

Development of Eye


Optic Cup and Lens Vesicle

- 22nd day embryo → eye begins as *shallow grooves* on the sides of the forebrain.
- With neural tube closure → grooves → form optic vesicles (outpocketings of forebrain).
- Induction of lens formation:
 - Optic vesicles contact surface ectoderm → induce ectoderm to form lens placode.
 - Lens placode invaginates → lens vesicle.
 - By 5th week → lens vesicle separates from surface ectoderm and lies within mouth of optic cup.
- Optic cup formation:
 - Optic vesicle invaginates → double-walled optic cup.
 - Initially, the intraretinal space separates inner & outer layers → disappears later → layers appose.

- Invagination also occurs inferiorly → forms choroid fissure.
 - Function → allows hyaloid artery to enter eye.
 - By 7th week → lips of fissure fuse.
 - Optic cup opening → becomes future pupil.

Retina

- Outer layer of optic cup → Pigmented layer of retina.
- Inner layer of optic cup → develops into neural retina:
 - Posterior 4/5 (pars optica retinae) → differentiates into:
 - Rods & cones (photoreceptors).
 - Mantle layer → gives neurons & supporting cells → forms:
 - Outer nuclear layer
 - Inner nuclear layer
 - Ganglion cell layer
 - Fibrous layer (nerve fiber layer) → axons converge → optic stalk → becomes optic nerve.

 *Exam Tip:* Light passes through these layers before

reaching rods & cones (often asked!).

- Anterior 1/5 (pars ceca retinae) → thin, 1-layered. Later forms:
 - Pars iridica retinae → inner layer of iris.
 - Pars ciliaris retinae → contributes to ciliary body.

Iris

- Composed of:
 - Outer pigmented layer (from optic cup).
 - Inner non-pigmented layer (from optic cup).
 - Vascular connective tissue (mesenchyme).
 - Muscles (sphincter pupillae & dilator pupillae):
 - Develop from neuroectoderm of optic cup (*exception: muscles usually mesodermal, but these are ectodermal* → very exam-favorite point).

Ciliary Body

- Pars ciliaris retinae → shows marked folding.
- Covered externally by mesenchyme → forms ciliary muscle.
- Connected to lens by zonular fibers (suspensory

ligament).

- Function: Contraction/relaxation changes lens curvature → accommodation reflex.

Clinical Correlations

- Coloboma iridis → failure of choroid fissure to close → notch in iris.
- Congenital cataract → lens opacity if lens development disturbed.
- Retinal detachment → persistence of intraretinal space between neural & pigmented layers.

Development of Lens, Choroid, Sclera, Cornea & Vitreous Body

Lens Development

- After lens vesicle formation:
 - Posterior wall cells → elongate anteriorly → form primary lens fibers.
 - By end of 7th week → fibers reach anterior wall → fill vesicle lumen.
- Growth continues → by addition of secondary lens

fibers throughout life → around central core.

🔑 *Exam Tip:* Lens keeps growing — new fibers are added; explains why old age → lens becomes denser → presbyopia/cataract.

Choroid & Sclera

- At end of 5th week → eye surrounded by loose mesenchyme.
- Differentiates into:
 - Inner layer (like pia mater) → vascular, pigmented → choroid.
 - Outer layer (like dura mater) → tough fibrous → sclera.
 - Sclera is continuous with dura mater of optic nerve.

Cornea & Anterior Chamber

- Anterior chamber forms → by vacuolization → mesenchyme splits into:
 - Inner layer → in front of lens & iris → iridopupillary membrane.
 - Outer layer → continuous with sclera → substantia propria (stroma) of cornea.

- Cornea composition:

1. Outer epithelium → surface ectoderm.

2. Substantia propria (stroma) → mesenchyme, continuous with sclera.

3. Inner epithelium (endothelium) → lines anterior chamber.

- Iridopupillary membrane → disappears later → clears anterior chamber.

- Posterior chamber: space between iris (anteriorly) and lens + ciliary body (posteriorly).

- Aqueous humor circulation:

- Produced by ciliary processes.

- Pathway → Posterior chamber → Pupil → Anterior chamber → Resorbed at Canal of Schlemm (scleral venous sinus).

- Function → nourishes avascular lens & cornea.

🔑 *Clinical* → Glaucoma = blockage of aqueous humor outflow at canal of Schlemm → ↑ intraocular pressure → optic nerve damage.

Vitreous Body

- Mesenchyme → enters optic cup via choroid fissure.
 - Forms:
 - Hyaloid vessels → supply lens & inner retina during fetal life.
 - Delicate network of fibers between lens & retina.
 - Interstitial spaces → fill with gelatinous substance → forms vitreous body.
- Later changes:
 - Hyaloid vessels regress → leave Hyaloid canal (Canal of Cloquet).

Exam Tip: Persistence of hyaloid artery → congenital anomaly → can cause visual defects.

Clinical Correlations

- Congenital cataract → due to lens fiber development abnormalities.
- Glaucoma → blockage at canal of Schlemm → raised intraocular pressure.

- Persistent hyaloid artery → may lead to congenital blindness.
- Coloboma (already mentioned previously) → failure of choroid fissure closure.

Optic Nerve Development & Eye Abnormalities

Optic Nerve Formation

- Connection:
 - Optic cup connected to brain via optic stalk.
 - Choroid fissure (ventral groove of stalk) → carries hyaloid vessels.
- Retinal nerve fibers:
 - Retina develops ganglion cells → axons run into optic stalk.
 - By 7th week → choroid fissure closes → stalk forms narrow tunnel.
- Maturation of optic nerve:
 - Inner wall of stalk thickens due to growing nerve fibers.
 - Inside and outside walls fuse.
 - Inner cells → form neuroglia to support fibers.

- Optic stalk → transformed into optic nerve.
- Vascular supply:
 - Hyaloid artery persists proximally → becomes central artery of retina.
- Coverings:
 - Continuous with meninges:
 - Pia → from choroid
 - Dura → from sclera

Clinical Correlations (Eye Abnormalities)

1. Coloboma

- Cause → failure of choroid fissure closure (7th week).
- Most common form → coloboma iridis (keyhole defect in iris).
- May extend to ciliary body, retina, choroid, optic nerve.
- Can also affect eyelids.

2. Iridopupillary membrane persistence

- Should disappear during anterior chamber

formation.

- Persistence → thin strands visible in front of lens.

3. Congenital Cataract

- Lens opacity.
- Causes:
 - Genetic defects.
 - Maternal rubella infection (critical between weeks 4-7).
 - Post-7th week rubella → no lens damage, but may cause deafness (cochlear anomalies).
- Prevented by MMR vaccination.

4. Hyaloid artery persistence

- Normally regresses (leaving hyaloid canal).
- Persistence → cord/cyst in vitreous.

5. Microphthalmia

- Eye abnormally small (2/3rd size).
- Often with other ocular anomalies.
- Associated with intrauterine infections: CMV, toxoplasmosis.

6. Anophthalmia

- Complete absence of eye (sometimes minimal tissue remains).
- Usually with severe cranial defects.

7. Congenital Aphakia

- Absence of lens.
- Very rare.

8. Aniridia

- Absence of iris.
- Due to PAX6 mutations.
- May also contribute to microphthalmia & anophthalmia.

9. Cyclopia / Synophthalmia

- Spectrum: single fused eye / partially fused eyes.
- Cause → failure of midline tissue development (days 14-21 or later).
- Associated brain anomaly → Holoprosencephaly (cerebral hemispheres fused).
- Risk factors:
 - Alcohol
 - Maternal diabetes
 - SHH mutations

- Defects in cholesterol metabolism (affects SHH signaling).