



#2

Pharmacology of drugs acting on the eye

Objectives:

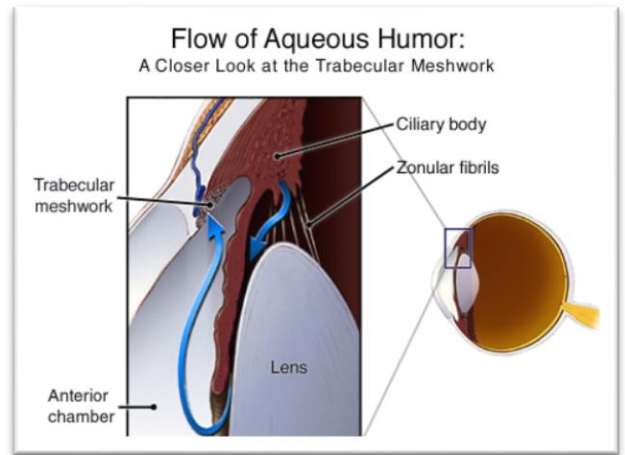
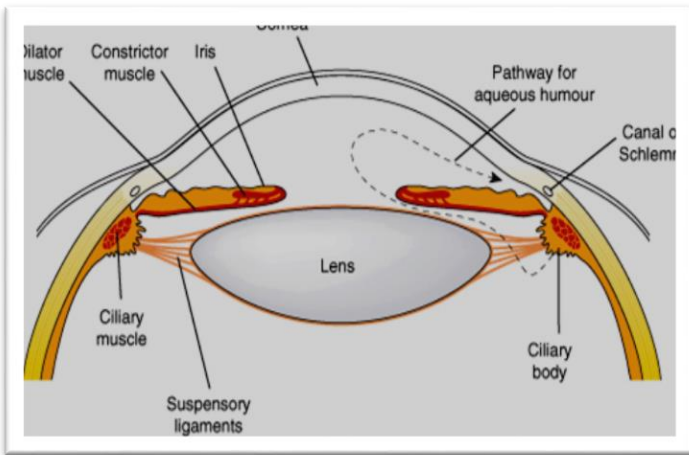
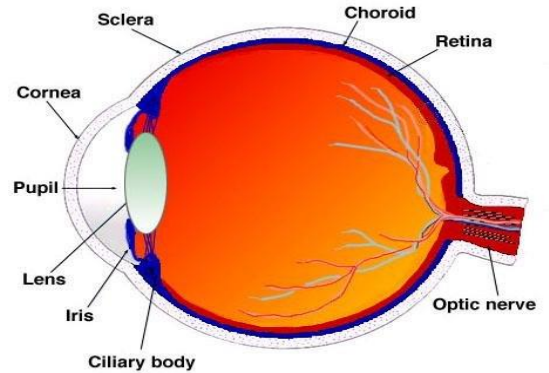
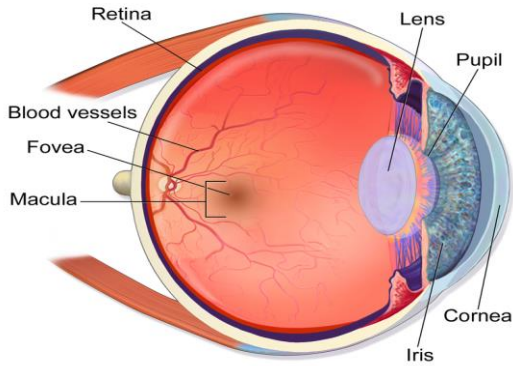
- Outline common routes of administration of drugs to the eye.
- Discuss the pharmacokinetics of drugs applied topically to the eye.
- Classify drugs used for treatment of disorders of the eye.
- Outline ocular toxicity of some drugs.
- Elaborate on autonomic drugs, anti-inflammatory drugs, and drugs used for glaucoma

Color index:

- Drugs names
- Doctors notes
- Important
- Extra

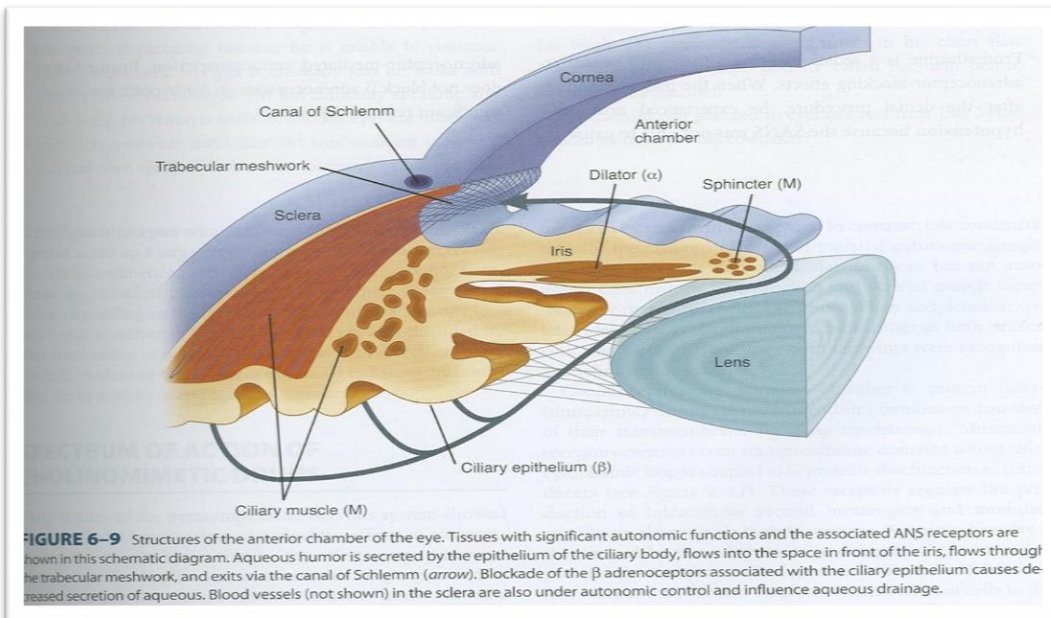
To understand better

Anatomy of the Eye:



The aqueous humor is secreted by the epithelium of ciliary body. Produced by a combination of active transport of ions and ultrafiltration of interstitial fluid. The fluid flows over the surface of the lens, out through the pupil into the anterior chamber. Flows through the trabecular meshwork into Schlemm's canal and is collected in the scleral veins.

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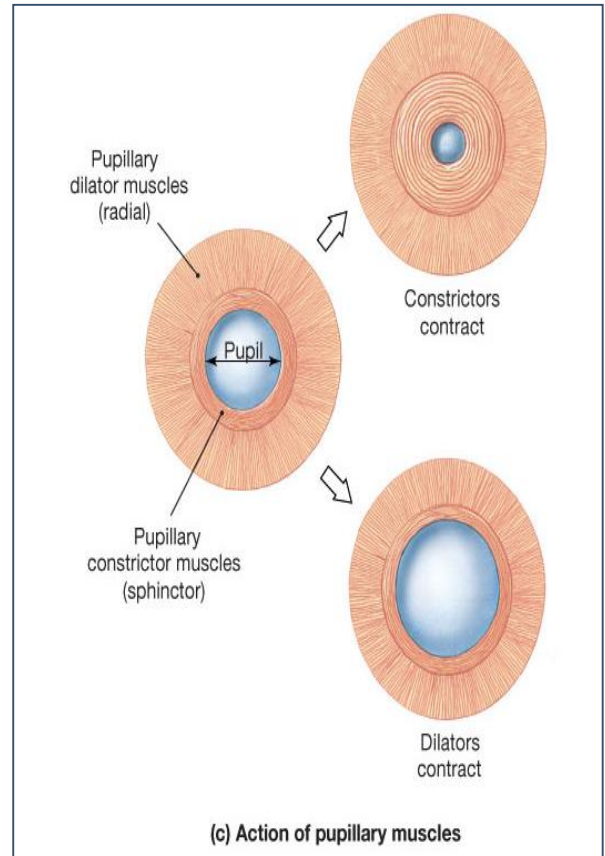
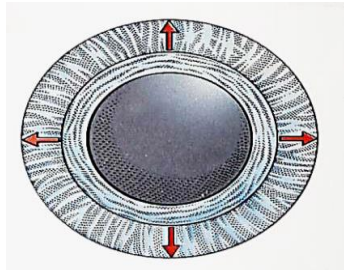
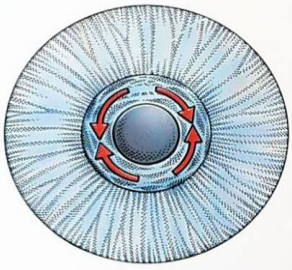
To understand better

Accommodation for **light intensity**:

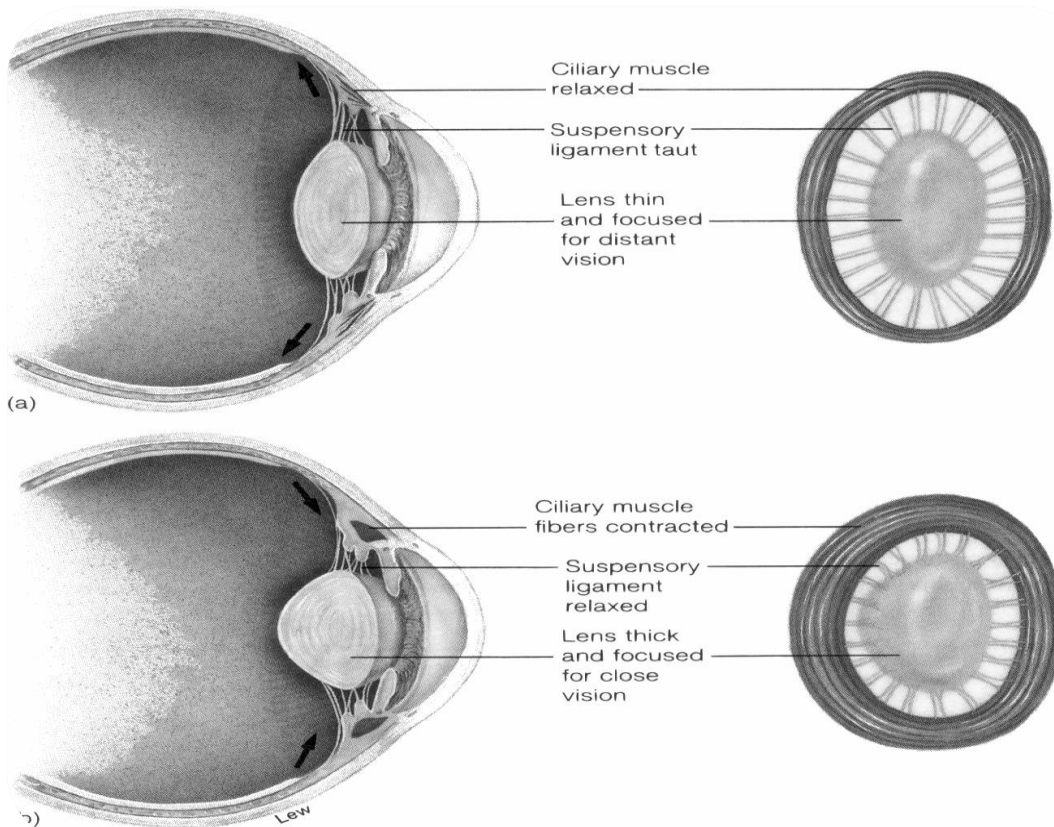
Pupillary Muscles

Miosis

Mydriasis



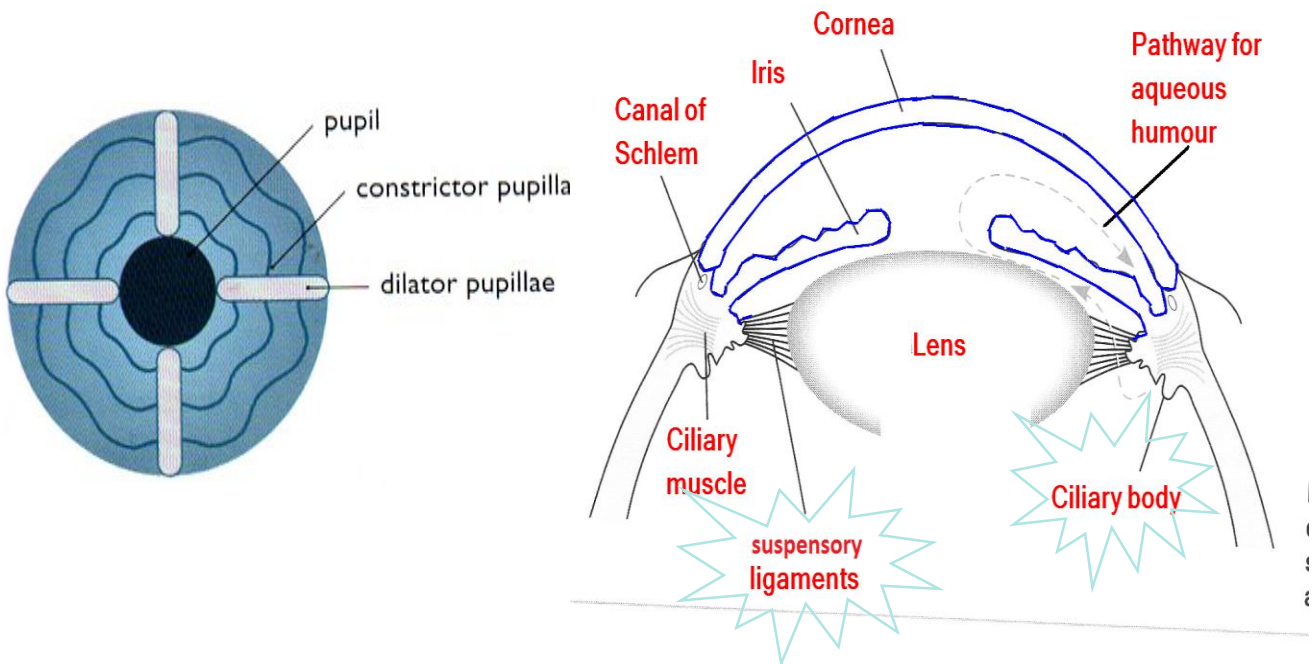
Accommodation for **near/far vision**:



Extra recall: 1st year, MSk block

❖ The iris has two muscles that control light intensity:

1. **Dilator pupillae**, a longitudinal radial muscle which **dilates** the pupil (mydriasis) in the dark, to allow as much as possible of light to enter the eye. It is innervated by **sympathetic** NS.
2. **Constrictor pupillae**, a circular muscle which **constricts** the pupil (miosis) in places with good lighting. It is innervated by **parasympathetic** NS.



❖ Accommodating the ciliary muscle for near vision:

- Parasympathetic activation contracts **the ciliary muscle**. (when ACh combines with muscarinic M3 receptor).
- Contraction of ciliary muscle pulls the **ciliary body** forward & inward, relaxing **the suspensory ligaments** of the lens (lens becomes spherical).

(Contraction of ciliary body = relaxation of the suspensory ligaments, and vice versa).

- The lens bulges more (increased curvature), this causes a decrease in focal length.
- This parasympathetic reflex is essential to accommodate for **near vision**.

Extra recall: 1st year, MSk block (cont.)

❖ Constrictor pupillae is important for:

- 1- Adjusting the pupil in response to change in light intensity.
- 2- Regulating the intraocular pressure.

Regulating the intraocular pressure:

In the healthy eye:

- Aqueous humour is secreted by the cells of the epithelium covering the ciliary body.
- Increased tension in the ciliary body removes the Aqueous humour continuously by drainage into the canal of Schlemm.
- Normal intraocular pressure is 10-15mmHg above atmospheric pressure.

In some people:

- Dilatation of their pupil will block canal of schlemm, therefore it impedes drainage of aqueous humour.
- The accumulation of aqueous humour leads to an increase in intraocular pressure.
- ↑ IOP may lead to glaucoma, and retinal detachment.

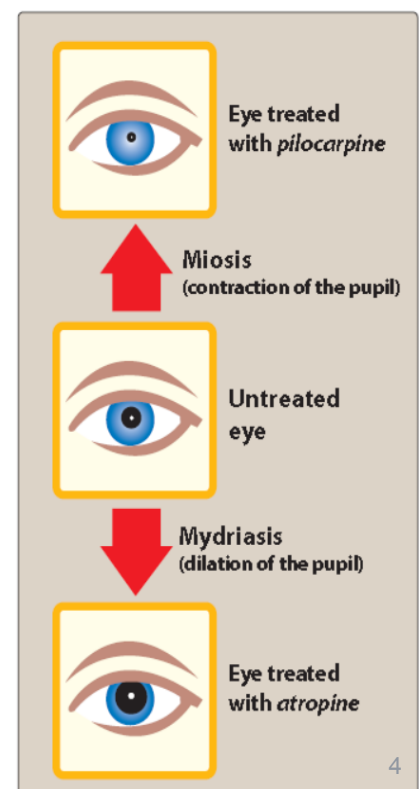
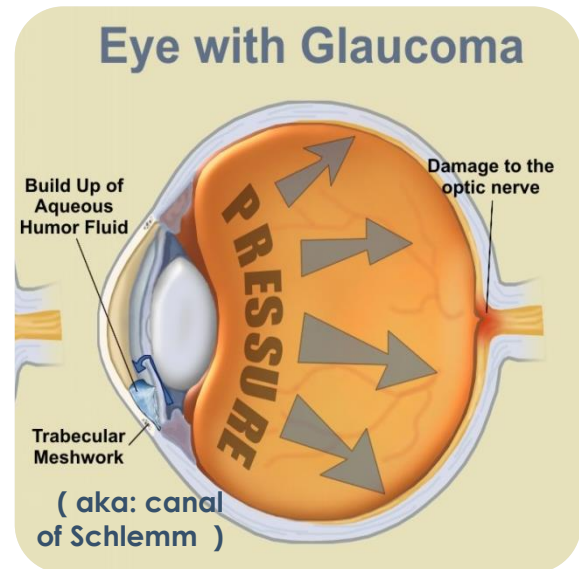
(Retinal detachment describes an emergency situation in which a critical layer of tissue (the retina) at the back of the eye pulls away from the layer of blood vessels that provides it with oxygen and nourishment).

Treatment:

When using **cholinergic drugs** (e.g. **pilocarpine**), constrictor pupillae causes miosis, which contracts the pupil away from canal of schlemm, leading to increased filtration of Aqueous humour.

Thus, activation of constrictor pupillae decreases intraocular pressure in patients with glaucoma.

Development of Glaucoma Animation.



To understand better

Autonomic Nerve supply of the Eye:

Parasympathetic N.S.

= Cholinergic drugs action

- **Constriction** of the **pupillary sphincter** "constrictor/circular" muscle (**miosis**)
- **Contraction** of the **ciliary muscle** (**accommodation for near vision**).
- Decrease in intraocular pressure
↓ **IOP.** → increases aqueous **outflow** through the trabecular meshwork into canal of Schlemm by ciliary muscle contraction.
- Increased lacrimation (التدمع)
- Conjunctival Vasodilatation

Sympathetic N.S.

- **Contraction** of dilator "radial" Pupillae (**Active mydriasis**)* → α_1 → mean the iris go to the back.
- Relaxation of ciliary muscles (**accommodation for far vision**) β_2 ** = reduce filtration angle.
- Increase in intraocular pressure
- Lacrimation α_1
- **Vasoconstriction** of conjunctival blood vessels α_1 . "α & β receptors in the blood vessels of the ciliary processes help in regulation of aqueous humour formation."

* Active vs. passive mydriasis:

- Atropine (anticholinergic): **Blocking** muscarinic receptors → **relaxing circular** muscles → **Passive Mydriasis**
- Sympathetic stimulation: **activation** of α receptors in **radial muscles** → **contraction** → **Active mydriasis**

** in the sympathetic system, activation of α receptors leads to smooth muscle contraction, and activation of β2 receptors leads to smooth muscle relaxation.

You have to know the effects of autonomic actions, because they are the same effects of autonomic (symp. & paraS.) drugs!

Eye		Parasympathetic N.S. (near vision)	Sympathetic N.S. (far vision)
Iris:	- radial muscle	No effect	Contraction (Mydriasis) (α1)
	- circular muscle	Contraction (miosis) (M3)	No effect
Ciliary muscle		Contraction (M3)	Relaxation (β2)
Lens		Thick, more convex	Thin, more flat
Conjunctival blood vessels		Conjunctival Vasodilatation	Conjunctival Vasoconstriction (α1)

Examples to help you remember:

- In a parasympathetic condition a person is in a "rest and digest" state, this person is for example laying on the couch and reading a book. His iris circular muscle is constricted because he is in a place with good lighting, and his ciliary muscle is also constricted to accommodate for near vision, his conjunctival blood vessels are relaxed and dilated.
- In a sympathetic condition "fight and flight", a person is being chased by a lion at dawn. Iris radial muscle is constricted to dilate the pupil and allow it to receive more light, and the ciliary muscle is relaxed to accommodate for far vision, the conjunctival blood vessels are constricted to shift the blood flow to his skeletal muscles.

Pharmacology of drugs acting on the eye

Drugs can be delivered to ocular tissue as:

Locally (Topically): *more common*	Systemically:
<ul style="list-style-type: none"> - Eye drops. - Ointments. - Injections: <ul style="list-style-type: none"> - Periocular injection. - Intraocular injection. <p>Advantages:</p> <ul style="list-style-type: none"> - Convenient (مريح), Economic (رخيص), Relatively safe. <p>Disadvantages:</p> <ul style="list-style-type: none"> - Compliance, Corneal & conjunctival toxicity. 	<ul style="list-style-type: none"> - Orally. - IV.

First – **locally:**

	Eye drops	Ointment (مرهم)
Definition	<ul style="list-style-type: none"> • Eye drops are saline-containing drops “liquid” • Most common route of administration. • One drop = 50 µl / 4 hours (usually) 	Ointment is a smooth oily preparation, As a rule of thumb, an ointment base is more occlusive and will drive the medication into the skin more rapidly than a solution or cream base.
Advantages		Increases the contact time of ocular medication to ocular surface → providing better effect .
Disadvantages	The contact time between the drug and the eye is low due to fast removal by tears. → Thus has to be used several times.	The drug has to be high lipid soluble to have the maximum effect as ointment.




Eye injections:

Intra-ocular injections

For anterior segment surgery, infections & retinitis


1- Intra-cameral:
"inside anterior or posterior chamber of the eye"

 0:23 min

E.g.

- Intracameral **acetylcholine** or **lidocaine** during cataract surgery.

2- Intra-vitreous
"inside the eye"

 3:06 min

E.g.

- Intravitreal **antibiotics** in cases of **endophthalmitis** (an inflammation of the internal coats of the eye)
- Intravitreal **steroid** in **macular edema** (the build-up of fluid in the **macula**, an area in the center of the retina.)


ADRs

- Retinal toxicity.
- Intraocular toxicity.
- Corneal toxicity.

Techniques

Peri-ocular injections

1- Subconjunctival

 0:4 min

2- Retro-bulbar
"behind the eyeball"

 0:34 min

3- Peri-bulbar
"above and below the orbit"

4- subtenon  0:41 min

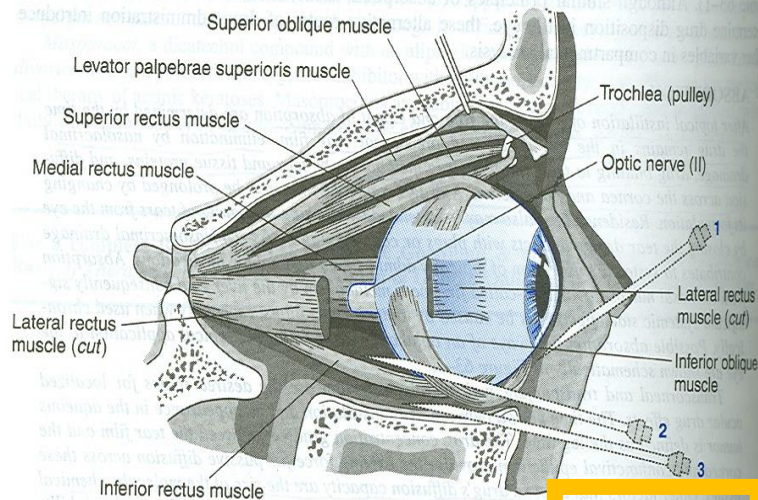


FIGURE 63-1 Anatomy of the globe in relationship to the orbit and eyelids. Various routes for anesthesia are demonstrated by the needle pathways numbered in blue.

1. Subconjunctival route
2. Retrobulbar route
3. Peribulbar route

Advantages:

- Reach behind iris-lens diaphragm **better than** topical application.
- **Drugs penetration is generally weaker for low lipid-soluble drugs**, however injections can bypass the conjunctival and corneal epithelium which is good for **drugs with low lipid solubility (e.g. penicillins)**
- **Steroid and local anesthetics** can be applied this way.
- Used for infection of anterior segment and inflammation of uvea.

Disadvantages:

- Local toxicity, tissue injury, globe perforation, optic nerve damage.

Pharmacokinetics of topical drugs:

Absorption

Drug residence time → the rate of absorption is determined by the time drug remains in cul-de-sac, tear. It can be prolonged by plugging tear ducts or change formulation. (residence time = the time in which drug will still in the eye).

Metabolism.

Elimination → by **nasolacrimal** drainage or binding to tear protein.

Diffusion → across cornea & conjunctiva.

Distribution

After corneal absorption → the drug accumulates in the **aqueous humor**, intraocular structures or systemically distributed.

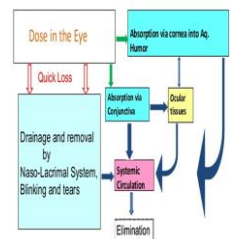
* Melanin binding prolongs the effect of **α-agonists** in patients with **dark pigmented iris**.

Metabolism

Significant biotransformation takes place in the eye.

Esterases activate pro-drugs, e.g.:

- **Dipivefrin** → (adrenaline)
- **Latanoprost** → (PGF2α)

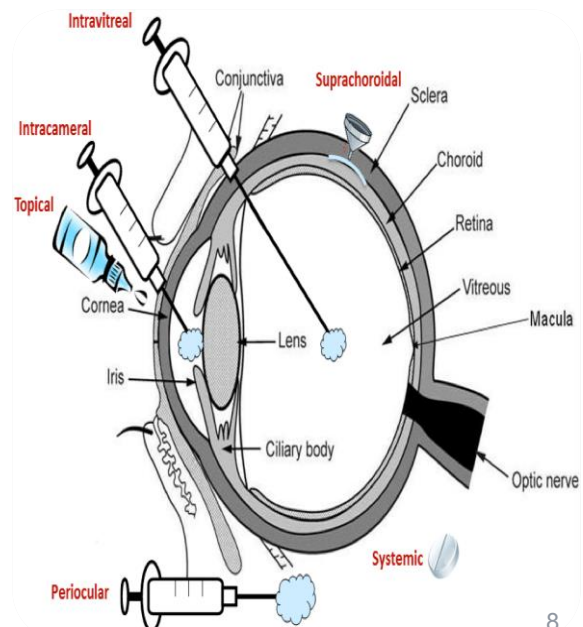


Second- systemically:

Oral or IV

- Factors influencing systemic drug penetration into ocular tissue:

- **lipid solubility of the drug:** more penetration with high lipid solubility
- **Protein binding:** more effect with low protein binding (inverse proportion)
- **Eye inflammation:** more penetration with ocular inflammation.



Treatment of **open angle glaucoma (chronic)**

The main goal is to **decrease IOP** by:



Decreasing production of aqueous humor.

- Beta blockers.
- Alpha-**2** agonists.
- Carbonic anhydrase inhibitors.

Increasing outflow of aqueous humor

- Prostaglandins.
- Adrenergic agonists, nonspecific.
- Parasympathomimetics.



Prostaglandins and Beta blockers are the most popular

Carbonic anhydrase inhibitors & prostaglanin analogues

Drug	Carbonic anhydrase inhibitors E.g. acetazolamide (oral) dorzolamide (topical) → <u>preferred</u>	Prostaglandin analogues E.g. latanoprost , travoprost
Mech. of action	- ↓ production of aqueous humor by blocking carbonic anhydrase enzyme required for production of bicarbonate ions → (transported to posterior chamber, carrying osmotic water flow).	- ↑ uveoscleral aqueous outflow. - Latanoprost is preferred due to lesser adverse effects. - They have <u>replaced beta blockers</u> . "Better than beta blockers because it has a long duration → use it once a day" They are used topically as eye drops & once a day.
Indications	open angle glaucoma	
ADRs	<ul style="list-style-type: none"> • Myopia (Nearsightedness) , malaise, anorexia, • GI upset, headache. Metabolic acidosis, renal stone. 	<ul style="list-style-type: none"> • Pigmentation of the iris (heterochromia iridis) • Intraocular inflammation. • Macular edema.
C.I	<ul style="list-style-type: none"> • Sulfa allergy → bc they are sulfa derivatives. • Pregnancy "Affects fetus" • Digitalis users. 	

Drugs acting on parasympathetic system

Cholinergic agonists

Drug	Direct agonists				Indirect agonists (anticholinesterases)				
	Methacholine	Ach	carbachol	 pilocarpine	reversible	Irreversible (phosphate ester)			
				physostigmine	demecarium	echothiophate	isofluorophate		
Indications	Specific uses	<ul style="list-style-type: none"> - Induction of miosis in surgery. - Open angle glaucoma. * Carbachol → causing pupillary contraction and a decrease in intraocular pressure. 		Open angle glaucoma * The drug of choice in <u>acute</u> attack (closed or open glaucoma)	<ul style="list-style-type: none"> • Glaucoma • Accommodative esotropia (نوع من الحول) → echothiophate.  0:23 min • In lice infestation of lashes → physostigmine. 				
	General uses	<ul style="list-style-type: none"> • Glaucoma (open & closed angle). • Counteract action of mydriatics. • To break iris-lens adhesions. • In accommodative esotropia (echothiophate). 							
ADRs	ocular	<ul style="list-style-type: none"> • Diminished vision (myopia). • Headache. 							

Cholinergic (muscarinic) antagonists

Drug	Natural alkaloids		Synthetic atropine substitutes		
	atropine	Scopolamine (Hyoscine)	homatropine	cyclopentolate	tropicamide
Duration of effect	7-10 days	3-7 days	1-3 days	24 hours	6 hours
	Long duration			Short duration	
actions	<ul style="list-style-type: none"> • Passive mydriasis → due to relaxation of circular muscles. (passive = without any effect of sympathetic) • Cycloplegia (loss of <u>near</u> accommodation) → due to relaxation of ciliary muscle. (This effect is due to blocking of paraS only!) • Loss of <u>light reflex</u>. • Increased IOP → glaucoma. (especially angle closure glaucoma) • Decreased lacrimal secretion → sandy eye. 				
Indications (uses)	<ul style="list-style-type: none"> • To prevent adhesion in uveitis & iritis. → bc they are doing mydriasis. • Funduscopy examination of the eye. • Measurement of refractive error. → problem with focusing of light on the retina due to the shape of the eye. 				
C.I	<ul style="list-style-type: none"> • Glaucoma (angle closure glaucoma) → Because there is no miosis → which makes the filtration easier → IOP may rise dangerously → acute attack of eye pain. 				

Adrenergic agonists

Drug	Non-selective agonists ($\alpha_1, \alpha_2, \beta_1, \beta_2$)		Selective α_1 agonists	Selective α_2 agonists
		epinephrine	Dipivefrin (pro-drug of epinephrine)	phenylephrine
Action / Mech. of action	- Increase uveoscleral outflow of aqueous humor.		- Active Mydriasis (without cycloplegia) . → bc their effect is on the radial muscle , not the ciliary muscle which is innervated by paraS.	- ↓ production of aqueous humor. - ↑ uveoscleral outflow of aqueous humor. - Inhibits sympathetic working.
Route of administration	Used locally as eye drops . → to minimize the ADRs			Eye drops
Indications	Open angle glaucoma.		<ul style="list-style-type: none"> Fundoscopy examination of the eye. To prevent adhesion in uveitis & iritis. Decongestant in minor allergic hyperemia of eye. 	<ul style="list-style-type: none"> Open angle glaucoma treatment Prophylaxis against IOP spiking after glaucoma laser procedures.
ADRs	<ul style="list-style-type: none"> Headache. Arrhythmia. Increased blood pressure. 		<ul style="list-style-type: none"> May cause significant increase in blood pressure. Rebound congestion. Precipitation of acute angle-closure glaucoma in patients with <u>narrow</u> angles. 	<ul style="list-style-type: none"> Headache. Bradycardia. Hypotension.
C.I	In patients with narrow angles (low drainage) as they may precipitate closed angle glaucoma. (α_1 effect) → bc it is doing <u>mydriasis</u> .			

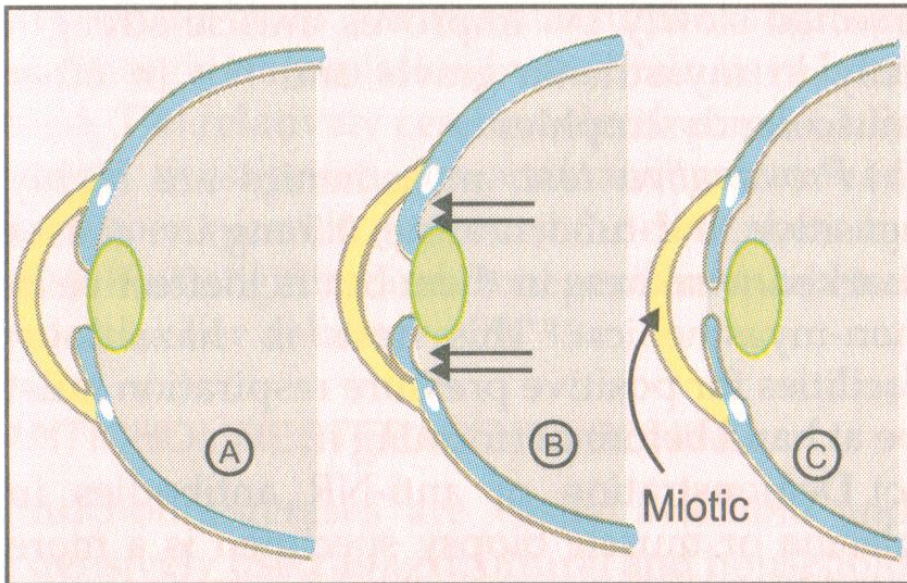
β Blockers

Drug	Non-selective		Selective β ₁ (cardio-selective)
		timolol	carteolol
MOA	<ul style="list-style-type: none"> - Act on ciliary body to ↓ production of aqueous humor. - Blocking of β₂ → blocking the relaxation effect on the ciliary muscle. 		
Route of administration	Given topically as eye drops .		
Advantages	Can be used in patients with hypertension & ischemic heart disease .		
Indications	<p>Open angle glaucoma.</p> <p>β-adrenergic blocker timolol, are effective in treating <u>chronic</u> glaucoma but are not used for emergency lowering of intraocular pressure.</p>		
ADRs	Ocular effects	Irritation.	
C.I	<ul style="list-style-type: none"> • In asthma patients. → bc the effect of β₂ → bronchospasm. • Patients with CVS disorders. → bc the effect of β₁ .on the heart. 		

B blockers are the most popular & effective treatment of open angle glaucoma AFTER **prostaglandins**.

Angle closure glaucoma

Development of angle **closure** glaucoma and it's reversal by miotics:



- **Mydriasis** occurs in an eye with **narrow iridocorneal angle**, and the iris makes contact with the lens blocking passage of the aqueous from the posterior to the anterior chamber.

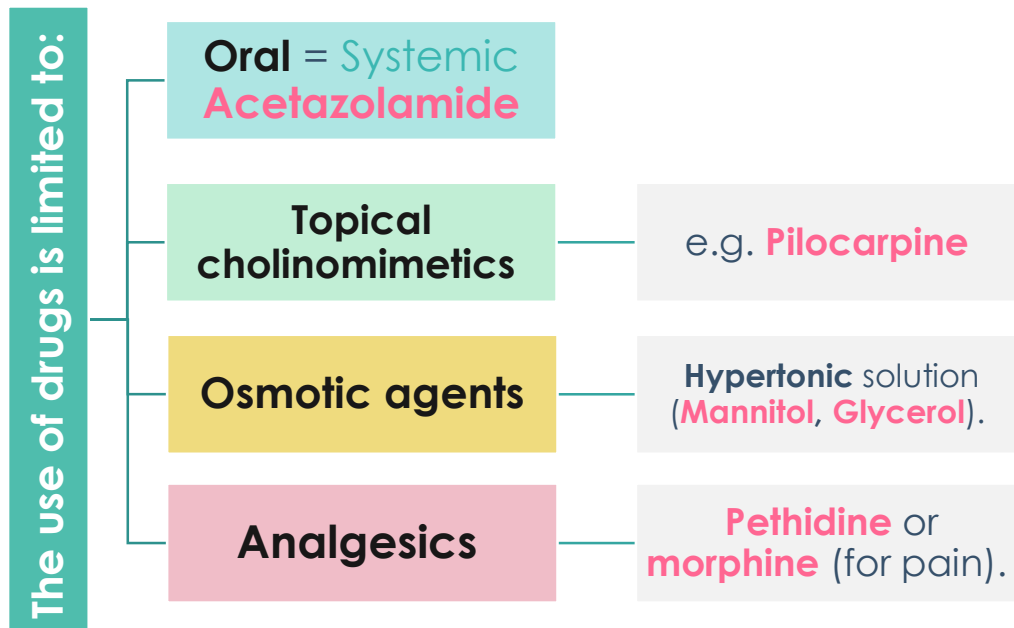
Possibly builds up behind the iris which bulges forward and closes the iridocorneal angle thus blocking aqueous outflow.

- **Miotic** makes the iris **thin** and pushes it away from the lens removing the pupillary block and restoring aqueous drainage.

▶ 1:03 min

Treatment of narrow **closed** angle glaucoma (**acute**)

- **Acute, painful** increases of intraocular pressure due to **occlusion** of the outflow drainage pathway.
- The only way to treat it → Surgery, but before surgery we give him treatment.
- emergency situation that require treatment before surgery (**Iridectomy**)



Osmotic agents → Systemic

Mech. of action	<p>Can rapidly ↓ IOP by ↓ vitreous volume.</p> <ul style="list-style-type: none"> - Glycerol 50% syrup, orally (cause nausea, hyperglycemia). - Mannitol 20% IV (cause fluid overload and not used in heart failure). - Dehydrate vitreous body which reduce IOP prior to anterior surgical procedures
Indications	<p>Used only in acute situations to temporarily reduce high IOP until more definitive treatments can be rendered.</p>
ADRs	<ul style="list-style-type: none"> - Diuresis, circulatory overload, pulmonary edema , heart failure, central nervous system effects <u>such as seizure, and cerebral hemorrhage</u>.

Anti-inflammatory drugs

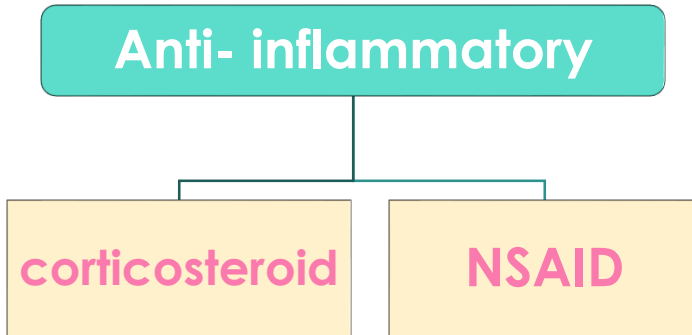
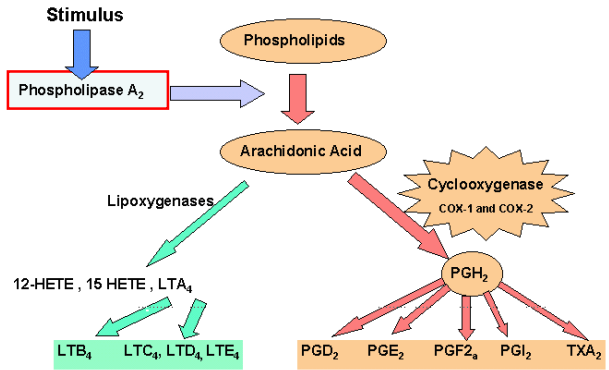


Figure 2 : Biosynthesis of eicosanoids



Corticosteroids

MOA

- Inhibition of arachidonic acid release from phospholipids by **inhibiting phospholipase A2**

Indications

Topical

E.g. **prednisolone**, **dexamethasone**, **hydrocortisone**

Systemic

E.g. **prednisolone**, **cortisone**


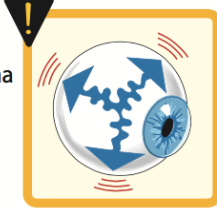
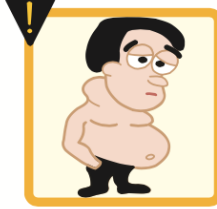



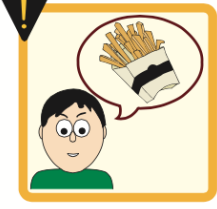

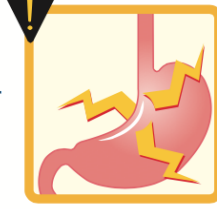
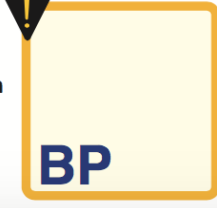


Uses: anterior uveitis, severe allergic conjunctivitis, scleritis, prevention and **suppression of corneal graft rejection**.


Uses:
- posterior uveitis.
- **optic neuritis**.

ADRs

- **Glaucoma**, cataract, mydriasis → especially if it used for a long time.
- Skin atrophy.
- Secondary infection.
- Delayed wound healing. → (healing is slow bc it is an immune suppression)

Corticosteroids side effect

Decreased growth in children		Glaucoma		Centripetal distribution of body fat	
Osteoporosis		Increased risk of infection		Hirsutism	
Increased appetite		Emotional disturbances		Peptic ulcer	
Hypertension		Peripheral edema		Hypokalemia	

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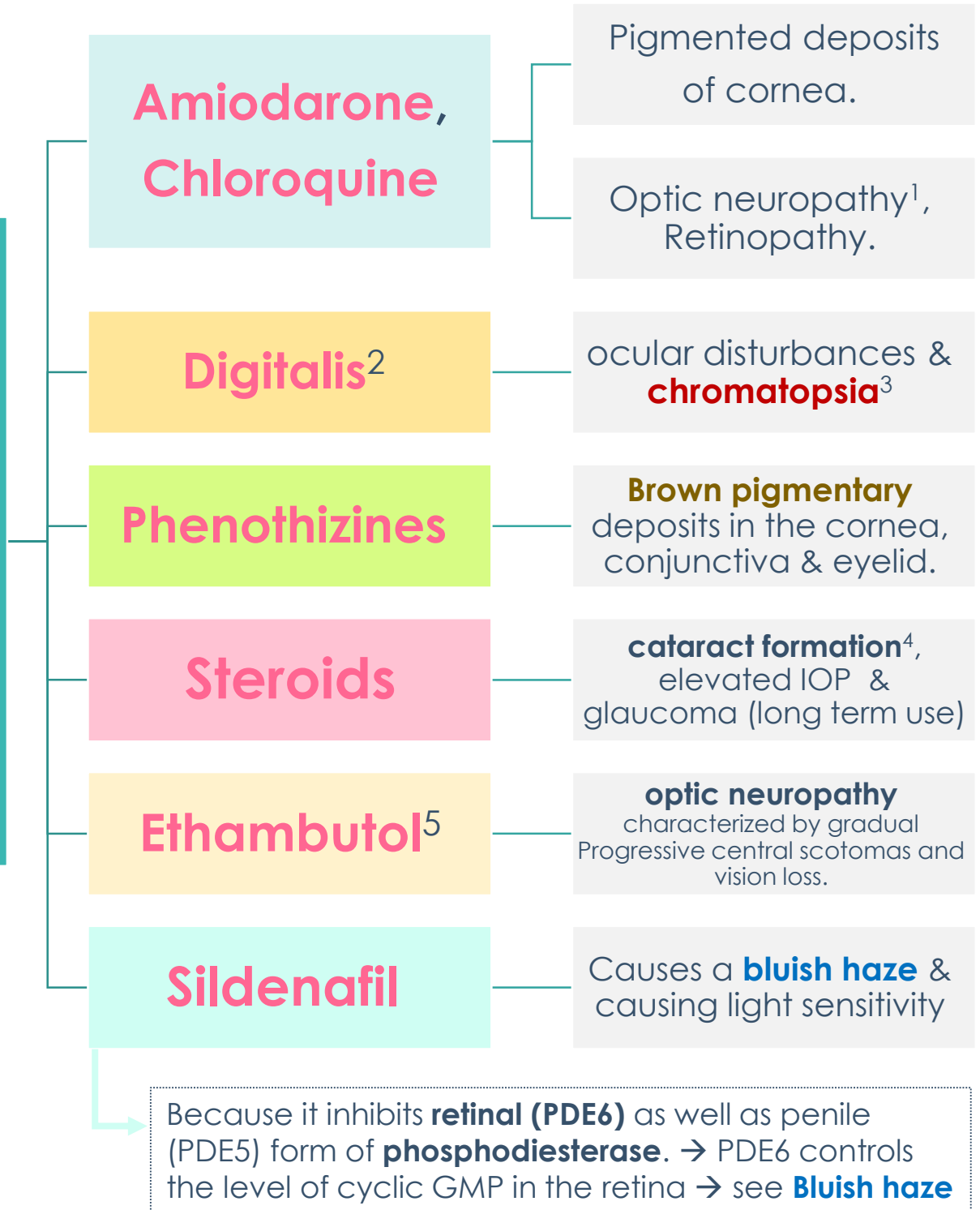
* This pic is extra.

NSAID

Drug	Ketorolac	diclofenac	Flurbiprofen
MOF	COX (cyclo-oxygenase) - inhibitors		
Indications	Cystoid macular edema occurring after cataract surgery.	postoperative inflammation, mild allergic conjunctivitis, mild uveitis.	Preoperatively to prevent miosis* during cataract surgery . * Bc they inhibit prostaglandins which produce miosis without action of cholinergic.
ADRs	Stinging (irritation), sterile corneal melt & perforation.		

Drugs causing corneal deposits

Drugs causing corneal deposits



1 **optic neuropathy**: mild decreased vision + visual field defects.

2 Cardiac failure drug

3 **chromatopsia** (objects appear **yellow**): overdosing can cause ocular disturbances.

4 [Click here to see a picture explains it.](#)

5 TB medication.

Summary-1

Drugs can be delivered to ocular tissue as: **Locally** (topically) or **systemically**

1- Locally

Eye drops	Ointment	Intra-ocular injections	Peri-ocular injections
<ul style="list-style-type: none"> - Most common rout of administration. - Has to be used several times (contact time is slow) 	<ul style="list-style-type: none"> - ↑ the contact time of the drug to ocular surface → better effect. - Has to be ↑ lipid soluble. 	<ul style="list-style-type: none"> - <u>Intra-cameral</u> e.g. Ach or lidocaine during cataract surgery - <u>Intra-vitrear</u>, e.g. <ol style="list-style-type: none"> 1- Antibiotics in case of endophthalmitis 2- Steroid in macular edema 	<p><u>Advantages:</u></p> <ul style="list-style-type: none"> - Reach behind iris-lens diaphragm better than topical app. - Good for drugs w\ ↓ lipid solubility (e.g. Penicillins) <p><u>Types:</u></p> <ol style="list-style-type: none"> 1- Sub-conjunctival. 2- Retro-bulbar. 3- Peri-bulbar.

2- Systemically

<ul style="list-style-type: none"> - Factors influencing systemic drug penetration into ocular tissue: - The more lipid solubility → the more penetration. - More effect w\ ↓ protein binding. - Eye inflammation → more penetration.
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Treatment of glaucoma

Open angle glaucoma	Narrow closed angle glaucoma
<ol style="list-style-type: none"> 1- ↓ <u>Production of aqueous humor</u>, by: <ul style="list-style-type: none"> - Beta-blockers. - α₂ agonists. - Carbonic anhydrase inhibitors. 2- ↑ <u>outflow of aqueous humor</u>, by: <ul style="list-style-type: none"> - Prostaglandins. - Adrenergic agonist. - Parasympathomimetics. <p>* Prostaglandins & Beta-blockers are the most popular.</p>	<ul style="list-style-type: none"> - Oral Acetazolamide. - Topical cholinomimetics - Osmotic agents - Analgesics.

Cholinergic agonists

Drug	Direct agonist	Indirect agonist
	Methacholine, Carbachol, Pilocarpine	<u>Reversible</u> → Physostigmine, demecarium <u>Irreversible</u> → Echothiophate, isofluorophate
Indications	<ul style="list-style-type: none"> - <u>Specific uses:</u> Induction of miosis in surgery (w\ Methacholine, Carbachol) - Open angle glaucoma. 	<ul style="list-style-type: none"> - Glaucoma. - Accommodative esotropia.
	<ul style="list-style-type: none"> - Glaucoma (open & closed angle). - In accommodative esotropia (echothiophate). 	<ul style="list-style-type: none"> - Counteract action of mydriatics. - To break iris-lens adhesions.
ADRs	- Ocular effects: Diminished vision (myopia) & headache.	

Summary-2

Cholinergic antagonists (muscarinic antagonists)

Drug	Natural alkaloids	Synthetic Atropine substitute
	Atropine → the lonest duration. Scopolamine	Homotropine, Cyclopentolate, Tropicamide → The shortest duration.
Action	- Passive mydriasis. - ↑ IOP → glaucoma.	- Cycloplegia. - Loss of light reflex. - ↓ lacrimal secretion (Sandy eye)
Uses	- Prevent adhesion in uveitis & iritis. - Measurement of refractive error.	- Funduscopy examination of the eye.
C.I	Glaucoma.	

Adrenergic agonists

Drug	Non-selective	Selective α_1 agonists	Selective α_2 agonists
	Epinephrine, Dipiverfin	Phenylephrine	Apraclonidine
Action	- ↑ uveoscleral outflow of aqueous humor.	- Mydriasis (without cycloplegia) - Decongestant.	- ↓ production of aqueous humor. - ↑ uveoscleral outflow of aqueous humor.
Indication	Open angle glaucoma.	- Funduscopy examination of the eye. - To prevent adhesion in uveitis & iritis. - Decongestant in minor allergic hyperemia of eye.	- Open angle glaucoma treatment - Prophylaxis against IOP spiking after glaucoma laser procedures.
ADRs	- Headache, Arrythmia, ↑ Bp.	- ↑ Bp, rebound congestion, precipitation of acute-angle glaucoma	- Headache, bradycardia, hypotension.
C.I	In patients with narrow angles as they may precipitate closed angle glaucoma.		

Beta blockers

Drug	Non-selective	Selective β_1
	Timolol, Cartelol	Betaxolol
General inf.	MOA: Act on ciliary body to ↓ the <u>production</u> of aqueous humor. Advantage: Can be used in pts with <u>hypertension</u> . Indication: Open angle glaucoma.	
ADRs	Irritation of eye. - C.I: Asthma pts.	

Summary-3

Drug	Mech. Of action	Indication	ADRs
Carbonic anhydrase inhibitors	e.g. Acetazolamide (oral) Dorzolamide (topical) - ↓ production of aqueous humor by blocking carbonic anhydrase enzyme required for production of bicarbonate ions .	Open angle glaucoma	- Myopia , malaise, anorexia, GI upset, headache. C.I.: Sulfa allergy, pregnancy & Digitalis user.
Prostaglandin analogues	- ↑ uveoscleral aqueous outflow. - Latanoprost is preferred due to lesser adverse effects. - They have replaced beta blockers . - They are used topically as eye drops & once a day .		- Pigmentation of the iris (heterochromia iridis). - Intraocular inflammation. - Macular edema .
Osmotic agents	Can rapidly ↓ IOP by ↓ vitreous volume. - Glycerol 50% syrup , orally (cause nausea, hyperglycemia). - Mannitol 20% IV (cause fluid overload and not used in heart failure).	Acute Situations	- Diuresis, circulatory overload, pulmonary edema , heart failure, seizure & cerebral hemorrhage .
Corticosteroids	- Inhibition of arachidonic acid release from phospholipids by inhibiting phospholipase A2 - Topical: E.g. prednisolone, dexamethasone, hydrocortisone - Systemic: E.g. prednisolone, cortisone	- Topical: severe allergic conjunctivitis, prevention and suppression of corneal graft rejection . - Systemic: posterior uveitis, optic neuritis .	- Glaucoma , cataract, mydriasis - Skin atrophy. - Secondary infections. - Delayed wound healing.
NSAID	COX (cyclo-oxygenase) – inhibitors.	- Ketorolac → Cystoid macular edema occurring after cataract surgery. - Diclofenac → Postoperatively. - Flurbiprofen → Preoperatively to prevent miosis during cataract surgery.	Stinging.

Drugs causing corneal deposits

Amiodarone, Chloroquine → Pigmented deposits of cornea, optic neuropathy.	Steroids → Cataract formation, ↑ IOP.
Digitalis → Chromatopsia .	Ethambutol → Optic neuropathy.
Phenothizines → Brown pigmentary .	Sildenafil → Bluish haze .



Thank you for checking our team!



Pharmacology 435

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Sources:

- 1- Dr.Hanan Hagar's lecture & Dr.AbduLatif Mahesar's lecture , 2016.
- 2- 435 MSK block
- 3- Wikipedia
- 4- Pharmacology (Lippincotts Illustrated Reviews Series), 5th edition.
- 5- Ganong's Review of medical physiology, 25th edition.

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