

Drug Used in Myasthenia gravis and Glaucoma



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Content of the Lectures

- 💡 Myasthenia gravis
- 💡 Drug Used in MG
- 💡 Drug used in glaucoma

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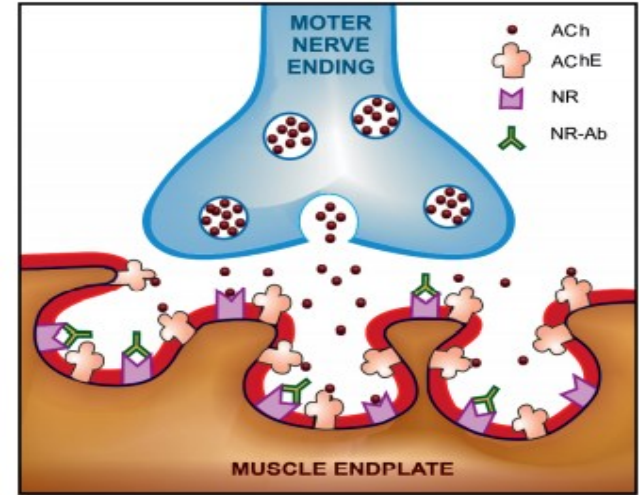
Myasthenia Gravis

- ❖ Myasthenia gravis is an **autoimmune disorder** affecting about 1 in 10,000 population,
- ❖ **Myasthenia gravis** (MG) is a long-term **neuromuscular disease** that leads to varying degrees of **skeletal muscle weakness**.
- ❖ The most commonly affected muscles are those of the eyes, face, and swallowing.
- ❖ It can result in double vision, drooping eyelids, trouble talking, and trouble walking.



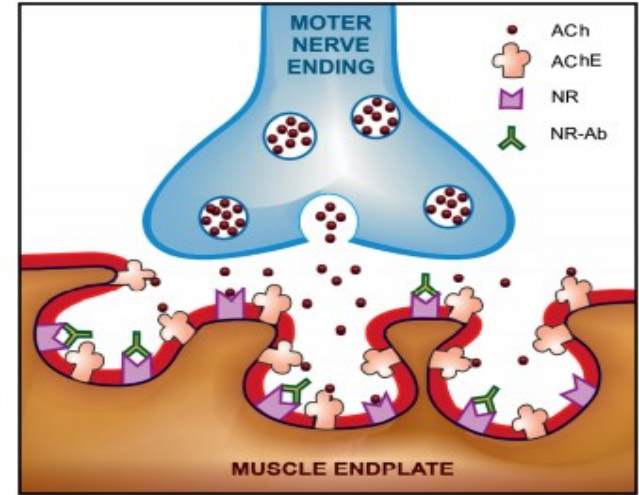
Myasthenia Gravis

- MG is due to development of **antibodies** that block or destroy **nicotinic acetylcholine receptors** (NR) at the muscle endplate and causes reduction in number of free NR cholinergic receptors to 1/3 of normal or less and structural damage to the neuromuscular junction
- This prevents **nerve impulses** from triggering muscle contractions
- Most cases are due to immunoglobulin G1 (IgG1) and IgG3 antibodies that attack nicotinic AChR in the postsynaptic membrane, causing complement-mediated damage and muscle weakness.



Myasthenia Gravis

- Babies of mothers with myasthenia may have symptoms during their first few months of life, known as neonatal myasthenia
- Diagnosis can be supported by blood tests for specific antibodies, the edrophonium test, or a nerve conduction study



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Drug Used in Myasthenia Gravis

1. **Acetylcholinesterase inhibitors:**
neostigmine and pyridostigmine.

- Treatment is usually started with **neostigmine 15 mg orally 6 hourly**; dose and frequency is then adjusted to obtain optimum relief from weakness
- Pyridostigmine is an alternative which needs less frequent dosing

2. **Immunosuppressants:** prednisone
(Corticosteroids), azathioprine

- Corticosteroids inhibit production of NR-antibodies and may increase synthesis of NRs

Drug Used in Myasthenia Gravis

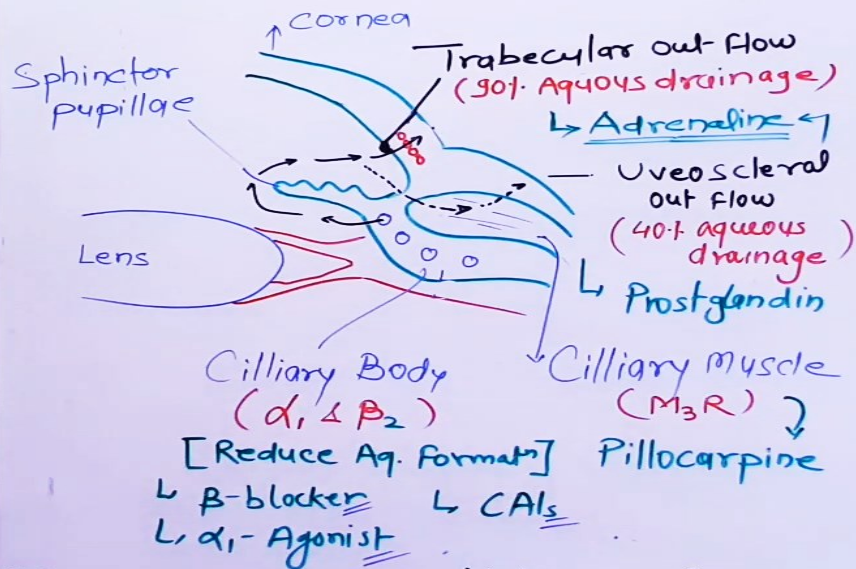
❖ azathioprine and cyclosporine also inhibit NR-antibody synthesis by affecting T-cells, but response to the former is slow in onset (takes upto 1 year), while that to the latter is relatively quick (in 1–2 months)

3. The **surgical removal** of the **thymus** may improve symptoms in certain cases.

4. **Plasmapheresis** and high-dose **intravenous immunoglobulin** may be used during sudden flares of the condition

ANTI-GLAUCOMA DRUGS

Glaucoma → It is an Ocular disorder characterized by optic nerve damage with raised IOP (Intra-ocular pressure)



OPEN-ANGLE GLAUCOMA - wide/chronic, Genetically affected, ↓ Trabecular Outflow

DRUGS: - I) **Miotics** → M_3 -Agonist (Topically)
 → Pilocarpine, Physostigmine
 → ↑ Trabecular Outflow
 → 3rd choice

II) **β -blocker** → Topically, ↓ Aqueous Secretion
 ↳ Timolol, Betaxolol, Levobunolol
 ↳ These drugs do not affect the pupil size
 ↳ 1st choice of drug

III) **α_1 Agonist** → Adrenaline / Dipivefrin
 ↳ ↑ outflow & ↓ Aq. Secretion ↳ Pro-drug of Adr. (Topically used)

IV) **α_2 -Agonist** - Apraclonidine (Topically)

↳ ↑ Uveoscleral outflow & ↓ Aq. Secretion

V) **Prostaglandin** - Latanoprost (Topically)

↳ ↑ Uveoscleral Outflow

VI) **CAIs** - Acetazolamide (orally)

Dorzolamide (Topically)

↳ ↓ Aq. Secretion

CLOSED ANGLE GLAUCOMA - Drugs -

① β -blocker

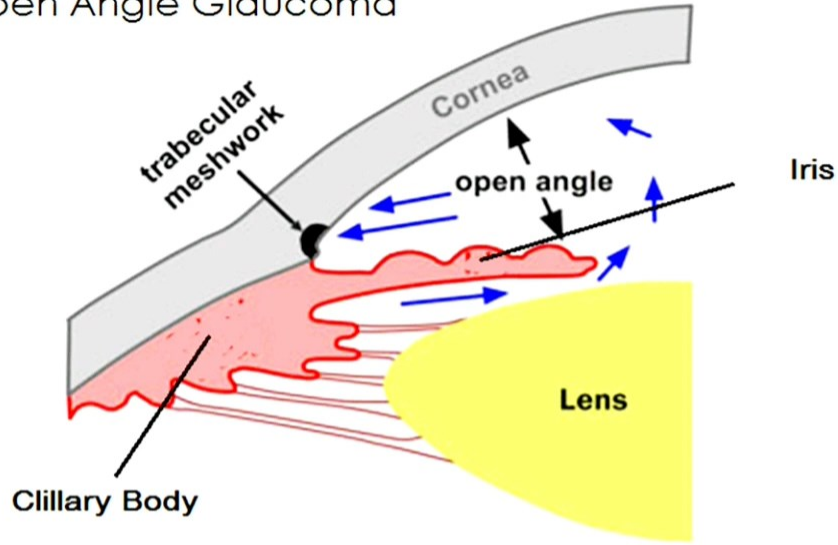
② Pilocarpine

③ CAIs

(IV) α_2 -Ag

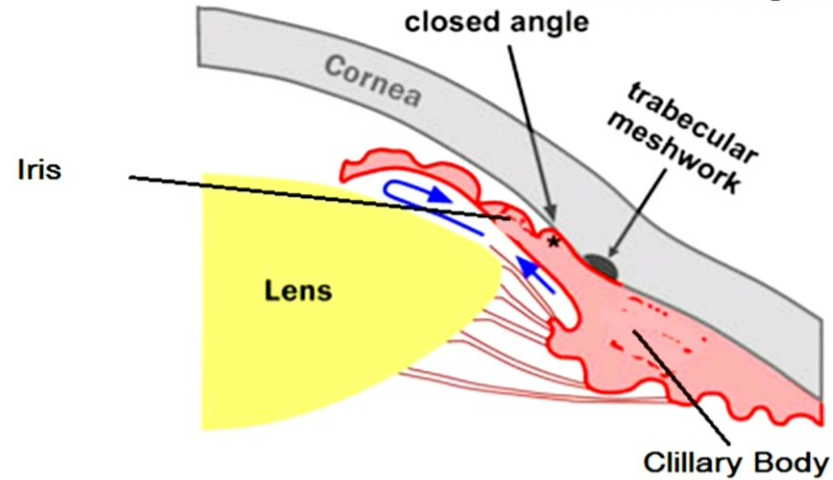
④ Hypertonic → Mannitol / Glycerol

Open Angle Glaucoma



- ✓ Wide/Open angle between Iris and Cornea
- ✓ Caused by **Slow** blockade of Drainage channel (Trabecular Meshwork)
- ✓ Increase in Intra Ocular pressure (IOP)
- ✓ Developed slowly and is a life long condition
- ✓ Most common case 90% of all Case
- ✓ Symptoms are not noticeable

Closed Angle Glaucoma



- ✓ Narrow/Closed angle between Iris and Cornea
- ✓ Caused by **rapid** blockade of Drainage channel (Trabecular Meshwork)
- ✓ Sudden Increase in Intra Ocular pressure (IOP)
- ✓ Developed rapidly
- ✓ Symptoms are noticeable
- ✓ Required immediate medication



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