NOTES
VISION DISORDERS

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Vision deficit disorders
- Correlate with anatomical lesions along visual pathway
  - Light → cornea → lens → media → retina → optic nerve → chiasmal decussation → optic radiations (parietal, temporal paths) → primary visual cortex in occipital lobe

CAUSES

- Mass effect → impingement of structures
- Vascular → brain parenchyma infarction along visual pathway

SIGNS & SYMPTOMS

- Impaired vision

DIAGNOSIS

OTHER DIAGNOSTICS

- History
- Physical/neurologic examination
- Cranial nerve (CN) testing
  - *CN II:* visual fields and acuity
  - *CN II/III:* pupillary reflex
  - *CN III/IV/VI:* ocular movement

TREATMENT

MEDICATIONS

- *Vascular:* thrombolytics

SURGERY

- *Masses:* resection

BITEMPORAL HEMIANOPSIA

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PATHOLOGY & CAUSES

- *Visional deficit:* lateral vision loss
  - Optic chiasm lesions (commonly)
- *Pathogenesis:* ↑ sellar mass size → presses optic chiasm → impinges decussating visual fibers (most medial) → bitemporal hemianopsia

CAUSES

- Pituitary enlargement
  - Hyperplasia (i.e. pregnancy or lactation); adenoma (specific, hormone-secreting pituitary hyperplasia); cyst; abscess
- Craniopharyngioma
- Meningioma (in sella turcica)
- Saccular aneurysm (anterior communicating artery)
- Primary malignancy
  - Germ cell tumor (AKA ectopic pinealoma); chordoma; central nervous system (CNS) lymphoma

COMPLICATIONS

- ↑ size → further impinges surrounding structures
  - Cavernous sinus impingement → CN III, IV, VI → diplopia → ophthalmoplegia
- Dorsal extension of mass → dorsal midbrain impingement → Parinaud's syndrome
  - Upgaze paralysis
  - Pinealomas (posterior, common)

SIGNS & SYMPTOMS

- Vision loss
  - Lateral fields, both eyes (may go unnoticed; chronic, progressive)
- Headache
- Diplopia
- Ophthalmoplegia (especially large mass lesions, pinealomas)
- Hormonal deficiency/excess (if pituitary growth is functional)

DIAGNOSIS

DIAGNOSTIC IMAGING

MRI

- Visualize mass at area of optic chiasm; gadolinium-enhanced images aid elucidating pituitary tissue (↑ gadolinium uptake in pituitary)

CT scan

- Less diagnostic; may reveal sellar calcification, mass

TREATMENT

MEDICATIONS

- Smaller, hormone-responsive adenomas (prolactinomas → dopamine agonists first-line therapy)

SURGERY

- *Neurosurgery:* nasal aperture, posterior nasopharynx, sublabial (upper lip) incision accesses inferior aspect of cerebrum
  - *Fluoroscopic visualization:* navigation, pituitary visualization
- First-line therapy for all other pituitary adenomas, sellar masses with meaningful visual field impingement/other symptom severity

COLOR BLINDNESS

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PATHOLOGY & CAUSES

- Altered color perception
- Pathogenesis
  - Atypical cone type(s) function → altered color hue → limited color discrimination (commonly)
  - Optic nerve/other retinal lesions (uncommon)

CAUSES

Congenital

- Three cone types (opsins)
  - *Red, green opsins (X-chromosome):* most inherited color blindness X-linked recessive → predominantly biologically-male individuals
  - *Blue opsin (VII-chromosome):* blue wavelength deficiency, very rare
- Associated with Turner syndrome

Acquired

- Optic neuropathies
  - *Optic neuritis:* persistent color blindness after visual deficit restoration; early multiple sclerosis symptom
  - *Diabetic retinopathy:* neoproliferation, microvascular disease → retinal dysfunction (glaucoma)
- Bilateral, ventral occipital stroke → cerebral achromatopsia (rare)

Iatrogenic

- Ethambutol → poor red-green discrimination
- Digoxin → yellowish hue disturbance
- Other
  - Ibuprofen, quinine, acetaminophen, sildenafil citrate, tobacco

COMPLICATIONS

- *Nyctalopia:* limited night vision

SIGNS & SYMPTOMS

- Limited color discrimination

DIAGNOSIS

- Family, medication history

OTHER DIAGNOSTICS

- *Ishihara plates:* visual stimuli, colors offer wavelength-specific stimulation for three cone types
  - Inability to perceive numbers/letters on plate → reveal cone type deficit(s)

TREATMENT

OTHER INTERVENTIONS

- No curative therapy
- Acquired disease
  - *Glaucoma:* regular eye examinations
  - *Diabetes:* glycemic control → ↓ microvascular disease; regular eye examinations
- Individual education → lifestyle adaptation → proper visual cue interpretation
  - Unable to perceive red vs. green light difference on traffic signals → location discrimination education → top vs. bottom light interpretation

CORTICAL BLINDNESS

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PATHOLOGY & CAUSES

- *Acquired blindness:* bilateral lesions to visual cortex in occipital lobe

Pathogenesis

- Vascular occlusion
  - Bilateral, distal posterior cerebral artery (PCA) occlusion; commonly embolic
  - Basilar artery occlusion → ↓ blood flow in bilateral distal PCAs
- Vascular flow dysregulation → posterior reversible encephalopathy syndrome (PRES)

CAUSES

- Primary visual cortex lesions (calcarine fissure in occipital lobe)
  - Neighboring lesions → similar anopsia

COMPLICATIONS

- Anton–Babinski syndrome (visual anosognosia)
  - Individual unable to perceive vision → blindness denial
  - Image confabulation common

SIGNS & SYMPTOMS

- Inability to perceive visual input
- *CN testing:* II/III preserved pupillary light reflex

DIAGNOSIS

DIAGNOSTIC IMAGING

MRI

- Some cases, detects cause (e.g. vascular occlusion, infarction)

OTHER INTERVENTIONS

History, physical examination

- Assess non-cortical functions: normal pupillary light reflex
  - Limited/no visual response with intact pupillary light reflex → blindness neurological not ocular

Fundoscopy

- Normal

TREATMENT

MEDICATIONS

- *Vascular occlusion:* thrombolysis
- *PRES:* emergent antihypertensives

OTHER INTERVENTIONS

- Spontaneous recovery
  - Visual defects may persist (e.g. prosopagnosia—inability to recognize faces)

HEMIANOPSIA

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PATHOLOGY & CAUSES

- Individual loses half of visual field, commonly due to retrochiasmatic lesion of visual tract

Pathogenesis

- Vascular
  - *Middle cerebral artery (MCA):* complete contralateral hemianopia
  - *Unilateral posterior cerebral artery (PCA):* contralateral hemianopia with macular sparing
- Mass
  - Visual pathway compression

CAUSES

- Unilateral optic tract lesion
- Large (complete) unilateral optic radiation lesion
- *Quadrantanopia:* sub-complete lesion, corresponds to lesioned optic radiation
  - Upper outer-quadrant deficit ("pie-in-the-sky" defect) → temporal lobe loop lesion
  - Lower inferior quadrant deficit → parietal lobe lesion
- Large, unilateral primary visual cortex lesion
  - Macular visual field spared
- Bilateral upper/lower visual cortex lesion → altitudinal hemianopia
  - Upper/lower field visual defect

COMPLICATIONS

- Vascular/mass effect territory-dependent
- PCA distribution
  - Diplopia, dizziness, balance issues
- Anterior cerebral artery (ACA)/MCA distribution
  - Ipsilateral motor and sensory symptoms
- Parietal lobe involvement → contralateral neglect
  - Lesion to non-dominant lobe → Gerstmann syndrome (finger agnosia, acalculia, agraphia, right-left visualization)
- Temporal lobe involvement → seizure

SIGNS & SYMPTOMS

- Visual field loss
  - *Unilateral hemianopia:* contralateral optic tract lesion (homonymous hemianopia); large, contralateral optic radiation lesion
  - *Superior quadrantanopia:* contralateral temporal lobe lesion of optic radiation loop
  - *Inferior quadrantanopia:* contralateral parietal lobe lesion of optic radiation loop
- Neurologic examination
  - *CN II testing:* visual field
  - Motor/sensory testing for concomitant symptoms

DIAGNOSIS

- History, physical examination

DIAGNOSTIC IMAGING

MRI

- Mass lesions/old stroke (preferred method)

CT scan

- Mass lesion and acute, hemorrhagic stroke

TREATMENT

SURGERY

- *Resection:* mass compressing the visual pathway

OTHER INTERVENTIONS

- Peripheral prism spectacles
  - High-power prism segments in regular spectacle lens → expands visual field up to 30°
- Saccadic eye movement training (scanning therapy)
  - Individual makes compensatory saccadic eye movements to side with lost visibility without moving head → ↑ function, injury prevention

HOMONYMOUS HEMIANOPSIA

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PATHOLOGY & CAUSES

- Lesion in optic tract → vision loss in each eye (corresponding halves of visual field)
- Pathogenesis
  - *Vascular:* large MCA/smaller anterior choroidal artery stroke
  - *Mass effect:* tumor, cyst, arteriovenous malformation (AVM)

CAUSES

- Unilateral optic tract lesion

SIGNS & SYMPTOMS

- Half of visual field lost
  - Not relieved by monocular vision (vision deficit persists despite closing one eye)

DIAGNOSIS

- History, physical examination

DIAGNOSTIC IMAGING

MRI

- Mass lesions/old stroke (preferred method)

CT scan

- Mass lesion/acute, hemorrhagic stroke

TREATMENT

SURGERY

- *Resection:* mass compressing the visual pathway

OTHER INTERVENTIONS

- Peripheral prism spectacles
- Saccadic eye movement training (scanning therapy)