

## Headmirror's ENT in a Nutshell

### Sleep: Obstructive Sleep Apnea In Adults

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**Definition: Obstructive sleep apnea in adults is a sleep disorder characterized by periodic, complete or partial upper airway obstruction during sleep that causes apneas or hypopneas despite ongoing respiratory effort.**

### Presentation (0:55)

- Symptomatology
  - Snoring (most common)
  - Choking and gasping in sleep causing recurrent awakenings
  - Unrefreshing sleep, daytime sleepiness
  - Multiple Long-term sequelae: cardiovascular disease, hypertension, cerebrovascular disease, and potentially dementia. All secondary to intermittent hypoxia.
    - Other Contributing factors:
      - Decreased Genioglossus muscle activation, pulls tongue out of pharynx
      - Ventilator instability, regulation of breathing. High loop gain
      - Low arousal threshold, awakes easily. Awakening with minor breathing pause
  - In the United States, estimated to that 5% women, 10% men have OSA
  - Differential diagnosis: other SDB such as primary snoring, upper airway resistance syndrome, obesity hypoventilation (linger beyond sleep period), central sleep apnea (brain not trigger), insufficient sleep, insomnia, depression, sleep timing disorders
  
- Factor contributing to OSA (4:15)
  - Anatomical: Crowded anatomy of oral cavity and oropharynx. Largest contributor
  - Physiological:
    - Increased collapsibility of tissues when sleeping
    - Loss of muscle tone: genioglossus m. relaxation
    - Blunting of tone when sleeping
  - Ventilatory instability: high loop gain or over/under correction of breathing
  - Low arousal threshold, awake with minor breathing pause and disrupt sleep
  
- History
  - Chief concern: snoring, sleepiness
  - Quantify symptoms: Epworth sleepiness scale (11:50)
    - 0-24 validated scale, level of sleepiness during the day
      - 0-10=normal
      - 10-16=suffer from increased daytime sleepiness
      - >16=dangerously sleepy
  - Family history of OSA can be predictive
  - Older age, more laxity

- Clinical Examination (13:50)

- Obese patients: Neck circumference >15.5 in. female, 17 in. male
- What anatomical factors are contributing?
  - Facial skeleton: micrognathia
  - Nasal obstruction: septum, turbinates, nasal valve collapse
  - Oral cavity: Large tongue, observe where it sits, look for scalloping
  - Oropharynx: Palatine Tonsils, lingual tonsils, soft palate (length/laxity/collapse), uvula
  - Pharyngeal collapse, redundant tissue
- Modified Mallampati
  - I: hard palate, soft palate, uvula, and tonsillar pillars visible
  - II: hard palate, soft palate and base of the uvula visible
  - III: hard and soft palate visible
  - IV: only hard palate visible
- Friedman Staging System: FTP + Tonsil + BMI. Score can predict UPPP results

**Table** Friedman staging system as determined by Friedman tongue position (FTP), tonsil size, and body mass index (BMI).

Stage	FTP	Tonsil size	BMI (kg/m <sup>2</sup> )
I	I, IIa, IIb	3 or 4	<40
IIa	I, IIa, IIb	0, 1, or 2	<40
IIb	III or IV	3 or 4	<40
III	III or IV	0, 1, or 2	<40
IV*	I-IV	0-4	>40

\*All patients with significant craniofacial or other anatomical abnormalities.

- Friedman tongue position: tongue in a neutral position
  - I: uvula and tonsils or pillars
  - IIa: most of uvula, no tonsil or pillars
  - IIb: all of soft palate (no uvula), no tonsil or pillars
  - III: some of soft palate with absent distal end
  - IV: only hard palate
- Tonsil hypertrophy
- BMI
- In general favorable results after UPPP: large tonsil, small tongue, not obese. 80% success with Friedman 1

**Workup (19:09)**

- Sleep testing (please see podcast on sleep studies)

- to diagnose and assess severity of OSA
  - Formal diagnosis: **5 or more obstructive events per hour**. Repeated choking or airway obstruction during sleep that disrupts sleep. Measure breathing.
    - Complete obstruction: apnea
    - Incomplete obstruction: hypopnea, but ramification of awakening/oxygen drop
- Treat (23:30):
  - OSA syndrome: 5 or more apnea/hypopnea per hour with symptoms
  - 15 or more w/ or w/o symptoms. Measurable increase in long term risk from epidemiology data
- Mild 5-15, mod 25-30, severe >30.

- O2 saturation fluctuation is linked to long-term risk factor for developing disease and death
  - Percentage of time low O2 predictive of long term outcomes. 15 or more per hour desaturations suggest poor long term health implications
  - Sleep test does predict long term outcomes health implication, not indicative of extent of symptoms
- Awake Endoscopy
  - for dynamic collapse: determine level of obstruction
  - Maneuvers while awake:
    - Lay supine to assess for BOT collapse
    - Muller maneuver (mimic hypotonia): inhale, exhale as much as possible, very end of exhale creates reflex that mimics hypotonia
- DISE (please see podcast on sleep studies)
  - Sleep endoscopy under sedation assess airway for treatment. Tailor surgical plan.
- Imaging
  - XR/CT for skeletal surgery, not routinely used

## Complications

- Effects of OSA: disruptive sleep day-to-day, long term increased sympathetic discharge, oxidative stress due to low O2 and intermittent hypoxia. Cardiovascular MI/HTN/arrhythmia/stroke. Potentially, dementia. May affect all organs secondary to intermittent hypoxia. Overall, shorter life spans with severe sleep apnea.

## Treatment (28:00)

- Multidisciplinary: Sleep medicine, Dental, Neurology, ENT.
  - Medical management: Do they need treatment?
    - Behavior modification, getting more sleep, avoid sedative/alcohol, weight loss, positional interventions/reduce supine sleep
    - Medications don't have a role directly, but hypnotic agent to decrease threshold of awakening. Stimulative for residual sleepiness, modafanil.
  - Mandibular advancement device (31:30)
    - Best for patients with mild OSA
      - Requires adequate teeth, one molar in each quadrant. Protrusive capacity required.
      - Class II malocclusion best, favorable movement of teeth. May mitigate even if not best candidate
  - Continuous positive airway pressure (CPAP) (34:30)
    - *Gold standard*: air pressure in the upper airway prevents collapsing.
      - Fixed pressure or auto adjust dependent on detection of collapse.
    - BiPAP adjust pressure insp/exp, often higher pressure.

- ASV gives a breath in central sleep apnea.
  - Requires efficacy – CPAP can obliterate sleep apnea and can completely treat, decrease symptoms, and improve long term health. Effectiveness in the real world not always 50% of people.
  - Adequate use: 4 hours per night, 5 nights per week (insurance standards over 30 day period) this cut off improves. Dose dependent.
  - Contraindications: intolerable side effects, skull base defect (pneumocephalus), some lung disease, recurrent sinusitis, air leak, aerophagia
- Surgery: address abnormal anatomy that is seen on exam (42:40)
- Goal: facilitate improvement or eliminate OSA or salvage improvement in OSA
1. Adjunct to CPAP/oral appliance. Example: nasal surgery to decrease obstruction and lower CPAP requirement
  2. Primary treatment. Young, not obese, >50% eliminate. (ie removal tonsils)
  3. Salvage, not able to tolerate full surgery, but something smaller to make improvement
    - Site specific to the anatomic abnormal. Often staged procedures
      - Nose: Turbinate, septoplasty.
      - Palate/velopharynx UPPP and modifications.
      - Uvula, stabilize palate, palatopharyngeus m. re-orientation, tonsillectomy.
      - Tongue: Tongue reduction, advancement genioglossus suspension, and hyoid suspension. Epiglottis arytenoids.
    - Global airway procedure: address multiple levels at once
      - Maxillomandibular advancement
    - Hypoglossal nerve stimulator: physiologic, activating hypoglossal nerve.
    - Tracheotomy: critical co-morbidities that make other treatments
    - Bariatric surgery. Class 2 obesity BMI >35-40
- Surgical outcomes:
- Goal for surgery 1) decrease symptoms (decrease Epworth sleep scale) 2) improve long term health. (Get sleep apnea into mild range)
  - Repeat sleep testing 4 months post op