



Medical and Surgical Management of Obstructive Sleep Apnea in the CPAP Intolerant Patient

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Disclosures

I have no conflicts of interest, financial or otherwise, relevant to this talk.

Learning Objectives

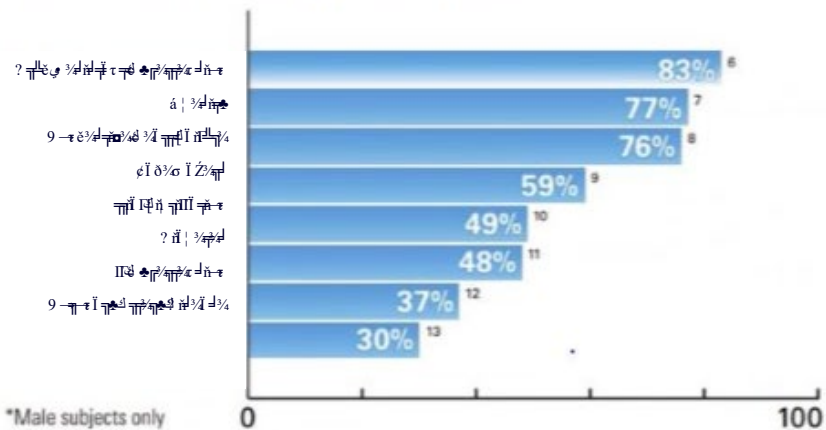
1. Provide an overview of OSA management .
2. Highlight Non-PAP OSA treatment strategies.
3. Discuss the approach to the CPAP intolerant patient.
4. Review the use of HGNS in the treatment of OSA

Prevalence of Sleep Apnea

Prevalence of Sleep Apnea

- Approximately 42M Adults have SDB
- An estimated 26% of adults have at least mild SDB
- 9% of Middle-aged women and 25% of middle-aged men suffer from OSA
- Prevalence is like asthma (20M) and diabetes (26M) of US population
- 75% of severe SDB cases remain undiagnosed

Prevalence of Sleep Apnea in Comorbidities



Obstructive Sleep Apnea

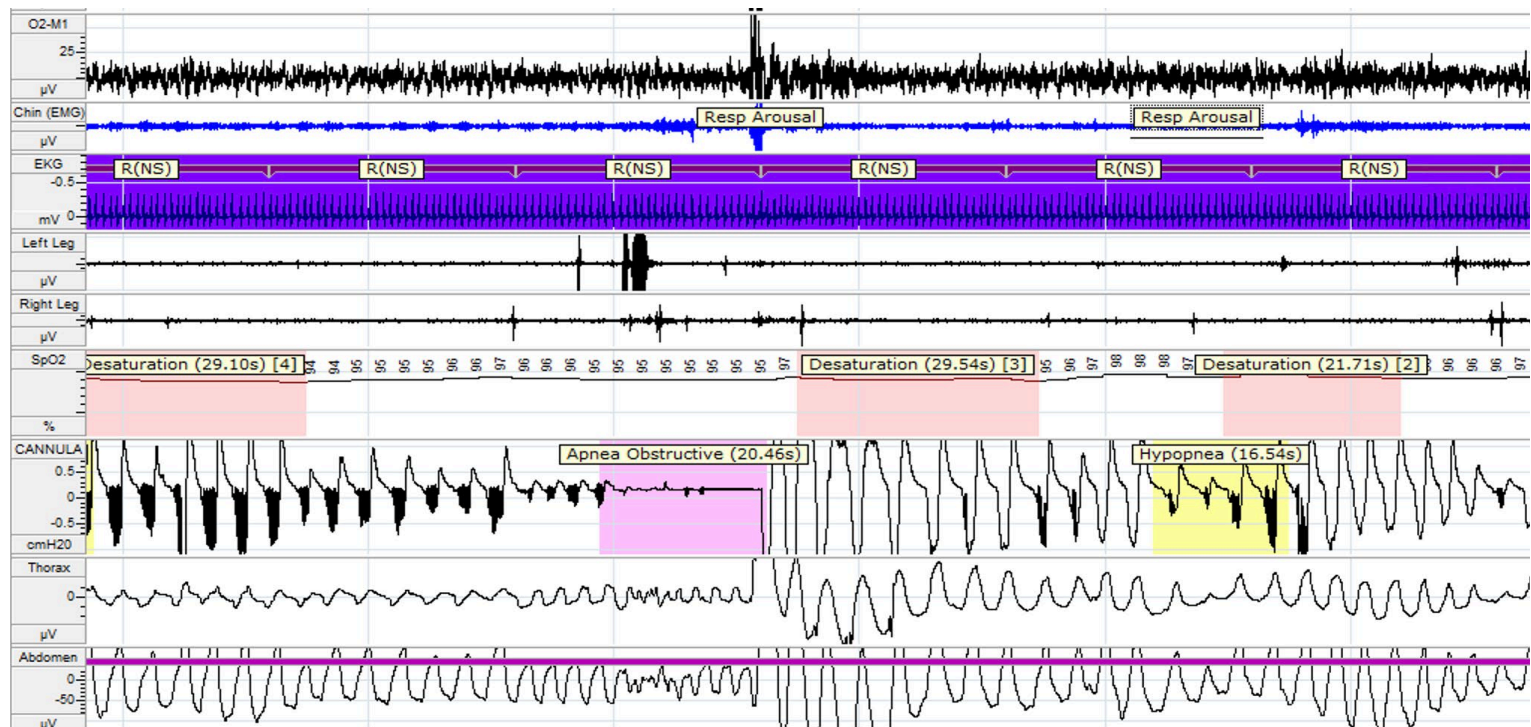
Repeated episodes of **upper airway collapse** during sleep that restrict/reduce airflow despite the effort to breathe.

- Associated with a reduction in blood oxygen saturation or an arousal from sleep
- Severity determined by the Apnea-Hypopnea Index (AHI)

Determination of AHI

Rate of apneas and hypopneas per hour

- **Obstructive Apnea:** >90% reduction in airflow for >10 seconds with associated respiratory effort



FLOW

EFFORT

Consequences of OSA

TABLE 1: SLEEP AND CARDIOVASCULAR DISEASE ASSOCIATIONS

Underlying condition	Elevates Risk for...
Obstructive sleep apnea	• Atherosclerosis • Atrial fibrillation • Congestive heart failure • Death • Metabolic syndrome • Myocardial infarction • Stroke • Systemic hypertension • Type 2 diabetes (a risk factor for cardiovascular disease)
Heart failure	• Paroxysmal nocturnal dyspnea from Cheyne-Stokes breathing
Sleep deprivation	• All-cause mortality • Myocardial infarction • Transient ischemic attacks and stroke • Weight gain
Snoring	• Early death after stroke • Poor prognosis after myocardial infarction

Adapted from references 3-20.

Consequences of OSA

OSA and Cardiac Arrhythmias; Atrial Fibrillation:

- Four times increased risk of AF in pts with OSA (AHI>30) (Sleep Heart Health Study 2006)
- Onset of >75% of persistent A fib episodes in pts with OSA occur at night (8pm-8am)
- A fib recurrence after cardioversion twice as high in untreated OSA
- Observational review over 17 yrs suggests that nocturnal hypoxemia influences the onset of A fib

Postgrad Med J 2008; 84:15-22
Proc Am Thorac Soc 2008; 5:200-206

Consequences of OSA

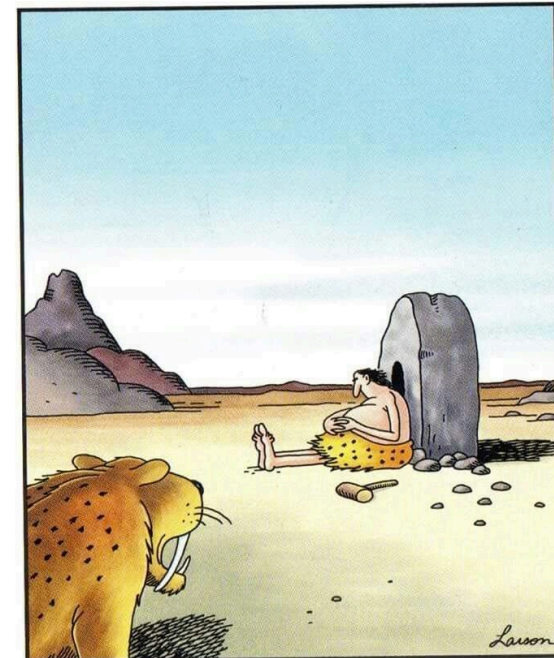
Excessive daytime sleepiness secondary to untreated OSA leads to increased accidents and lost workplace productivity totaling \$119.6 billion

Undiagnosed Sleep Apnea: *A Hidden Health Crisis*

In the U.S. the estimated economic cost of undiagnosed obstructive sleep apnea was nearly \$150 billion in 2015.



Source: American Academy of Sleep Medicine, 2016 | www.sleepeducation.org



Thag Anderson becomes the first fatality as a result of falling asleep at the wheel.

OSA in Surgical Patients

Direct influence on post-operative outcomes

- Increased rate of cardiac events
- Increased rate of respiratory events
- Increased ICU transfer rate
- Increased Infection Rate in OHNS Patients

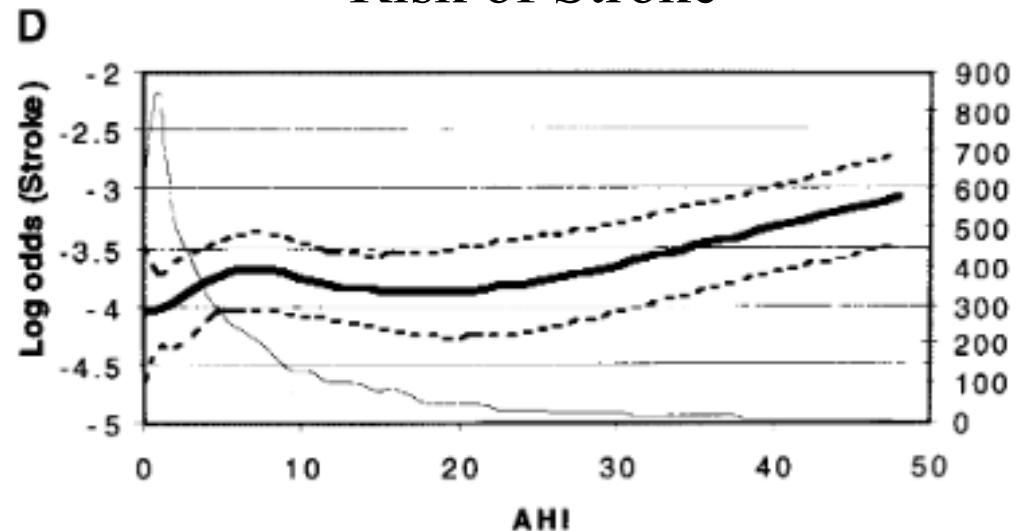
Assessing Risk in the OSA patient

Mild OSA: AHI 5-15/hr

Moderate OSA: AHI 15-30/hr

Severe OSA: AHI >30/hr

Risk of Stroke



Why do we treat OSA?

1. Risk Mitigation

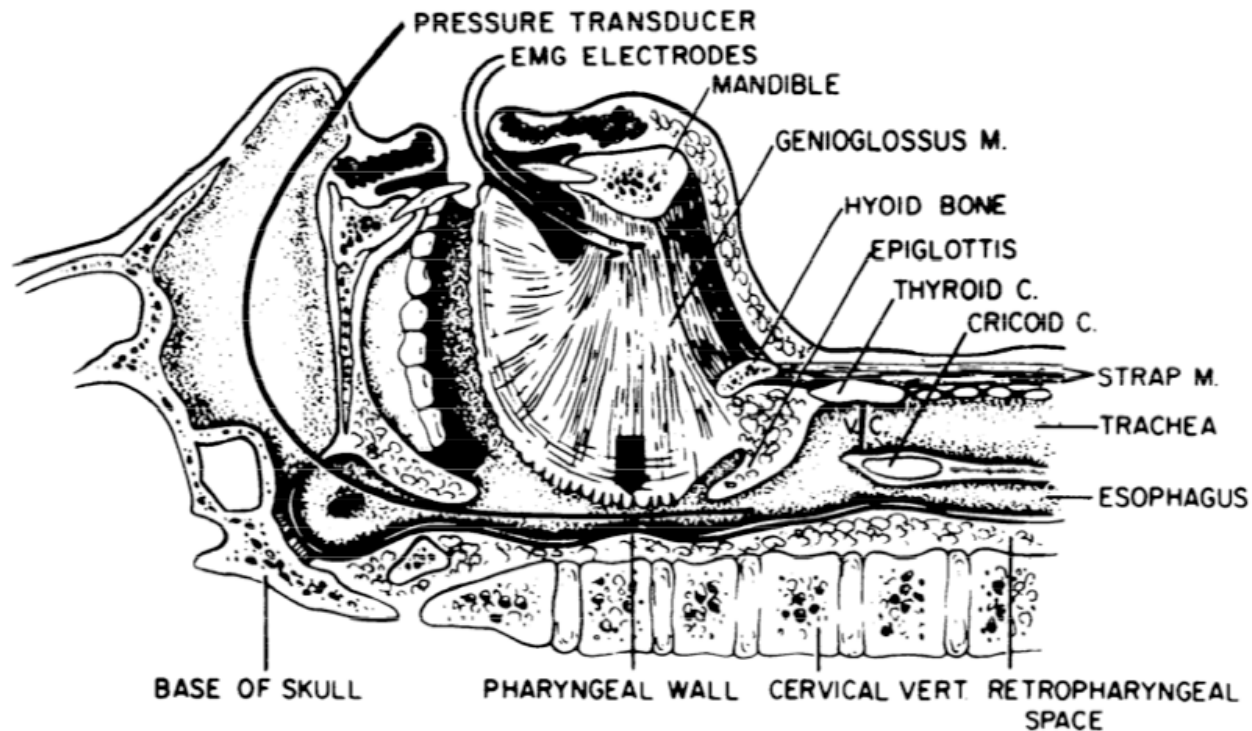
- Attempt to mitigate the known cardiometabolic sequelae.

2. Sleep Quality

- Attempt to improve sleep quality related to OSA.

Treatment of OSA

Goal: Preventing upper airway collapse to stabilize breathing during sleep.



CPAP is First Line Therapy for OSA

Continuous Positive Airway Pressure (CPAP)

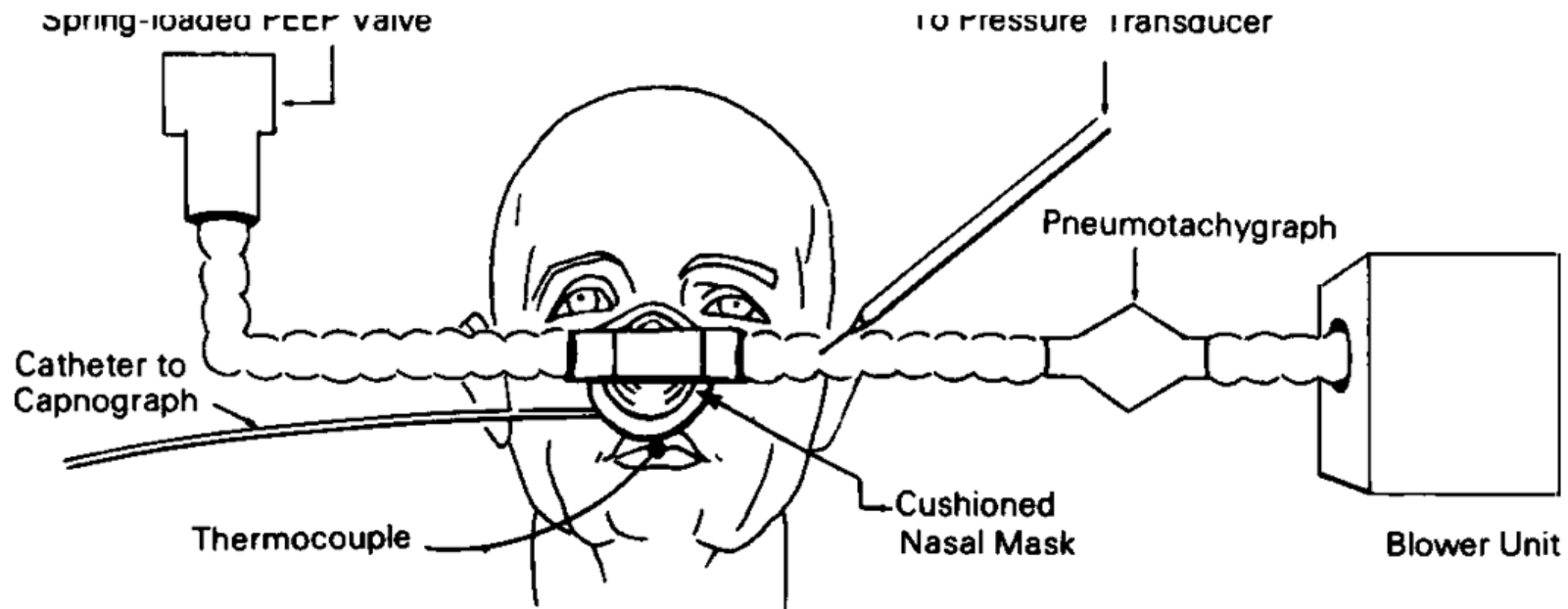
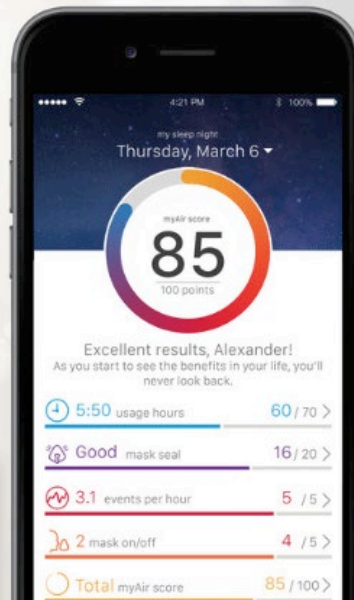


FIGURE 1. Diagram of the nasal CPAP apparatus employed.

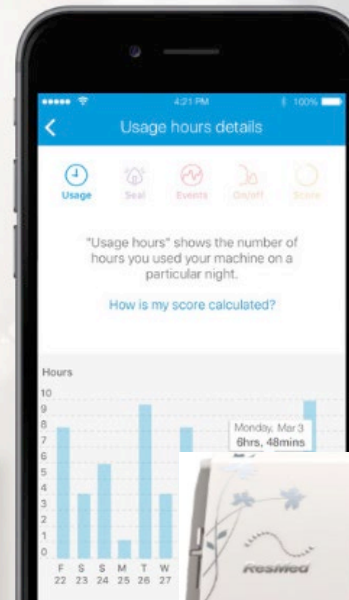
CPAP therapy has evolved



A daily sleep score shows how you did on treatment last night



Track your treatment quality and progress over time



CPAP Compliance is Poor

CPAP non-compliance rates between 35-50% 3 years after OSA diagnosis.

- Large population still subjected to OSA risks
- Effective non-PAP treatment strategies are needed.

Compliance:

>4hrs on 70% of nights

~35% of total sleep



Non-PAP alternatives for OSA

- Positional Therapy
- Oral Appliance Therapy
- Weight Loss
- UPPP surgery
- Hyoid Suspension
- Maxillary expansion
- Maxillomandibular advancement
- Advancement genioplasty
- Tongue base reduction
- Midline glossectomy
- Transpalatal advancement
- Hypoglossal Nerve Stimulation

Limited rates of success for non-PAP alternatives

Oral Appliance: 29-71%

Hyoid myotomy and suspension: 17-61%%

Uvulopalatalpharyngoplasty: 40-70.8%

Expansion pharyngoplasty: 82%

Midline glossectomy: 42-77%

Genioglossus advancement: 39-78%

Maxillomandibular advancement: 80-92%

Hypoglossal Nerve Stimulation (Inspire): 66-85%

Multi-level Surgery improves success rate



Pharyngoplasty + Tongue base Excision: 82.3%

Pharyngoplasty + Hypoid Myotomy and Suspension: 76.9%

Pharyngoplasty + Hypoglossal nerve stimulation: 100% (limited patient cohort)

***Sher Criteria: AHI <20 and 50% AHI reduction*

OSA Surgery Carries Risk

- Bleeding
- **Pain**
- Dysphagia
- Nasopharyngeal reflux
- Malocclusion
- Respiratory failure
- Death
- Treatment failure

Finding treatment solutions for the CPAP intolerant patient

1. Acknowledging the risk of untreated OSA.
2. Accepting risk related to treatments.
3. Understanding the risk of treatment failure.
4. Reassurance that we will find a solution(s) that works for them.



Providing tailored treatment for the CPAP intolerant patient

Characterization

Patient ~~selection~~ is critical:

- The correct patient for treatment.
- The correct treatment for that patient.

Determining the Correct Patient

1. Assessing OSA Severity and Risk

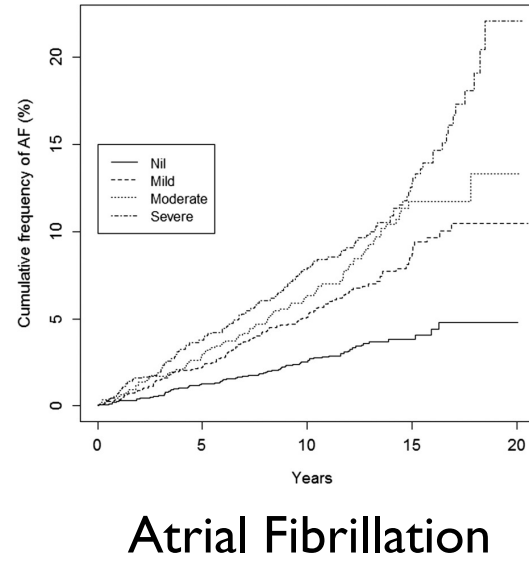
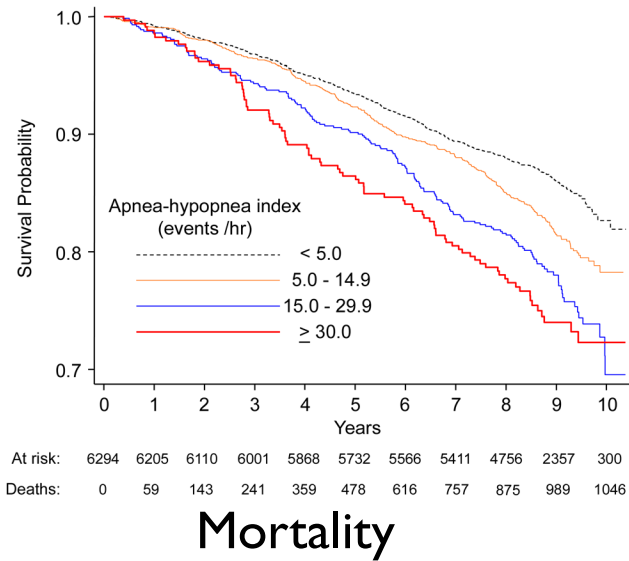
- Does it medically need treated?
 - What risk does it pose to the patient?
 - Age of patient
 - Medical Comorbidities
 - Patient reported symptoms
 - Daytime sleepiness

2. CPAP Averse or CPAP Intolerant

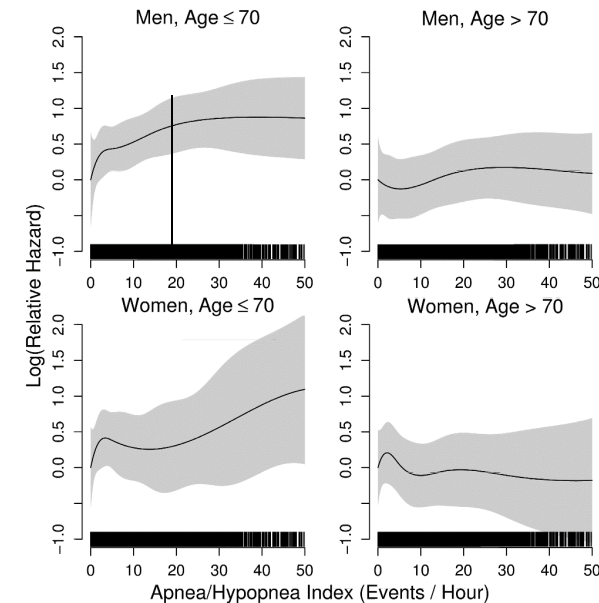
3. Define the Expected Outcome

What OSA requires treatment?

Risk by Severity



Risk by Age



High AHI (>20)

Younger Age

Presence of OSA associated comorbidities

Accurate characterization of OSA severity is critical

Multiple platforms for assessing AHI severity exist

- In-lab Polysomnography
- Home sleep testing
- WatchPAT testing

Disclaimer: Not all sleep testing is created equal

Table 2: CMS Classification of Sleep Tests

STUDY TYPE	PARAMETERS MONITORED
I	Minimum of 7 to include EEG, EOG, chin EMG, airflow, respiratory effort, oxygen saturations, and ECG. Attended by a sleep technician.
II	Minimum of 7 to include EEG, EOG, chin EMG, airflow, respiratory effort, oxygen saturations, and ECG. Unattended by a sleep technician.
III	Minimum of 4 channels to include ECG/HR, oxygen saturations, two channels of respiratory effort or one respiratory effort channel and one airflow channel. Attended or unattended by a sleep technician.
IV	Minimum of 3 channels, one of which is airflow or include actigraphy, oxygen saturations, and peripheral arterial tone. Attended or unattended by a sleep technician.

EMG = electromyogram; EOG = electrooculogram; HR = heart rate.
Source: CMS Manual System. Pub 100-03 Medicare national coverage determinations. Transmittal 86. Centers for Medicare & Medicaid Services website. <https://www.cms.gov/transmittals/downloads/R86NCD.pdf>. Accessed March 29, 2015.

Heterogeneity in sleep testing is prevalent



- Night to night variability
 - *Dependent on body position and sleep stage*
- Variability in scoring algorithms (CMS vs AASM)
 - *AHI differences >20 events/h in >10% of patients*
- Difference in testing platforms (In-lab PSG vs Home sleep testing)
 - *Home sleep testing does not monitor sleep/wake and underpredicts severity*

Determining the Correct Patient

1. Assessing OSA Severity and Risk

2. CPAP Averse or CPAP Intolerant?

1. **CPAP Intolerant:** Has tried and is consistently unable to wear CPAP

→ *Needs an alternative treatment to mitigate OSA risk*

2. **CPAP Averse:** Tolerates CPAP, but is looking for QOL improvement

→ *Consider alternative treatment if favorable outcome is likely*

3. Define the Expected Outcome

CPAP is First Line Treatment



Understanding the nature of the patients
CPAP non-compliance is critical.

- CPAP usage patterns
- Claustrophobia, PTSD, mask leak, insomnia
- Efforts should be made to help patients become compliant with CPAP before non-PAP alternatives are explored

Determining the Correct Patient

1. Assessing OSA Severity and Risk
2. CPAP Averse or CPAP Intolerant
- 3. Define the Expected Outcome**

What is the likelihood of reducing the patients OSA severity to a range that improves symptoms and does not impose cardiometabolic risk?

Who is going to respond to treatment?

Response rates to Oral Appliance therapy

	All	Success criteria			
		Criterion 1 AHI < 5	Criterion 2 AHI < 10	Criterion 3 AHI < 15	Criterion 4 AHI reduction rate ≥ 50%
All patients - n (%)	442	148 (33.5%)	293 (66.3%)	356 (80.5%)	280 (63.3%)
OSA severity					
Mild - n (%)	133	72 (54.1%)	112 (84.2%)	124 (93.2%)	75 (56.4%)
Moderate - n (%)	224	63 (28.1%)	145 (64.7%)	180 (80.4%)	143 (63.8%)
Severe - n (%)	85	13 (15.3%)	36 (42.4%)	52 (61.2%)	62 (72.9%)
Obesity severity					
Underweight - n (%)	17	9 (52.9%)	14 (82.4%)	16 (94.1%)	11 (64.7%)
Normal range - n (%)	291	104 (35.7%)	203 (69.8%)	245 (84.2%)	191 (65.6%)
Pre-obese - n (%)	108	31 (28.7%)	64 (59.3%)	81 (75.0%)	65 (60.2%)
Obese - n (%)	21	1 (4.8%)	7 (33.3%)	9 (42.9%)	9 (42.9%)
Age Group					
Young - n (%)	69	30 (43.5%)	53 (76.8%)	63 (91.3%)	49 (71.0%)
Middle - n (%)	268	96 (35.8%)	184 (68.7%)	223 (83.2%)	176 (65.7%)
Elderly - n (%)	105	22 (21.0%)	56 (53.3%)	70 (66.7%)	55 (52.4%)

AHI, apnoea and hypopnea index; mild, $5 \leq \text{AHI} < 15$; moderate, $15 \leq \text{AHI} < 30$; severe, $\text{AHI} \geq 30$. Underweight, $\text{BMI} < 18.5$; normal range, $18.5 \leq \text{BMI} < 25.0$; pre-obese, $25.0 \leq \text{BMI} < 30.0$; obese, $30 \leq \text{BMI}$. Young, $20 \leq \text{Age} < 40$; middle, $40 \leq \text{Age} < 65$; elderly, $65 \leq \text{Age}$.

Success with HGNS therapy

TABLE 3

Logistic regression univariate analysis of predictors of obstructive sleep apnoea treatment success at the final visit (median time 373 days)

	OR (95% CI)	p-value
Age (baseline) years	1.04 (1.01–1.08)	0.01
Sex (female <i>versus</i> male)	2.62 (0.88–7.78)	0.08
BMI (baseline) kg·m⁻²	0.91 (0.83–0.99)	0.03
AHI (baseline) events·h⁻¹	1.00 (0.98–1.03)	0.88
Therapy hours per week at post-titration visit	1.04 (0.99–1.67)	0.25
Therapy hours per week at final visit	1.02 (0.99–1.04)	0.22
Bilateral protrusion <i>versus</i> right protrusion	2.08 (0.96–4.51)	0.37
Other <i>versus</i> right protrusion	2.03 (0.50–8.21)	0.61

4% increase per year

9% change per BMI point

What factors influence the success of Non-PAP therapies

1. OSA Severity
2. Body Mass Index
3. Age

Case 1

39 y/o M with HTN and severe OSA (AHI4: 59, O2 nadir: 74, Time <88% SpO2: 92.3 minutes) presents for HGNS consult as due to failing CPAP.

- BMI 33, Neck Circumference 18 in, MMP 4, with 1+ tonsils and tongue ridging.
- Gained 25 pounds over the last 2 years
- PMHx: HTN poorly controlled on HCTZ

Management

~~Drug-induced sleep endoscopy~~ **NO!**

1. Determine the nature and reason the patient failed PAP therapy.

- Type of mask
- Type of CPAP
- Insomnia
- Anatomic factors (nasal obstruction)

CPAP Optimization

- Trial of nasal masks
- PAP/BIPAP titration
- Management of comorbid sleep disordered (RLS/Insomnia/CRD)
- Treatment of nasal obstruction

CASE 1

The patient reports trying only a full facemask, endorses frank left sided nasal obstruction secondary to prior lacrosse injury, and has a hard time falling asleep due to work and family stress.

- Obtain nasal mask
- Start Flonase and azelastine
- Manage insomnia

CASE 1

He returns with mild improvement in CPAP utilization but struggles to tolerate the mask due to nasal obstruction.

- Due to daytime nasal obstruction, as well as difficulty with CPAP, he elects to undergo septoplasty and turbinate reduction.

Nasal Surgery and OSA

Nasal surgery can improve CPAP compliance, daytime sleepiness, snoring intensity but does not change OSA severity

Table 2. Extracted Data from Studies.

Authors and Year	Apnea Hypopnea Index		Epworth Sleepiness Scale		Respiratory Disturbance Index		Number of Subjects
	AHI Pre	AHI Post	ESS Pre	ESS Post	RDI Pre	RDI Post	
Powell et al, 2001 ^{16,a}			10.5 (4.8)	8.6 (1.8)			17
Verse et al, 2002 ¹⁷	31.6 (25.6)	28.9 (24.7)	11.9 (4.7)	7.7 (5.0)			26
Kim et al, 2004 ¹³					39.0 (14.3)	29.1 (14.4)	21
Virkkula et al, 2006 ¹⁸	13.6 (15.8)	14.9 (19.3)					40
Nakata et al, 2008 ¹⁵	44.6 (22.5)	42.5 (22.0)	10.6 (4.1)	4.5 (2.6)			49
Koutsourelakis et al., 2008 ^{14,a}	31.5 (16.7)	31.5 (18.2)	13.4 (2.9)	11.7 (3.4)			27
Bican et al, 2010 ¹⁰	43.1 (27.1)	24.6 (22.2)					20
Park et al, 2014 ²⁰	23.9 (14.9)	12.2 (6.4)	9.7 (4.7)	5.8 (2.1)	28.8 (14.4)	17.1 (7.5)	25

Abbreviations: AHI, Apnea Hypopnea Index; ESS, Epworth Sleepiness Scale; RDI, Respiratory Disturbance Index.

^aRandomized controlled trials; only treatment arms presented.

Case 1

8 weeks s/p septoplasty and turbinate reduction – using CPAP nightly for 7 hours

OR

Breathing well through his nose, but still cannot initiate and maintain sleep with CPAP

Moving past CPAP

~~**SURGERY!!!!**~~ STILL NO

Exhaust all non-surgical options first

- Oral appliance therapy
- Positional therapy
- Weight loss
 - Pharmacological, Surgical, Behavioral

Weight loss, GLP1, and OSA

Table 3

Baseline and post intervention values for primary outcomes among completers

Variable	Baseline	Post Intervention	p-value
Body composition			
Weight, kg	97.5 (82.0 to 114.2)	88.4 (78.4 to 105.3)	<0.01
BMI, kg/m ²	33.8 (32.6 to 36.9)	31.6 (30.3 to 34.7)	<0.01
Waist Circumference, cm	108 (101 to 114)	102 (97 to 113)	<0.01
Total Body Fat, %	42.2 (38.3 to 47.6)	39.9 (34.4 to 46.9)	<0.01
Trunk Fat, %	46.6 (41.9 to 49.4)	43.0 (37.9 to 48.2)	<0.01
Aerobic capacity			
VO ₂ max ml/kg/min	22.3 (18.8 to 25.4)	26.8 (23.1 to 28.6)	<0.01
* VO ₂ max L/min	1.91 (1.79 to 2.80)	2.17 (2.05 to 2.97)	<0.01
Vascular parameters			
SBP, mmHg	129 (114 to 147)	123 (112 to 131)	0.20
DBP, mmHg	69 (65 to 76)	67 (65 to 72)	0.50
AI, AU	36 (22 to 44)	26 (10 to 49)	0.84
Sleep parameters			
Total Sleep Time, min	389 (323 to 401)	416 (395 to 444)	<0.01
Mean SaO ₂ , %	94.9 (94 to 95.4)	95.2 (94.4 to 95.7)	0.02
Average Low SaO ₂ , %	89.9 (88.5 to 91.5)	91.0 (89.4 to 92)	0.02
AHI, events/hour	22 (14 to 44)	12 (5 to 26)	0.03

Modest changes in BMI can influence the AHI dramatically.

SURMOUNT-OSA: study design

Two randomized, placebo-controlled phase 3 trials under one master protocol (NCT05412004)

469

adults with obesity and moderate-to-severe OSA, randomized

52 wk

weekly subcutaneous tirzepatide, 10 or 15 mg, vs placebo

51.5

mean baseline AHI (events/h); mean BMI 39.1 kg/m²

Trial 1: not on PAP

n = 234 (114 tirzepatide, 120 placebo)

Primary endpoint: change in AHI from baseline at week 52

Trial 2: on PAP

n = 235 (120 tirzepatide, 115 placebo)

PAP use for at least 3 consecutive months before screening

Efficacy at week 52

Tirzepatide vs placebo; AHI fell by 20 to 24 events/h more with tirzepatide in both trials

Endpoint	Trial 1 · no PAP	Trial 2 · on PAP
Mean AHI change (events/h)	-25.3 vs -5.3	-29.3 vs -5.5
Treatment difference (95% CI)	-20.0 (-25.8 to -14.2)	-23.8 (-29.6 to -17.9)
AHI change from baseline	-50.7% vs -3.0%	-58.7% vs -2.5%
AHI reduction of 50% or more	61.2% vs 19.0%	72.4% vs 23.3%
Disease resolution*	42.2% vs 15.9%	50.2% vs 14.3%
Body weight change	-17.7% vs -1.6%	-19.6% vs -2.3%

*AHI below 5, or 5 to 14 with Epworth Sleepiness Scale score of 10 or less. Systolic BP also fell more with tirzepatide (-9.5 and -7.6 mm Hg vs -1.8 and -3.9 mm Hg) alongside hypoxic burden and hsCRP.

Source: Malhotra A et al. N Engl J Med 2024;391:1193-1205

Safety and clinical takeaways

Safety

Most common adverse events, % (Trial 1 / Trial 2); mostly mild to moderate

Event	Tirzepatide	Placebo
Diarrhea	26.3 / 21.8	12.5 / 8.8
Nausea	25.4 / 21.8	10.0 / 5.3
Vomiting	17.5 / 9.2	4.2 / 0.9
Constipation	15.8 / 15.1	2.5 / 4.4

Stopped for adverse events: 4.4% / 3.4% on tirzepatide vs 1.7% / 7.0% on placebo

Two confirmed acute pancreatitis cases, both Trial 2 tirzepatide arm

Takeaways

- FDA approved in December 2024: first drug for moderate-to-severe OSA in adults with obesity
- AHI fell with and without background PAP, alongside 18 to 20% weight loss
- Disease resolution in 42 to 50%, so many patients still have residual OSA
- AHI is a surrogate; links to long-term clinical outcomes remain unclear

Case 1

Achieved a 30-pound weight loss over 7 months.

BMI now 29.8

Titrated to 7.5mg – did not tolerate higher doses.

Repeat HST demonstrated an improvement in AHI4 to 29.8

Case 1:

Patient is counseled on the need for additional OSA treatment and is referred for oral appliance therapy.

Background: oral appliance therapy

Mandibular advancement devices are the main oral appliance used for OSA

Mandibular advancement

Advancing the mandible pulls the tongue and soft palate forward and enlarges the pharyngeal airway, mostly laterally, which lowers upper airway collapsibility.

Tongue-retaining

Hold the tongue forward by suction. Rarely used; discomfort drives higher discontinuation.

Palatal lift

Elevate the soft palate. Virtually obsolete for OSA because of poor long-term tolerance.

Mechanism

Advancing the mandible pulls the tongue and soft palate forward and enlarges the pharyngeal airway, mostly laterally, which lowers upper airway collapsibility.

Care pathway

1. Initial assessment and diagnosis of OSA. 2. Patient education and counseling. 3. Selection of appropriate oral appliance. 4. Fabrication and fitting of the appliance. 5. Follow-up and titration. 6. Long-term monitoring and maintenance.

Sources: AASM/AADSM guideline 2015; Sleep Med Res 2023 review; ADA evidence brief

Efficacy: effect on AHI

CPAP lowers AHI more, but sham-controlled trials show a real oral appliance effect

Mean AHI on an active device (events/h)

Marklund 2015



Durán-Cantolla 2015



Response is variable; no factor consistently predicts success

46.2%

reached a 50% or greater AHI reduction with an active device, vs 18.4% with sham

6.24

events/h lower AHI with CPAP than with an oral appliance (95% CI 4.34 to 8.14)

Sources: AASM/AADSM guideline 2015; ADA evidence brief (Marklund 2015; Durán-Cantolla 2015)

Indications and contraindications

Indications

- Adults with primary snoring who request treatment
- OSA with CPAP intolerance, or a preference for an alternative
- Often cited as favorable: mild to moderate OSA, younger age, lower BMI; none reliably predicts success
- Severe OSA: AASM2015 offers CPAP first, while AADSM2025 standards list mild, moderate and severe OSA

Contraindications

- Too few teeth, severe decay, loose teeth or active periodontitis
- Temporomandibular joint disease
- Inadequate mandibular protrusion or severe micrognathia
- Relative: nasal obstruction, large tonsils, long palate or uvula
- Follow-up: every 6 months in year 1, then yearly, watching for bite change

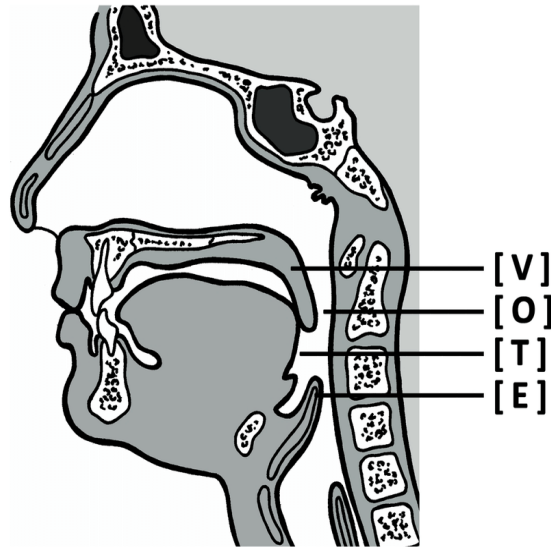
Case 1:

Patient is successful treated with a custom fabricated MAD

However, he experiences severe TMJ pain 3 months into therapy and discontinues therapy.

He wishes to pursue surgical therapy and is recommended DISE

Drug-Induced Sleep Endoscopy (DISE)



First described in 1991

Obstructive Sleep Apnea Evaluation

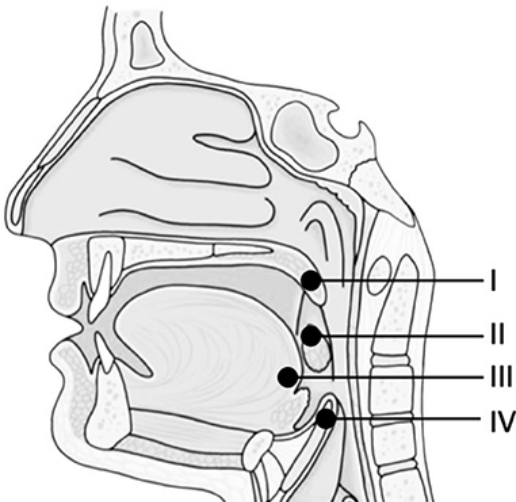
- PSG: AHI
- DISE

DISE Limitations

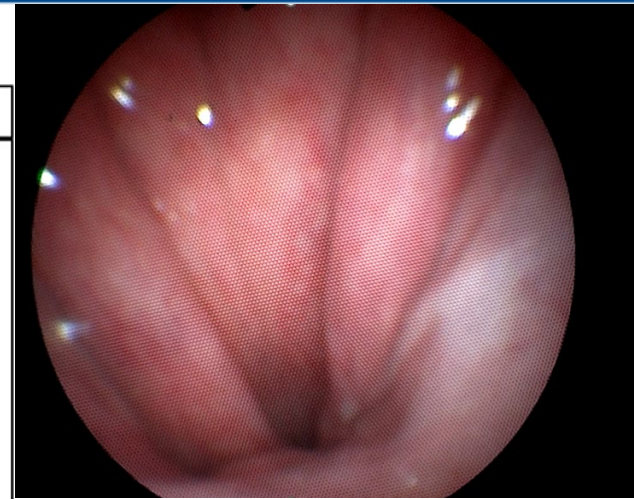
- Subjective
- Visual impression of site of collapse
- Limited predictive capacity

Physiologic measures of airflow reduction, the gold standard for diagnosis of OSA, are not included

Drug-Induce Sleep Endoscopy



VOTE classification system			
Level	Degree	Direction	
I: Velum	None	Antero-posterior	
II: Oropharynx	Partial	Lateral	
III: Tongue base	Complete	Concentric	
IV: Epiglottis			
HMS classification system			
Level	Degree	Direction	Tongue position
I: Isolated palate	None	Antero-posterior	Vallecula entirely visible
II: Lateral walls	Partial	Lateral	Posteriorly located tongue:
III: Tongue-related	Complete	Concentric	- Vallecula obscured
IV: Epiglottis		Transversal	- Vallecula and glottis obscured



What DISE predicts, by treatment

Treatment	Finding that supports it	Finding that predicts failure
Palate surgery	Anteroposterior velum collapse	Complete concentric velum, lateral wall or tongue base collapse
Hypoglossal nerve stimulation	Complete tongue base obstruction; palatal opening pressure under 8 cmH2O	Palatal complete concentric collapse (excluded in STAR); lateral wall collapse
Tongue base surgery	Tongue base collapse, which is often missed on awake exam	Oropharyngeal lateral wall collapse
Epiglottis procedures	Epiglottic collapse, seen in 15 to 74% of OSA patients	No guideline defines clinically significant collapse
Oral appliance	Tongue base collapse; airway opens with Esmarch maneuver	Complete concentric collapse

Hypoglossal nerve stimulation outside the STAR criteria: patients with complete concentric collapse did not respond (non-responder AHI 39.4 to 45.2)

Sources: Curr Otorhinolaryngol Rep 2025; Univ. of Iowa DISE protocol; J Laryngol Otol HGNS review; Curr Otorhinolaryngol Rep 2019

Lateral wall collapse and HGNS

Complete lateral wall collapse lowers HGNS response but is not a formal contraindication

OR 0.43

pooled odds of HGNS success, complete vs non-complete collapse (95% CI 0.28 to 0.64)

58–67%

HGNS response with complete collapse, vs 74–85% without (4 studies)

4–41%

graded complete collapse across cohorts; inter-rater kappa was 0.48 in one study

Not a formal contraindication

- Largest study (n = 369): no longer significant after adjustment (OR 0.57, 95% CI 0.26 to 1.22)
- Partial collapse shows no consistent effect; evidence certainty is low

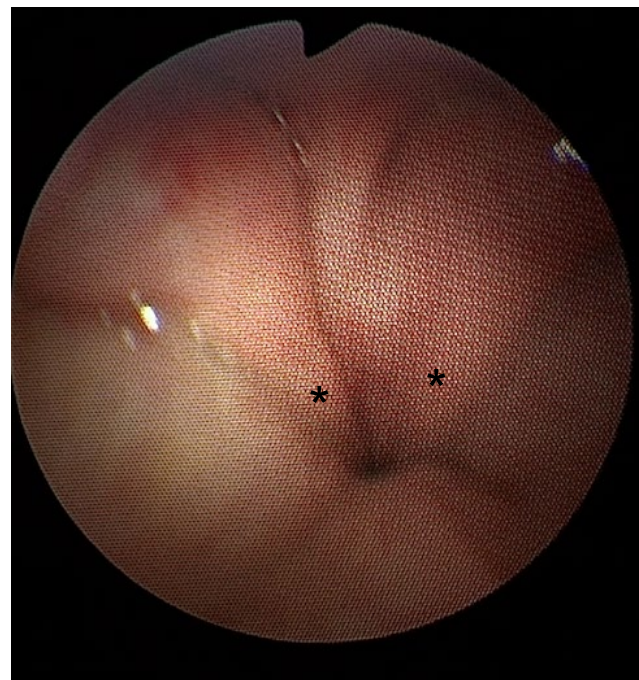
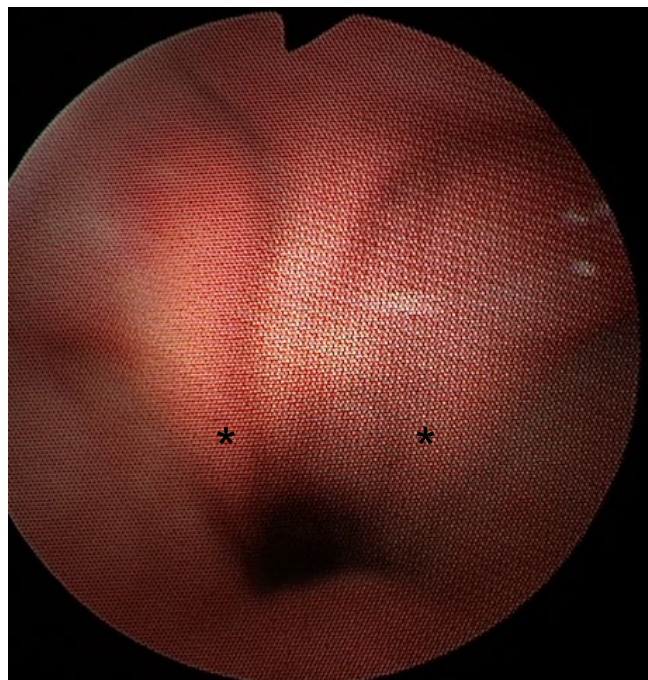
If present: consider tonsillectomy

- Small tonsils (n = 97): adding tonsillectomy raised response odds 8.6-fold (95% CI 1.7 to 43.4)
- Response reached 79%, vs 86% in patients without lateral wall collapse

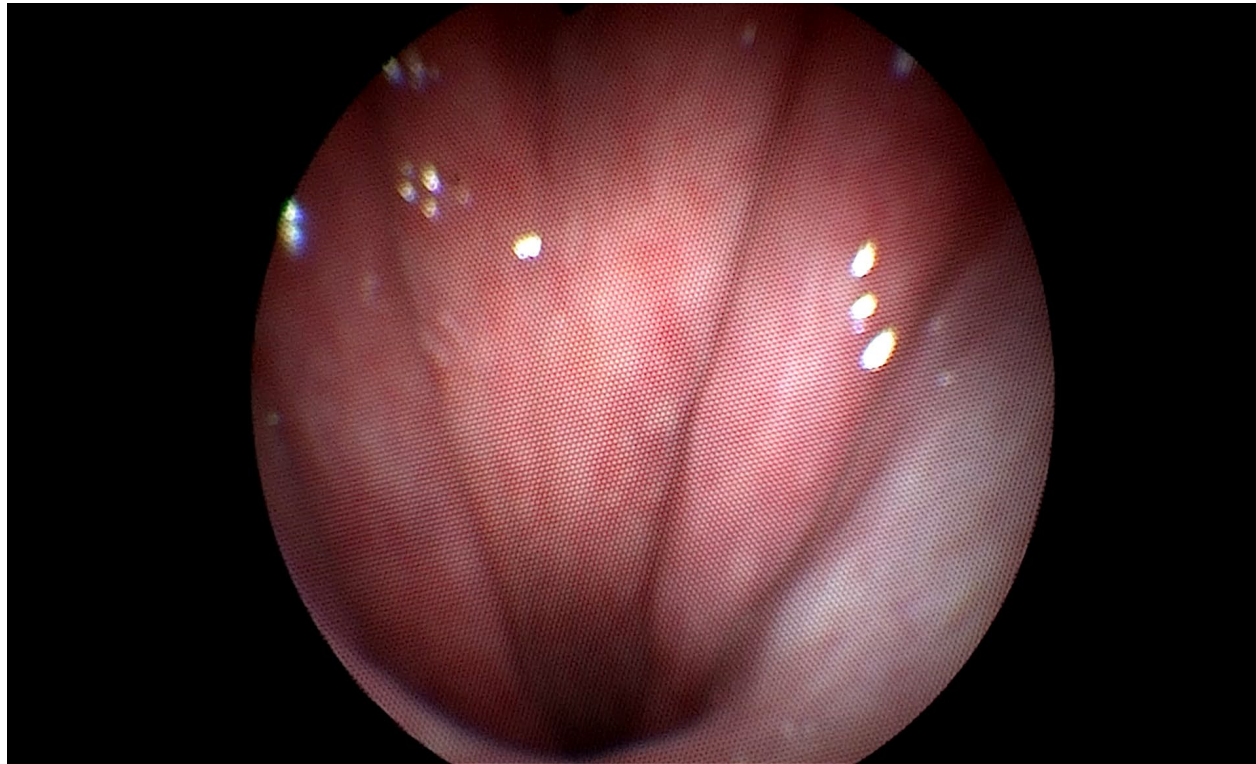
Sources: Diagnostics 2026 systematic review (8 studies, 1,051 patients); Patel et al., Ear Nose Throat J 2026 meta-analysis

Assessing the soft palate, oropharynx, and velopharyngeal airway

Assessing the lateral pharyngeal walls



Assessing the Velopharynx during DISE



Case 1:

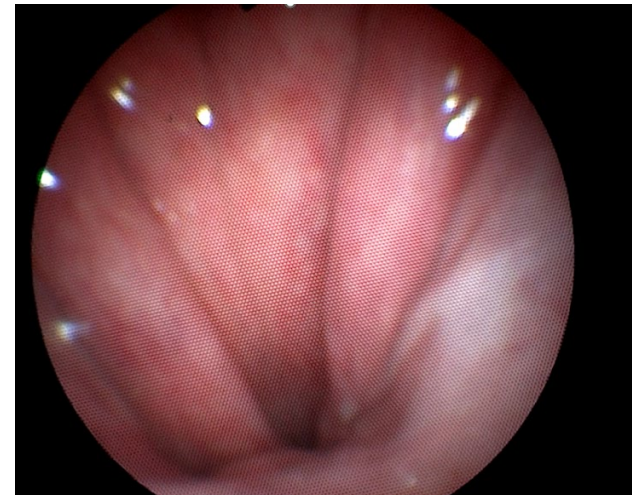
Patient undergoes DISE exam with Propofol

V:1 (AP with 50% lateral wall collapse)

O:2L

T:2

E:0



Limits of DISE prediction

Does DISE raise success?

- Recent meta-analyses: no difference in surgical success, DISE vs non-DISE
- Randomized trial (n = 63, severe OSA, BMI 35 or less): no difference, p = 0.69
- One study cited in a DISE guide: 84% vs 52% success with DISE guidance
- Most dependable use: flagging poor candidates, such as concentric collapse for HGNS

Why prediction is imperfect

- Whether sedated collapse matches natural sleep is still being studied
- No agreed interpretation protocol; 2 of 8 comparative studies used VOTE
- Only 1 of those 8 repeated DISE after surgery
- Oral appliance studies are small, retrospective and skew to milder OSA

DISE guides which site to treat and which patients to avoid; it does not reliably predict surgical success

Sources: Curr Otorhinolaryngol Rep 2025; randomized trial (n = 63); Sleep Med Res DISE guide; Medicina 2023 review; Univ. of Iowa DISE protocol

Case 1:

Patient is counseled he is a candidate for HGNS but does have evidence of lateral wall collapse and may want to consider a lateral wall stabilizing procedure prior to HGNS.

Advised given tongue base collapse on DISE, he may still need HGNS therapy to treat tongue base obstruction.

UPPP and expansion sphincter pharyngoplasty

Both treat retropalatal obstruction; they differ in how they handle the lateral wall

UPPP: removes tissue

- Tonsillectomy with trimming of the uvula and distal soft palate
- Closure of the tonsillar pillars
- Removes redundant tissue; does not directly tension the lateral wall

ESP: repositions muscle

- Bilateral tonsillectomy; inferior palatopharyngeus transected horizontally
- Muscle rotated superolaterally, sutured to the palate, still attached to the constrictor
- Partial uvulectomy and pillar closure
- Tensions the lateral wall instead of only removing bulk

2007 ESP trial profile: BMI under 30, Friedman II or III, small tonsils, lateral wall collapse

Outcomes: UPPP vs ESP

Randomized trial (Pang 2007): 22 UPPP vs 23 ESP, mean follow-up 6.5 months

Outcome	UPPP	ESP
Mean AHI, baseline to follow-up	38.1 to 19.6	44.2 to 12.0
Success: AHI down 50% and below 20	68.1%	82.6%
Success: AHI down 50% and below 15	45.5%	78.2%
Lowest SaO2, baseline to follow-up	75.1% to 86.6%	78.4% to 85.2%

17.2 vs 19.8

mean AHI fall, UPPP vs lateral or expansion pharyngoplasty (59 papers, 2,715 adults)

65–90%

ESP success across comparative studies, vs 39–59% for UPPP (8 studies, 614 patients)

Caveats

Small trials, short follow-up, mixed success definitions; ESP tied to more early pain and bleeding

Sources: Pang 2007; palate surgery meta-analysis, Eur Arch Otorhinolaryngol 2018; Medicina 2023 systematic review

Why use them: DISE-guided selection

Anteroposterior velum collapse

The usual match for UPPP-type soft palate surgery.

Lateral wall or complete concentric collapse

UPPP tends to do worse. PAP is preferred; if declined, ESP and other lateral pharyngoplasties add lateral wall tension.

Tongue base or hypopharyngeal collapse

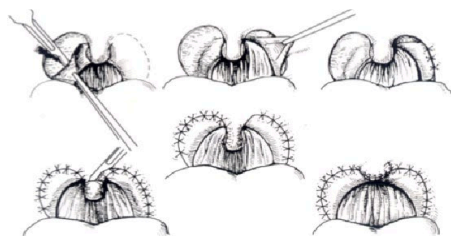
Linked to lower odds of response, so palate surgery alone may fall short. DISE mainly changes the plan at the tongue base.

Evidence is mixed

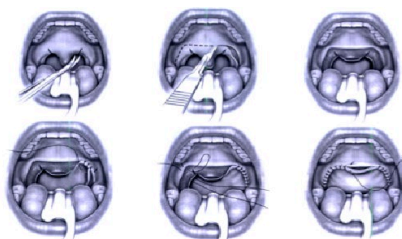
One study reported 84% vs 52% surgical success with vs without DISE guidance, yet a systematic review found DISE changed plans in about 50% of cases with little gain in success. Only 2 of 8 comparative velopharyngeal studies used VOTE.

Sources: Sleep Med Res DISE guide; Curr Otorhinolaryngol Rep 2019; Medicina 2023 systematic review

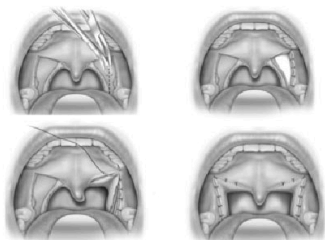
Surgically addressing the velopharynx in OSA



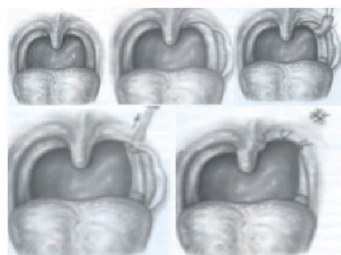
FUJITA TECHNIQUE



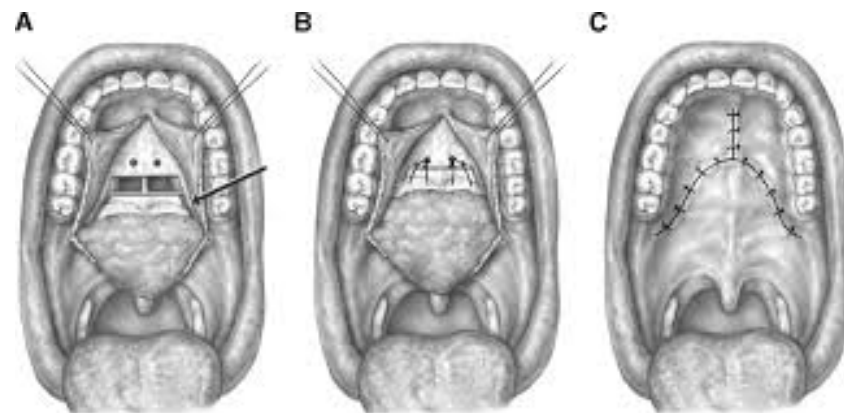
FAIRBANKS TECHNIQUE



**LATERAL PHARYNGOPLASTY
TECHNIQUE**



**EXPANSIVE PHARYNGOPLASTY
TECHNIQUE**



Case 1:

Does well with ESP surgery, snoring is improved and at 3 months post surgery his AHI4 is reduced to 17.8 events/hr.

He is counseled on additional weight loss or HGNS therapy. He elects to proceed with HGNS.

Hypoglossal nerve stimulation: overview

An implanted neurostimulation therapy for OSA in patients who do not tolerate CPAP

2014

FDA approval of Inspire; Genio (bilateral)
approved August 2025

66%

STAR responders at 12 months (AHI
down at least 50% and below 20)

29.3 → 9.0

median AHI at 12 months; Epworth score
11 to 6

How it works

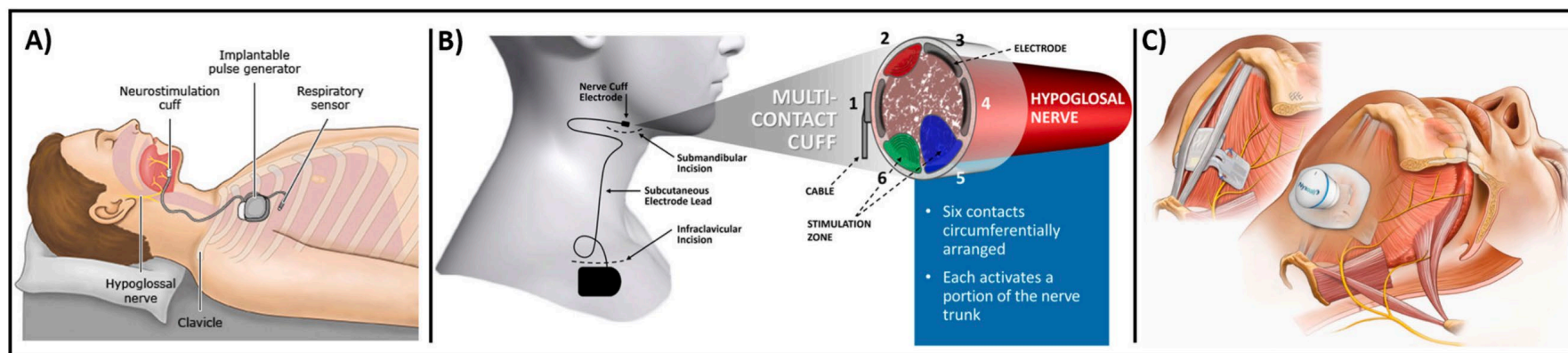
- Stimulates hypoglossal nerve branches, causing tongue protrusion and airway opening
- Inspire: unilateral, synchronized with breathing; Genio: bilateral, asynchronous

Inspire indications

- Age 18 or older (13 or older with Down syndrome)
- AHI 15 to 100 and BMI 40 or less; Genio: AHI 15 to 65

Sources: AASM overview of FDA-approved HGNS devices; AASM news, Genio FDA approval (Aug 2025)

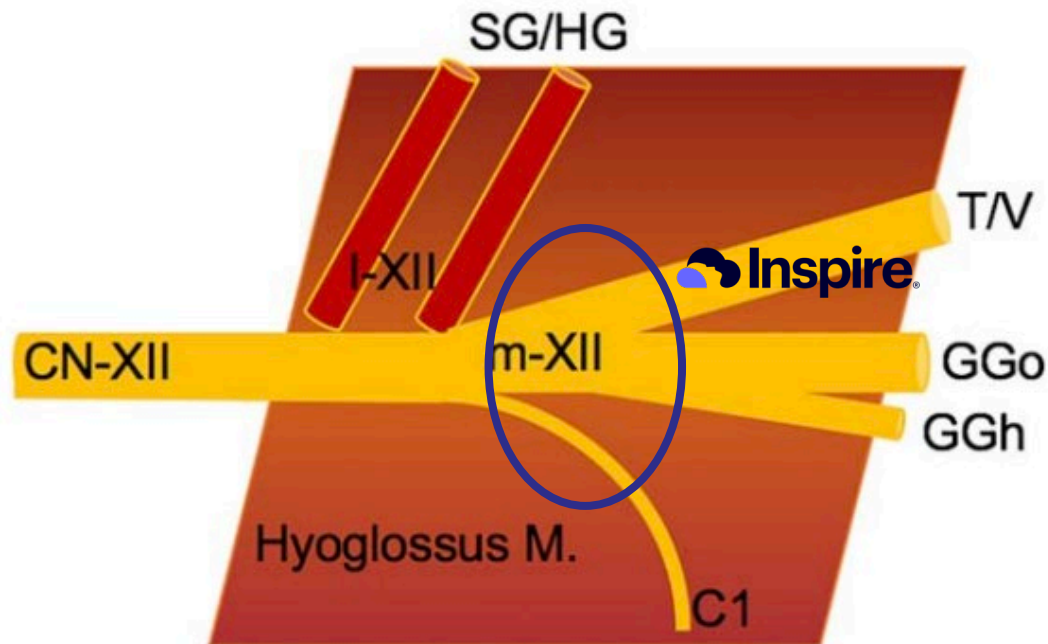
Devices for Hypoglossal Stimulation



Hypoglossal Stimulation Devices

Indication	Inspire	Nyxoah Genio	LivaNova aura6000
AHI (<25% CAI/MAI)	15-100/hour	15-65/hour	20-65/hour; AI < 30; <15/hr with SpO2 decrease >10%
BMI	<40 kg/m2	<32 kg/m2	<35 kg/m2
DISE	No CCC	Under investigation for CCC	Not required
Implant	2 incisions, 10 year battery	1 incision, external battery	2 incisions, 10-15 year battery
MRI	Up to 1.5T	Up to 3.0T	Not compatible

Hypoglossal Nerve Anatomy

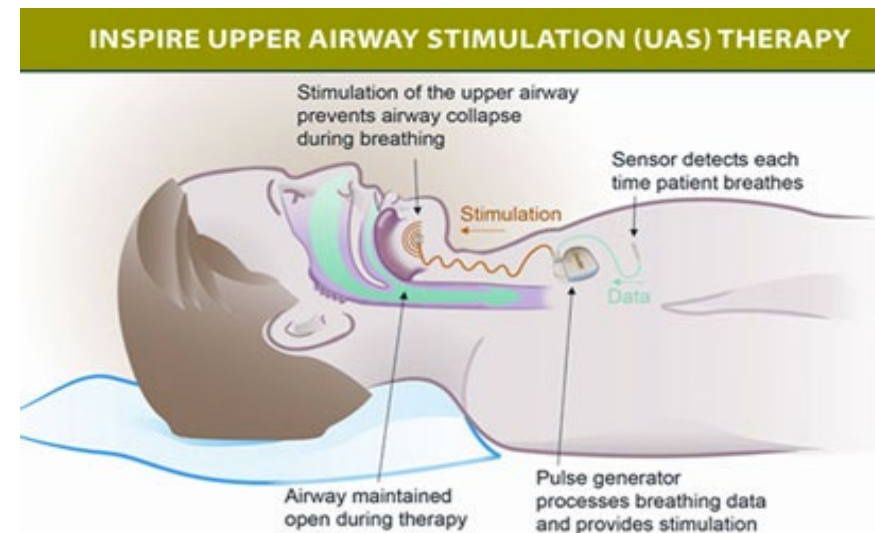


FDA Indications

	Inspire (Adult)	Inspire (Pediatric)
Age	18+	13+ (Down syndrome)
BMI	<40	<40
AHI	$15 \leq \text{AHI} \leq 100$	$10 \leq \text{AHI} \leq 50$
CAI/MAI	<25%	<25%
Treatment	PAP intolerance	PAP intolerance; after T&A
DISE	No circumferential palatal collapse	No circumferential palatal collapse

Inspire Therapy

- Unilateral HGNS with IPG and Respiratory lead
- **First FDA approved device in USA**



Retractors



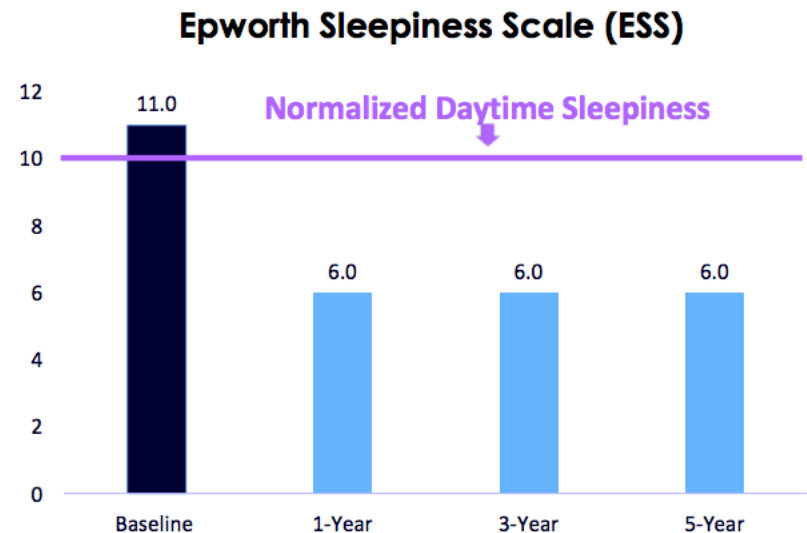
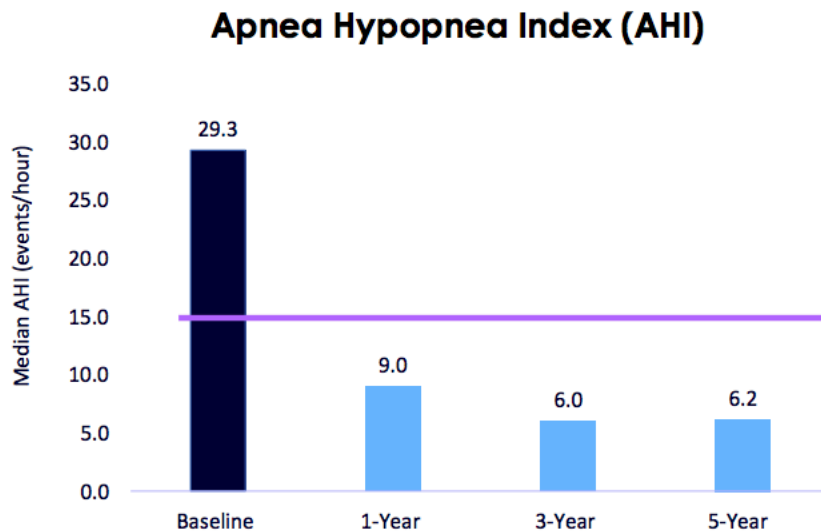
Protrusors

Inspire HGNS: Background

- **STAR Trial (2010-2013)** --*Strollo et al. 2014 NEJM*
 - 126 patients with OSA
 - BMI <32, AHI: 20-50 (<25% CA and non positional)
 - No complete concentric collapse on DISE

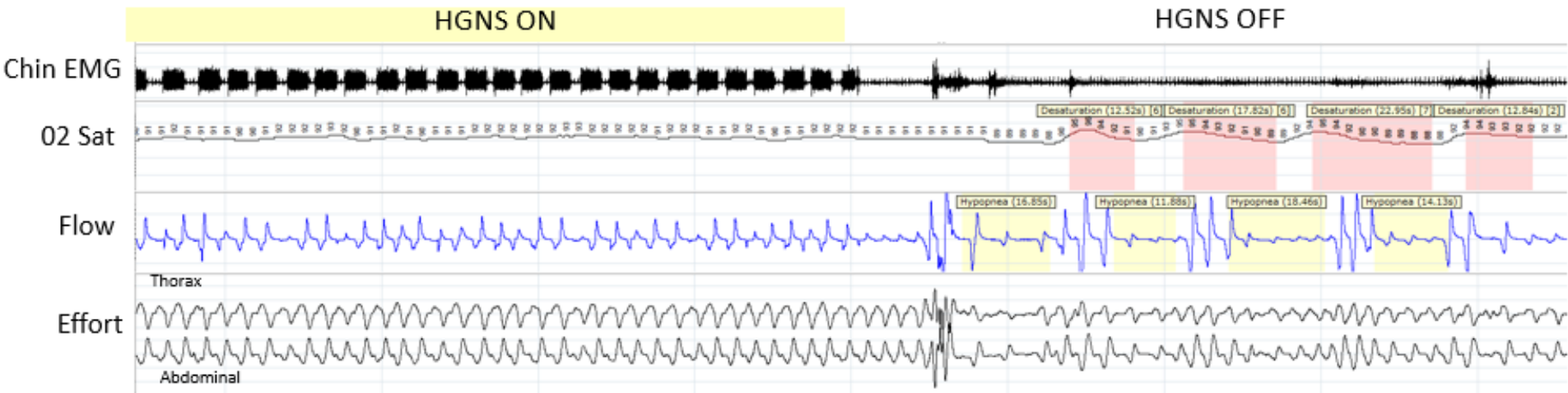
Inspire HGNS: Background

STAR Trial Overview: 5-Year Follow-Up Complete

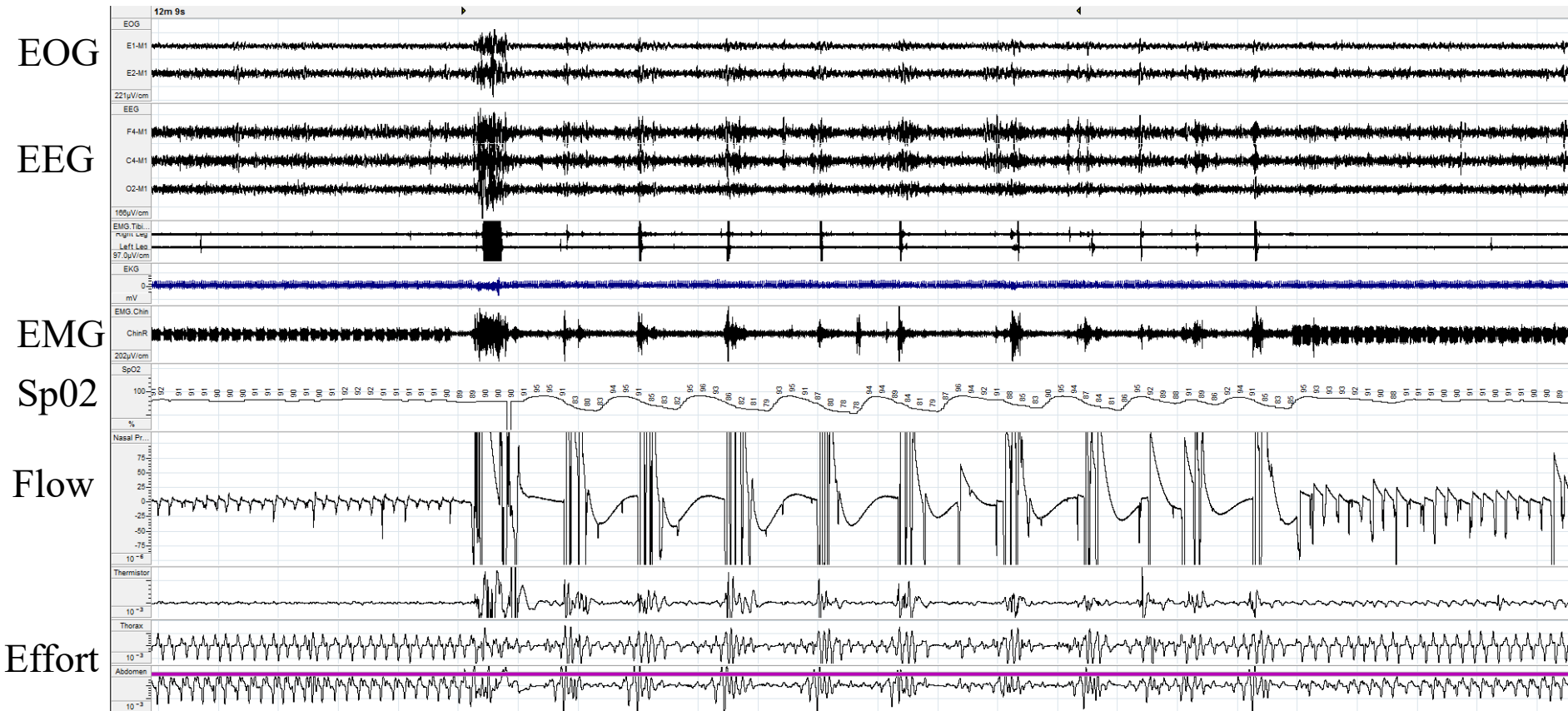


Multi-Center Prospective, 126 Patients, Randomized Control Tx, Withdrawal ARM

Effect of Inspire Therapy on Reversing Obstructive Events



Effect of Inspire Therapy on Reversing Obstructive Events



Case 1: Key Take Aways

Well that was a lot.....

1. No therapy is perfect (accept it)
2. CPAP is still the gold standard
3. Try to exhaust non-surgical therapies
4. Acknowledge the need for multi-modal treatment/surgery

How do we get better?

Development a systematic approach for characterizing upper airway collapse during sleep that allows for the delivery of tailored treatment and prognosticates outcomes in OSA surgery.

What does this even look like????

Fundamentals of OSA Treatment

Successful OSA surgery is predicated on overcoming upper airway collapse to restore airflow during sleep.

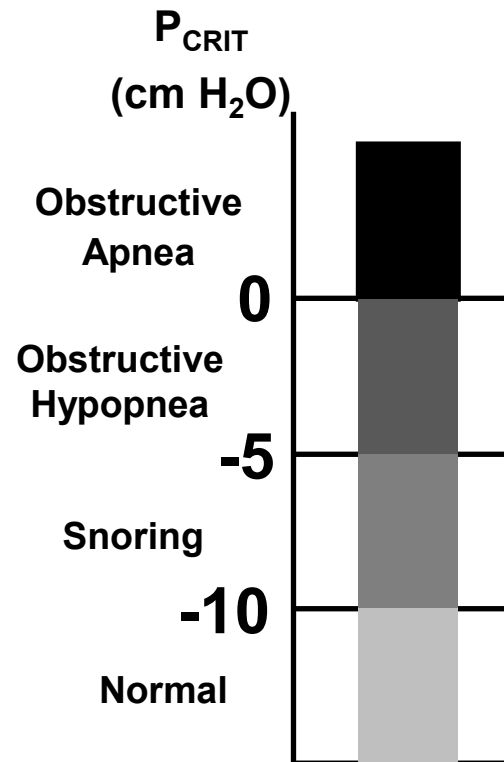
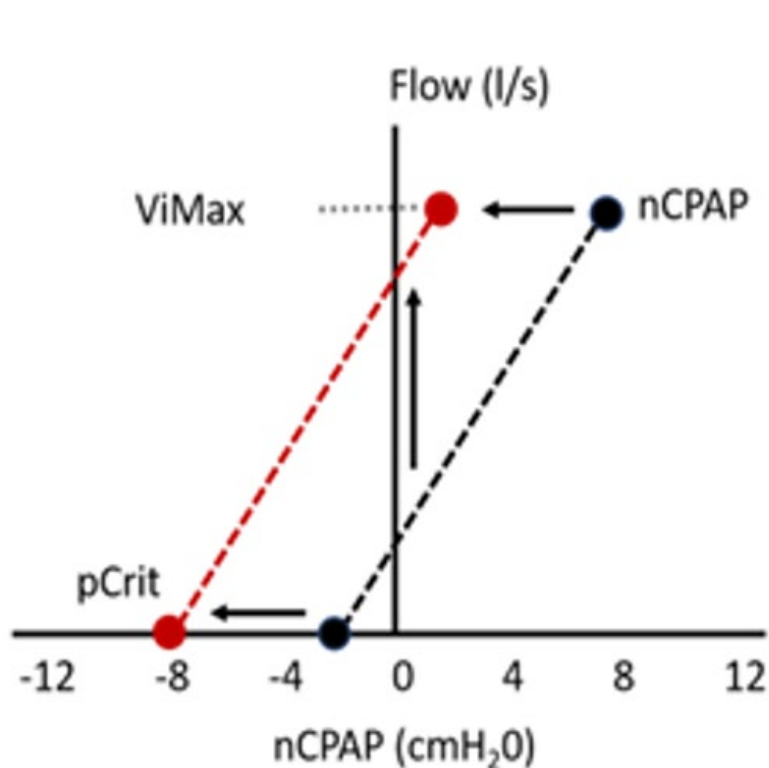
Interventions for OSA must stabilize the upper airway from collapse.

1. How collapsible is the airway?
2. How much can my intervention change that collapse?

This is critical to how we talk to patients about treatment

Methods for measuring airway collapsibility?

Airway critical closing pressure (PCrit): the pressure at which the airway closes (no flow).



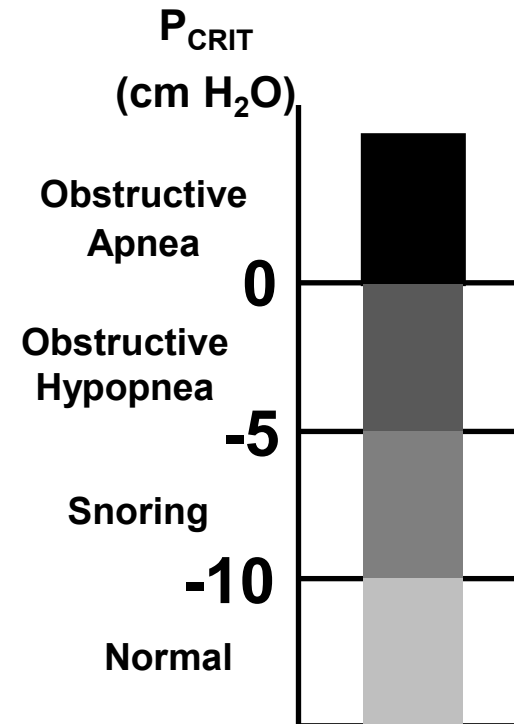
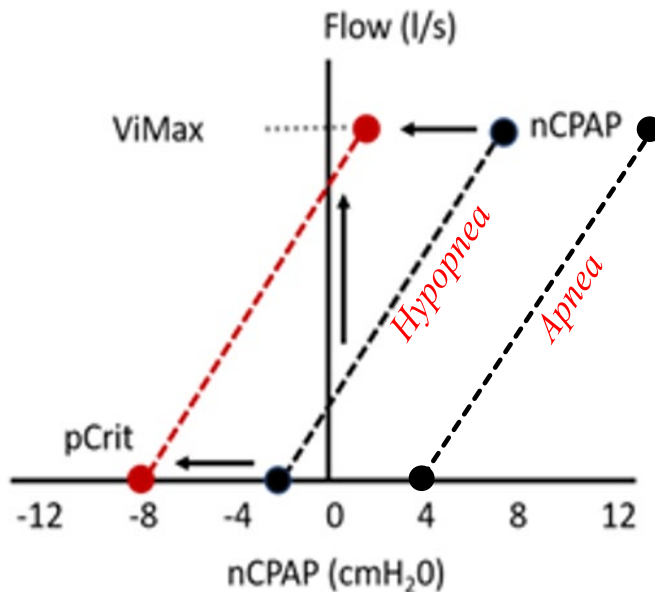
Nasal CPAP pressure is surrogate for PCrit

Therapeutic Nasal CPAP pressure is the pressure (cmH₂O) required to abrogate airway collapse.

- **Inverse of PCrit:**(Open pressure vs. Closing pressure)

Hypopnea:Apnea ratio (HAR) is a surrogate for PCrit

Higher HAR is associated with lower (more negative) PCrit and Lower therapeutic nasal CPAP pressure



Nasal CPAP pressure predicts response to HGNS therapy

Table 2. Comparison of DISE and Sleep Study Data in HGNS Responders and Nonresponders^a

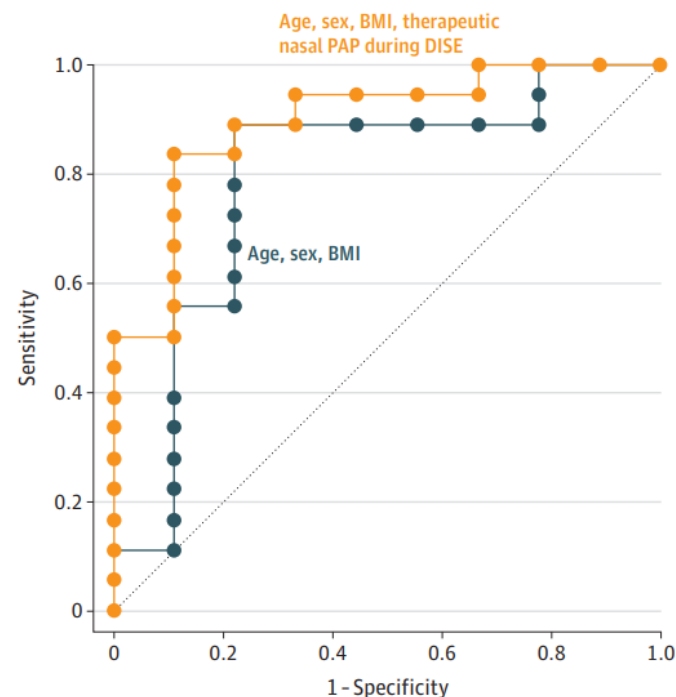
Measure	Mean (SD)		Effect size (95% CI) ^b	Adjusted effect size (95% CI) ^{b,c}
	Responders (n = 18)	Nonresponders (n = 9)		
DISE data				
Velum % collapse	92.2 (20.5)	100 (0)	-7.8 (-22.0 to 6.4)	-6.5 (-20.9 to 8.0)
Velum opening pressure, cm H ₂ O	5.0 (2.8)	9.2 (3.7)	-4.2 (-6.8 to -1.6)	-3.5 (-6.7 to -0.4)
Sleep study data, events/h				
Baseline AHI	40.5 (20.6)	31.2 (17.6)	9.3 (-7.3 to 25.8)	10.4 (-5.9 to 26.8)
Treatment AHI	9.3 (8.8)	34.5 (14.8)	-25.2 (-34.5 to -15.9)	-26.3 (-36.6 to -15.9)
Absolute AHI change	-31.2 (14.2)	3.3 (12.8)	-34.4 (-46.0 to -22.9)	-36.7 (-48.7 to -24.7)
% AHI change	-78.8 (11.3)	24.2 (56.7)	-103.0 (-131.1 to -74.9)	-103.1 (-137.7 to -68.5)

Nasal CPAP pressure predicts HGNS success

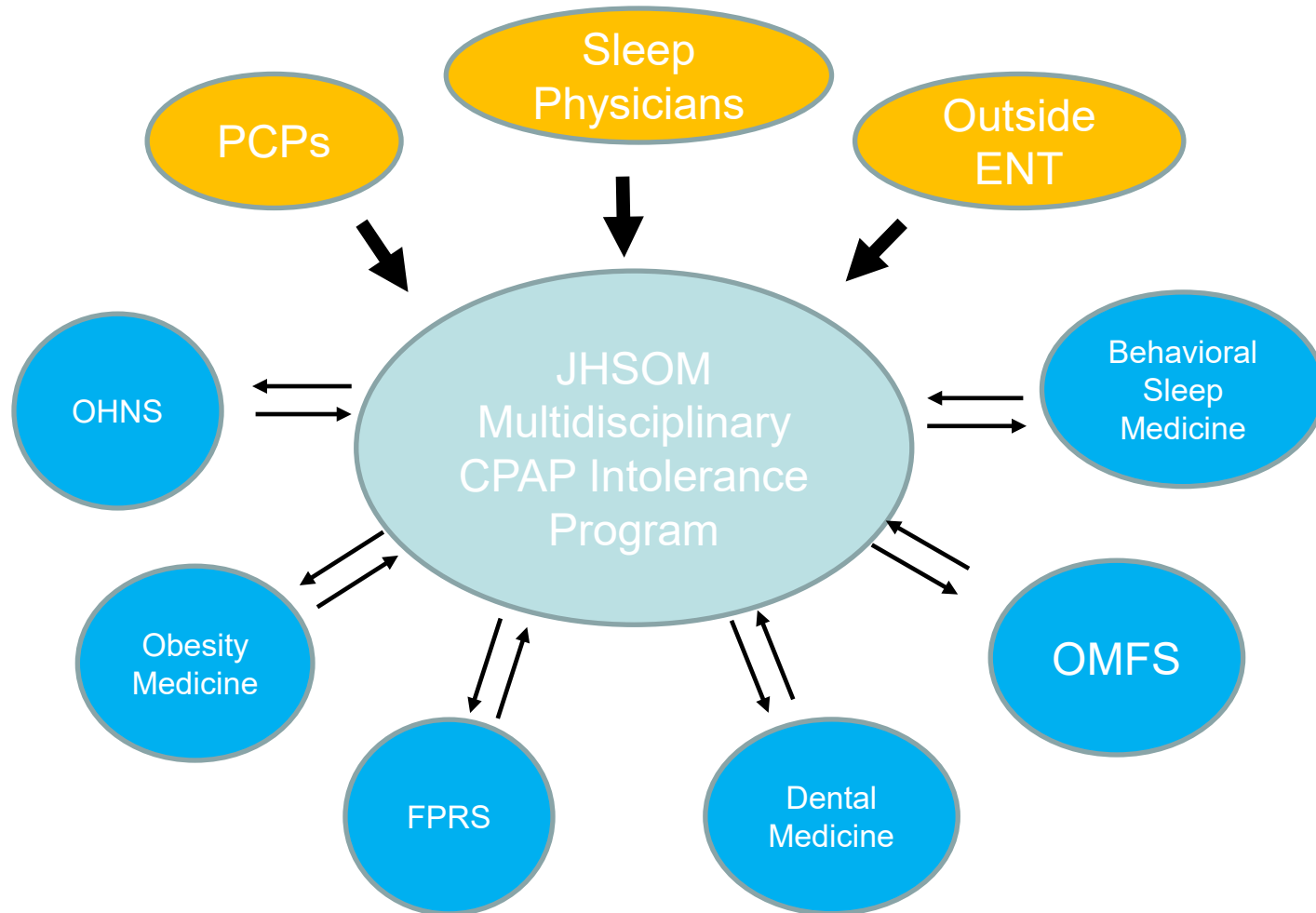
Table 4. Area Under the Receiver Operating Characteristic Curve for Clinical Factors and Therapeutic Nasal PAP During DISE in Prediction of Response to HGNS^a

Model	AUC (95% CI)
Individual measures	
Age	0.559 (0.330-0.787)
Male sex	0.694 (0.510-0.879)
BMI	0.630 (0.404-0.855)
Nasal PAP during DISE	0.818 (0.629-1.000)
Combined measures	
Age, male sex, and BMI	0.790 (0.578-1.000)
Age, male sex, BMI, and nasal PAP during DISE	0.895 (0.766-1.000)

Figure 2. Therapeutic Nasal Positive Airway Pressure (PAP) During Drug-Induced Sleep Endoscopy (DISE) as a Predictor for Hypoglossal Nerve Stimulation Response, Defined as a 50% Reduction in Overall Apnea-Hypopnea Index (AHI) Level



Multidisciplinary Team



Questions?
