INTERNATIONAL CONGRESS OF THE

EGYPTIAN

OPHTHALMOLOGICAL SOCIETY

EOS 1902

In collaboration with: MEACO

Pediatric Conditions difficult to manage!

Why?

How to proceed?

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Cairo University

Delayed Visual Development in an Infant

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Patients presenting as early as the first month and during the first year of life

Signs

- Searching movements of the eyes
- Poor pupillary constriction
- Inability to fix or follow

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Etiology

Identifiable causes by ocular examination

- Severe ocular malformations (microphthalmia)
- ROP
- Dense bilateral media opacities
  - Congenital cataract
  - Advanced Primary Congenital glaucoma
  - AS dysgenesis
- Retinal dysplasia and severe retinal dystrophies (FEVR)

Etiology

Identifiable causes by ocular examination

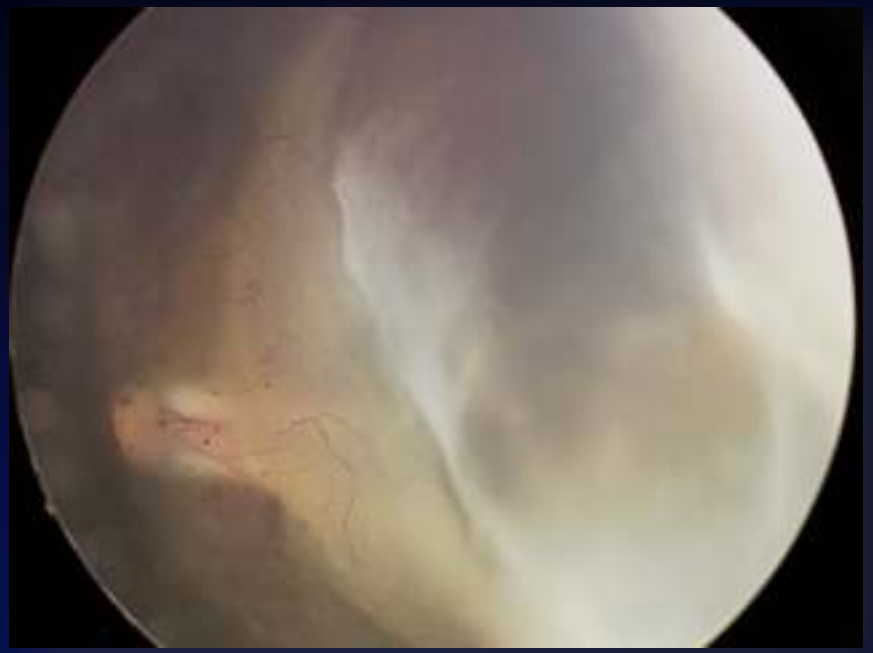
- Aniridia
- Optic nerve anomalies
  - Hypoplasia
  - Coloboma
  - Atrophy (COA)
- Albinism and foveal hypoplasia
- Shaken Baby syndrome
- Congenital idiopathic nystagmus
- Extreme refractive errors

Etiology

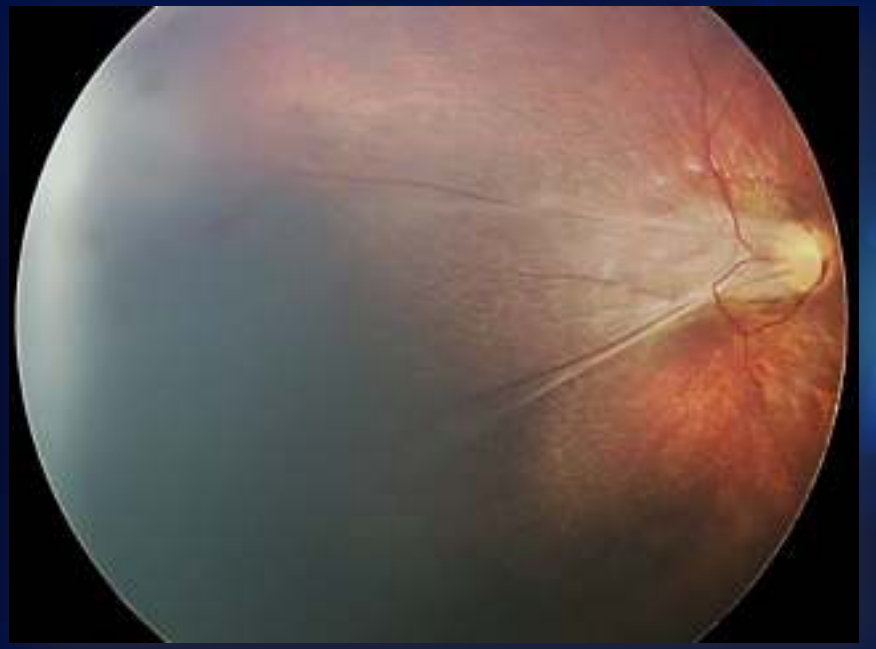
With normal ocular examination

- Leber Congenital amaurosis
- IRDs (Rod/cone Dystrophy)
- Diffuse cerebral dysfunction
- Delayed maturation of visual system

ROP



Stage 4



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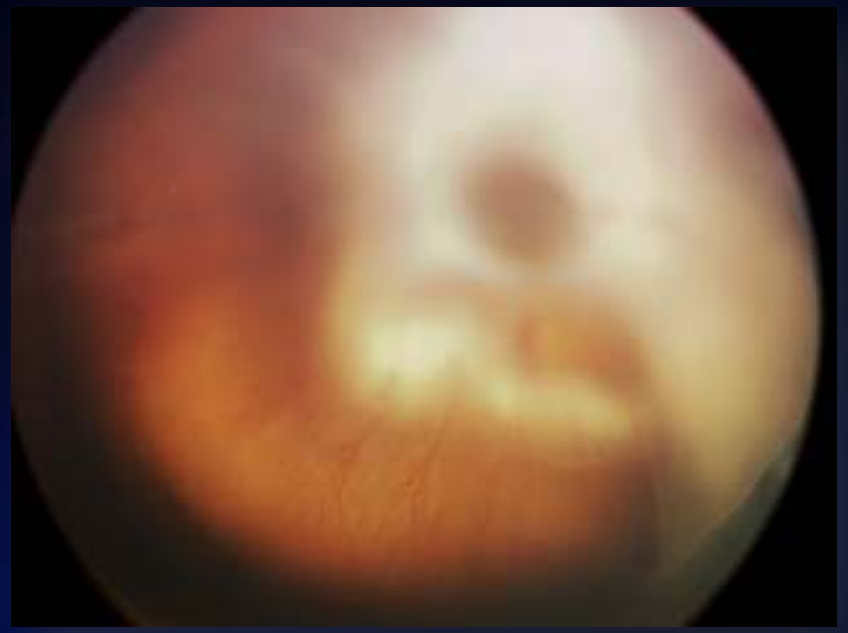
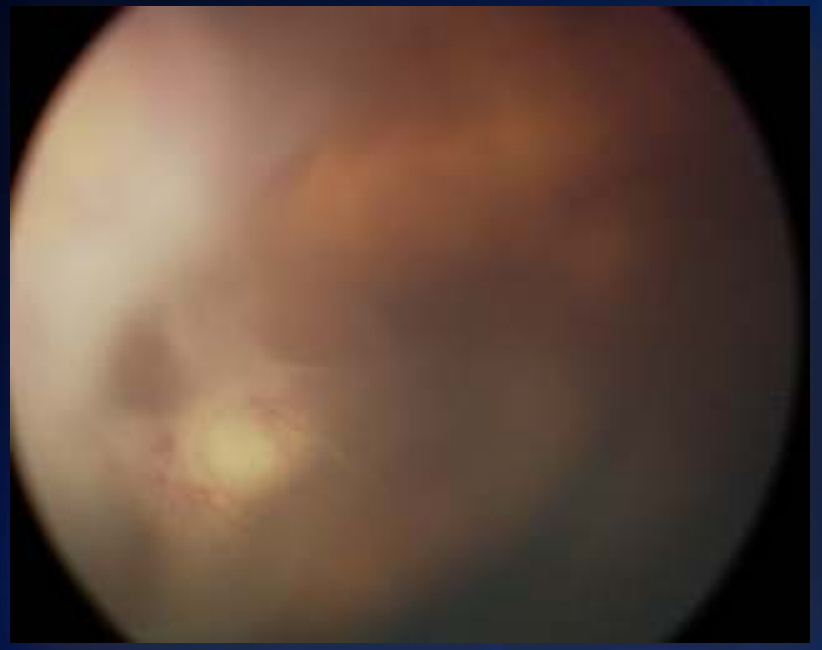
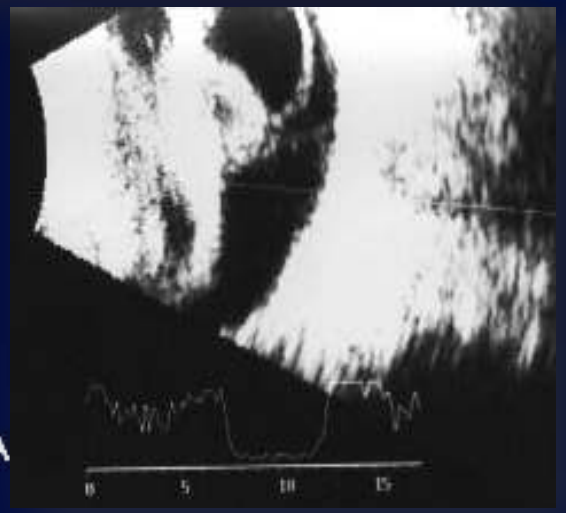
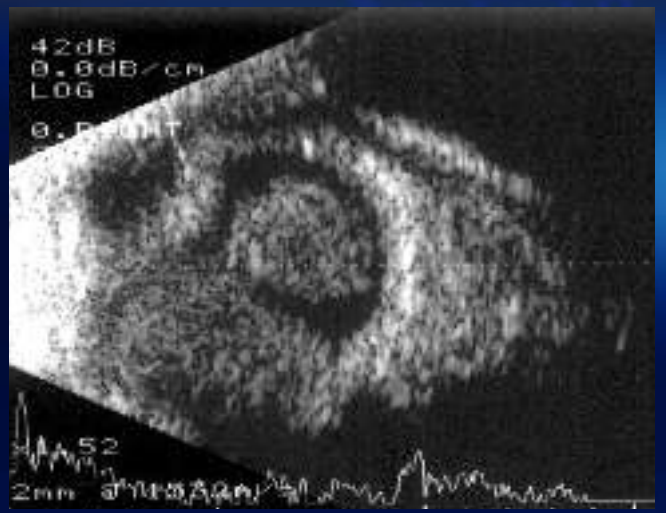
ROP





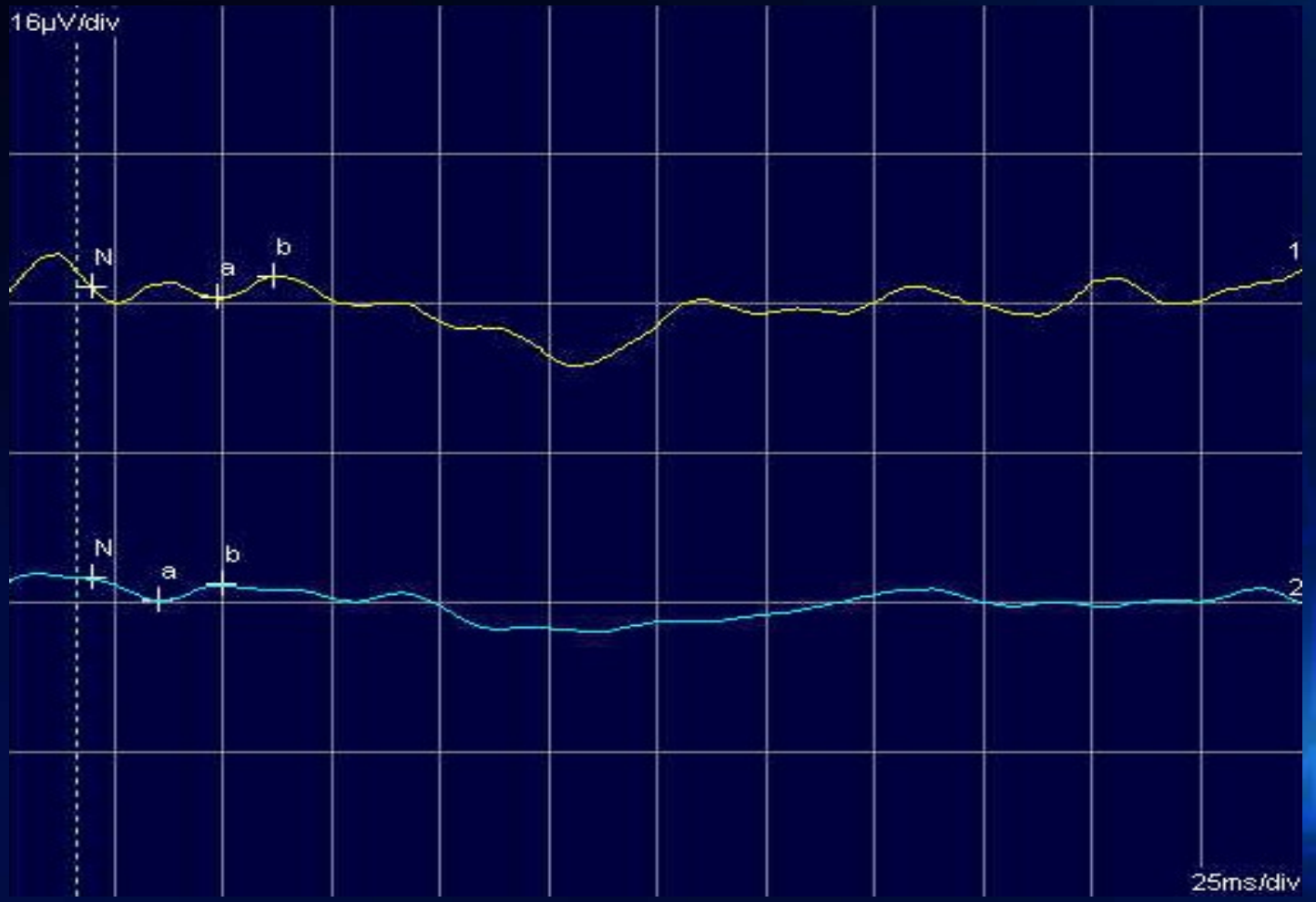
Stage 5



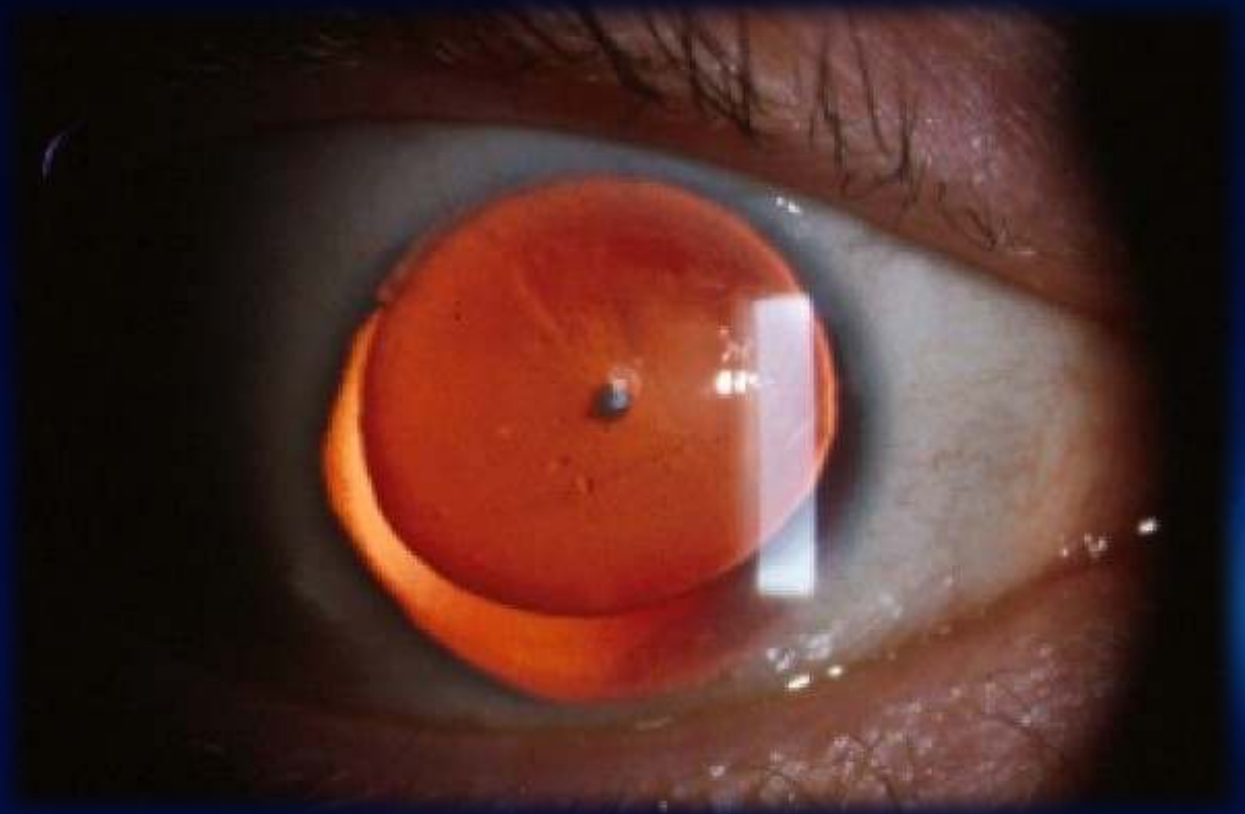


ROP

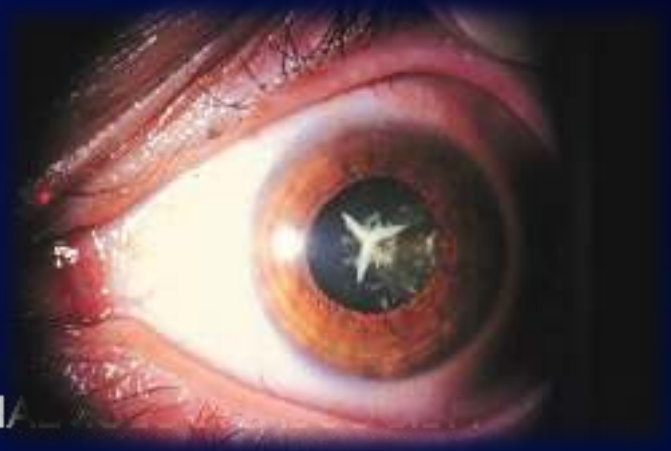
Flash ERG of small amplitude



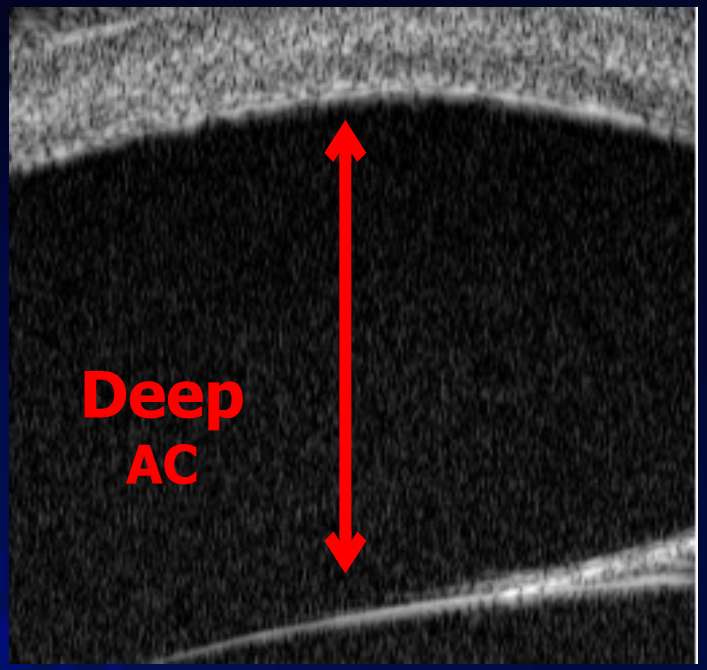
Aniridia



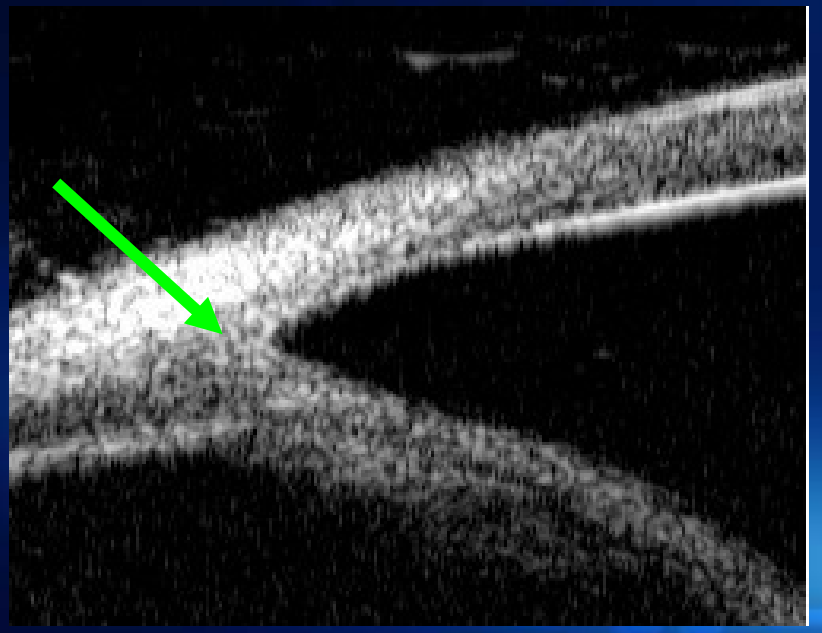
Congenital Cataract



Primary Congenital Glaucoma

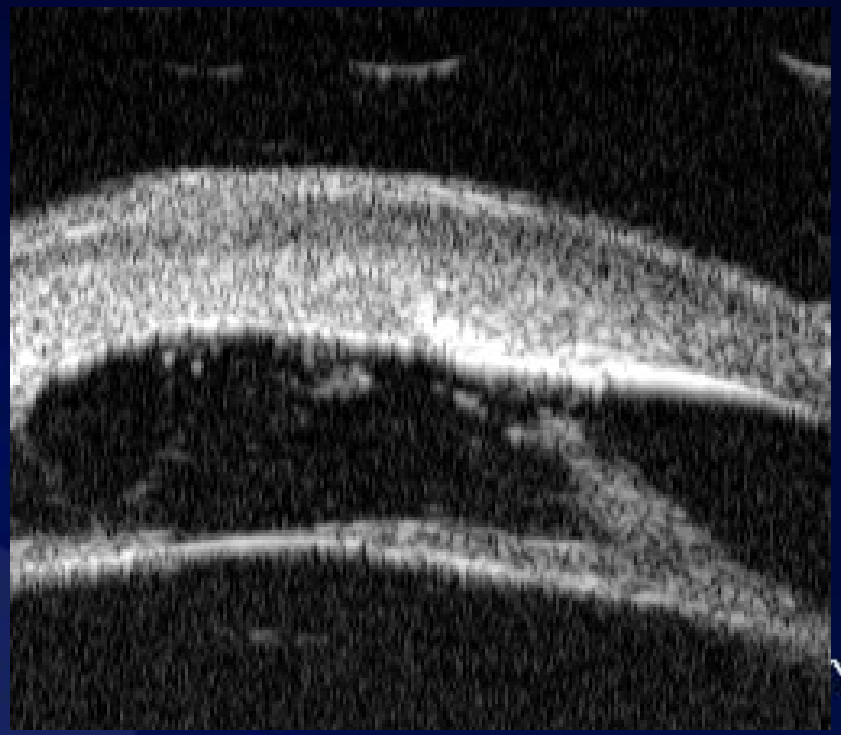


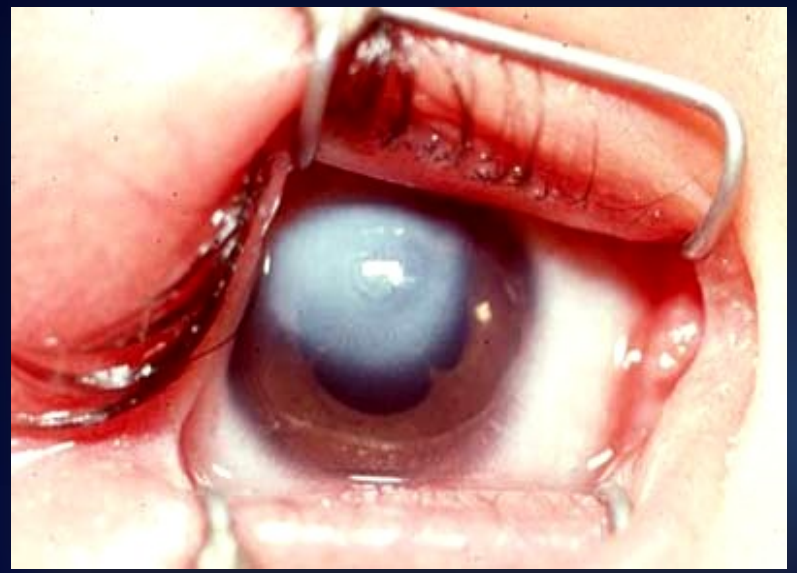
Iris covers the scleral spur

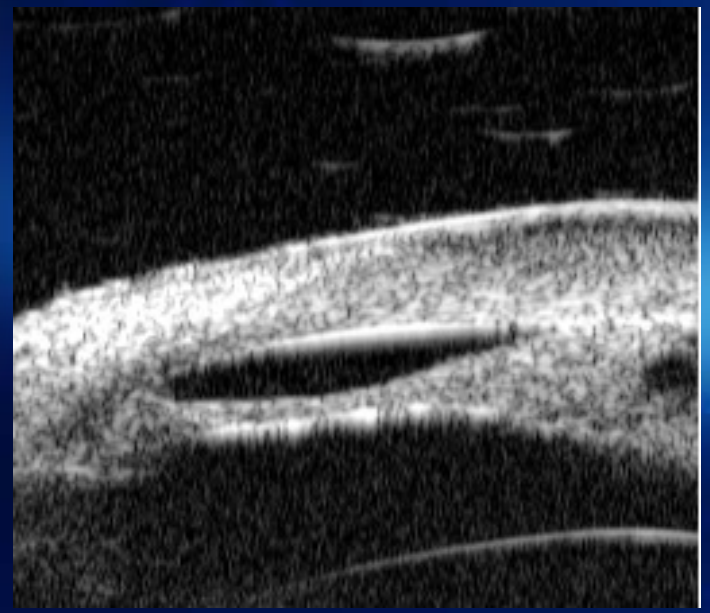


Anterior Segment Dysgenesis

Peter's Anomaly: Type 1

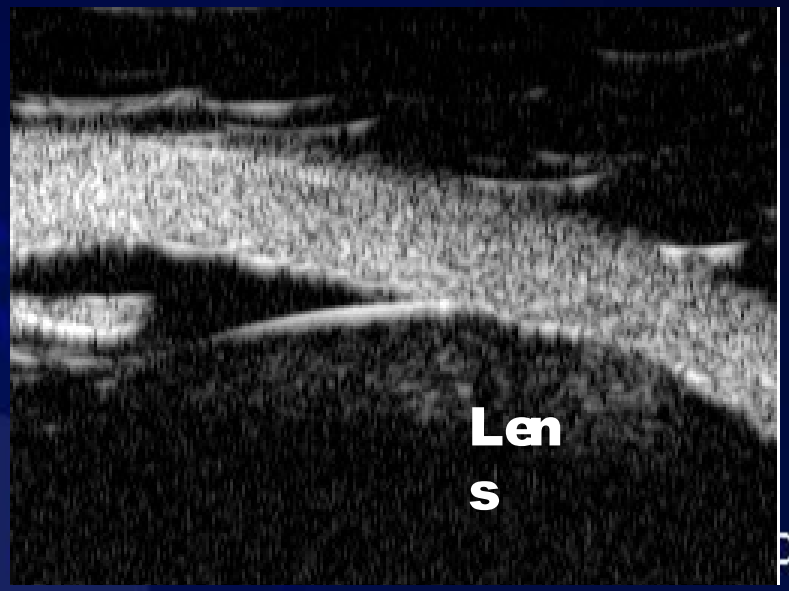




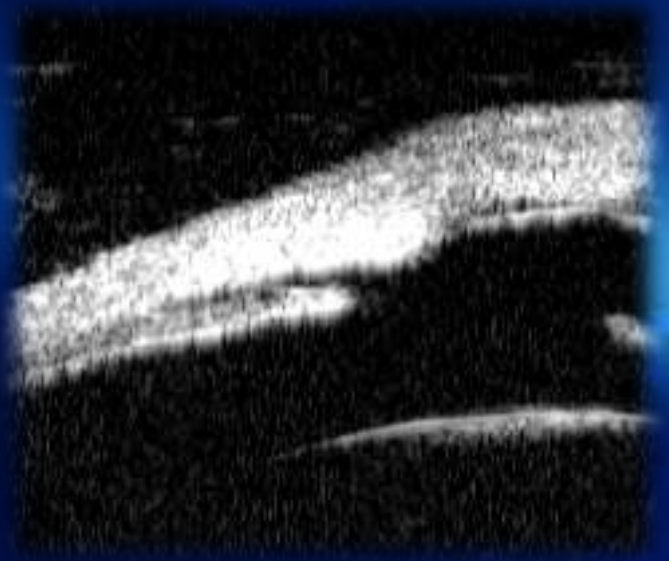


Anterior Segment Dysgenesis

Peter's Anomaly : Type 2

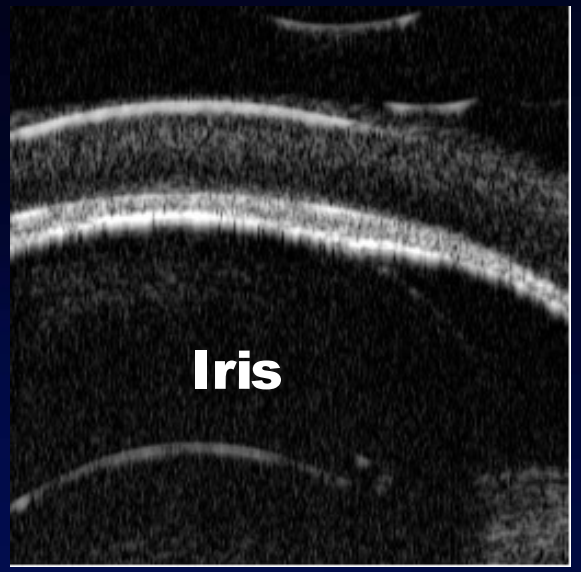


CIETY

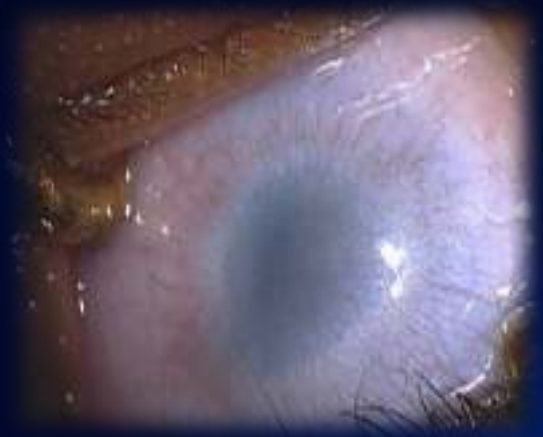


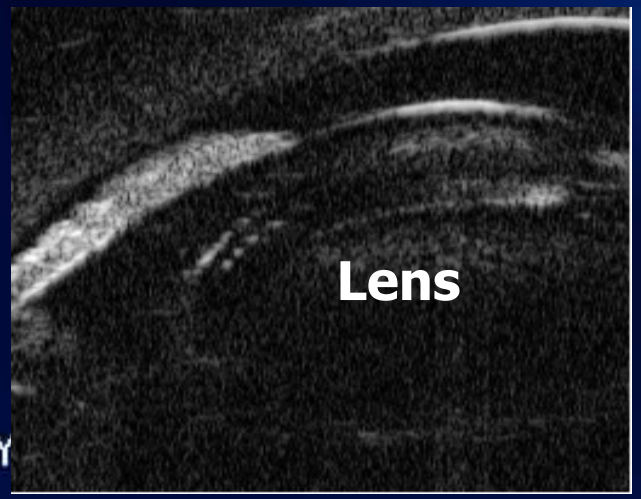
Anterior Segment Dysgenesis

Sclerocornea

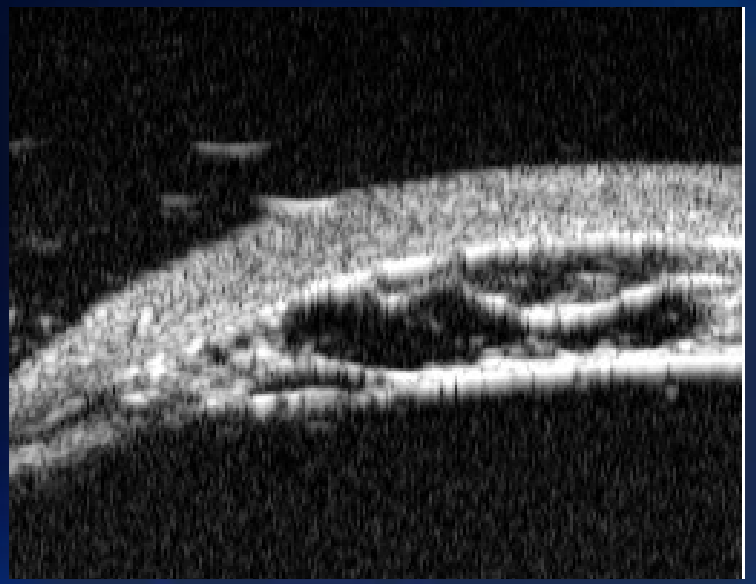


Total Iridocorneal adhesion and Aphakia





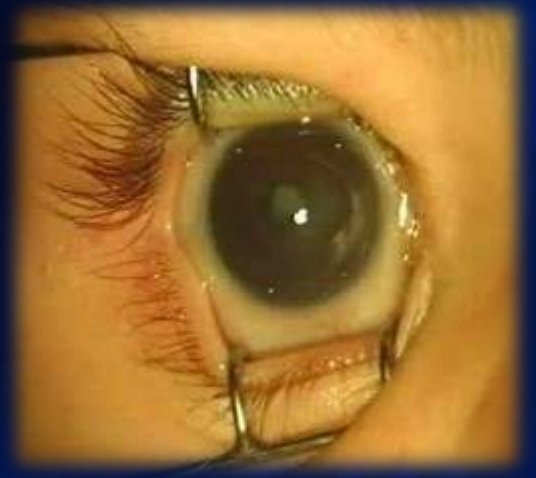
Spherophakia

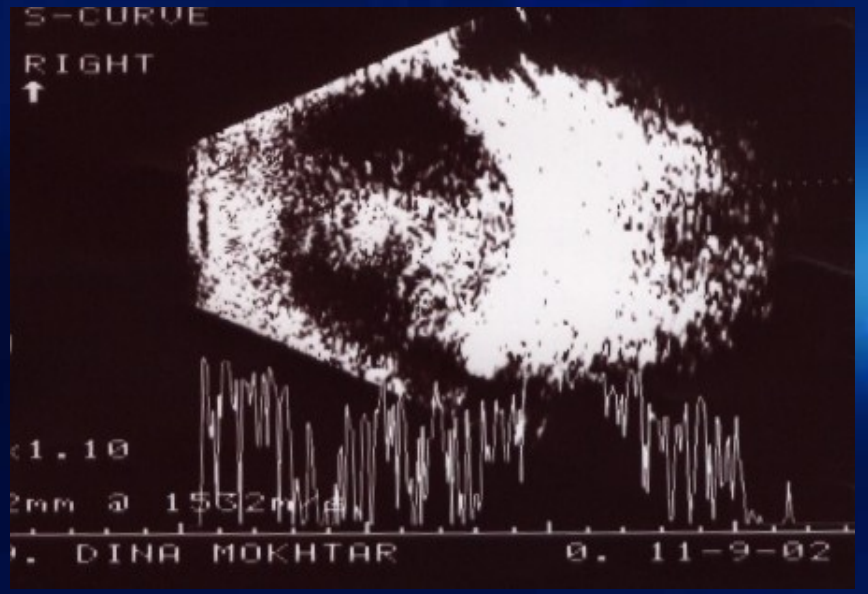


Rudimentary Iris shreds

Retinal dysplasia

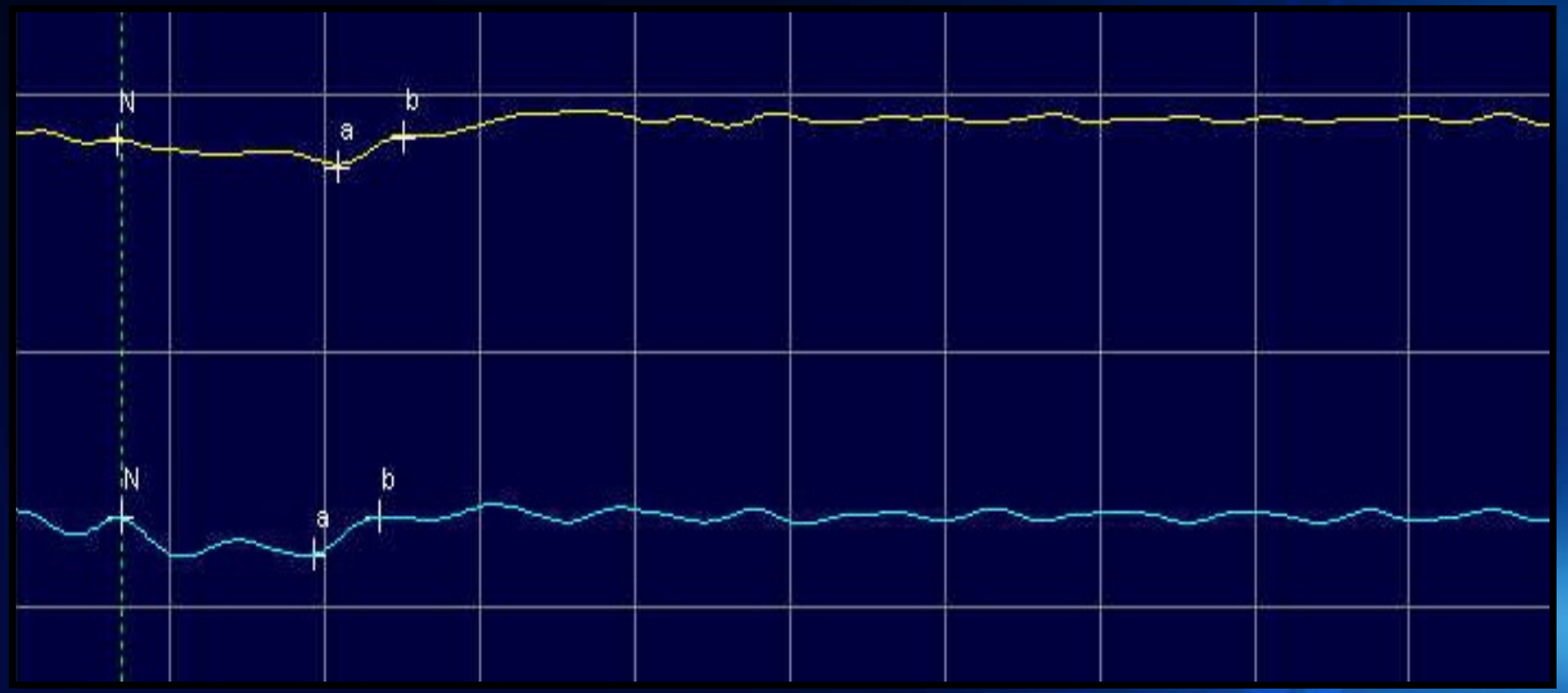
Presence of primitive retinal structure as an epiretinal mass composed of portions of retina & cortical vitreous with primitive vascular structures



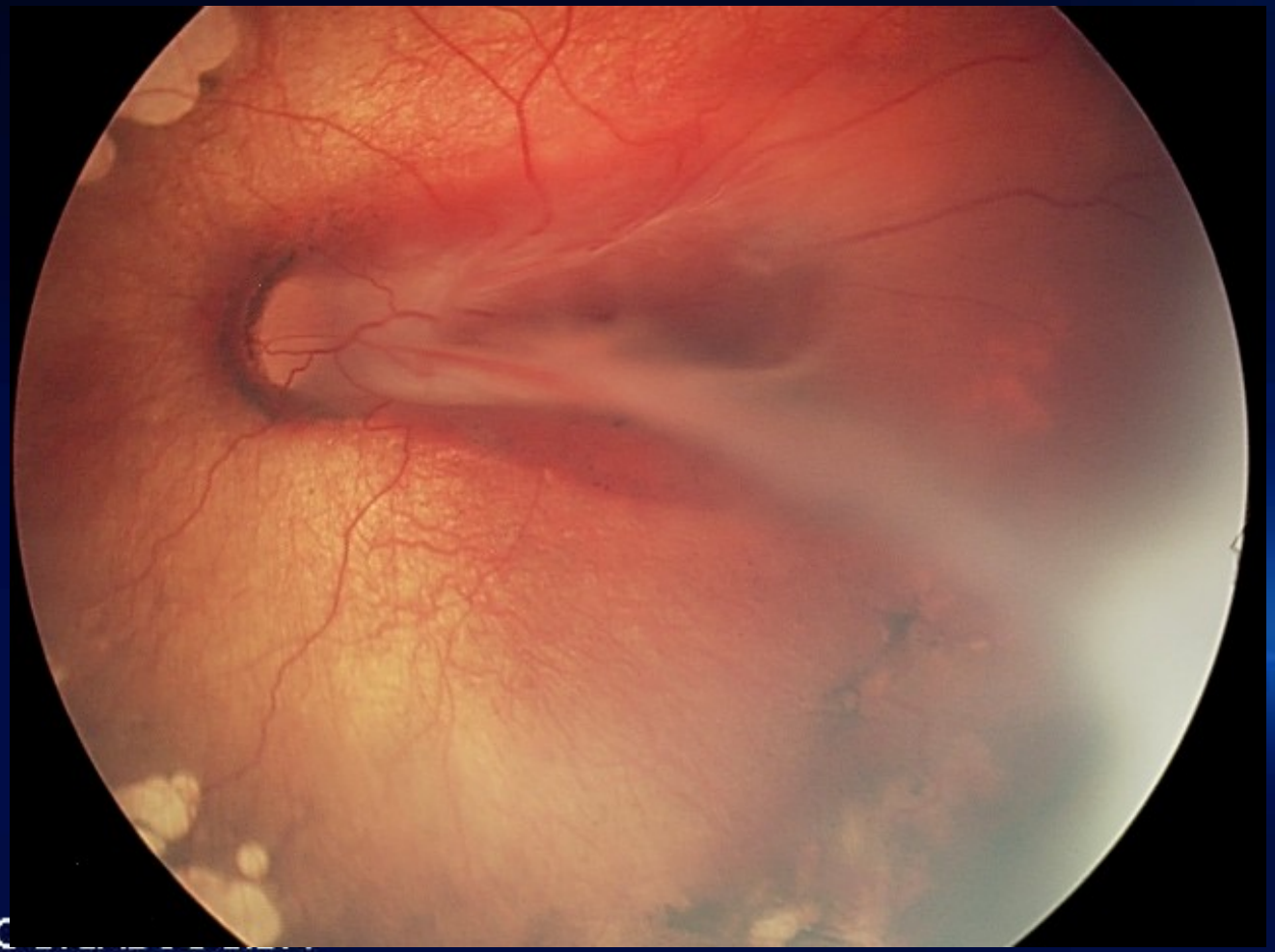


Retinal dysplasia

Marked affection of flash ERG

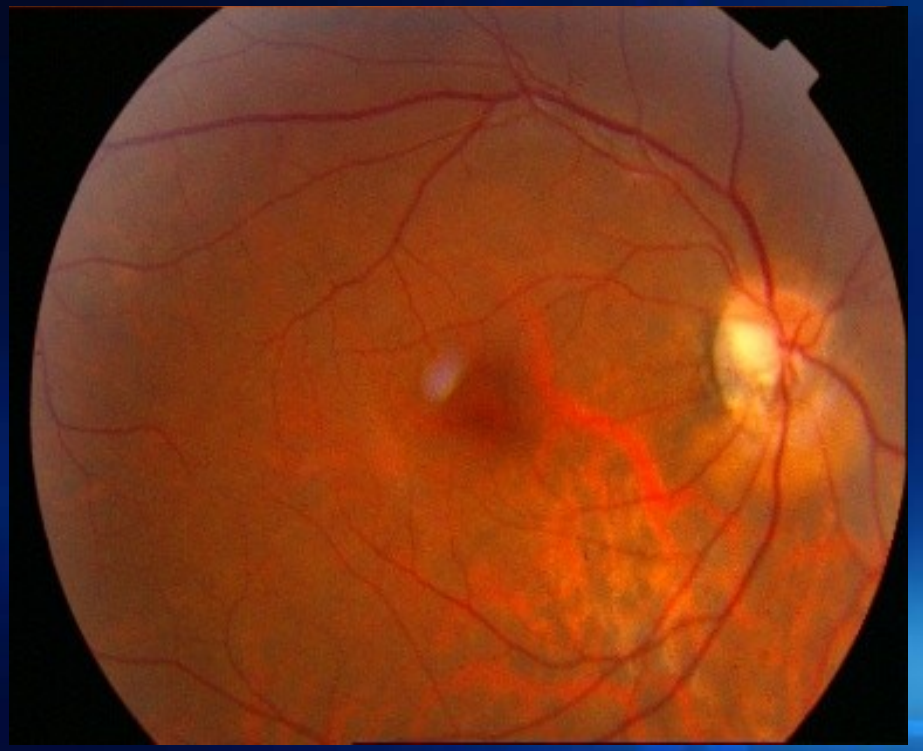


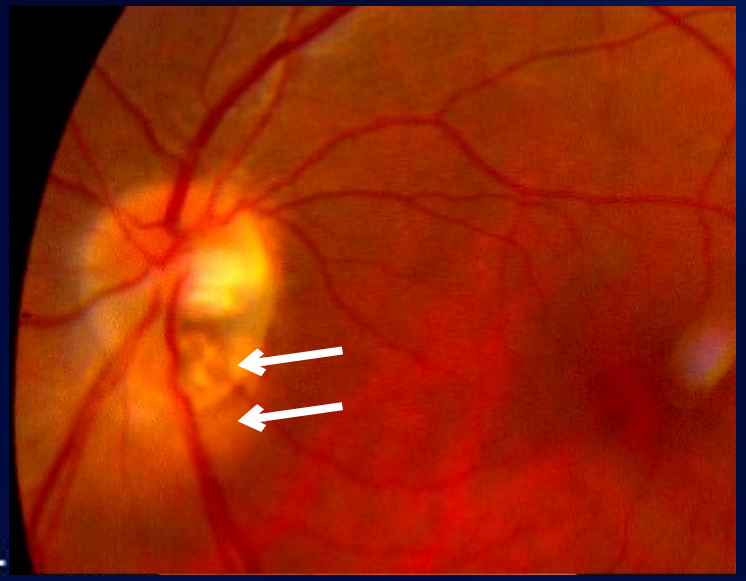
Familial Exudative Vitreo-retinopathy
FEVR



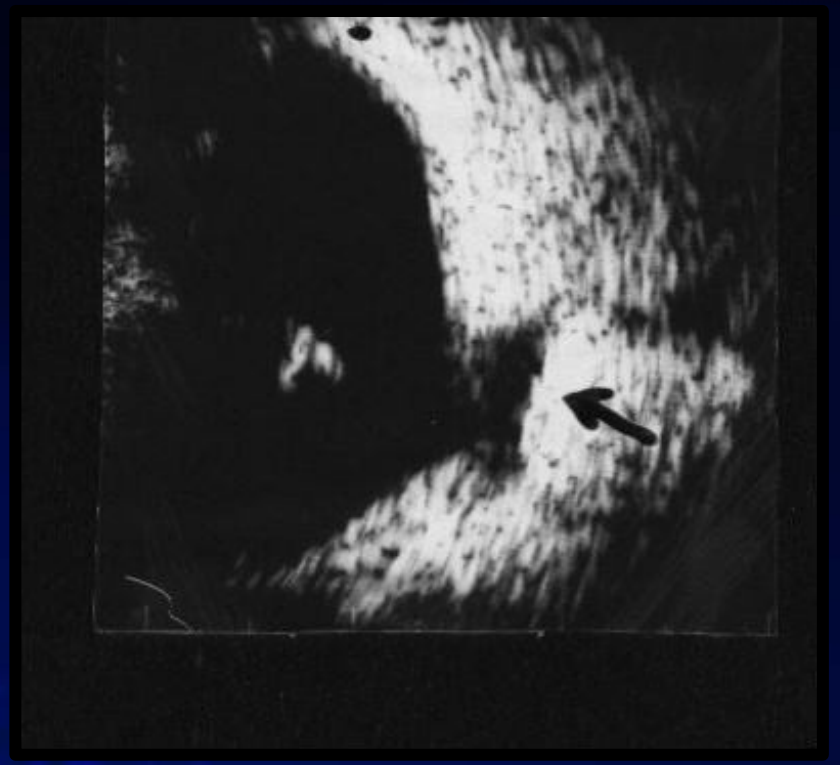
Optic Nerve Hypoplasia

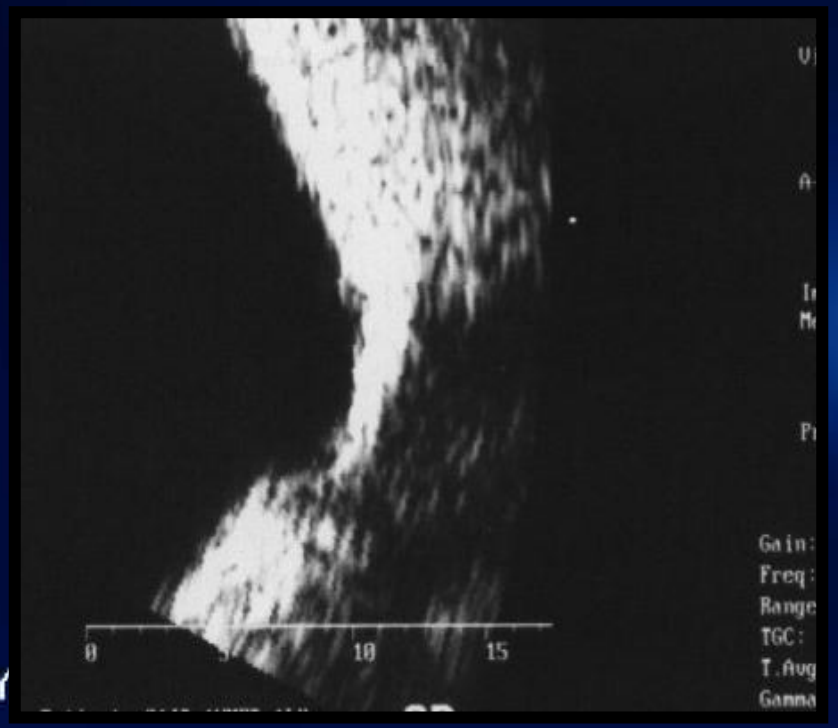
- An abnormally small optic nerve head
- Optic nerve pallor
- The ”double-ring sign”

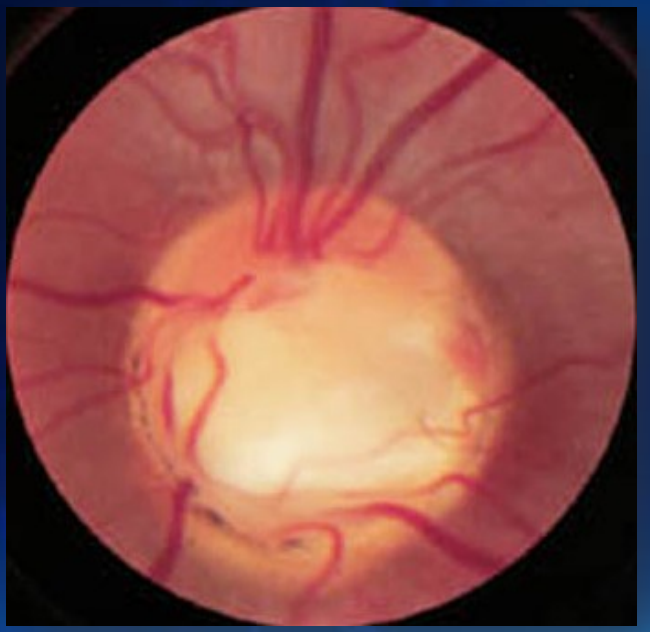




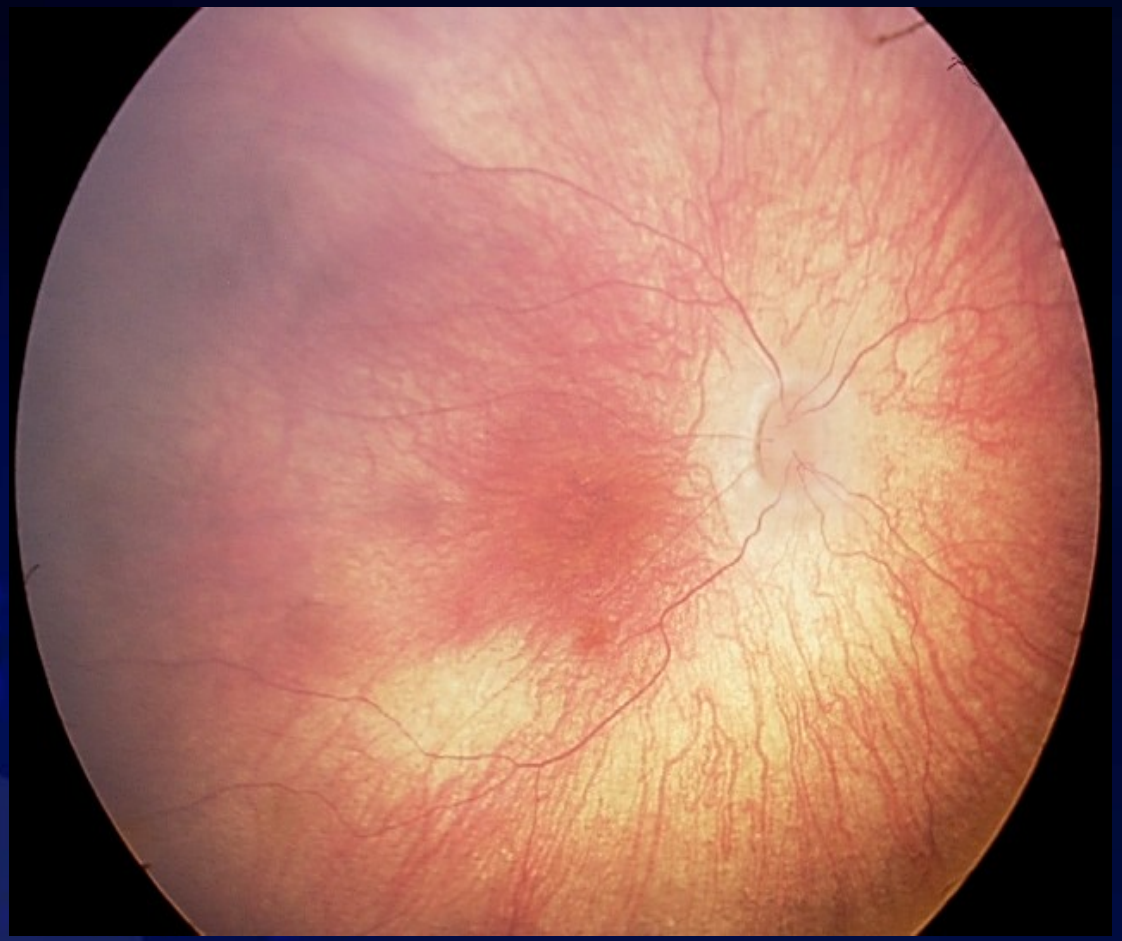
Optic nerve Coloboma

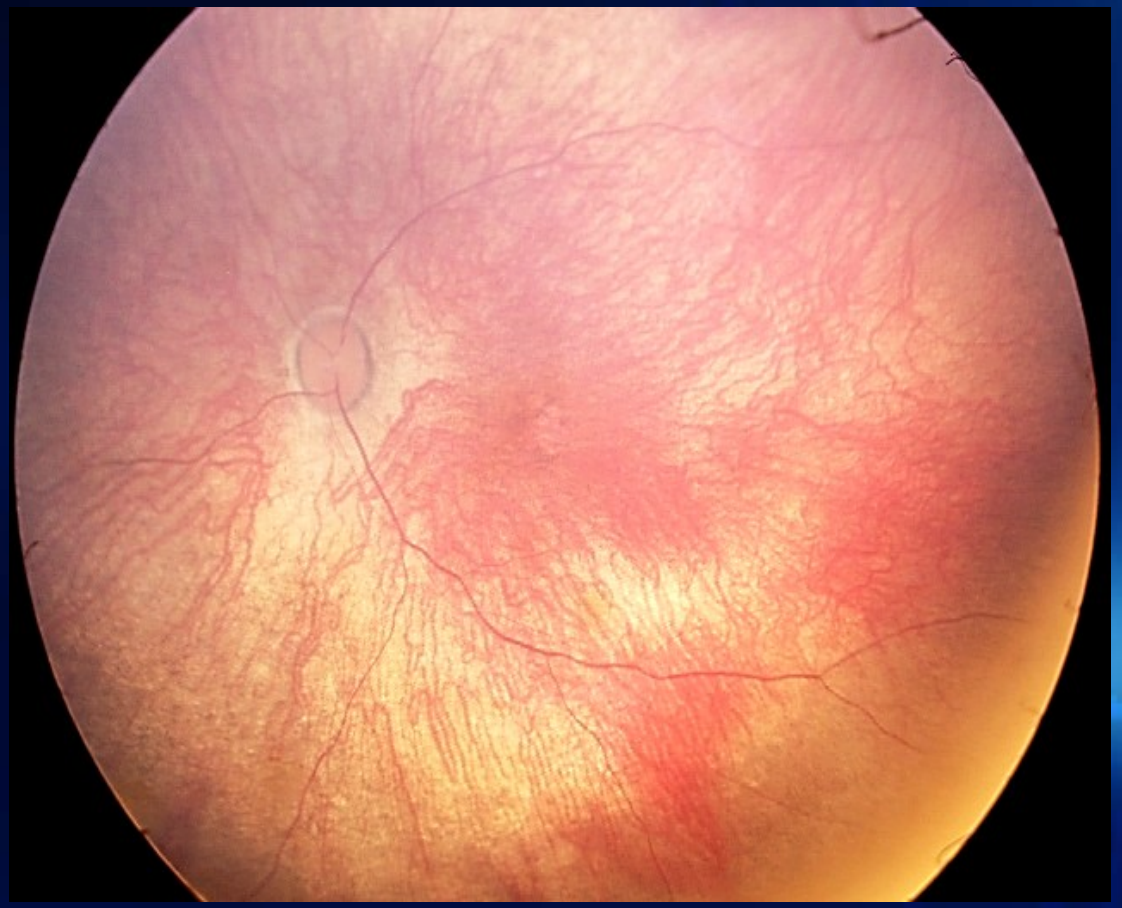




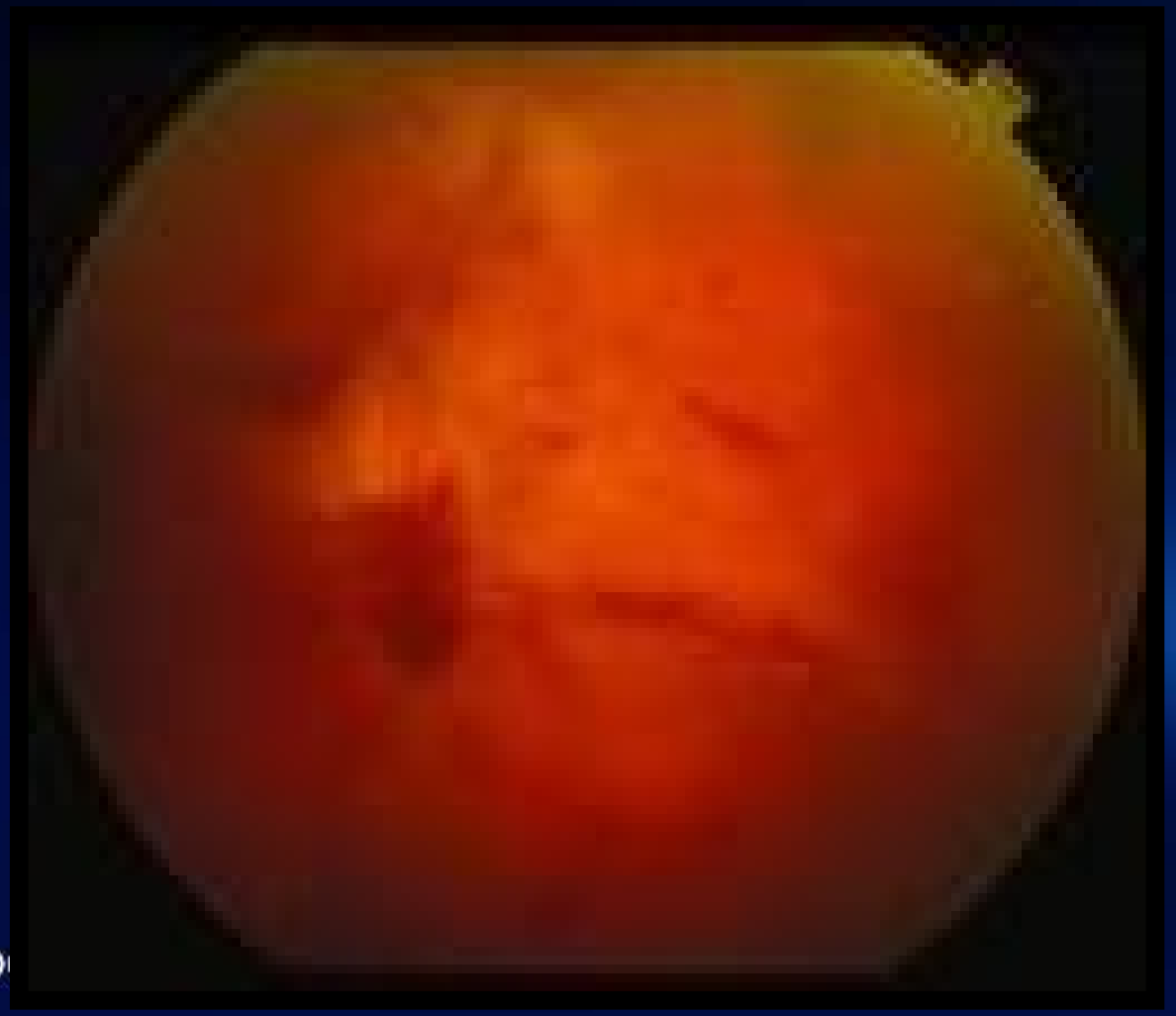


Foveal Hypoplasia



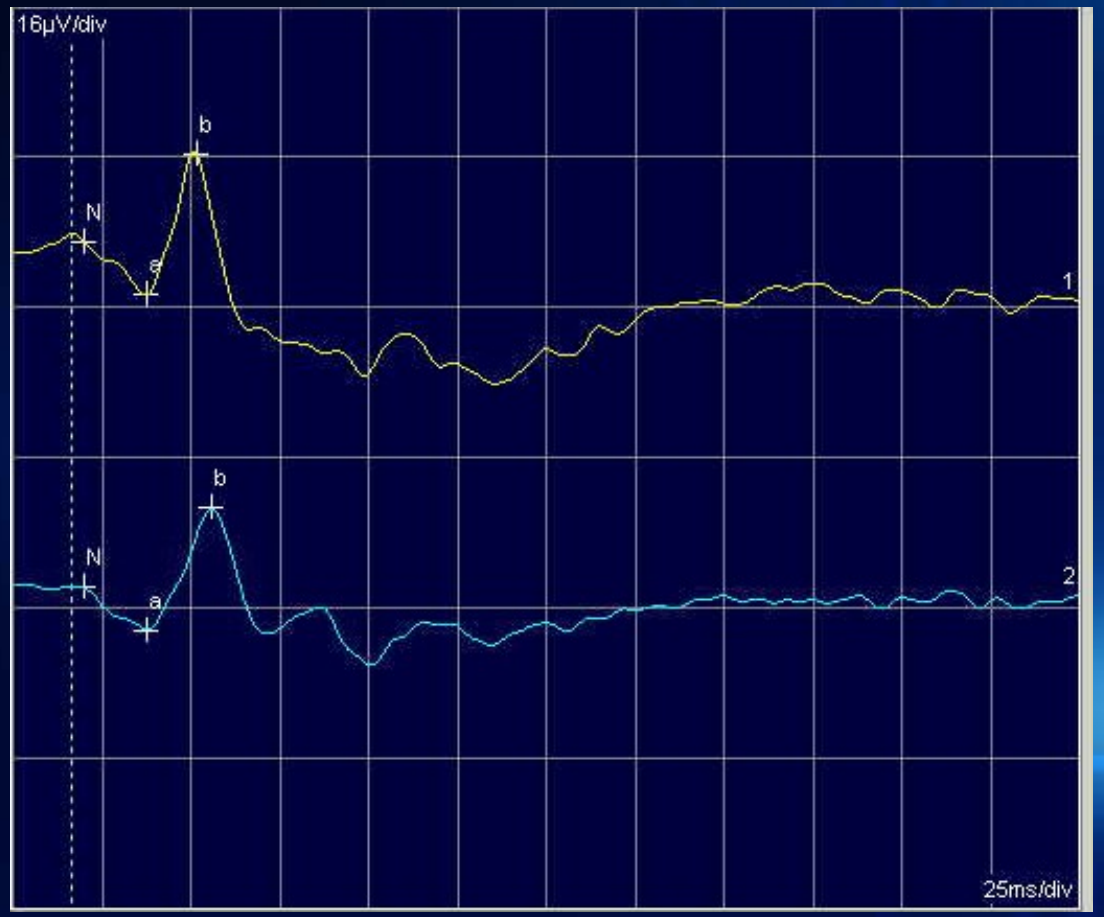


Vitreous hge (Shaken Baby Syndrome)



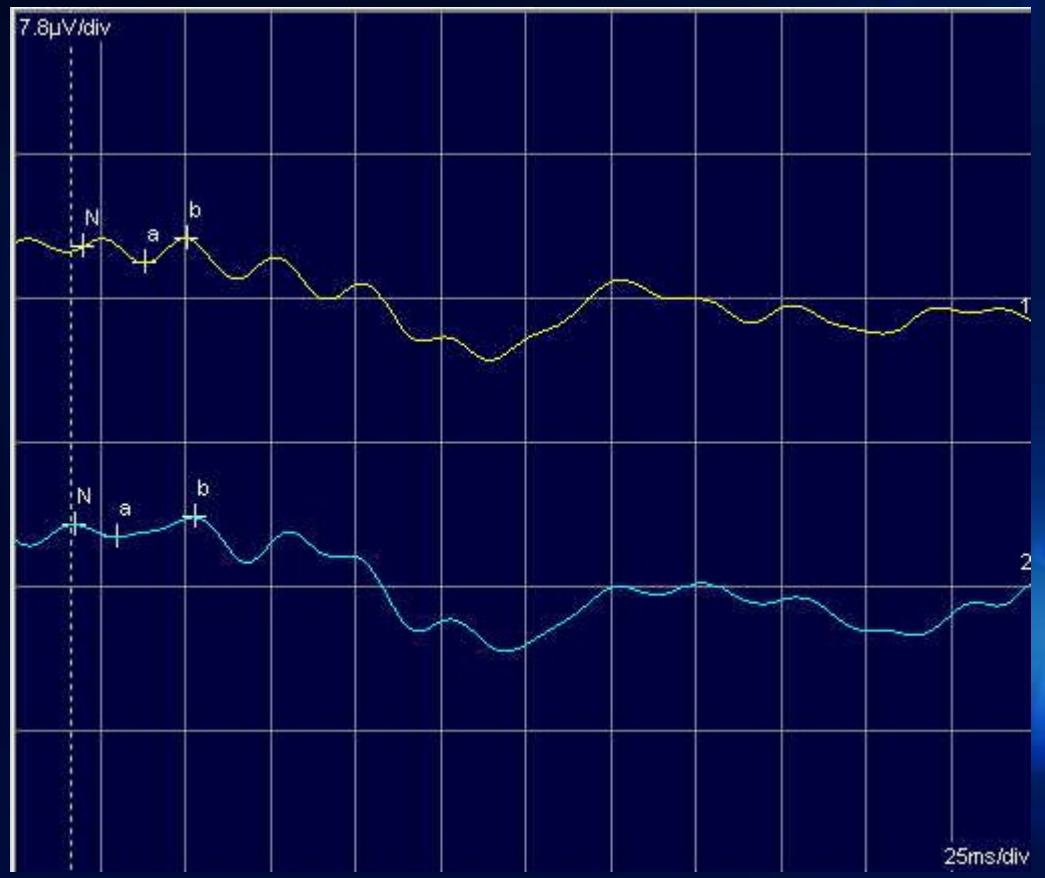
Retinal development

- With intense stimuli, both photopic and scotopic ERG components can be recorded within 14 h of birth
- Photopic components achieve adult values at about 2 months
- Under dark adapted conditions adult values are reached at 1 year



Rod/cone Dystrophy

Diminished cone response



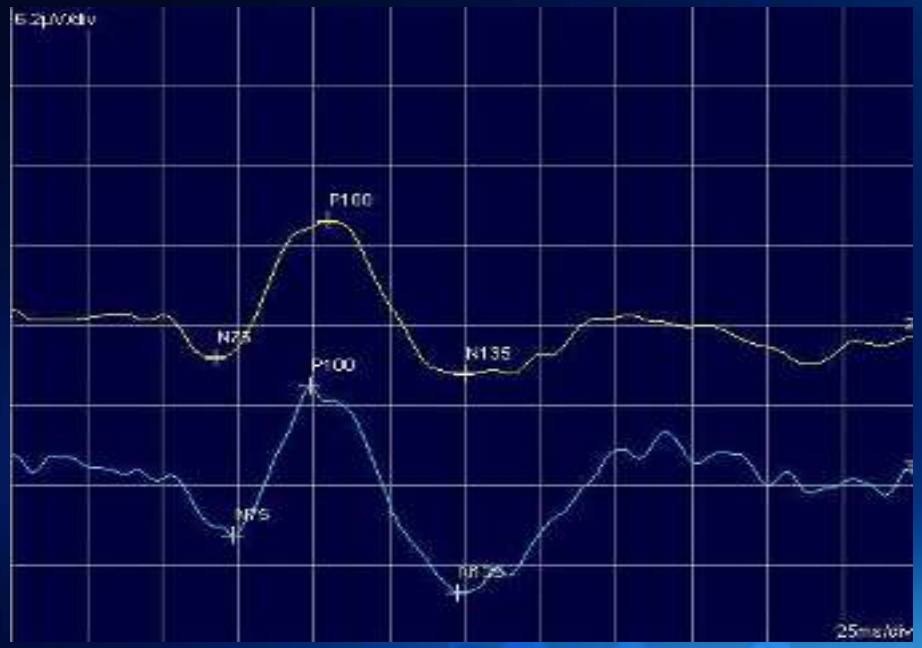
Diffuse Cerebral Dysfunction

Characterized by:

- Severe visual impairment
- Normal pupillary responses
- Normal fundus
- No nystagmus
- Usually with developmental delays and neurologic abnormalities
- VEPs ranged from normal to non-detectable
- Either abnormal flash or pattern VEPs or both

Delayed Visual Maturation

- Flash VEPs are reduced in amplitude and delayed in latency
- Delayed myelination of the optic nerve may underlie the flash VEP abnormalities
- Pattern VEPs are normal in cases of delayed visual maturation



Normal pattern VEP in delayed visual maturation

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