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#### OSA is:

- a sleep discorder
- with nocturnal pharyngeal collapse
- hypopneic or apneic events during sleep

#### Causes for ocular manifestations are:

- vascular
- mechanical



- Floppy eyelid syndrome
- Retinal Conditions
  - retinal vein occlusions central serous retinopathy diabetic retinopathy
- NAION (nonarteritic ant. ischemic optic neuropathy)
- Papilledema
- Glaucoma



- Floppy eyelid syndrome
- Retinal Conditions
   Retinal vein occlusions
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# Floppy eyelid syndrome (Signs and symptoms)

eyelid hyperlaxity and easily everted eyelids

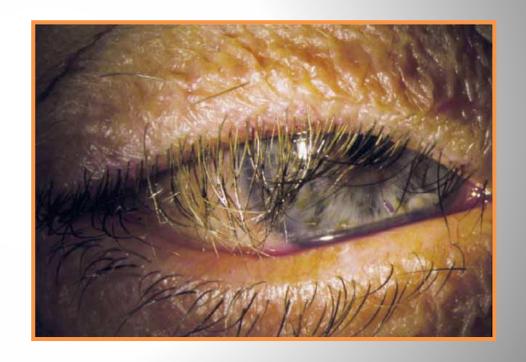
chronic irritation, discomfort, foreign body sensation, dryness specially by awakening





# Floppy eyelid syndrome (Signs and symptoms)

eyelash ptosis with lashes displaced downward leading to possible corneal erosions, keratitis, even perforation

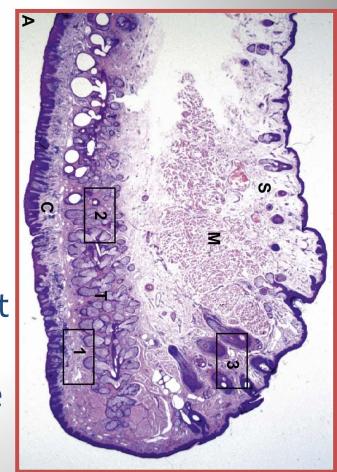




# Floppy eyelid syndrome (etiology)

Decreased elastin and increased matrix metalloproteinase acitivity leading to a weak tarsal plate (connective tissue weakness)

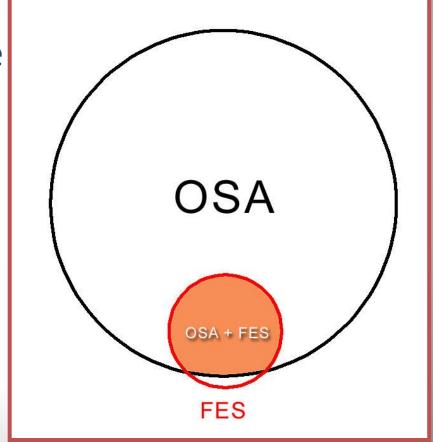
mechanical pressure and transient ischemia in lid tissue due to reduced arousability and pressure from the pillow





### Floppy eyelid syndrome

- 10%-20% of OSA patients have FES (40% with severe OSA)
- 96% of FES patients have
   OSA



Surv Ophthalmol 2010;55:35-46



# Floppy eyelid syndrome (therapy)

 Artificial tears and ointments to relieve the dryness and protect cornea from exposure

CPAP

No Surgical correction without treating OSA!!
 (high rate of recurrence)



### Floppy eyelid syndrome

Ask your OSA patients if they experience:

- Ocular discomfort
- Excessive tearing
- Blurry vision upon awakening



- Floppy eyelid syndrome
- Retinal Conditions
   retinal vein occlusions
   central serous retinopathy
   diabetic retinopathy
- NAION (nonarteritic ant. ischemic optic neuropathy)
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#### **Retinal conditions**

### Lack of OXYGEN!!!

#### **OSA** promotes:

- Virchows triad: hypercoagulability, hemodynamic changes, endothelial dysfunction
- injury and imperfect repair of blood vessels
- increased levels of VEGF



# Retinal conditions (RVO: Retinal vein occlusions)

- Second most common cause of vascular blindness
- BRVO: branch retinal vein occlusion
- CRVO: central retinal vein occlusion

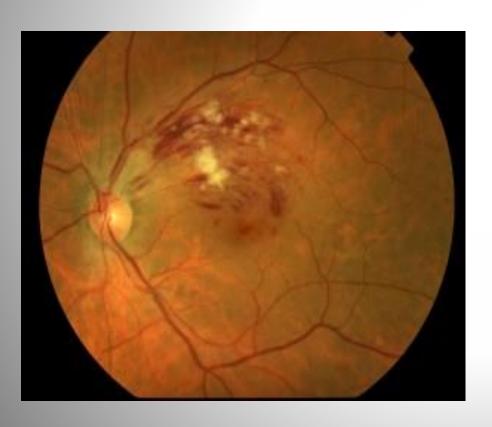


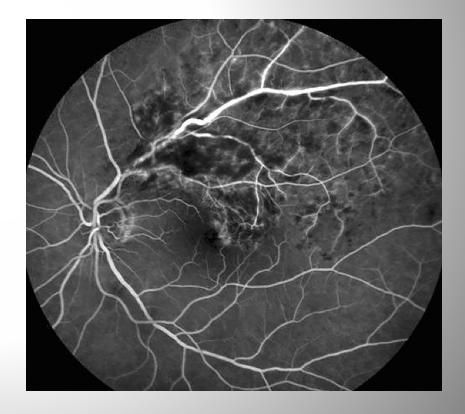
## Retinal conditions (RVO: Retinal vein occlusions)

- OSA prevalence of 77% among RVO-Patients (Glacet-Bernard et al.)
- related to OSA induced changes on bloodflowautoregulation and microvasculature
- presence of hypoxemia and nocturnal ICP elevations
- hemodynamic changes in CRA due to hypercapnia leading to a compression of RV



# Retinal conditions (BRVO: Branch retinal vein occlusion)

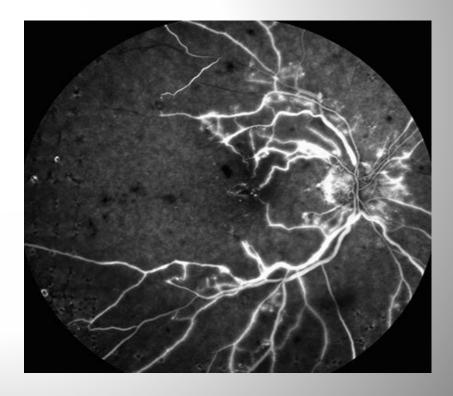






# Retinal conditions (CRVO: central retinal vein occlusion)

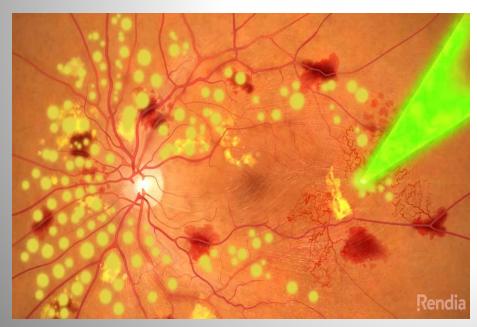


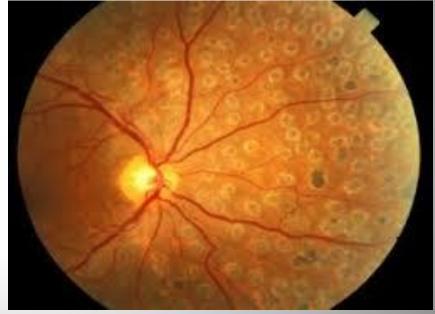




# Retinal conditions (CRVO and BRVO: treatment)

### Argon-Laser coagulation Anti-VEGF-Injections







## Retinal conditions (CRVO and BRVO: treatment)

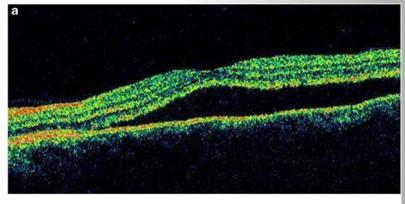
Ask your OSA patients about:

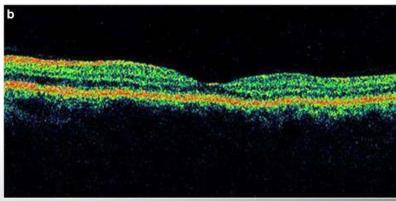
- visual symptoms
- transient loss of visual fields



# Retinal conditions (CRS: central serous retinopathy)

- idiopathic retinal detachment
- visual impairment
- metamorphopsia







## Retinal conditions (CRS: central serous retinopathy)

- association between CRS and OSA (Huon et al.)
- increased levels of epinephrine and norepinephrine known in OSA
- endothelial dysfunction in blood-retina-barrier
- subretinal fluid accumulation



## Retinal conditions (CRS: central serous retinopathy)

Ask your OSA patients about:

- visual abnormalities (darkening, image magnification, reduction of VA)
- seeing straight lines as wavy



Nr. 1 cause of vascular blindness

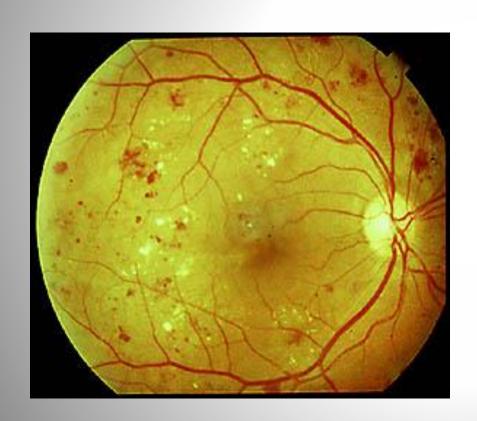
- microvasculopathy, increased VEGF
- clinical presentation:

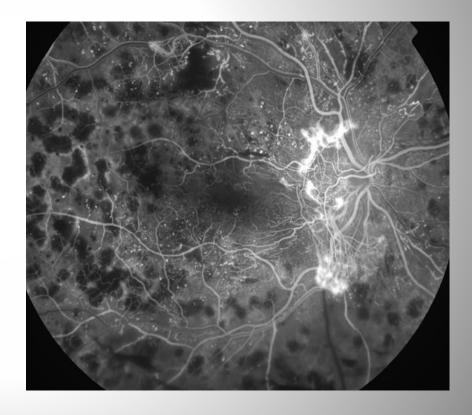
non-proliferative DRP proliferative DRP diabetic maculopathy



- common and severe in patients with OSA
- OSA is an independent risk factor for progression (Quratul et al.)
- CPAP, slows progression by minimizing nocturnal hypoxia

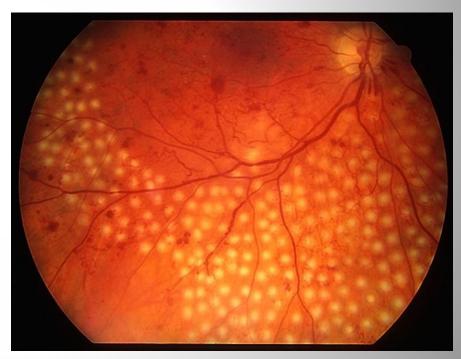








- controlling systemic diseases: blood sugar, hypertension, screen for OSA!!!!!
- Argon-Laser-Coagulation
- Injection of Anti-VEGF



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Ask your OSA patients with known diabetes:

Do you visit an ophthalmologist on a regular basis?



- Floppy eyelid syndrome
- Retinal Conditions
   retinal vein occlusions
   central serous retinopathy
   diabetic retinopathy
   age-related macular degeneration
- NAION (nonarteritic ant. ischemic optic neuropathy)
- Papilledema
- Glaucoma



#### NAION

(nonarteritic anterior ischemic optic neuropathy)

- acute, painless, irreversible visual loss
- upon awakening
- ischemia in the post.
   short cilliary arteries
- 15% risk for the contralateral eye





### (nonarteritic anterior ischemic optic neuropathy)

- Impaired blood flow autoregulation and increased intracranial pressure during apneic episodes limit optic nerve perfusion
- 16% higher risk of development of a NAION in patients with untreated OSA (Stein et al.)
- urgent need to alleviate risk factors like hypertension diabetes OSA!!!!



#### NAION

(nonarteritic anterior ischemic optic neuropathy)

Ask your OSA patients about:

- history of a sudden visual loss in one eye upon awakening
- history of an "optic nerve infarction"

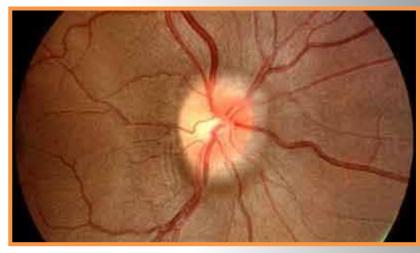


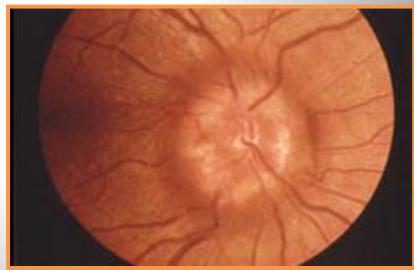
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### Papilledema

- disc swelling associated with increased ICP
- symptoms of elevated ICP:
   headache, tinnitus, trans.
   obscuration of vision
- chronic papilledema
   may lead to optic
   atrophy and vision loss







### Papilledema

 Persons with OSA have 30% to 100% increased risk of developing papilledema (Stein, 2011)

 Hypercapnia induces cerebral vasodilatation, which elevates ICP leading to papilledema

• Intermittent (nocturnal) increase in ICP can cause sustained papilledema (Parvin, 2000)



### Ask your OSA patients about:

• headache, tinnitus, trans. obscuration of vision

progressive decrease in vision



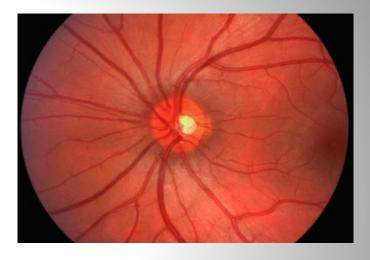
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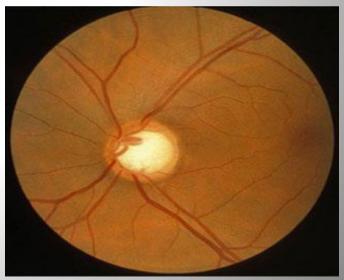


- chronic progressive optic neuropathy
- second leading cause of blindness worldwide

### Types:

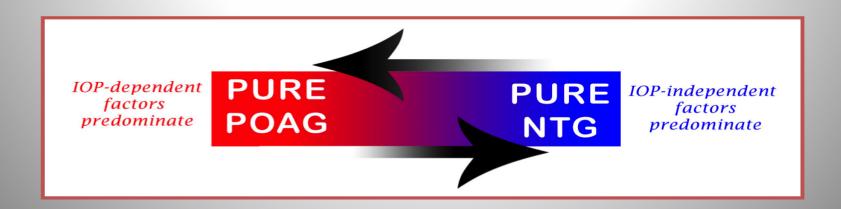
- open angle glaucoma (OAG)
- closed angle glaucoma (CAG)
- normotensive glaucoma (NTG)







- OAG and CAG are associated with an increased IOP leading to a mechanical compression of optic nerve
- NTG patients have normal IOP levels but a characteristic glaucoma damage to optic nerve



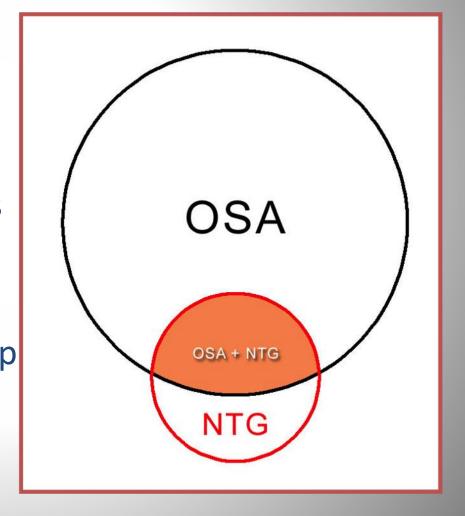


- OSA contributes to the pathogenesis of NTG through:
  - -vascular effects (optic nerve ischemia)
  - episodes of hypoxia
  - hemodynamic changes
  - oxidative stress
- Patients with OSA may have optic nerve heads more susceptible to mechanical stress due to poor perfusion



- 5%-10% of OSA patients have NTG (<3% general pop)
- up to 50% of NTG patients have OSA

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





Ask your OSA patients if they:

Have glaucoma

Note: There are no symptoms until late in the course of the disease.

Refer every OSA patient for a glaucoma screening.



# Ocular manifestations of OSA (conclusions)

- Remain alert to the potential ocular sequelae of OSA to prevent permanent vision loss in these patients!
- Increased referrals between sleep specialists and ophthalmologists improves vascular and ocular health of patients with OSA



### Thank you for your attention!

