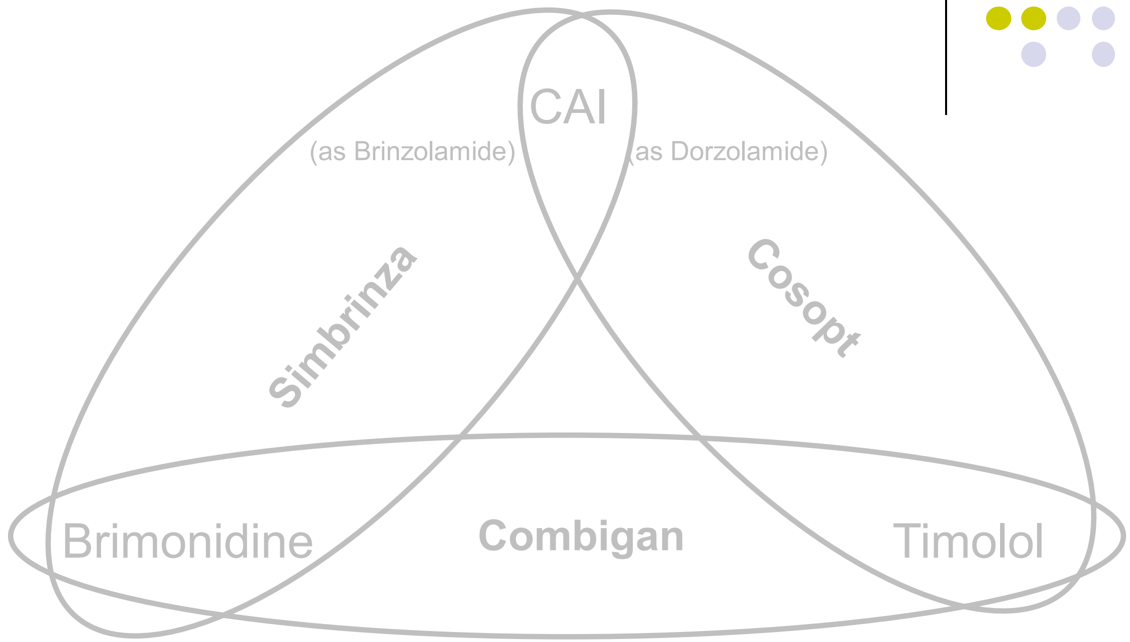

Latanaprost — *What is the brand name of the Latanaprost/Netarsudil combo drop?* **Rocklatan** — Netarsudil

Q

Ocular Hypotensives: List the common agents

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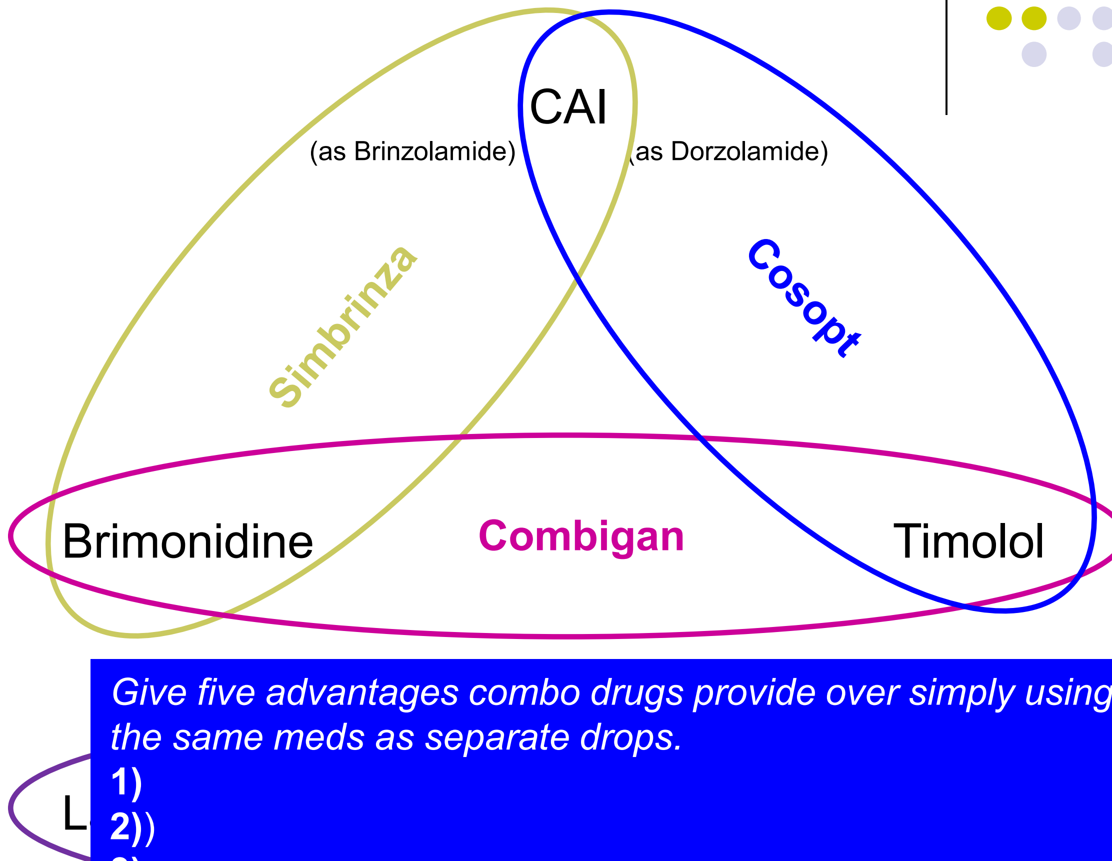

Give five advantages combo drugs provide over simply using the same meds as separate drops.

1)
2))
3)

4)
5)

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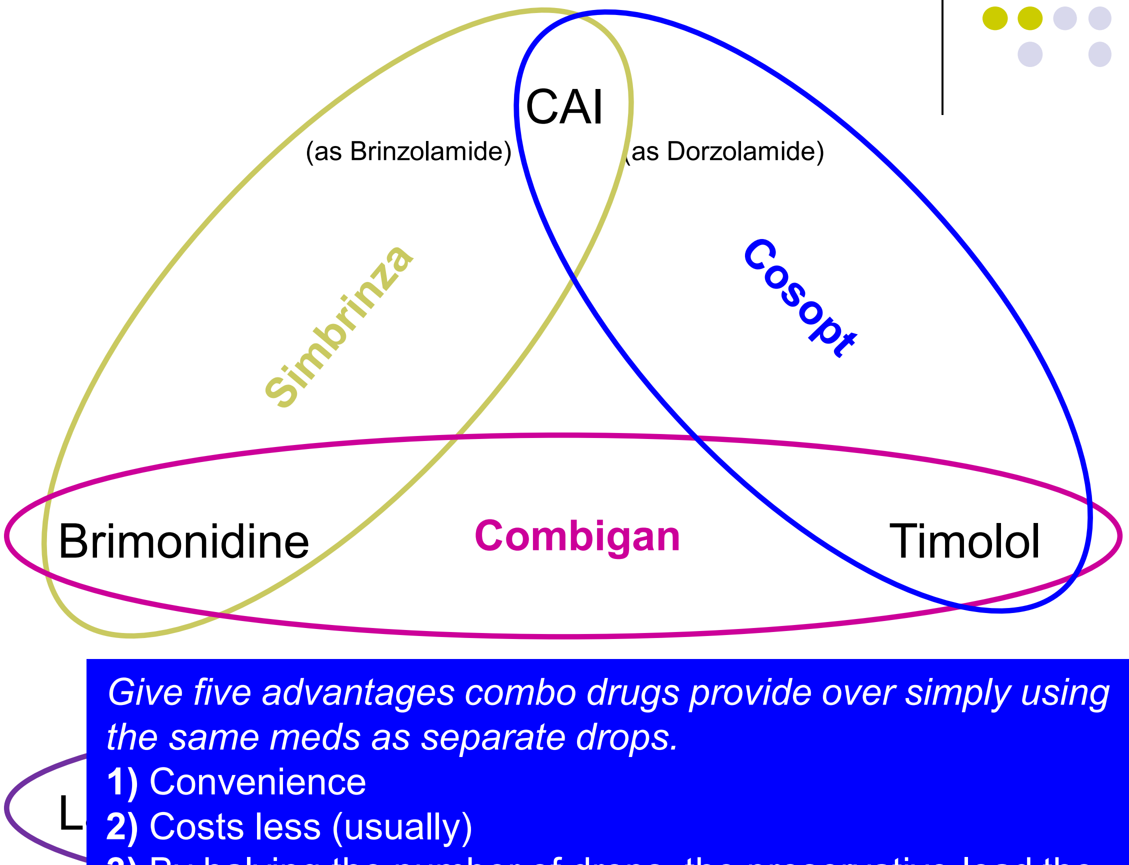

Give five advantages combo drugs provide over simply using the same meds as separate drops.

1) Convenience
2) Costs less (usually)
3) By halving the number of drops, the preservative-load the ocular surface must endure is halved as well, thus making irritation less of an issue
4) Improved **c**ompliance
5) Eliminates washout (ie, when an impatient pt instills their second drop too soon after the first, thereby washing it out)

Q

Ocular Hypotensives: List the common agents

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  - Timolol
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- *Rho kinase inhibitor*
  - **Netarsudil**

What is the standard dosing frequency for latanaprost?

Latanaprost Rocklatan Netarsudil

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - **Latanaprost**
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  - Apraclonidine
  - Brimonidine
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What is the standard dosing frequency for latanaprost?
Daily

Latanaprost Rocklatan Netarsudil

Q

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  - Brinzolamide
  - Acetazolamide
- *Selective α agonists*
  - Apraclonidine
  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - **Netarsudil**

What is the standard dosing frequency for latanaprost?
Daily

What is the standard dosing frequency for netarsudil?

Latanaprost Rocklatan **Netarsudil**

A

Ocular Hypotensives: List the common agents

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

Latanaprost   Rocklatan   **Netarsudil**

Q

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**

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?

Latanaprost Rocklatan Netarsudil

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
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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

Latanaprost Rocklatan Netarsudil

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
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  - Brimonidine
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  - 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?

Latanaprost Rocklatan **Netarsudil**

A

Ocular Hypotensives: List the common agents

- *β blockers*
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  - Apraclonidine
  - Brimonidine
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  - 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**

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  - **Netarsudil**

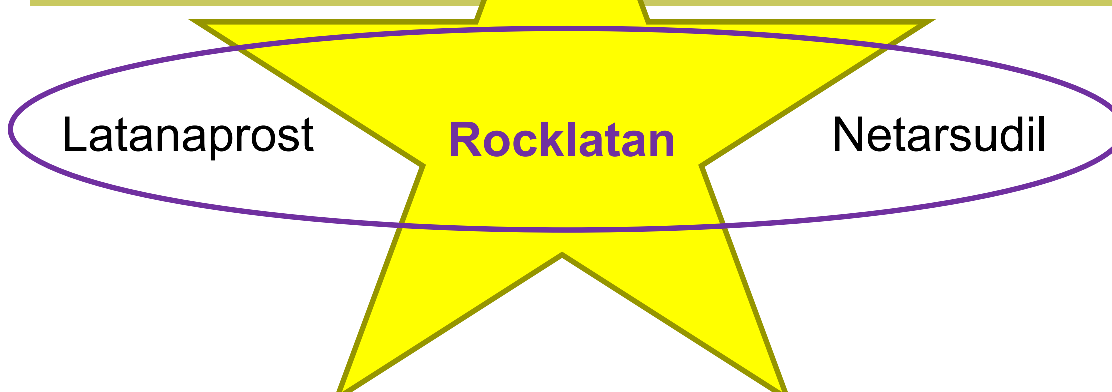
What is the standard dosing frequency for latanaprost?
Daily

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….



Q

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

A

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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?

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So if a pt is on brimonidine, what drop ***should*** *you use to blunt a post-procedure IOP spike?*

A

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

1)

2)

A

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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

Q

Ocular Hypotensives: List the common agents

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What is the feared side effect of pilo?

A

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Retinal tears

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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

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- *β blockers*
  - Timolol
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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

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

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What are these two α receptor subtypes called?
They are called α_1 and α_2

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

α_1:
--?
--?
--?

α_2:
--?

tors

What are these two α receptor subtypes called?
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Q/A

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α_1:

--Vasoconstriction

--Pupil one word

--Eyelid one word

α_2:

--?

What are these two α receptor subtypes called?

They are called **α_1 and α_2**

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

α_1:
--Vasoconstriction
--Pupil mydriasis
--Eyelid retraction

α_2:
--*?*

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--Pupil mydriasis

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α_2:

--Reduced acronym

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--Pupil mydriasis

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--Reduced IOP

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Note: The *Glaucoma* book states that **neuroprotection** is another ‘possible’ effect of α_2 stimulation

--Pupil mydriasis
--Eyelid retraction

α_2:
--Reduced IOP *and ‘neuroprotection’?*

- *Nonselective α/β* ... *tors*
  - Epinephrine
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Note: The *Glaucoma* book states that **neuroprotection** is another 'possible' effect of α_2 stimulation. That said, it doesn't elaborate on this claim, or explain what is meant by 'neuroprotection' (in fact, the term doesn't even appear in the index)

--Pupil mydriasis
--Eyelid retraction

α_2:
--Reduced IOP *and 'neuroprotection'?*

- *Nonselective α/*
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Which agent is notoriously allergenic?

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

Which agent is ***notoriously*** *allergenic?*

Iopidine

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

*Which agent is **notoriously** allergenic?*
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There are two classic manifestations of iopidine sensitivity—what are they?

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Q/A

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--[two words] of the lid and periorbital skin

--[type (one word)] conjunctivitis

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--Toxin
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Q/A

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

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A high propensity for the development of tachyphylaxis

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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 y
(ie, cau
'toxin' such as iopidine. What are the other two?
--Toxin
--Viral infection
--Chlamydia infection

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

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?*

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

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

Q

Ocular Hypotensives: List the common agents

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

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

?

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

*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

A

Ocular Hypotensives: List the common agents

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

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

Yes

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

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

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

*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?

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

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%

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

*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

If a pt is known to be allergic to iopidine, is it a given that s/he will be allergic to brimonidine?

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

*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

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?

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** (and methazolamide)
- *Selective α agonists*
  - Apraclonidine
  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

Which of these is available PO?

Acetazolamide and **methazolamide**

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?
Acetazolamide and **methazolamide**

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

--?
--?

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

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:
--
two specific issues
--
--Bitter classic descriptor taste
--Nephrolithiasis

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

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?
Acetazolamide and **methazolamide**

What are the common systemic side effects of PO CAIs?
--Malaise/fatigue/depression
--Paresthesias

How do the parasthesias typically manifest?

--Bitter ('metallic') taste
--Nephrolithiasis

Q/A

Ocular Hypotensives: List the common agents

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

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

What are the common systemic side effects of PO CAIs?
--Malaise/fatigue/depression
--Paresthesias

How do the parasthesias typically manifest?
As tingling of bodypart , bodypart and area

--Bitter ('metallic') taste
--Nephrolithiasis

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
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  - Latanaprost
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- *Rho kinase inhibitor*
  - Netarsudil

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

What are the common systemic side effects of PO CAIs?
--Malaise/fatigue/depression
--Paresthesias

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

--Thrombocytopenia
--Bitter ('metallic') taste
--Nephrolithiasis

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
- *Nonselective α/β agonist*
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  - Acetazolamide
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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?*

A

Ocular Hypotensives: List the common agents

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  - Timolol
  - Betaxolol
  - Carteolol
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  - Epinephrine
  - Dipivefrin
- *CAI*
  - **Dorzolamide**
  - **Brinzolamide**
  - Acetazolamide
- *Selective α agonists*
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  - 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?*

Q

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  - **Dorzolamide**
  - Brinzolamide
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  - 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

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

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

A

Ocular Hypotensives: List the common agents

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

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

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

Q

Ocular Hypotensives: List the common agents

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

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

Why does it sting?

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

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
- *Nonselective α/β agonist*
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  - 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

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

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

Q

Ocular Hypotensives: List the common agents

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

Topical dorzolamide is notorious for a particular adverse effe
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)?

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?*

A

Ocular Hypotensives: List the common agents

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

Topical dorzolamide is notorious for a particular adverse effe
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?*

Q

Ocular Hypotensives: List the common agents

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

Which two drugs lowers episcleral venous pressure (EVP)?

A

Ocular Hypotensives: List the common agents

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

Which two drugs lowers episcleral venous pressure (EVP)?
Iopidine and netarsudil (maybe)

Q

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

Are beta blockers known to cause significant ocular side effects?

A

Ocular Hypotensives: List the common agents

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

Are beta blockers known to cause significant ocular side effects?

No; beta blocker side effects of concern are *systemic*, not ocular

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
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  - Brimonidine
- *Miotics*
  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

Are beta blockers known to cause significant ocular side effects?

No; beta blocker side effects of concern are *systemic*, not ocular

What systemic side effects are of particular concern?

1)

2)

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

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)

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

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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?
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In population-based studies, which prostaglandin analogue has the best tolerability?

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Ocular Hypotensives: List the common agents

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They are all of very similar tolerability

So, does this mean they are all therapeutically equal?

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

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

  - Apraclonidine
  - Brimonidine
- *Miotics*
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Q

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

A

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

Q

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

1) ***Eyelash growth***

What's the $2 term for eyelash growth?

A

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

1) ***Eyelash growth***

What's the $2 term for eyelash growth?

'Hypertrichosis'

Q

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What's the $2 term for eyelash growth?
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Other deleterious eyelash changes may occur—what, specifically?

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Trichiasis and distichiasis

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What's the difference between trichiasis *and* distichiasis*?*

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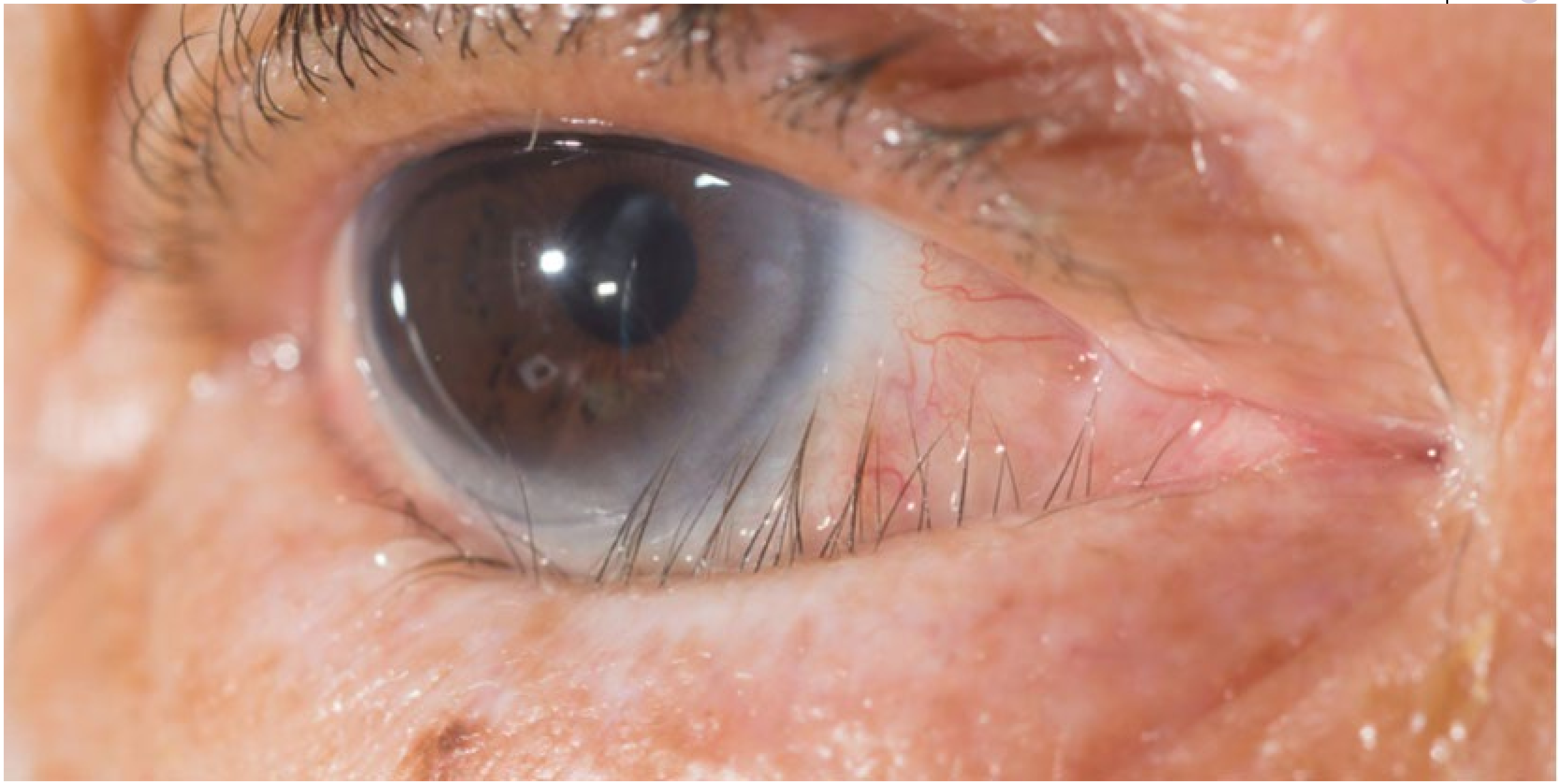
Ocular Hypotensives: List the common agents



Trichiasis

Q

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  - Carteolol
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In contrast, *distichiasis* refers to lashes abutting the surface that are growing from an abnormal location (specifically, the orifices of the two words).

A

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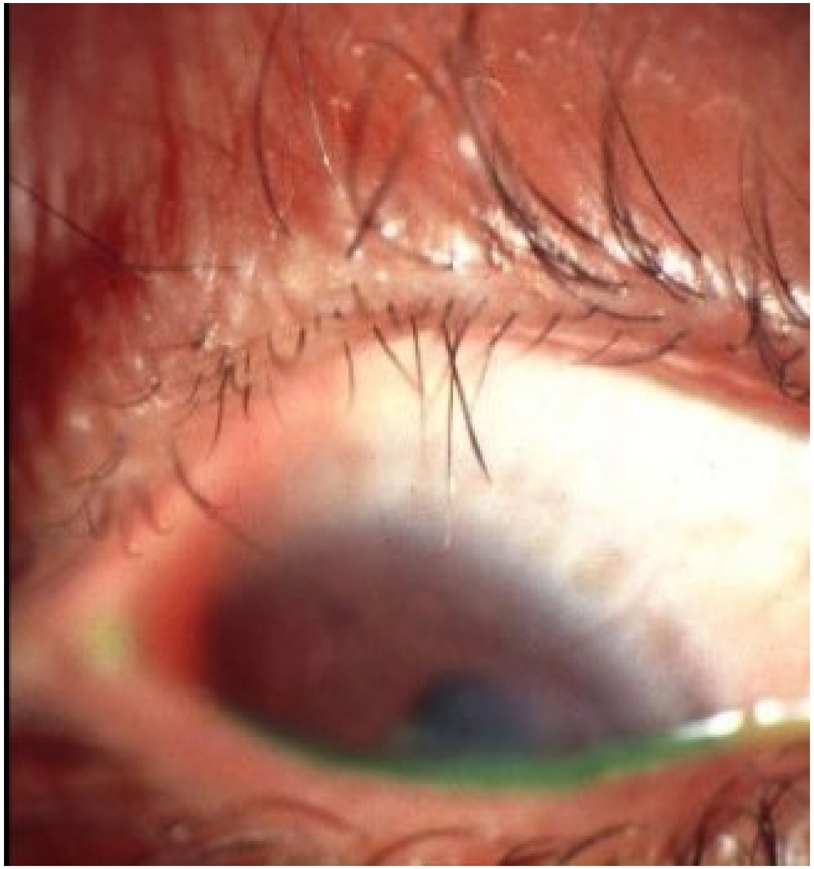
Ocular Hypotensives: List the common agents



Distichiasis: Lashes arising from MG orifices

Q

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  - Di
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:
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What's the $2 term for eyelash growth?
Hypertrichosis

Is the side effect of eyelash growth universally unwelcomed?

What's the difference between trichiasis *and* distichiasis*?*
Trichiasis refers to lashes directed against the ocular surface *<u>that originate from their normal anatomic location on the lid margin</u>*. In contrast, *distichiasis* refers to lashes abutting the surface that are growing from an abnormal location (specifically, the orifices of the meibomian glands).

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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:
1) ***Eyelash growth***

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

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

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1) ***Eyelash growth***

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

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

What's the difference between trichiasis *and* distichiasis*?*
Trichiasis refers to lashes directed against the ocular surface *that originate from their normal anatomic location on the lid margin*. In contrast, *distichiasis* refers to lashes abutting the surface that are growing from an abnormal location (specifically, the orifices of the meibomian glands).

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

1) ***Eyelash growth***

What's the $2 term for eyelash growth?

Hypertrichosis

Is the side effect of eyelash growth universally unwelcomed?

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

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Ocular Hypotensives: List the common agents

Latisse

Ocular Hypotensives: List the common agents

(Note the active ingredient)

Latisse

Q

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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

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

A

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4) Cystoid macular edema (CME)

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How can you minimize the cosmetic impact of acute hyperemia?

Q/A

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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 daily event, when cosmesis is not an issue

A

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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

Q

Ocular Hypotensives: List the common agents

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1) Eyelash growth

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3) ***Darkening of irides***

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

- Netarsudil

Q/A

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- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost

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

1) Eyelash growth
2) Conjunctival hyperemia
*3) **Darkening of irides***

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

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

- Netarsudil

A

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- Netarsudil

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Green-brown and **yellow-brown** (aka ***hazel***)

(Full disclosure: Being significantly red-green colorweak myself, I deferred to my wife to select the font color best representing 'hazel.' So if you disagree, take it up with her.)

Q

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Q/A

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

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3) ***Darkening of irides***
4) Cystoid macular edema (CME)

What proportion of pts will experience darkening of their irides after 5 years of PGA use?
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How likely are **green**-**brown** and ***hazel*** *irides to darken?*
Very—80%+ will do so after 5 years

Q

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What about ***blue eyes****—are they at high risk as well?*

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

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Q

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

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The periocular skin

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Overal … ained)

In one word (ending with –tic), what sort of process is responsible for the darkening of the irises and/or periocular skin?

Will the …

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What specific aspect of the melanocytic process is responsible?
Before we answer, let's sidebar to review this process…

Q

Ocular Hypotensives: List the common agents

What cell is responsible for melanocytic processes?

A

Ocular Hypotensives: List the common agents

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Hurr durr, Imma guess **melanocytes**?

Q

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Let's consider the embryology of melanocytes. From which primordial cell do they derive?

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Let's consider the embryology of melanocytes. From which primordial cell do they derive?
Neural crest cells (NCCs)

Q

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Briefly, what's the backstory on neural crest cells?

Q/A

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NCCs are a subtype of embryo cell type cells

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Briefly, what's the backstory on neural crest cells?
NCCs are a subtype of neuroectodermal cells. Early v Late in embryogenesis, some of the neuroectodermal cells located along the dorsal v ventral aspect of the structure (two words) are induced to transition into NCCs.

A

Ocular Hypotensives: List the common agents

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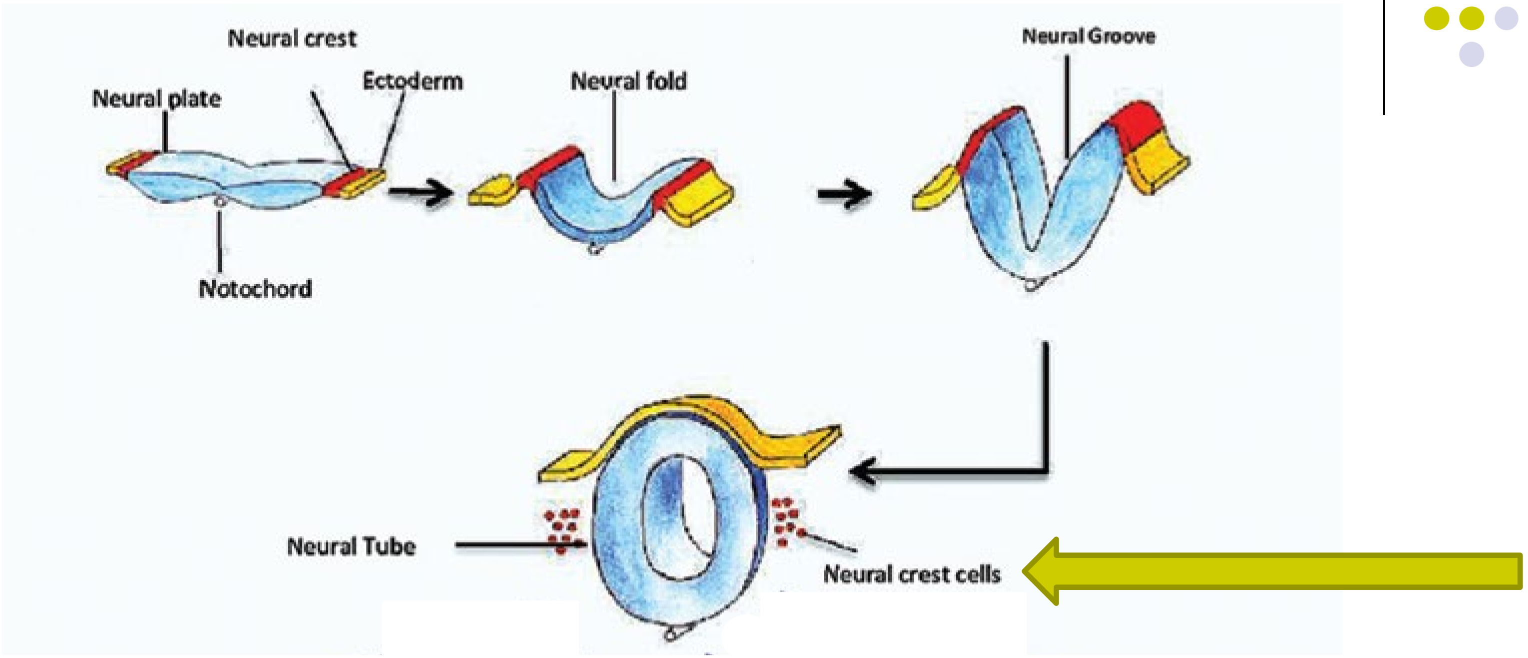
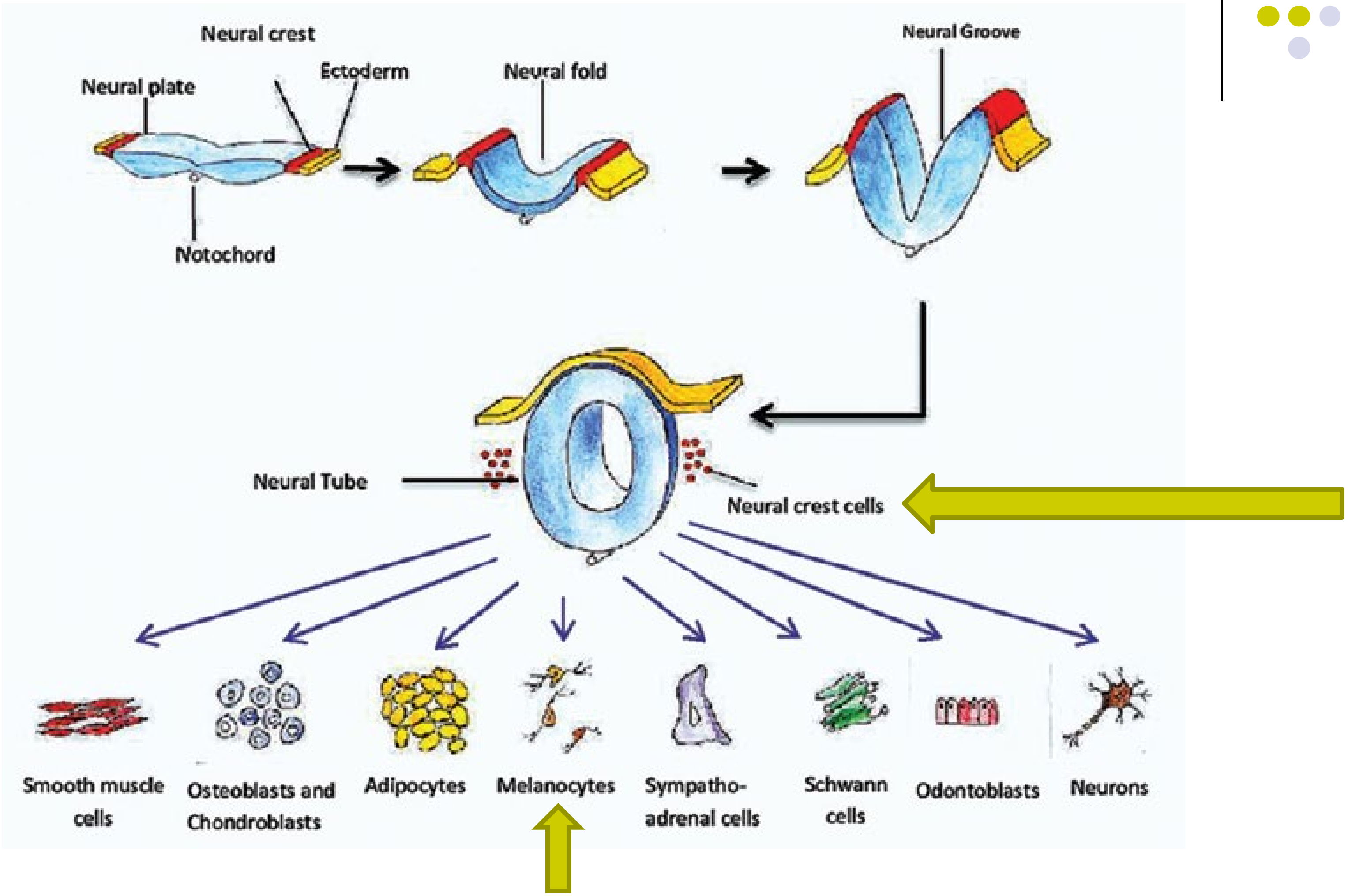
Briefly, what's the backstory on neural crest cells?
NCCs are a subtype of neuroectodermal cells. Early in embryogenesis, some of the neuroectodermal cells located along the dorsal aspect of the neural tube are induced to transition into NCCs. NCCs then migrate widely across the embryo, and upon arriving at their destination they proliferate and differentiate into specialized tissues and cells, including melanocytes.

Ocular Hypotensives: List the common agents



Neural crest cells…

Ocular Hypotensives: List the common agents



Neural crest cells…and their derivatives

Q

Ocular Hypotensives: List the common agents

Next let's consider the function *of surface melanocytes. What do they do?*

Q/A

Ocular Hypotensives: List the common agents

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A surprising number of things, but for our purposes their function is manufacturing
, the main pigment of the body surface

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Do melanocytes hang onto their melanosomes?

Q/A

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Do melanocytes hang onto their melanosomes?
No—once packaged in melanosomes, melanin is transferred to neighboring cells (eg, skin melanocytes transfer their melanin to nearby cell type)

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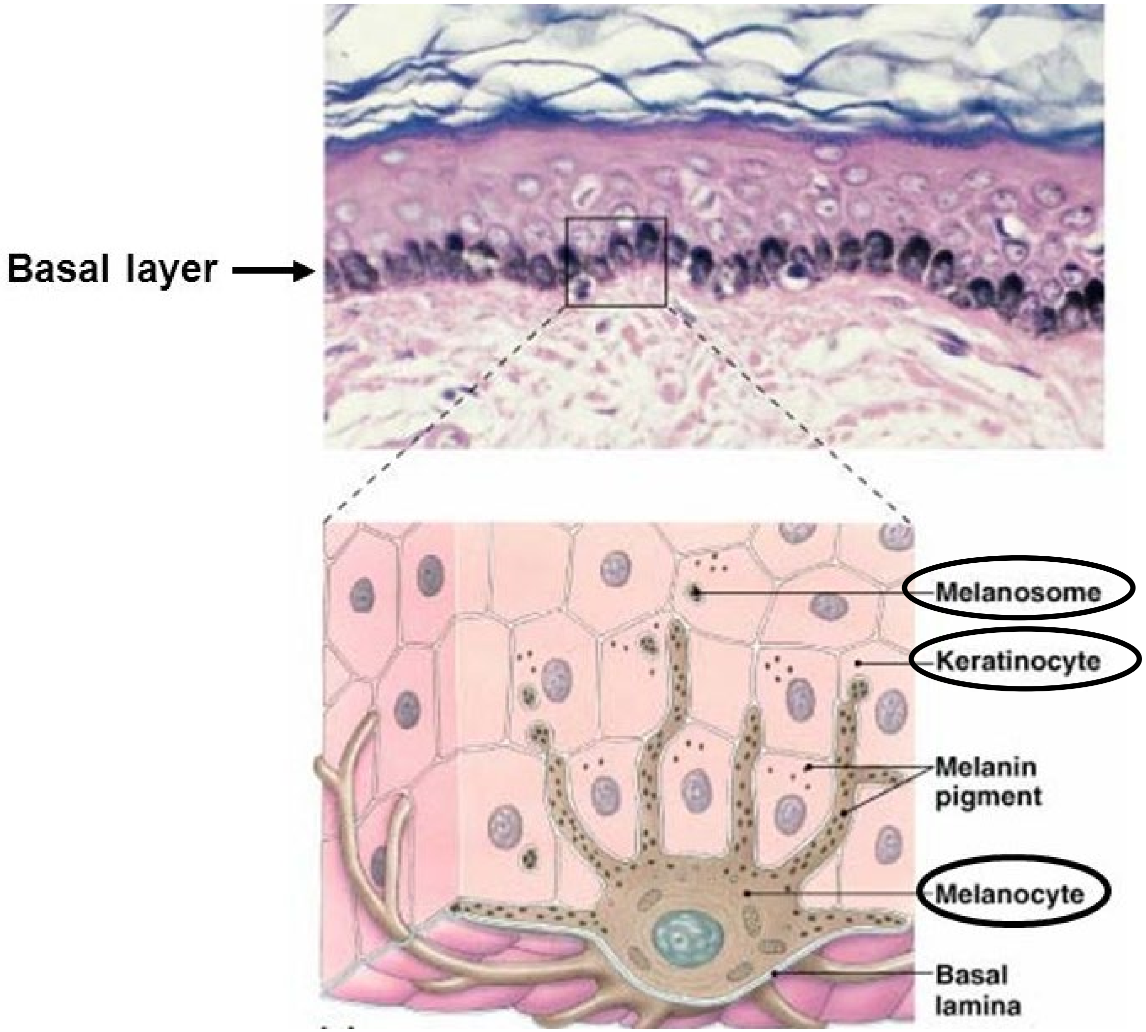

Melanocyte and its keratinocytes

Q

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Some people have darker skin than others. (Thanks, Captain Obvious.) Is it the case that darker-complected individuals have more melanocytes?

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No, the number of melanocytes does not vary with degree of pigmentation. People with darker complexion have more melanin in their keratinocytes.

Q

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2) Conjunctival hyperemia
3) ***Darkening of irides***

What p... PGA use?
Overal... ...ined)

In one word (ending with –tic), what sort of process is responsible for the darkening of the irises and/or periocular skin?
It is a melanocytic process

What specific aspect of the melanocytic process is responsible?
Before we answer, let's sidebar to review this process…

Will the...
It wi...

Now for the 'higher risk' issue: Of the myriad colors the human iris can assume, the
B...
W...
G...

Now that the melanocytic process *sidebar is complete, let's answer this question: What specific aspect of the melanocytic process is responsible for the darkening of the iris and periocular skin 2ndry to PGA use?*

In... ...of PGA use—what is it?
The periocular skin

- Netarsudil

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
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Increased numbers of melanosomes within melanocytes

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Increased numbers of melanosomes within melanocytes. Worth emphasizing what's **not** the cause, and that's melanocyte proliferation (which doesn't occur).

Q

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Does all this melanocytic mischief put PGA users at an increased risk of developing melanoma of the iris and/or periocular skin?

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No it doesn't

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Does all this melanocytic mischief put PGA users at an increased risk of developing melanoma of the iris and/or periocular skin?
No it doesn't, and this shouldn't come as a surprise given that melanocytic proliferation is **not** a component of the darkening response.

- *In... of PGA use—what is it?*
  The periocular skin
-
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  -
  -
- *CA*
  -
  -
  -
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  - Apraclonidine
  - Brimonidine
- *Miotics*
  - Pilo
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Prostaglandin analogues have a number of notable side effects. Identify 5 of them:

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

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

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What renders a pseudophakic eye even more likely to PGA-associated CME?

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An open posterior capsule (s/p either intra-op rupture, or YAG)

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Does aphakic status also convey an increased risk of CME?

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Does aphakic status also convey an increased risk of CME?
Indeed it does

Q

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What is Prostaglandin-Associated Periorbitopathy *(PAP)?*

Q/A

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A constellation of orbital/periorbital changes 2ndry to

4ish words

A

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What are the classic/typical manifestations of PAP?
--***?***
--***?***
--***?***
--***?***
--***?***

Q/A

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What are the classic/typical manifestations of PAP?
--Enopth並thalmos
--Deepening of the lower vs upper -lid sulcus
--*?*
--*?*
--*?*

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--*?*
--*?*

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--Deepening of the upper -lid sulcus
--Ptosis of the lower vs upper lid
--?
--?

A

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--*?*
--*?*

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--Ptosis of the upper lid
--Inferior two words
--*?*

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--Ptosis of the upper lid
--Inferior scleral show
--***?***

Q

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--A 'one word' orbit

A

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--Ptosis of the upper lid
--Inferior scleral show
--A 'tight' orbit

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

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 2ndry to atrophy of periorbital fat

What are the classic/typical manifestations of PAP?
--Enopthhalmos
--Deepening of the upper -lid sulcus
--Ptosis of the upper lid
--Inferior scleral show
--A 'tight' orbit

Is PAP reversible with cessation of PGA therapy?

A

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Is PAP reversible with cessation of PGA therapy?
As of this writing, this remains unsettled

Q

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Note that of the five side effects identified, four are related to ***cosmesis****. This implies that caution should be exercised in long-term use of PGAs in one group of pts (other than supermodels). Which pts are these?*

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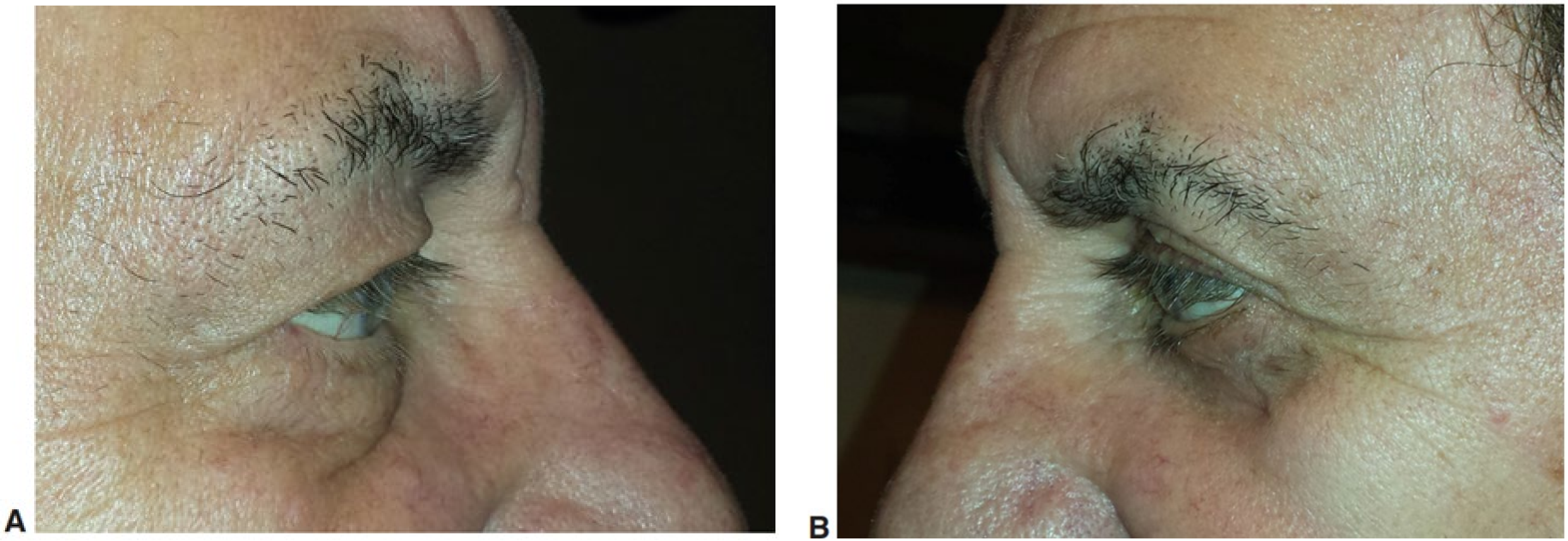
Those with **unilateral** glaucoma

Ocular Hypotensives: List the common agents

Unilateral hypertrichosis following latanoprost use OS only

Ocular Hypotensives: List the common agents



The right (A) and left (B) eyes of a patient on unilateral treatment with a topical prostaglandin analogue for the left eye. Left-sided periorbital skin hyperpigmentation, hypertrichosis, deepening of the superior eyelid sulcus, and loss of periorbital fat are evident.

Q

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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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Uveitis

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Another commonly-used med on the list is notorious for causing a granulomatous anterior uveitis. Which one?

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What corneal condition is a strong contraindication to PGA use?

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What corneal condition is a strong contraindication to PGA use?
HSV keratitis

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Are we talking about active dz only, or does this apply also to a history *of HSV keratitis?*

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Why the contraindication?

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What corneal condition is a strong contraindication to PGA use?
HSV keratitis

Are we talking about active dz only, or does this apply also to a history of HSV keratitis?
Both

Why the contraindication?
PGA use has been associated with prolongation and/or recurrence of HSV keratitis

Q

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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)

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Which class must be used cautiously in patients who take MAOIs and/or tricyclics?
The selective α agonists

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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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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.

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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.

Q

Ocula

What is the 'nonresponder' rate for the β blockers, ie, what percent of pts will not manifest a meaningful decrease in IOP?

- ***β blockers***
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  - **Betaxolol**
  - **Carteolol**
- *Prostaglandin analo*
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Ocular

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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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What is the 'nonresponder' rate for the β blockers, ie, what percent of pts will not manifest a meaningful decrease in IOP?
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What is a well-known cause of nonresponding that should probably keep you from trying a β blocker in the first place?
If the pt is on a **systemic** β blocker (eg, for HTN). In such pts, a topical β blocker may not move IOP much.

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With respect to pregnancy, and under the former system of classifying drugs:

(No question yet—proceed when ready)

Q

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Which are Class A?

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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?

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:

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Which are Class B?

Brimonidine. (The rest are all Class C.)

Q

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OK then, how should glaucoma be managed during pregnancy?

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Ocular Hypotensives: List the common agents

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With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A?
None of them

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

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

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Ocular Hypotensives: List the common agents

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  - Timolol
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With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A?

None of them

What treatment options fall under 'none'?

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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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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

What treatment options fall under 'none'?

--Suspend all treatment during pregnancy--just monitor the pt, and resume tx after delivery

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--?

OK then, how should glaucoma be managed during pregnancy?

With as few meds as possible (preferably **none**)

Q

Ocular Hypotensives: List the common agents

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  - Timolol
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  - Latanaprost
  - Travaprost
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  - Apraclonidine
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- *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

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 abb.
--?

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

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
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  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A?

None of them

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

--?

OK then, how should glaucoma be managed during pregnancy?

With as few meds as possible (preferably **none**)

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
- *Nonselective α/β agonist*
  - Epinephrine
-
  - 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

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 two words

OK then, how should glaucoma be managed during pregnancy?

With as few meds as possible (preferably **none**)

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
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  - Pilo
- *Rho kinase inhibitor*
  - Netarsudil

With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A?

None of them

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**)

Q

Ocular Hypotensives: List the common agents

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

Q/A

Ocular Hypotensives: List the common agents

- β blockers
  - **Timolol %?**
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
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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 % strength rather than the usual %

A

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
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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%

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - **Timolol 0.25**
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
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With respect to pregnancy, and under the former system of classifying drugs:

Which are Class A?
None of them

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

You probably know that the cap color for $T_{.5}$ is…

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

If meds are to be used, which is the best option?
Most experts would probably recommend timolol, but at the 0.25% strength rather than the usual **0.5%**

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With respect to pregnancy, and
former system of classifying dru

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%**

Q

Ocular Hypotensives: List the common agents

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Which are Class A?
None of them

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*You probably know that the cap color for $T_{.5}$ is…***yellow**

But do you know the cap color for $T_{.25}$?

d glaucoma be managed during pregnancy?
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None of them

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*You probably know that the cap color for $T_{.5}$ is…***yellow**

But do you know the cap color for $T_{.25}$? **Light blue**

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Won't the 0.25 strength be only half as effective as the 0.5?

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Far from it. In fact, in many pts, it works just as well

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Which are Class A?
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What about nursing mothers—should $T_{.25}$ be used for them as well?

...ged during pregnancy?
With as few meds as possible (preferably **none**)

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Far from it. In fact, in many pts, it works just as well

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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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What about nursing mothers—should $T_{.25}$ be used for them as well?
No, because β blocker metabolites get concentrated in breast milk

...ged during pregnancy?
With as few meds as possible (preferably **none**)

Won't the 0.25 strength be only half as effective as the 0.5?
Far from it. In fact, in many pts, it works just as well

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Why not use a PGA in pregnant women?

Which are Class B?

Brimonidine. (The rest are all Class C.)

Q/A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
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Why not use a PGA in pregnant women?
Reach way back to your Ob/Gyn rotation and recall that prostaglandins are involved in two words.

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

Q/A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
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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 .

Which are Class B?
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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.

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

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
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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?

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
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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 *Glaucoma* book states flatly that "oral CAIs should not be used by women in their childbearing years."

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
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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 *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 abb., a condition most commonly found in women in their childbearing years. Caveat emptor.)

A

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
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  - Latanaprost
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- *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?
CAIs have been shown to be teratogenic in mice. For this reason, the *Glaucoma* book states flatly that "oral CAIs should not be used by women in their childbearing years."
(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.)

Q

Ocular Hypotensives: List the common agents

- *β blockers*
  - Timolol
  - Betaxolol
  - Carteolol
- *Prostaglandin analogues*
  - Latanaprost
  - Travaprost
  - Bimataprost
- *Nonselective α/β agonist*
  - Epinephrine
  - Dipivefrin
- *CAI*
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What does IIH *stand for in this context?*
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As for **topical** CAIs in pregnancy, the *Glaucoma* book doesn't address them directly.