Sleep Apnea & the Eye

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Sleep Apnea & the Eye

- Sleep Apnea
  - Clinical consequences
  - Diagnosis
  - Treatment
- Ocular Manifestations
  - Asthenopia
  - CPAP assoc red eye
  - Floppy eyelid syndrome
  - Diabetic retinopathy
  - NAION
  - Papilledema
  - Normal tension glaucoma

Online Resources

- Lecture Notes
  - http://richardtrevino.net
- Powerpoint Slides
  - http://slideshare.net/rhodopsin
- Free Texts

Sleep Disorders

OSA is the “most physiologically disruptive and dangerous of the sleep-related disorders.”

- Sleep apnea
- Insomnia
- Narcolepsy
- Restless leg syndrome
- Parasomnias
- Circadian disorders
- Drug side effects
- Shift work

Sleep Architecture

Obstructive Sleep Apnea

Any Condition that Causes or Contributes to Upper Airway Narrowing is a Risk Factor for OSA
- Obesity
- Enlarged Tonsils
- Anatomical Malformations
- Neoplasms
- Edema of the pharynx
- Lymphoid Hypertrophy
- Pharyngeal Muscle Weakness
- Dysooordination of Respiratory Muscles

Bernoulli Effect
Obstructive Sleep Apnea

Polysomnography (PSG)

- Excessive daytime sleepiness
  - Most common symptom
- Disruptive snoring
  - Also gasping/snorting during arousals
- Apneic events witnessed by bed partner
  - Disruptive snoring + witnessed apneas: 94% specificity
- Obesity
  - 30% of pts with a BMI > 30 have OSA, and 50% of pts with a BMI > 40 have OSA.
- Neck circumference
  - ≥ 40 cm had a sensitivity of 61% and a specificity of 93% for OSA
  - Correlates better than BMI
- Male
  - 2-3x more common than female
- Family history of OSA
  - Relatives have 2-4 fold risk

Clinical Characteristics

- Excessive daytime sleepiness
- Disruptive snoring
- Obstructive Sleep Apnea
- Obese
- Neck circumference
- Male
- Family history of OSA

Clinical Consequences

- Cardiovascular Disease
  - HTN, CAD/MI, CHF, Arrhythmia
- Stroke
- Obesity
- Metabolic Syndrome
- Other Diseases
  - Morning headache, Eye, Liver, Kidney, others
- Cognitive and Emotional
  - Impaired mental functioning
  - Depression
  - Mood alteration
  - Effects on bed partners
    - Disruptive snoring
    - Accidents
    - Drowsy driving
    - Workplace

Epworth Sleepiness Scale

- How likely are you to doze off or fall asleep in the following situations?
  - 0 = No chance, 1 = Slight chance, 2 = Moderate chance, 3 = High chance
  - Sitting and reading
  - Watching TV
  - Sitting inactive in a public place (theater, meeting)
  - As a passenger in a car for an hour without a break
  - Lying down to rest in the afternoon when circumstances permit
  - Sitting and talking to someone
  - Sitting quietly after a lunch without alcohol
  - In a car, while stopped for a few minutes in traffic
Obstructive Sleep Apnea

- **Treatment Options**
  - **Behavioral:** Weight loss, EtOH avoidance, nonsupine position
  - **Positive Airway Pressure:** CPAP, Provent, others
  - **Mandibular advancement device**
  - **Surgery:** UPPP, Tonsillectomy, Tracheostomy

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Provent

- Provent is a relatively new FDA approved proprietary device for treating OSA.
  - It is a 'one-way valve' that is taped into the nostrils, so that the seal is airtight.
  - By inhibiting the outflow of air, positive pressure in the airway is achieved

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OSA & the Eye

- **Ocular Manifestations of Sleep Apnea**
  - Asthenopia
  - CPAP-associated Red Eye
  - Floppy Eyelid Syndrome
  - Diabetic Retinopathy
  - NAION
  - Normal Tension Glaucoma

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Asthenopia

- **Common OSA-associated asthenopic symptoms**
  - Unexplained symptoms of blur
    - Trouble “focusing eyes”
    - Vision is 20/20 but the patient is c/o blur
  - Misinterpreting what is seen
    - Incorrect recording or copying
    - Work-related errors
  - Eye strain and/or fatigue
  - Headaches
    - Worse in the morning

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Asthenopia

- **If OSA is in the medical history**
  - Be on the lookout for sx of fatigue
  - Possibly due to poor compliance or residual fatigue
  - Offer supportive management (eg. CPAP compliance)

- **If OSA is not in the medical history**
  - High index of suspicion whenever the chief complaint is fatigue or asthenopia
  - Especially if habitus is Pickwickian
  - Be prepared to screen for sleepiness
CPAP-associated Red Eye

- Clinical Problems
  - Dry eye syndrome
  - EXW CL intolerance
  - Recurrent Conjunctival Erosion
  - Infectious conjunctivitis

- Causes
  - Air leaks
  - Retrograde air flow thru nasolacrimal apparatus

- Treatment
  - Lubricating ointments HS, punctal plugs
  - CPAP refitting: adjust headgear and pressure

Persons with OSA generally have greater ocular discomfort than controls, but is greatest among persons that are noncompliant with CPAP

Floppy Eyelid Syndrome

- Clinical Characteristics
  - Eyelid hyperlaxity
    - Rubbery, easily ejected upper eyelids
    - Eyelash ptosis with loss of parallelism
  - Papillary conjunctivitis
    - Chronic ocular irritation, worse upon waking
    - SPK, mucoid discharge common
    - Rubbing on pillow case

- Eyelash ptosis
  - Downward displacement of eyelashes
  - Lashes may point in various directions
  - Loss of parallelism
  - Ps may trim with scissors
**Floppy Eyelid Syndrome**

**Etiopathogenesis**
- Loss of elastic fibers in tarsus and upregulation of elastase MMP
- Likely caused by repeated mechanical trauma, possibly eye rubbing or sleeping with the face buried in the pillow
- May represent an adaptive response that allows tensional homeostasis to be maintained at the high levels of tissue stress experienced in FES
- FES strongly associated with keratoconus, reinforcing suspected role of mechanical trauma

**Treatment**
- CPAP therapy
  - Treatment of OSA can improve symptoms of FES
- Protect eye during sleep
  - Ointments HS
  - Patching, taping, sleep mask
- Surgical therapy is considered the definitive treatment
  - Greatest success with medial canthus/lateral canthus plication and upper lid lateral tarsal strip procedures
  - 25-50% failure rate within 2yrs

**Relation to OSA**
- 5-15% pts with OSA have FES
- 96% pts with FES have OSA
- OSA tends to be more severe in pts with FES
- FES strongly associated with OSA even after adjusting for weight

**OSA & the Eye**

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**Diabetic Retinopathy**
- OSA associated with higher risk of PDR, independent of other risk factors
- Risk of progression associated with severity of OSA
- OSA increases risk of NVG in patients with PDR
- CPAP may prevent progression of diabetic retinopathy by minimizing nocturnal hypoxia
- Diabetics with OSA should be screened for retinopathy and encouraged to be compliant with CPAP

**NAION**

- Clinical Characteristics
  - Most common acute optic neuropathy in pts ≥50yo
  - Sudden painless visual loss, usually upon awakening
  - Nerve fiber bundle VF defects
  - Diffuse or sectoral disc edema
  - Disc at risk: small, crowded
    - Mean C/D = 0.2
    - All ≤ 0.4
NAION

Pathophysiology
- Idiopathic ischemic process
- Disorder of posterior ciliary artery circulation
- Transient poor circulation in the ONH
- Trigger Event: Fall in blood pressure below a critical level
- There is no actual blockage of the posterior ciliary arteries

Cascade Effect
- Mechanical crowding caused by small crowded disc
- Ischemia → Swelling → Compression → Ischemia

Diagnosis: Must exclude GCA in every case
- ESR
- C-Reactive Protein
- Positive acute-phase protein
- Levels increase in presence of inflammation
- Upper limit normal does not rise with age
- Platelets
  - Secondary thrombocytosis due to chronic inflammation

Treatment
- Aspirin
  - Decreases incidence in fellow eye at 2 years, but not at 5 years
- Surgical decompression
  - No benefit (Ischemic Optic Neuropathy Decompression Trial)
- Control of predisposing systemic disease
  - May slow progression or reduce incidence in fellow eye
  - Hypertension, Diabetes, Hyperlipidemia, OSA
- Avoid phosphodiesterase 5 inhibitors (Viagra, Levitra, Cialis)
  - May increase risk of NAION in fellow eye

Medicolegal obligation to inform pts of risk to fellow eye

Relation to OSA

<table>
<thead>
<tr>
<th>NAION Patients with OSA</th>
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<tr>
<td>Mojon (2002)</td>
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<td>Palombi (2006)</td>
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<td>Li (2007)</td>
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- Conclusions
  - OSA may play an important role in pathogenesis of NAION
  - OSA may the systemic disorder most frequently associated with NAION
  - Patients with NAION should be screened for OSA

Clinical Characteristic
- Disc swelling associated with increased ICP
- Symptoms of elevated ICP: Headache, tinnitus, TOV
- Chronic papilledema (months) may lead to optic atrophy and vision loss
Papilledema

- **Work-up**
  - Urgent MRI or CT scan
  - Lumbar puncture if imaging normal

- **Idiopathic Intracranial Hypertension**
  - “Pseudotumor cerebri”
  - Syndrome of elevated ICP, papilledema, normal MRI/CT, normal CSF
  - Secondary pseudotumor cerebri syndromes
    - Venous sinus thrombosis, vitamin A toxicity, COPD, OSA
  - Tx: Diamox 250mg po QID, Underlying cause if known

- **Papilledema**

  - **Relation to OSA**
    - Stein (2011)
      - Reviewed 2.3 million insurance company billing records
      - Persons with OSA have 30% to 100% increased risk of developing papilledema
    - Parvin (2000)
      - 4 pts with unexplained papilledema that resolved with successful tx of OSA
      - ICP is normal during the day but elevated at night
      - Intermittent ↑ ICP can cause sustained papilledema
      - Hypercapnia-induced cerebral vasodilatation elevates ICP

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Normal Tension Glaucoma

- **Clinical Characteristics**
  - Probably a variant of POAG
  - IOP is never documented above 21 mmHg
  - Peripapillary hemorrhages may be more frequent
  - Peripapillary atrophy may be more marked
  - VF defects tend to be deeper and more localized

- **Diagnosis**
  - R/O other glaucomas
  - Diurnal IOP fluctuation
  - IOP normalization (burnt-out glaucoma, pseudophakia, steroids)
  - R/O other optic neuropathies
    - NAION, space-occupying lesions, congenital anomalies
    - When to order neuroimaging:
      - Younger age (<50 yrs)
      - Reduced VA (<20/40)
      - Vertically aligned VF defects
      - Neuroretinal rim pallor
Normal Tension Glaucoma

Relation to OSA

<table>
<thead>
<tr>
<th>Patients with OSA</th>
<th>Glaucoma Patients with OSA</th>
<th>OSA Patients with Glaucoma</th>
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6%-10% of OSA patients have NTG

50% of NTG patients have OSA

Treatment of OSA may help stabilize NTG (Kremmer, 2003) and improve VF performance (Sebastian, 2006)

CPAP Increases IOP

Kiekens (2008)

Average IOP and diurnal fluctuation higher with CPAP

30 min after CPAP cessation a significant decrease in IOP was recorded

Speculate that CPAP elevates intrathoracic pressure, leading to higher central venous pressure, and ultimately higher IOP

Recommend regular screening of VF and the optic disc for all patients with OSA, especially those treated with CPAP

Source: Arch Ophthalmol 2010;128:1257-1263

OSA May Cause NFL Loss Without Glaucoma

NFL thinning may place patients with OSA at increased risk of glaucoma

Kargi (2005)

Comparing NFL in 105 pts with OSA and 22 controls using OCT

NFL thickness was reduced in patients with OSA

Thinning was correlated to severity of OSA (AHI)

Pts with VF defects were excluded from the study

Lin (2010)

Comparing NFL in 105 pts with OSA and 22 controls using OCT

NFL thickness was reduced in patients with OSA

Thinning was correlated to severity of OSA (lowest oxygenation saturation on PSG)

Pts with OSA did not have an increased prevalence of VF defects ("silent optic neuropathy")
Normal Tension Glaucoma

Conclusions & Recommendations
- Persons with OSA should be screened for glaucoma
- Risk of glaucoma is correlated with severity of OSA
- Patients with NTG should be screened or at least questioned about OSA to determine if that is part of the explanation for their glaucomatous damage at normal IOPs
- Treatment of uncontrolled OSA may help stabilize glaucoma and improve VF performance
- Initiation of CPAP therapy may increase nocturnal IOP
- The clinical significance of this is unknown