Full Spectrum of OSA: From Infant to Elderly – Cardiovascular to Neurologic Consequences and The New Approaches to Management

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www.CSMA.clinic

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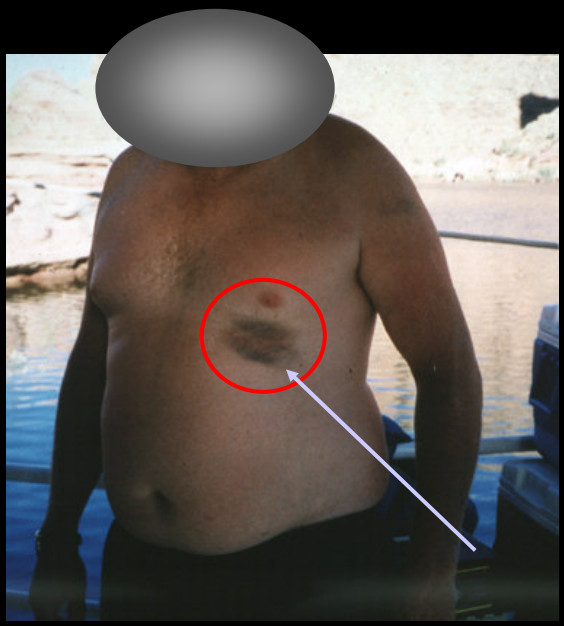
62 y/o male presents with tenderness under his left breast region where an area of echymosis has developed overnight. He is a deep sleeper and has no clear recollection of what may have precipitated the lesion.

Further questioning demonstrates excessive daytime sleepiness, most noted when sitting in meetings at work. Typically he keeps himself busy during the day and when first asked "are you sleepy during the day?" his response is "no". Not until further questioning does his difficulties with daytime sleepiness become apparent.



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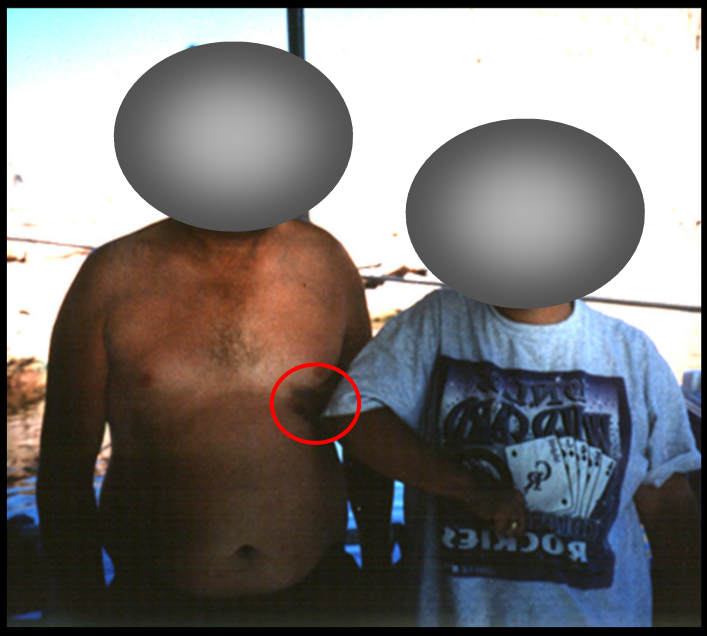
On questioning of his wife it became clear that she induced the lesion by striking him in the chest with her elbow in the middle of the night in attempts to stop his snoring that kept her awake.



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SNORING
AND
SLEEP APNEA

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How do you explain OSA to your patients?

- Do you base your discussion on **oxygen** levels?
- **Don't center your explanation around oxygen levels!!!!!!!!**
- **WHY NOT?**
- What if the study doesn't show any drop in oxygen, yet the patient has a host of symptoms suggesting OSA?
- Currently, Home Sleep Apnea Testing (HSAT) is being used frequently to assess for OSA, however, it may come back with a false negative result. The oxygen does not need to drop in have OSA.
- As you improve your ability to identify patients with sleep related obstructive breathing, you are more likely to suspect patients who have more subtle obstructive events, who do not desaturate SaO2 levels and as a result the HSAT is more likely to be falsely normal (false negative test).

You are best to describe OSA in terms of abnormal negative pressures in the airway.

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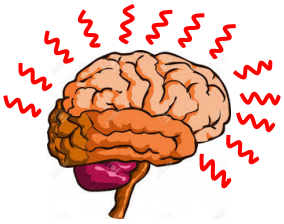
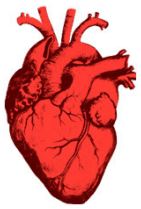
The Full Spectrum of Obstructive Breathing During Sleep

Snoring **Gasping** **Pauses in breathing**

< - Bruxing / Clenching During Sleep - >

Normal Breathing → Respiratory Effort Related Arousals of Sleep (RERAS) → Hypopneas with normal SaO2 (Hypopnea Rule 1A) → Hypopneas with SaO2 desaturations (Hypopnea Rule 1B) → **Complete Obstructive Apnea**





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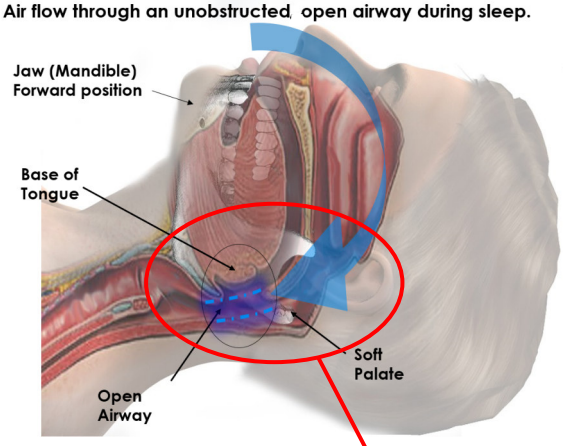
The Physics Behind Obstructive Breathing During Sleep

Normal Open Airway During Sleep

Air flow through an unobstructed, open airway during sleep.



Bernoulli effect from the flow of air produces a negative pressure in the back of the airway

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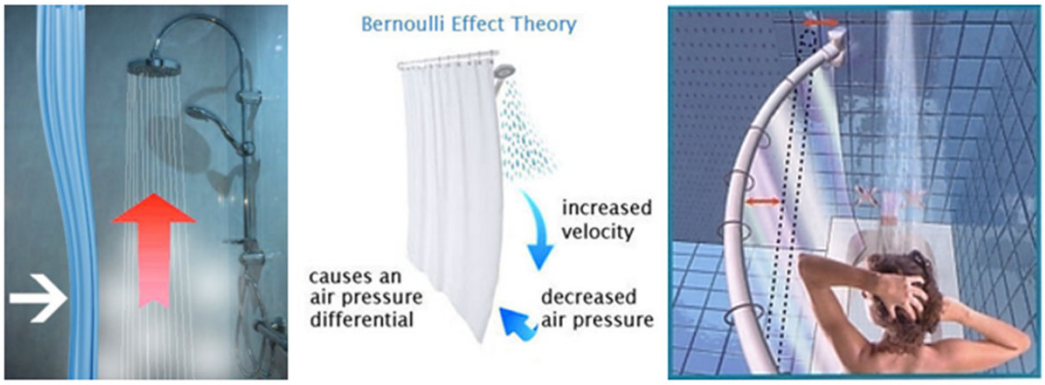
Flow and its relation to negative pressure (vacuum) – *The Bernoulli Effect*



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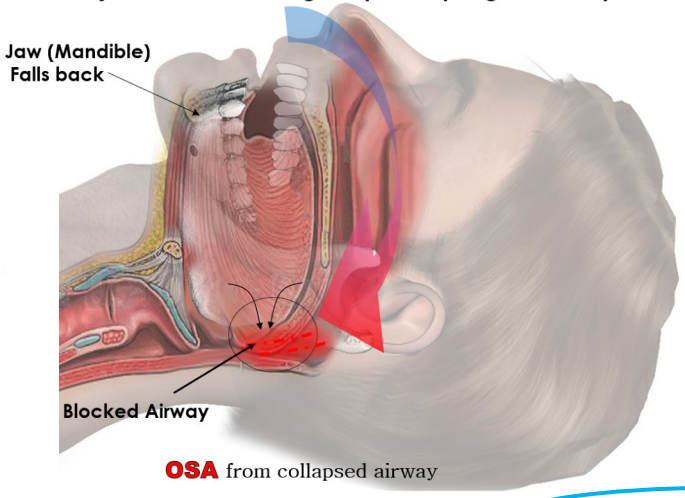
Abnormal Obstructed Airway

The jaw falls back during sleep, collapsing the airway



Obstructive Sleep Apnea is an anatomical / pressure problem, NOT an oximetry problem!!!!
Otherwise, we would call it Desaturation Sleep Apnea, but we call it OSA because of obstruction

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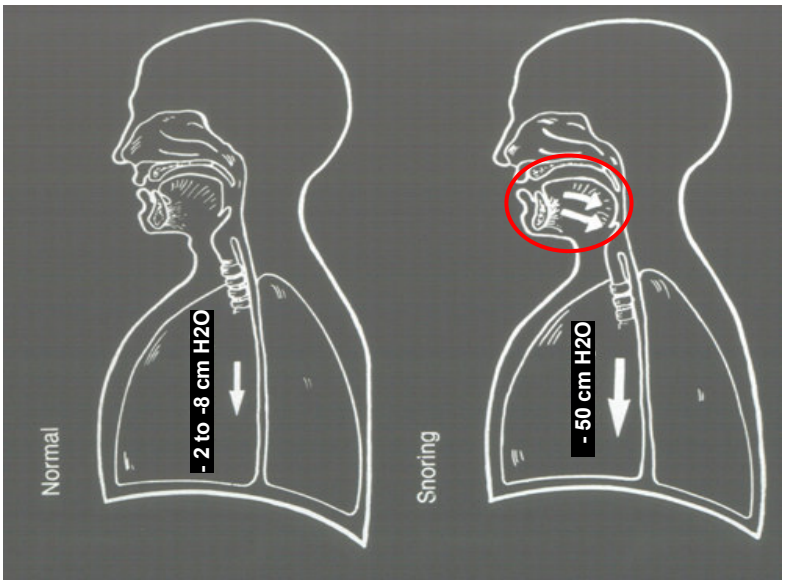
OSA is a pressure problem at its origin, not an oxygenation problem.
Oxygenation issues occur down stream, but not at its origin.
Thus, the term "obstructive" sleep apnea is used be cause of airway obstruction.



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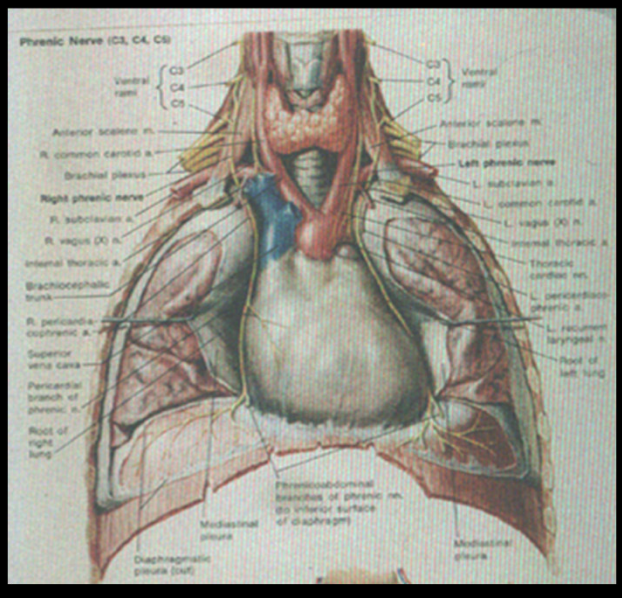
The heart has to function inside of a vacuum chamber in patients with OSA



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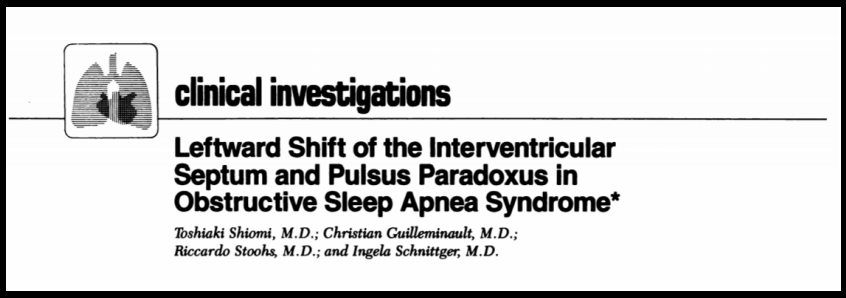

clinical investigations

Leftward Shift of the Interventricular Septum and Pulsus Paradoxus in Obstructive Sleep Apnea Syndrome*

Toshiaki Shiomi, M.D.; Christian Guilleminault, M.D.; Riccardo Stoohs, M.D.; and Ingela Schnittger, M.D.

Chest. 1991 Oct;100(4):894-902.

Abstract

Echocardiograms were taken from the parasternal long axis view during nocturnal sleep in ten patients diagnosed with OSAS. A table designed to support the echocardiographic probe prevented significant sleep disturbances during monitoring and allowed continuous data collection with and without nasal CPAP administration. In five of ten patients, there was before CPAP treatment a diastolic LSIVS during NREM sleep, inducing a flattening of the left ventricle. Arterial blood pressure recordings showed pulsus paradoxus when LSIVS was occurring. Nasal CPAP led to normal, unobstructed breathing, significant decrease in Pes nadir and disappearance of LSIVS and pulsus paradoxus. Increase in left ventricular afterload and increase in total peripheral resistance could lead to hypertrophy and hypertension in some OSAS patients. The presence of pulsus paradoxus in OSAS indicates a marked increase in Pes nadir, and its disappearance with nasal CPAP may be one of the signs of effective treatment of OSAS.

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Interventricular Septum

Esophageal Pressure

Interventricular Septum

Esophageal Pressure

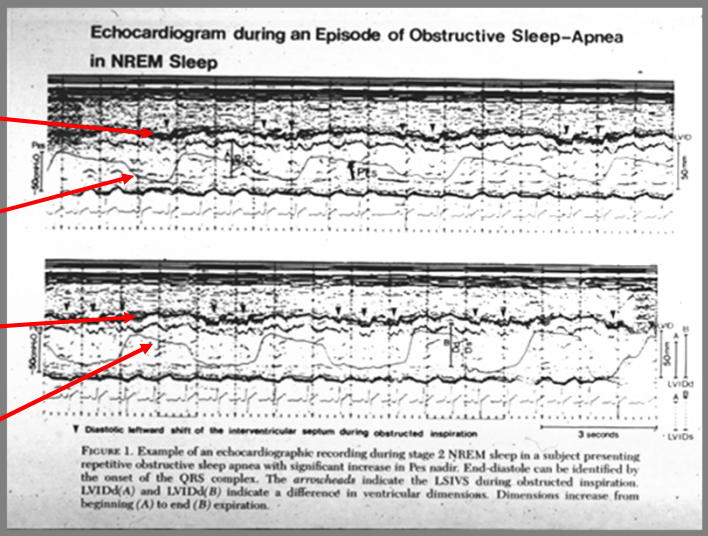

FIGURE 1. Example of an echocardiographic recording during stage 2 NREM sleep in a subject presenting repetitive obstructive sleep apnea with significant increase in Pes nadir. End-diastole can be identified by the onset of the QRS complex. The *arrowheads* indicate the LSIVS during obstructed inspiration. LVIDd(*A*) and LVIDd(*B*) indicate a difference in ventricular dimensions. Dimensions increase from beginning (*A*) to end (*B*) expiration.

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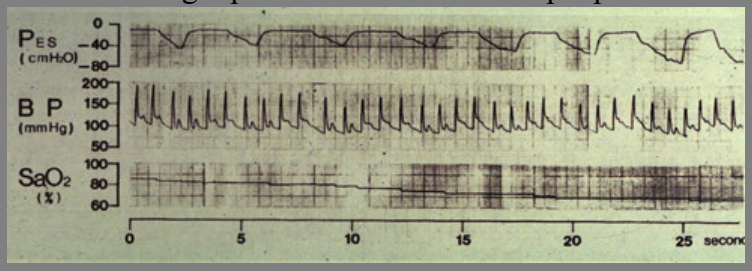
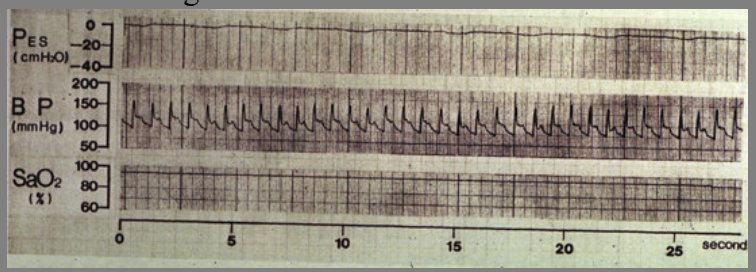
During repetitive Obstructive Sleep Apnea



During treatment with nasal CPAP



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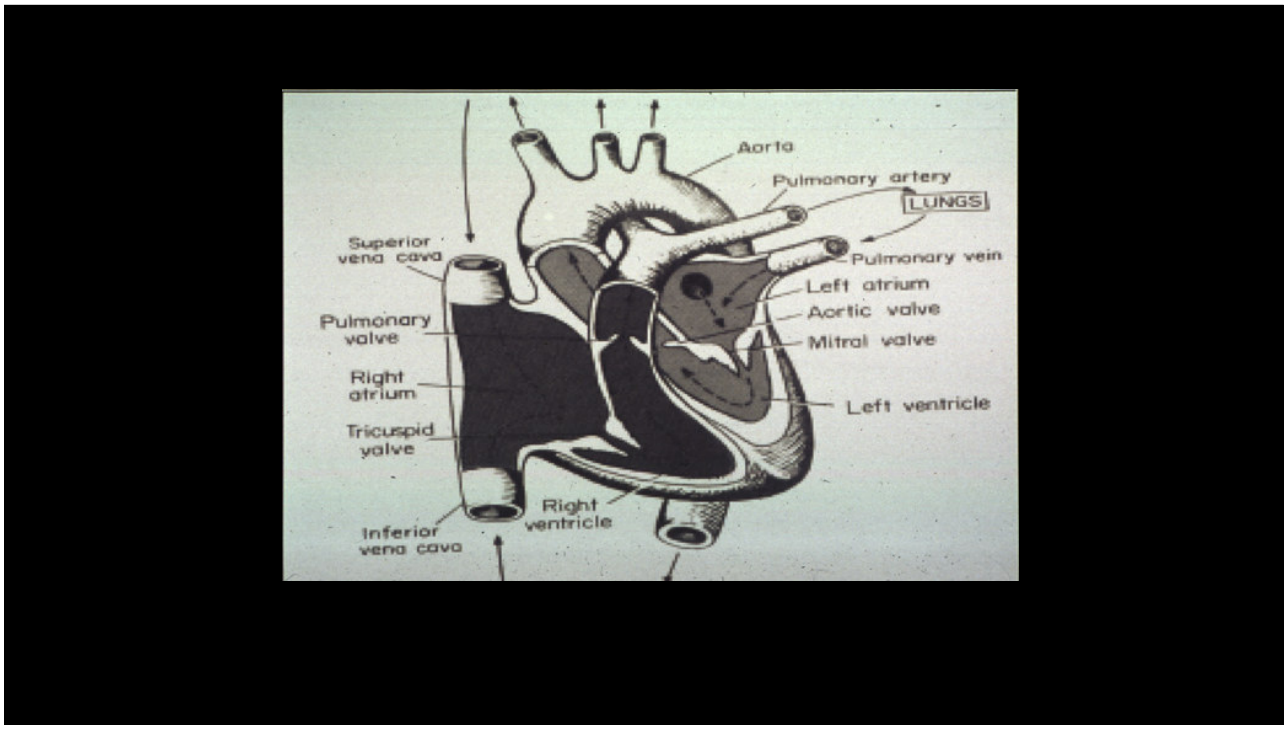

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Taking a Sleep History

"I'm having trouble sleeping at Night"

- The differential diagnosis starts at this point:

- Two major categories of difficulty sleeping:
  - Difficulties falling asleep (sleep initiation insomnia)
  - Difficulties staying asleep (sleep maintenance insomnia)

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Co-Morbid Insomnia and Sleep Apnea (COMISA): Prevalence, Consequences, Methodological Considerations, and Recent Randomized Controlled Trials

by Alexander Sweetman [1,*], Leon Lack [2] and Célyne Bastien [3]

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[2] The Adelaide Institute for Sleep Health: A Flinders Centre of Research Excellence, College of Education Psychology and Social Work, Flinders University, Adelaide 5042, South Australia, Australia
[3] School of Psychology, Félix-Antoine-Savard Pavilion, 2325, rue des Bibliothèques, local 1012, Laval University, Quebec City, QC G1V 0A6, Canada
* Author to whom correspondence should be addressed.

Brain Sci. **2019**, *9*(12), 371; https://doi.org/10.3390/brainsci9120371

Submission received: 15 November 2019 / Revised: 9 December 2019 / Accepted: 10 December 2019 / Published: 12 December 2019

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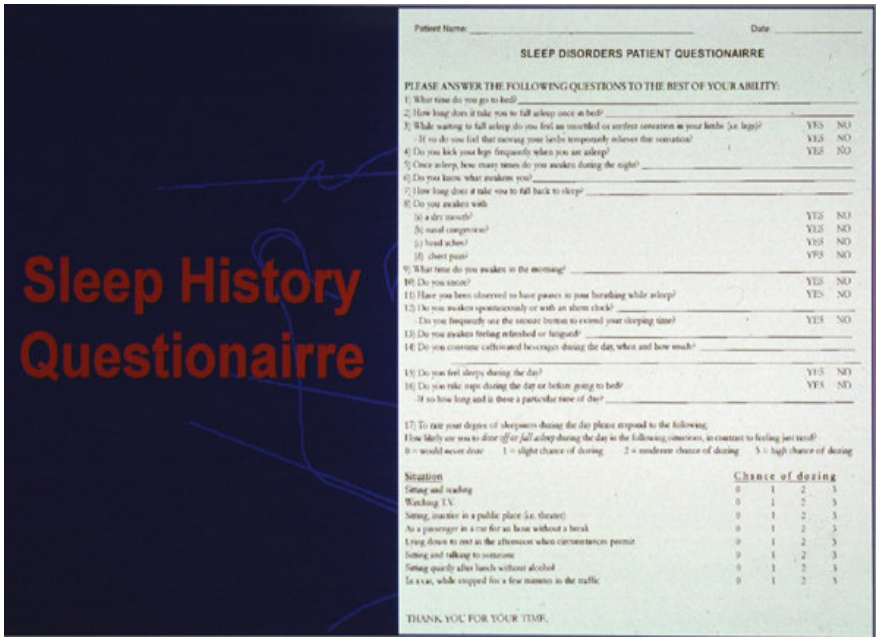
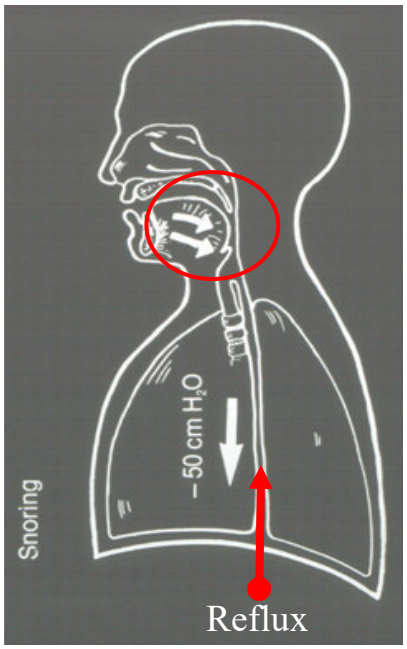


20

Nocturnal GERD

Simmons, J.H., Mann, C., Banerji, S. Intrathoracic Pressure Monitoring During CPAP Titration in Patients with Esophageal Reflux and OSA. Sleep Research Vol 26, 1997.



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Atrial Natriuretic Peptide increases during OSA

Respiration. 1992;59(3):164-8.

Changes of circulating atrial natriuretic peptide and antidiuretic hormone in obstructive sleep apnea syndrome.

Ichioka M, Hirata Y, Inase N, Tojo N, Yoshizawa M, Chida M, Miyazato I, Taniai S, Marumo F.

Second Department of Internal Medicine, Tokyo Medical and Dental University, Japan.

Abstract

Patients with obstructive sleep apnea (OSA) syndrome are known to exhibit nocturnal natriuresis/diuresis. We studied plasma and urinary levels of atrial natriuretic peptide (ANP), a potent natriuretic hormone released from the heart, and plasma antidiuretic hormone (ADH) levels in patients with OSA during awake and sleeping periods, to compare with those of normal subjects. Seven patients with OSA and 6 normal subjects were studied. Arterial blood samples were drawn during the awake and the sleeping period, while in patients with OSA, blood samples were obtained during the apneic period. Urine samples were collected over two 12-hour periods (9 a.m.-9 p.m. and 9 p.m.-9 a.m.) In patients with OSA, plasma ANP as well as urinary ANP excretion increased during the apneic period compared with the awake period. There was a significant negative correlation between plasma levels of ANP and ADH in patients with OSA. On the other hand, normal subjects had no apparent differences in plasma and urinary ANP levels between the two periods. It is suggested that nocturnal increase in ANP and decrease in ADH are responsible for the nocturnal diuresis and natriuresis associated with OSA.

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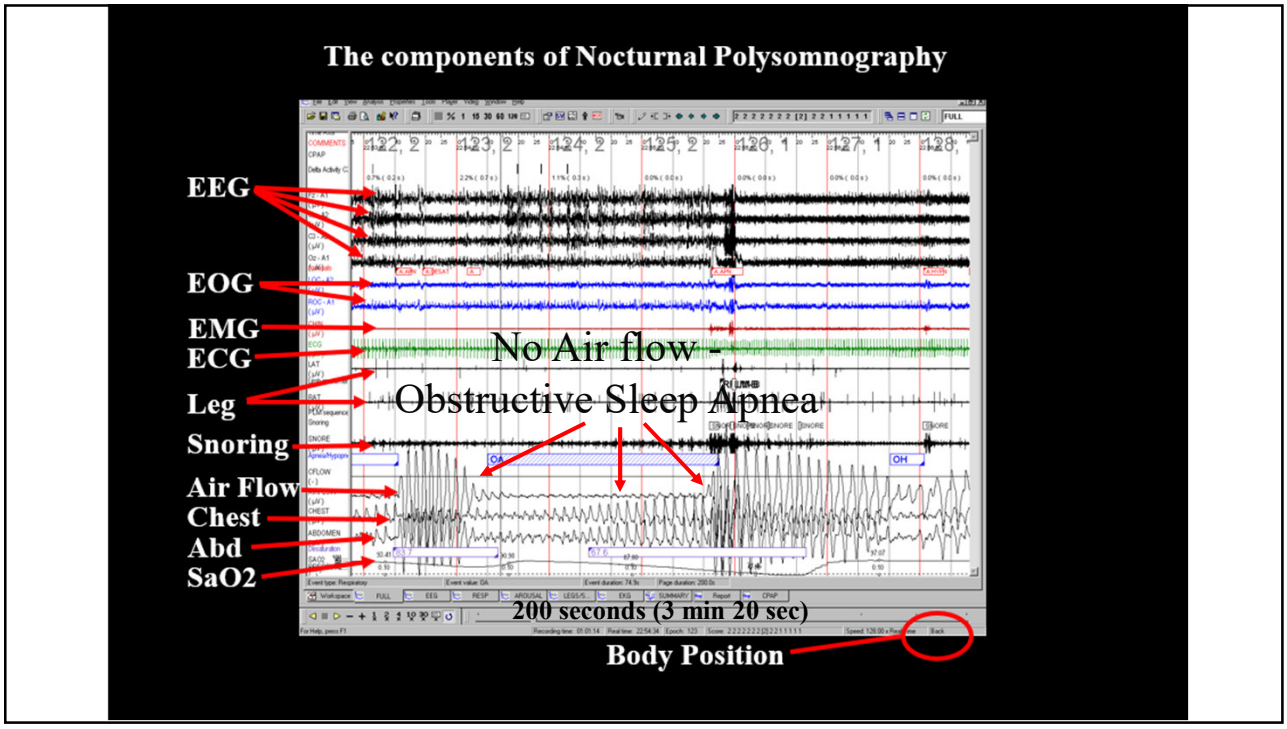
The components of Nocturnal Polysomnography
EEG
EOG
EMG
ECG
Leg
Snoring
Air Flow
Chest
Abd
SaO2
No Air flow -
Obstructive Sleep Apnea
200 seconds (3 min 20 sec)
Body Position

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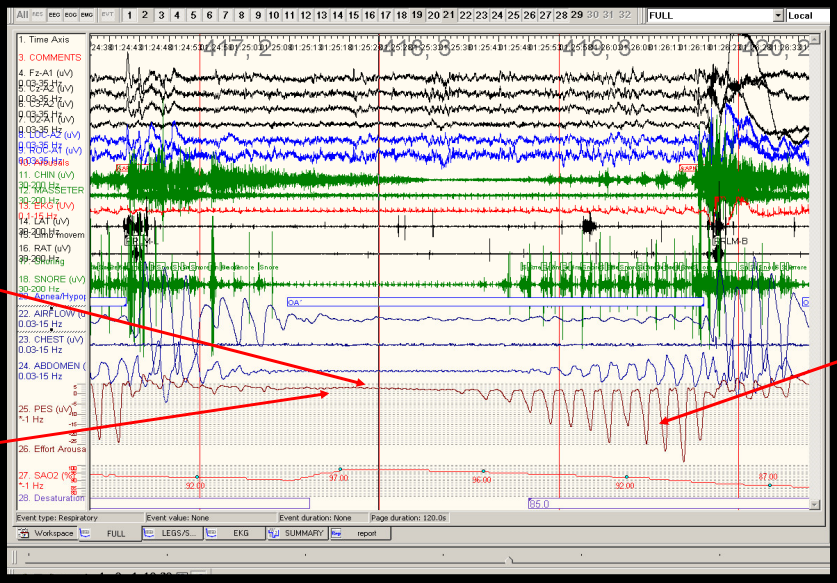
Mixed Apnea (both central and obstructive components)



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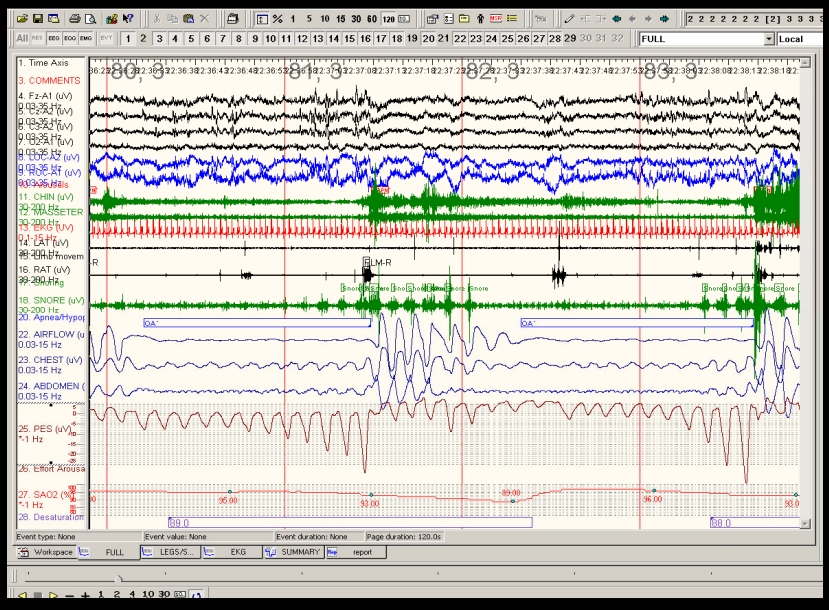
Two back-to-back Obstructive Apnea events



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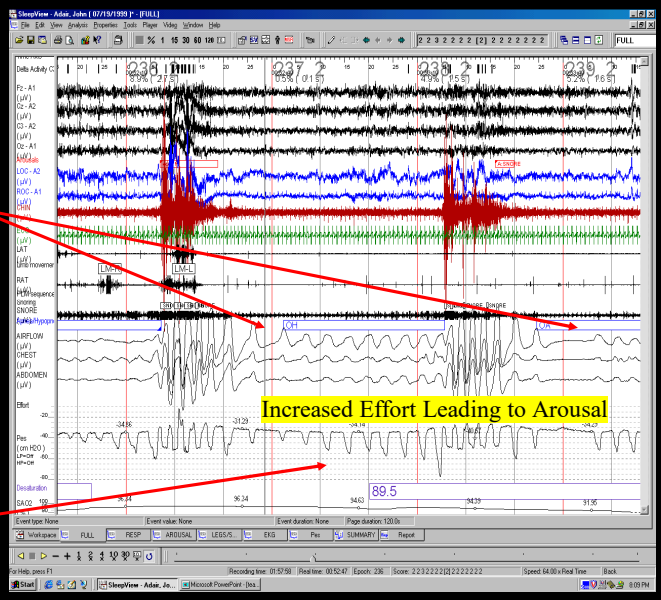
120 seconds of data showing HYPOPNEA



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Hypopnea Scoring Rules implemented by the AASM in 2013

Two different scoring rules currently exist, causing confusion.

Rule 1A (Recommended rule)
Score a respiratory event as a hypopnea if ALL of the following criteria are met:

a. The peak signal excursions drop by ≥30% of pre-event baseline
b. The duration of the ≥30% drop in signal excursion is ≥10 seconds.
c. There is a ≥3% oxygen desaturation from pre-event baseline or the event is associated with an arousal. Note: No SaO2 desaturation required.

Rule 1B (Acceptable but not the recommended rule) Unfortunately many sleep facilities use this rule. Medicare only recognizes this rule.

Score a respiratory event as a hypopnea if
a. The peak signal excursions drop by ≥30% of pre-event baseline
b. The duration of the ≥30% drop in signal excursion is ≥10 seconds.
c. There is a ≥ 4% oxygen desaturation from pre-event baseline Note: No mention of arousals. Arousals are not part of 1B Hypopneas.

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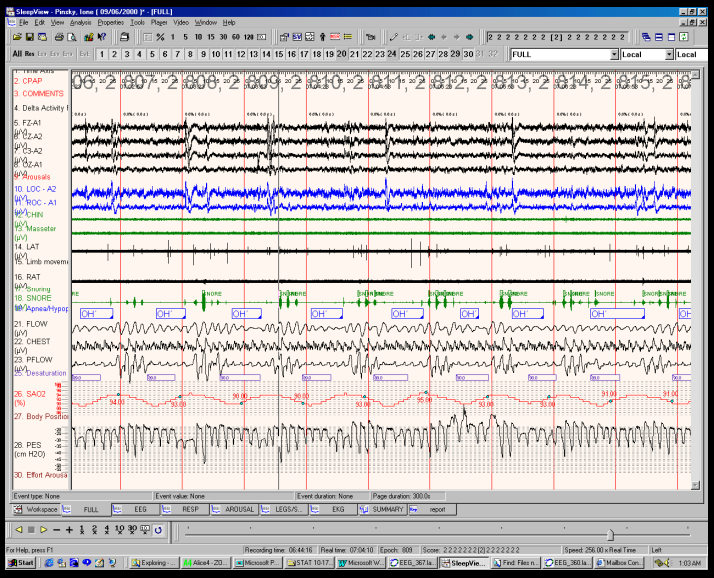
Repetitive Hypopnea over a 5 minute period



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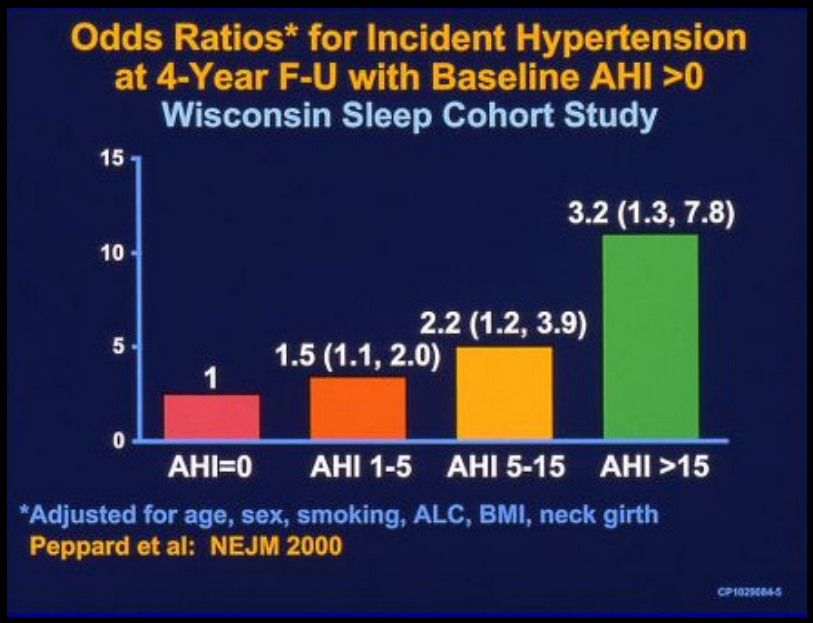

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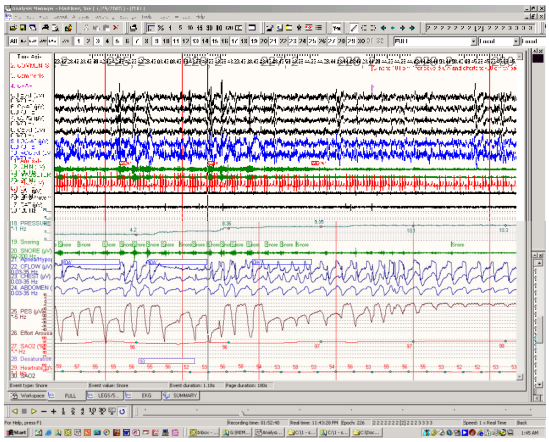
Drastic Improvement in the Intra-thoracic Pressures with CPAP



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Effect of Nasal Continuous Positive Airway Pressure Treatment on Blood Pressure in Patients With Obstructive Sleep Apnea

Heinrich F. Becker, MD; Andreas Jerrentrup, MD; Thomas Ploch, Dipl Psych; Ludger Grote, MD; Thomas Penzel, PhD; Colin E. Sullivan, MD; J. Hermann Peter, MD

Background—There is increasing evidence that obstructive sleep apnea (OSA) is an independent risk factor for arterial hypertension. Because there are no controlled studies showing a substantial effect of nasal continuous positive airway pressure (nCPAP) therapy on hypertension in OSA, the impact of treatment on cardiovascular sequelae has been questioned altogether. Therefore, we studied the effect of nCPAP on arterial hypertension in patients with OSA.

Methods and Results—Sixty consecutive patients with moderate to severe OSA were randomly assigned to either effective or subtherapeutic nCPAP for 9 weeks on average. Nocturnal polysomnography and continuous noninvasive blood pressure recording for 19 hours was performed before and with treatment. Thirty two patients, 16 in each group, completed the study. Apneas and hypopneas were reduced by ≈95% and 50% in the therapeutic and subtherapeutic groups, respectively. Mean arterial blood pressure decreased by 9.9±11.4 mm Hg with effective nCPAP treatment, whereas no relevant change occurred with subtherapeutic nCPAP (P=0.01). Mean, diastolic, and systolic blood pressures all decreased significantly by ≈10 mm Hg, both at night and during the day.

Conclusions—Effective nCPAP treatment in patients with moderate to severe OSA leads to a substantial reduction in both day and night arterial blood pressure. The fact that a 50% reduction in the apnea-hypopnea index did not result in a decrease in blood pressure emphasizes the importance of highly effective treatment. The drop in mean blood pressure by 10 mm Hg would be predicted to reduce coronary heart disease event risk by 37% and stroke risk by 56%. (***Circulation***. **2003;107:68-73.**)

Conclusion:

1. **Effective CPAP treatment substantially reduced BP.**
2. **In patient with a 50% reduction in the Apnea Hypopnea Index who still had abnormal AHI's did not demonstrate improvement in BP.**
3. **In the Effectively treated group, the reduction in mean BP of 10 mm Hg is predicted to reduce coronary heart disease event risk by 37% and stroke risk by 56%.**

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Current PAP machines are sit comfortably on the nightstand.

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Positive Airway Pressure (PAP)

CPAP = Continuous

APAP = Auto-titrating

BiPAP = BiLevel
IPAP = Inspiratory
EPAP=Expiratory

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Now there are CPAP machines that can fit in the palm of your hand.

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New Mask Technologies Improve Patient Compliance

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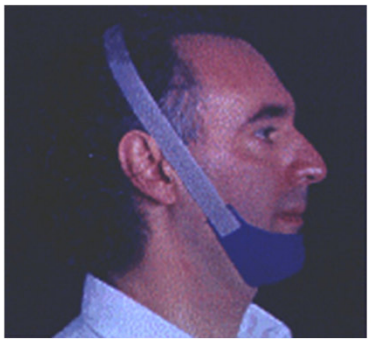
A Chin Strap May Be Needed To Keep The Mouth Closed



Controlling the mandible is the key to success!

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Full Face Mask

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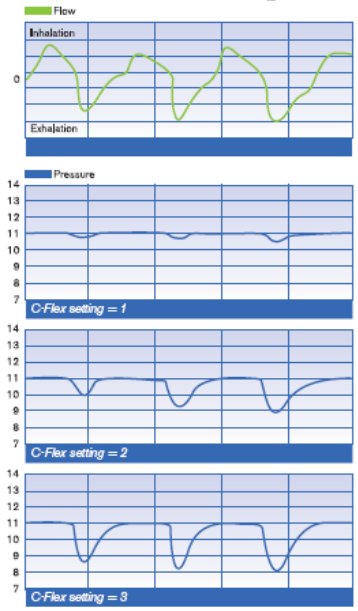
EPR (Expiratory Pressure Relief)



- Transiently lowers the pressure on expiration by 1,2 or 3 cm H2O
- Can improve comfort and help overcome difficulties with CPAP acclimation
- Clearly NOT for everyone and can be disruptive to some patient
- Lowering the pressure can induce events in some patients

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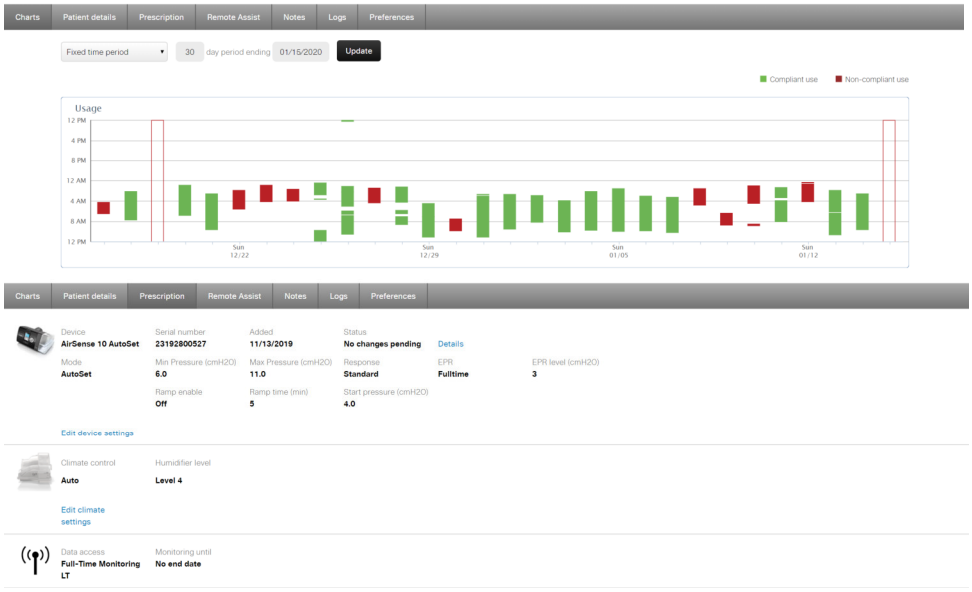
CPAP Compliance Data obtained remotely from the cloud.



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Proper CPAP / BiPAP care

- Change Filter
- Clean Mask Cushion
- Clean Hoses
- Proper adjustment of straps
- Proper Hose placement

Dirty CPAP Filter

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Keeping the humidifier clean is important. Moisture can become a place for mold to grow causing pulmonary infections.

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Small Machines and Comfortable Masks

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Terms use for describing different types of positive pressure treatments

The unit of measure is cm H2O. Typically, 4 – 20 cm H2O

Continuous Positive Airway Pressure = (CPAP)

BiLevel Positive Airway Pressure = (BPAP)

High pressure in inspiration (IPAP) / Low pressure on expiration (EPAP)

PAP = Either of these (Positive Airway Pressures)

APAP = Auto (adjusting) Positive Airway Pressure

This is the same as CPAP but the machine will adjust the pressures in response to OSA (if detected). The output is like CPAP not BiPAP

ERP = Expiratory Pressure Relief

(Used with CPAP or APAP. The pressure drops on expiration by either 1,2 or 3 cm H2O. Similar to BPAP but Max = 3)

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Indications for CPAP and other modes of OSA Tx

Centers for Medicare and Medicaid Services Guidelines

- AHI $\geq$ 15

  or

- AHI $\geq$ 5 with and of the following
  - HTN
  - Stroke
  - EDS (Excessive Daytime Sleepiness – i.e. Epworth > 8)
  - Ischemic Heart Disease
  - Insomnia
  - Mood disorder

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Treatment Options

- CPAP / BiPAP
- Oral Appliance Therapy (OAT)
  - Mandibular Advancing Dental Appliances
  - Tongue Retaining Appliances
- Surgical Intervention
  - Soft tissue reduction (UPPP, Tongue base reduction .etc.
  - Sinus surgery
  - Maxilla Mandibular Advancement
  - Hypoglossal Nerve Stimulator (Inspire)
- Weight loss in selected patients
- Orthodontics / Restorative dentistry
- Myofunctional Therapy
- Changes in habits – improved sleep hygiene, alcohol reduction etc.

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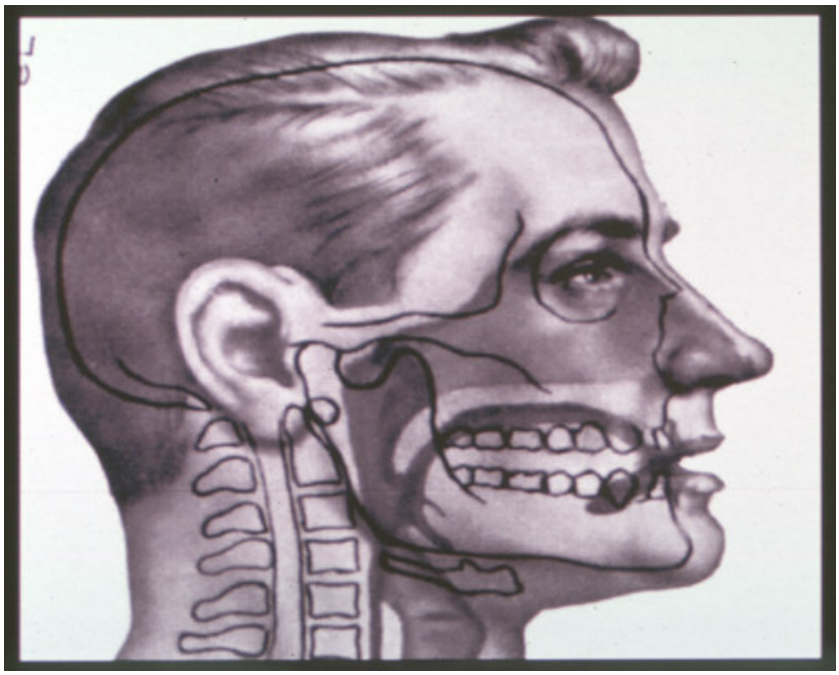

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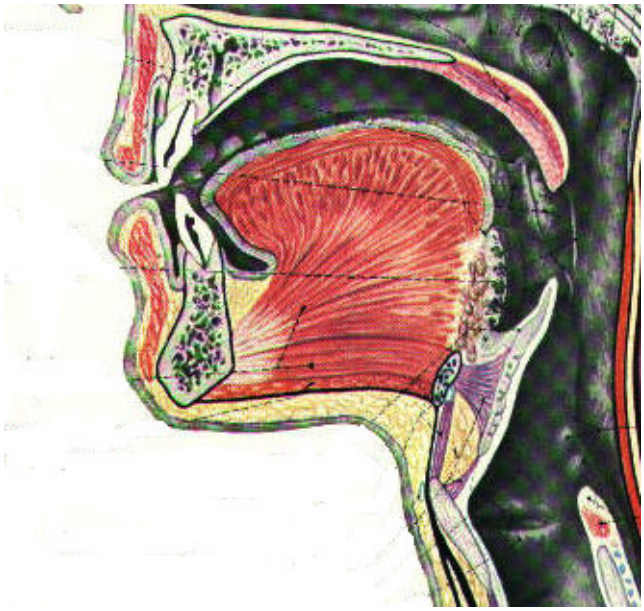

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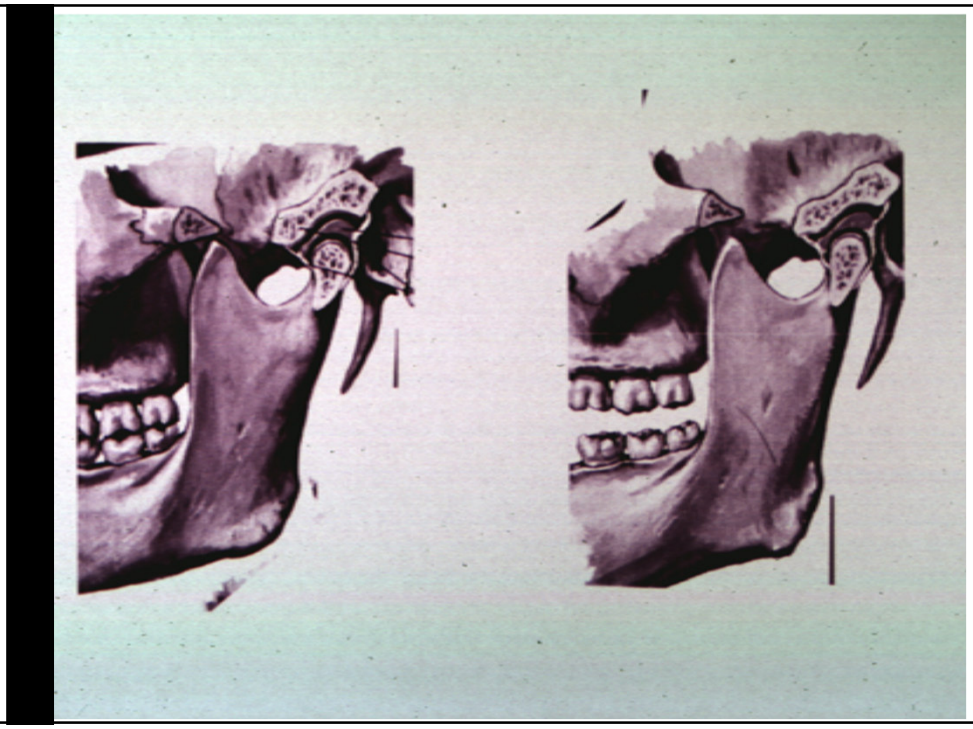

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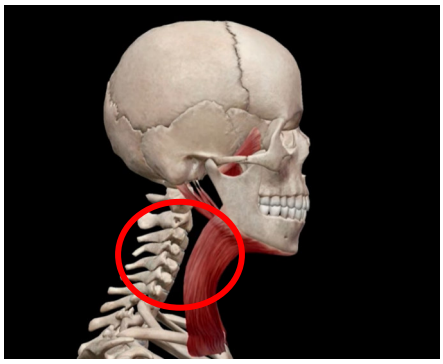
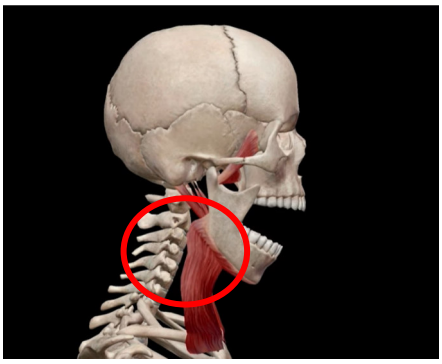
As the mandible opens and closes, the airway space changes





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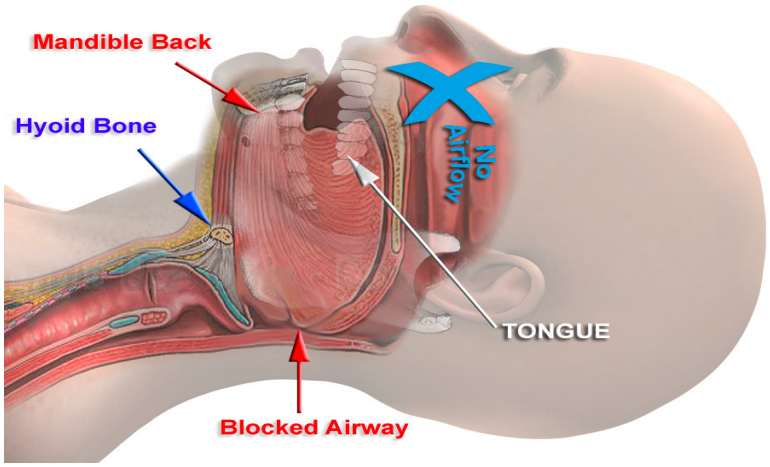
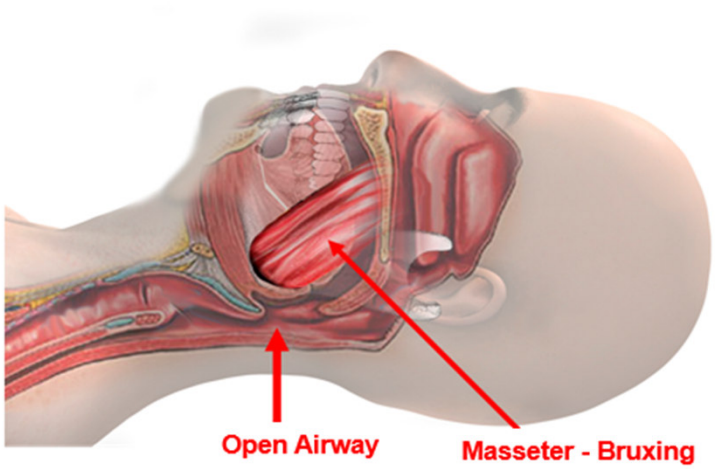
What happens with the Masseter, pterygoid muscles, genioglossus and other muscles that influence the upper airway .
Are these respiratory muscles? Some would say YES.

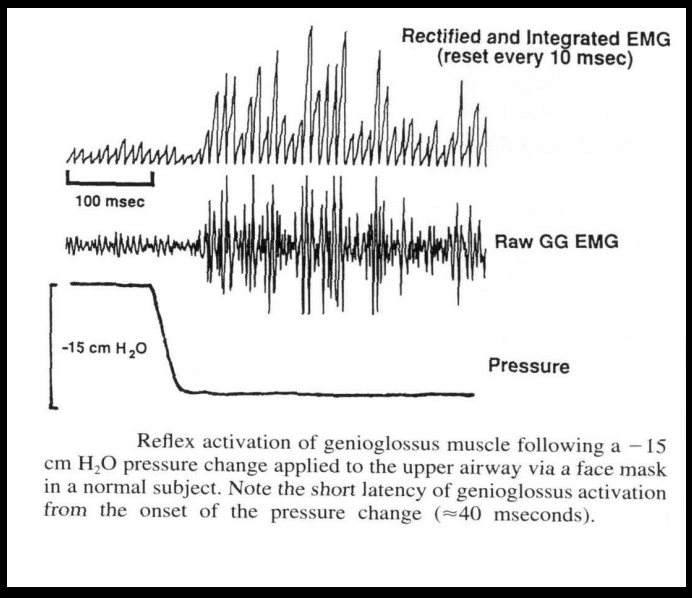


52

Increased masseter, genioglossus and pterygoids activity puts the TMJ into occlusion which hold the mandible, and thus the genioglossus muscle forward (preventing retraction), which helps keep airway open

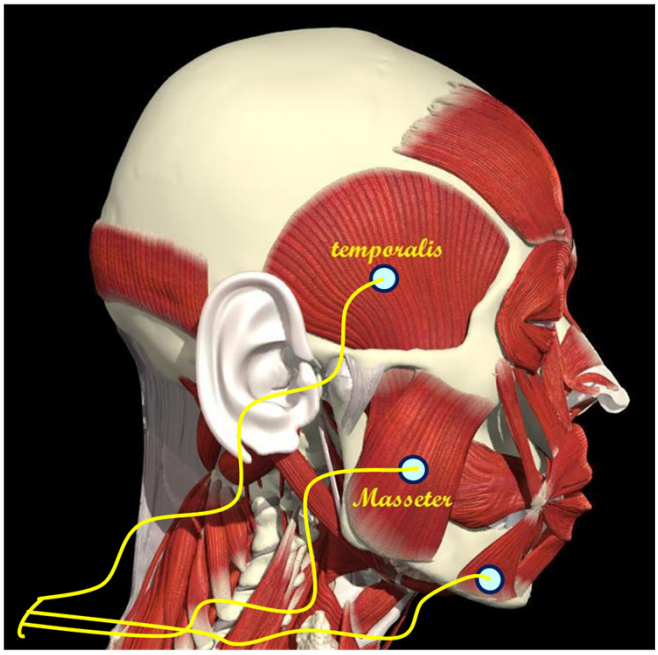


53



Reflex activation of genioglossus muscle following a −15 cm H_2O pressure change applied to the upper airway via a face mask in a normal subject. Note the short latency of genioglossus activation from the onset of the pressure change (≈40 mseconds).

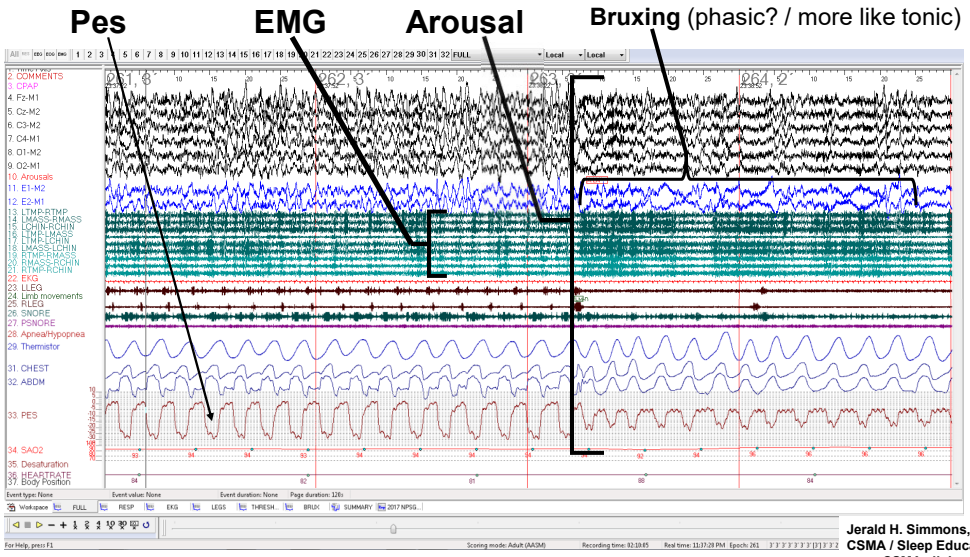
54



55

Confounding factors in the body of published literature on Sleep Bruxism:

1) Most Sleep Bruxing studies utilized older definitions of OSA (4% rule), not considering RERAS / UARS or the new AHI3% 1A definition.
2) None of the other clinical studies utilized esophageal pressure monitoring to characterize airway pressures to assess these changes.



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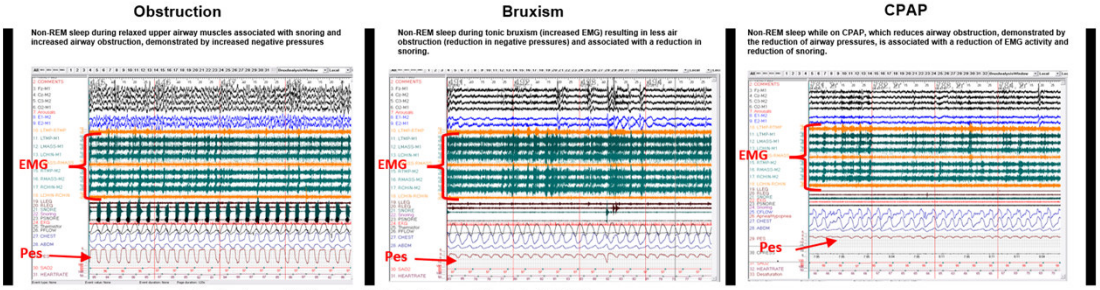
Tonic Bruxism Protects the Airway
CPAP Protects the Airway, Relieving the Need to Brux

Sleep Bruxism protects the upper airway against obstruction in patients with TMJ dysfunction and is reduced by PAP therapy:



Demonstration of Sleep Bruxism protecting the airway, which is not needed when the airway is treated with CPAP.
Pes monitoring (measuring pressure within the esophagus) provides the most objective assessments of airway obstruction. The above 3 segments, all from the same patient, demonstrate the following: Left image - increased obstruction (increased negative pressure) with snoring when the facial EMG activity is reduced (muscle relaxation). Middle image - Reduced airway obstruction (reduced Pes) when bruxing keeps the airway open. Right image – with CPAP treatment the airway is protected thus allowing the facial muscles, including the masseter, to relax.
Conclusion: 1) Sleep Bruxism is a protective compensatory process aimed to improve airway obstruction and 2) treatment with positive airway pressure improves Sleep Bruxism, most likely by eliminating the need for the protection Sleep Bruxism provides. This new methodology of tabulating EMG on polysomnography that we implemented may find a wider utilization however, further assessment is needed.

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International Journal of Pediatric Otorhinolaryngology
Volume 68, Issue 4 , Pages 441-445, April 2004

Improvement of bruxism after T & A surgery

Renata C. DiFrancesco, Paula Andreya S. Junqueira, Priscilla Maria Trezza, Maria Estela J. de Faria, Ronaldo Frizzarini, Fabio Elias Zerati

Received 9 May 2003; received in revised form 21 November 2003; accepted 23 November 2003.

Abstract

Bruxism or tooth grinding is an oral habit that frequently occurs during sleep. Some authors suggest it is associated to sleep apnea. ***Objective:*** The main goal of this study is compare the incidence of bruxism before and after adenotonsillectomy (T & A surgery) in children with sleep-disordered breathing. ***Methods:*** This is a prospective study in which we evaluated 69 consecutive children from the Otolaryngology Department of the University of São Paulo Medical School in pre- and post-surgical periods of adenotonsillectomy. Before and after surgery parents answered a questionnaire about sleep-disturbed breathing and bruxism. Children were submitted to E.N.T. examination and speech pathologist evaluation. The orthodontist inspected malocclusion. Before surgery all the 69 children presented sleep apnea and 45.6% presented bruxism. Malocclusion could be found in 60.71%. Three months after surgery none of the children presented breathing problems and only 11.8% presented bruxism. There was no difference in malocclusion. ***Conclusion:*** This study suggests that there is a positive correlation between sleep-disordered breathing and bruxism. There was an important improvement of bruxism after T & A surgery. Otolaryngologists must be aware that this pernicious sleep disorder is associated to airway obstruction and so, it must be considered when evaluating T & A hyperplasia.

Keywords: Bruxism, Apnea, Adenoids, Tonsil, Adenoidectomy, Tonsillectomy

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International Journal of Pediatric Otorhinolaryngology (2008) 72, 509–511

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International Journal of Pediatric Otorhinolaryngology

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Bruxism and adenotonsillectomy

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Received 13 October 2007; received in revised form 4 January 2008; accepted 5 January 2008
Available online 20 February 2008

KEYWORDS
Adenoidectomy;
Tonsillectomy;
Bruxism;
Snoring;
Mouth breathing

Summary

Objective: The main goal of this study is to assess the effect of adenotonsillectomy on bruxism in children with obstructive symptoms due to adenotonsillar hypertrophy.
Patients and methods: In a prospective study, 140 children aged between 4 and 12 years with obstructive symptoms due to adenotonsillar hypertrophy were evaluated. With a questionnaire existence of bruxism was evaluated before and after adenotonsillectomy and the results were compared with each other.
Results: The prevalence of bruxism was 25.7% before surgery and 7.1% after it. The difference was significant by p value of 0.02. There was not any significant difference between male and female dominancy.
Conclusion: This study suggests that adenotonsillectomy could improve bruxism significantly in children who have obstructive symptoms due to adenotonsillar hypertrophy.

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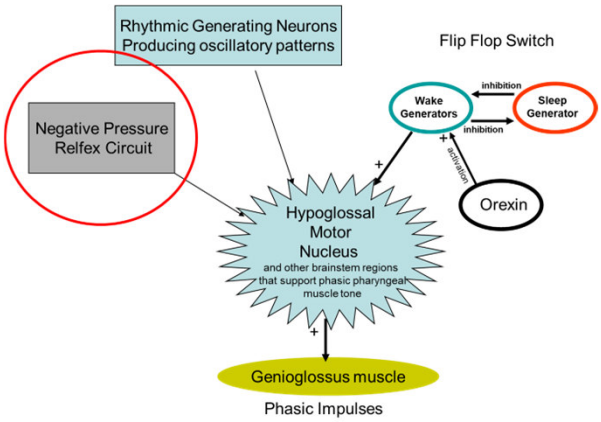
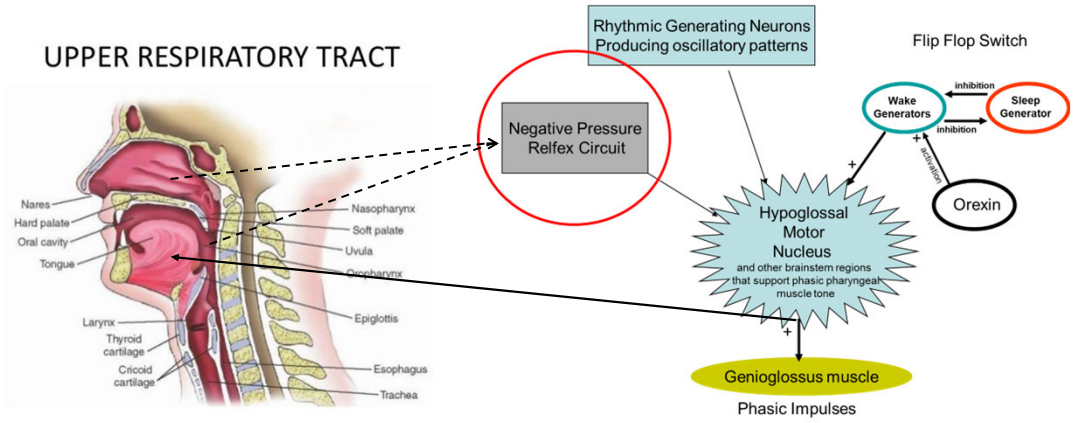
Brainstem regulation of Upper Airway Muscle Control of Respiration



60

Brainstem regulation of Upper Airway Muscle Control of Respiration



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Brief Communications

Local Mechanisms Drive Genioglossus Activation in Obstructive Sleep Apnea

ATUL MALHOTRA, ROBERT B. FOGEL, JILL K. EDWARDS, STEVEN A. SHEA, and DAVID P. WHITE

Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts

Individuals with obstructive sleep apnea (OSA) require increased pharyngeal muscle dilator activation during wakefulness to maintain upper airway patency. Negative pressure is one potential stimulus for this neuromuscular compensation. Individuals with OSA who have previously undergone tracheostomy provide an opportunity to study upper airway physiology in both the presence and absence of upper airway respiratory stimuli. If negative pressure (or another local airway stimulus) were important in driving pharyngeal dilator muscle activation, one would predict that during nasal breathing, the pharynx of a tracheostomized patient would be exposed to negative pressure, and that high levels of muscle activation would therefore be measured. Conversely, during breathing by the patient through the tracheal stoma, one would expect low levels of muscle activation in the absence of local stimuli. We measured a number of respiratory variables, including genioglossus activation under both nasal and tracheal stomal breathing conditions, in five patients. In all five patients there was a significant and substantial decrease in both peak phasic (100 ± 0 to 53.4 ± 9.2 arbitrary units [mean ± SEM], $p < 0.01$) and tonic genioglossus activation (36.3 ± 5.3 to 20.7 ± 3.9 arbitrary units, $p < 0.05$) during stomal breathing as compared with nasal breathing. We conclude that local upper airway respiratory stimuli, possibly negative pressure, are important in mediating the increased pharyngeal dilator muscle activation seen in sleep apnea patients during wakefulness.

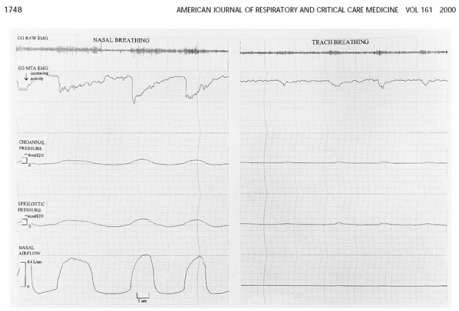

Figure 1. Both the raw and MTA GG EMG from one patient fell during tracheal stomal (trach) breathing. Note that both epiglottic pressure and inspiratory flow were substantially attenuated following the transition from nasal to tracheal breathing.

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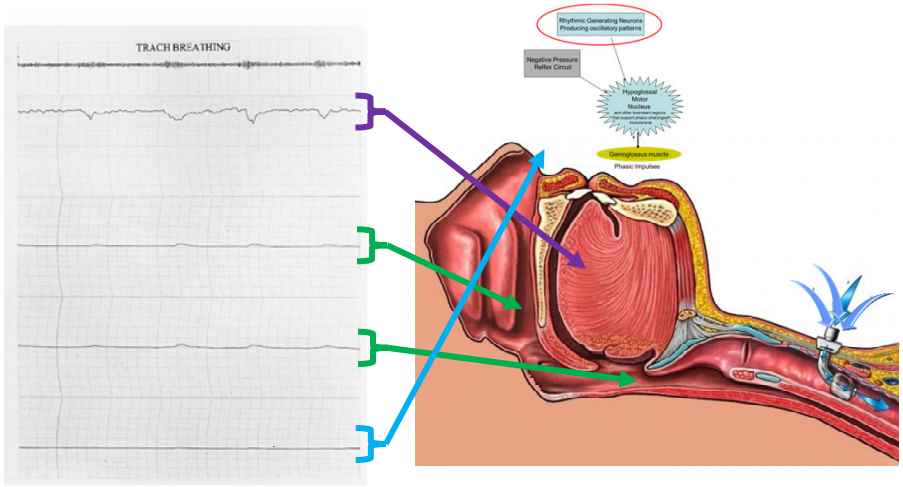
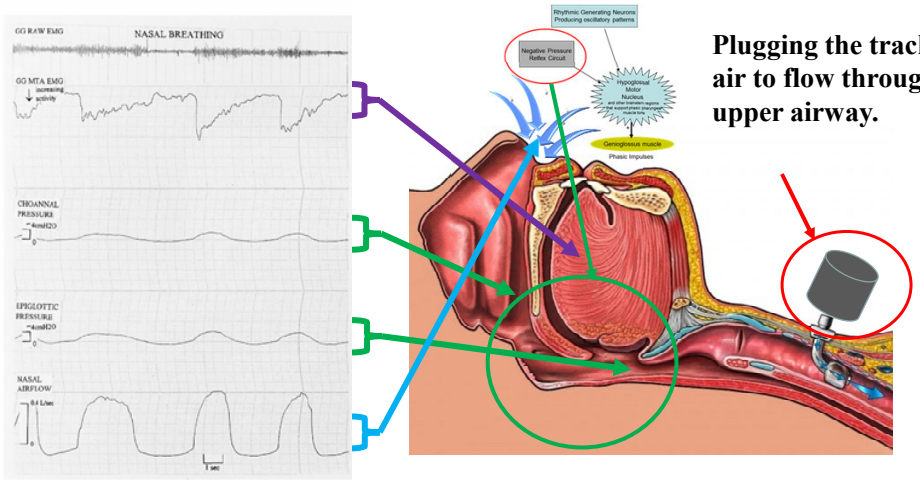


63

Increased phasic tongue (genioglossus) muscle occurs when there is flow generated negative pressure in the upper airway.



Plugging the trach requires air to flow through the upper airway.

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Trach Closed **Trach Open**

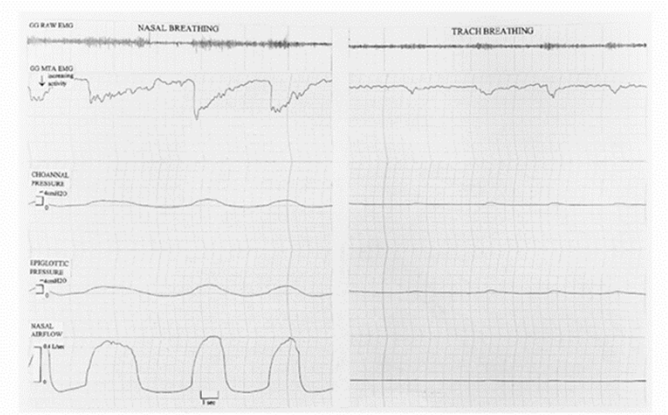
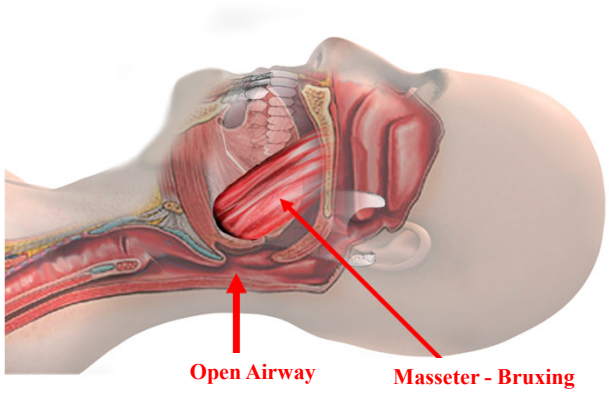


Figure 1. Both the raw and MTA GG EMG from one patient fell during tracheal stomal (trach) breathing. Note that both epiglottic pressure and inspiratory flow were substantially attenuated following the transition from nasal to tracheal breathing.

65

Increased Masseter activity opens the airway by putting the TMJ into occlusion and holding the genioglossus muscle forward



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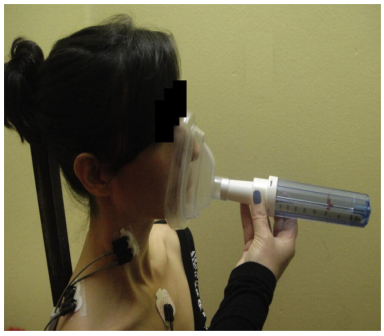

Breathing against increasing resistive loads (through a mask with progressive narrowing of the lumen of the intake hose) resulted in a correlated increase in masseter muscle activity.

Assessments of masseter EMG were obtained with pharyngeal pressures of -10, -20,-30,-40,-50 and -60 cm H2O

The masseter EMG increase progressively, and the increased EMG activity began about 100msec before the increase in negative pressure.

The same pattern was present in the Alae nasi muscles and the chin (genioglossus, geniohyoid, myohyoid and digastric muscles), all of which provide airway dilatation and facilitate respiration. Therefore, it is reasonable to conclude that the Masseter functions in the same fashion as other phasic Upper Airway dilators.

Hollowell, Suratt, American Physiological Society 1989

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Activation of masseter muscles with inspiratory resistance loading

D. E. Hollowell and P. M. Suratt

Abstract

Closure of the jaw exerts traction on muscles that insert on the hyoid bone and that may stabilize or expand the pharyngeal airway. We postulated that the masseter muscles, which close the jaw, would be activated when the patency of the pharyngeal airway is threatened. We therefore measured electromyographic activation of the masseters during inspiratory resistance loading and compared it with activation of chin muscles and alae nasi in 10 normal subjects. We observed no masseter activation during quiet unloaded breathing, but as pharyngeal pressure became lower there was a significant increase in masseter activation in all subjects. The change in masseter activation relative to pharyngeal pressure was similar to that of chin muscles and alae nasi. Activation of the masseter preceded the fall in pharyngeal pressure as also occurred in the chin muscles and alae nasi. We conclude that the masseters are activated by inspiratory resistance loading and have respiratory activity similar to pharyngeal airway muscles.

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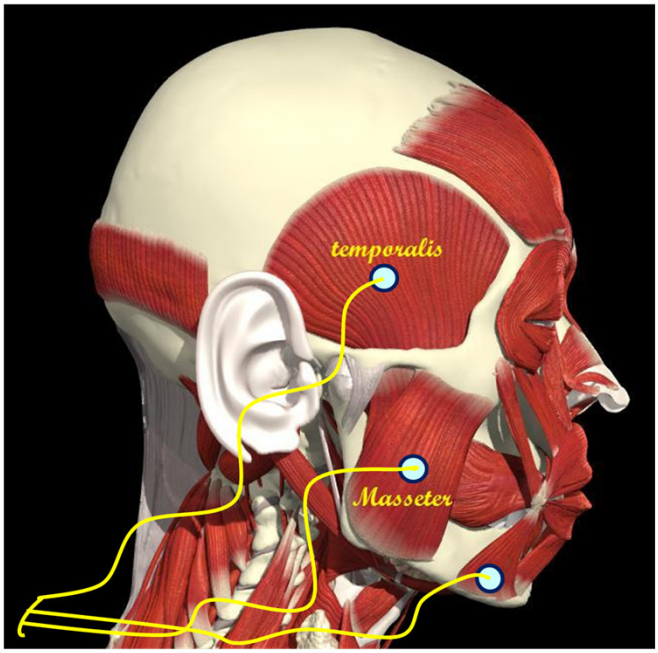
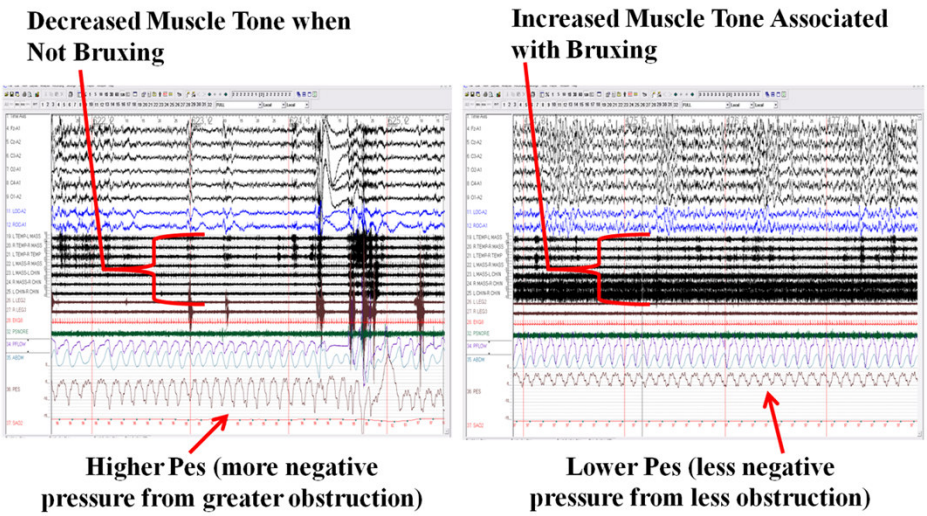


70

Muscle Tone Inversely Related to Upper Airway Obstruction



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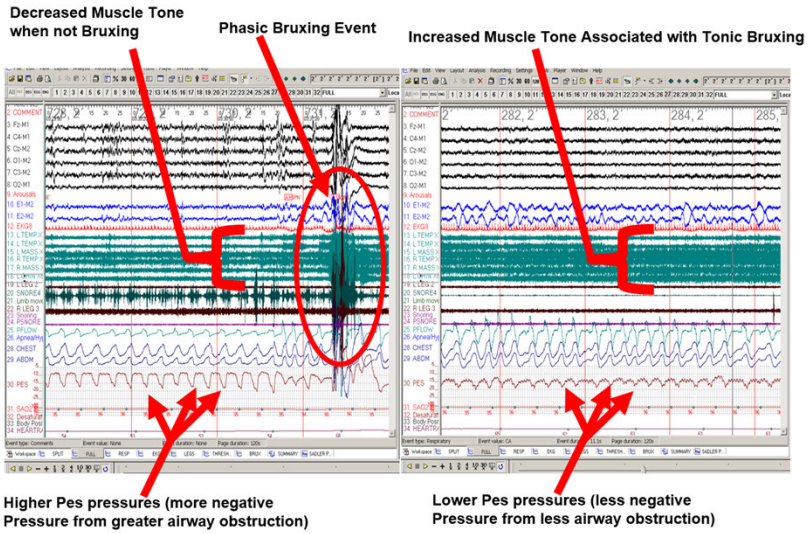
Muscle Tone Inversely Related to Upper Airway Obstruction



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Obesity

body mass index (BMI) : >30 kg/m^2

BMI FORMULA

USA **BMI=** $703 \times \frac{\text{weight (lb)}}{\text{height}^2\ (\text{in}^2)}$

METRIC **BMI=** $\frac{\text{weight (kg)}}{\text{height}^2\ (\text{m}^2)}$

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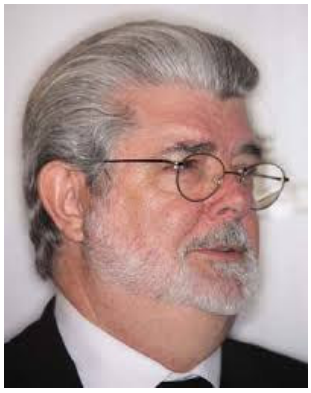
Neck Circumference

An enlarged neck circumference highly associated with OSA
men: >43 cm [17 in];
women: >37 cm [15 in])



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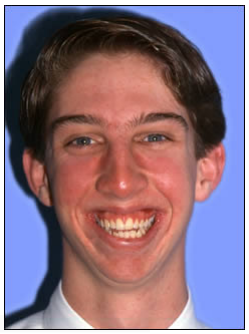
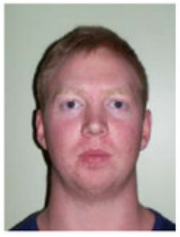
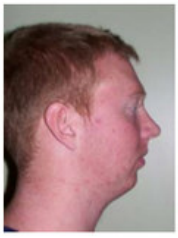
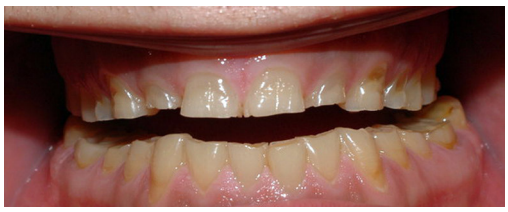
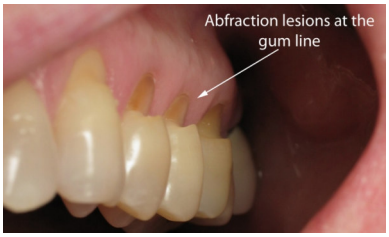
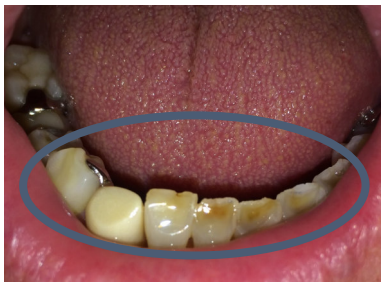
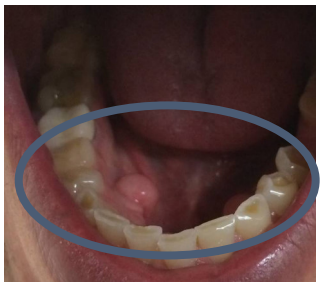
OVERBITE
Retrognathia or Micrognathia

75

Some of the signs of Bruxism at the time of presentation to the sleep center (This should be part of the Neurologic Exam)

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SCIENTIFIC INVESTIGATIONS

JCSM
Journal of Clinical Sleep Medicine
pii: jc-00375-14
http://dx.doi.org/10.5664/jcsm.4602

Frequency of Obstructive Sleep Apnea Syndrome in Dental Patients with Tooth Wear

Joaquín Durán-Cantolla, PhD[1,2,3,4,5]; Mohammad Hamdan Alkhraisat, PhD[5]; Cristina Martínez-Null, BSc[1,2,3,4]; Jose Javier Aguirre, MD[5]; Elena Rubio Guinea, DDS[5]; Eduardo Anitua, PhD[1,5]

[1]Sleep Disorders Unit, Clínica Eduardo Anitua, Vitoria, Spain; [2]Sleep Disorders Unit, Hospital Universitario Araba, Vitoria, Spain; [3]Research Service, BioAraba Project, Hospital Universitario Araba, Vitoria, Spain; [4]Centro de Investigación Biomédica en Red Enfermedades Respiratorias, (CIBERES), ISCIII, Madrid, Spain; [5]Eduardo Anitua Foundation, Vitoria, Spain; [6]Faculty of Medicine, University of País Vasco UPV/EHU, Biscay, Spain

Study Objectives: To estimate the frequency of obstructive sleep apnea syndrome (OSAS) in dental patients with tooth wear, and to assess the role of dentists in the identification of patients at risk of OSAS.

Methods: Dental patients with tooth wear and treated with occlusal splint were prospectively recruited to perform sleep study. The severity of tooth wear was established by the treating dentist before patient referral to sleep disorders unit. Sleep questionnaires, anthropometric measurements, and validated respiratory polygraphy were performed.

Results: All patients with dental wear were offered a sleepiness analysis. Of 31 recruited patients, 30 (77% males) participated in this study. Patients' mean age was 58.5 ± 10.7 years (range: 35–90 years) and the body mass index was 27.9 ± 3.4 kg/m^2. Tooth wear was mild in 13 patients, moderate in 8 and severe in 9. The mean apnea-hypopnea index (AHI) was 32.4 ± 24.9. AHI < 5 was reported in 2 patients, AHI of 5–29 in 17, and AHI ≥ 30 in 11. A statistically significant association was found between AHI severity and tooth wear severity (Spearman R = 0.505; p = 0.004).

Conclusions: Tooth wear could be a tool to identify those patients at risk of having OSAS. This highlights the importance of dental professionals to identify and refer patients with OSAS.

Keywords: apnea-hypopnea index, AHI, obstructive sleep apnea, bruxism, tooth wear

Citation: Durán-Cantolla J, Alkhraisat MH, Martínez-Null C, Aguirre JJ, Guinea ER, Anitua E. Frequency of obstructive sleep apnea syndrome in dental patients with tooth wear. *J Clin Sleep Med* 2015;11(4):445–450.

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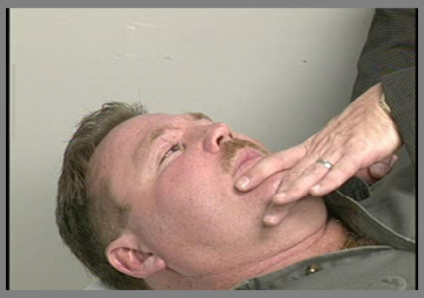
Examination of the Upper Airway in a Dynamic Fashion Chin-Press / Chin-Press Tongue-Curl Maneuver



Simmons, J.H., Mann, C.A., Gulliminault, C. **"The Chin Press/Tongue Curl Maneuver as Part of the Physical Exam on Patients Suspected of Sleep Related Obstructive Respirations."** Somnologie (1997) 1 (Suppl. 2), 1-56.

Simmons, J.H., Mann, C., Lebby, R. **The Chin Press Maneuver: A Method of Evaluating the Upper Airway During the Physical Exams,** Sleep Research Vol 26, 1997.

Simmons J., Mann C., Zuberi N., Lebby R., and Shellock F., Abstract - **"Evaluating the Upper Airway During the Physical Exam with the Chin Press Maneuver".** Sleep Research, 1996 Vol. 24 p. 269.

Meskill, G., Kincheloe, K., Simmons, J, Meskill, S. Abstract - **Simmons Chin Press and Tongue Curl (SCPTC) Maneuver is a Reproducible Objective Physical Exam Finding to Screen for Obstructive Sleep Apnea (OSA) Associated with Cardiovascular Morbidities and All-Cause Mortality** Sleep 2018 Vol. 41, 2018 p A187

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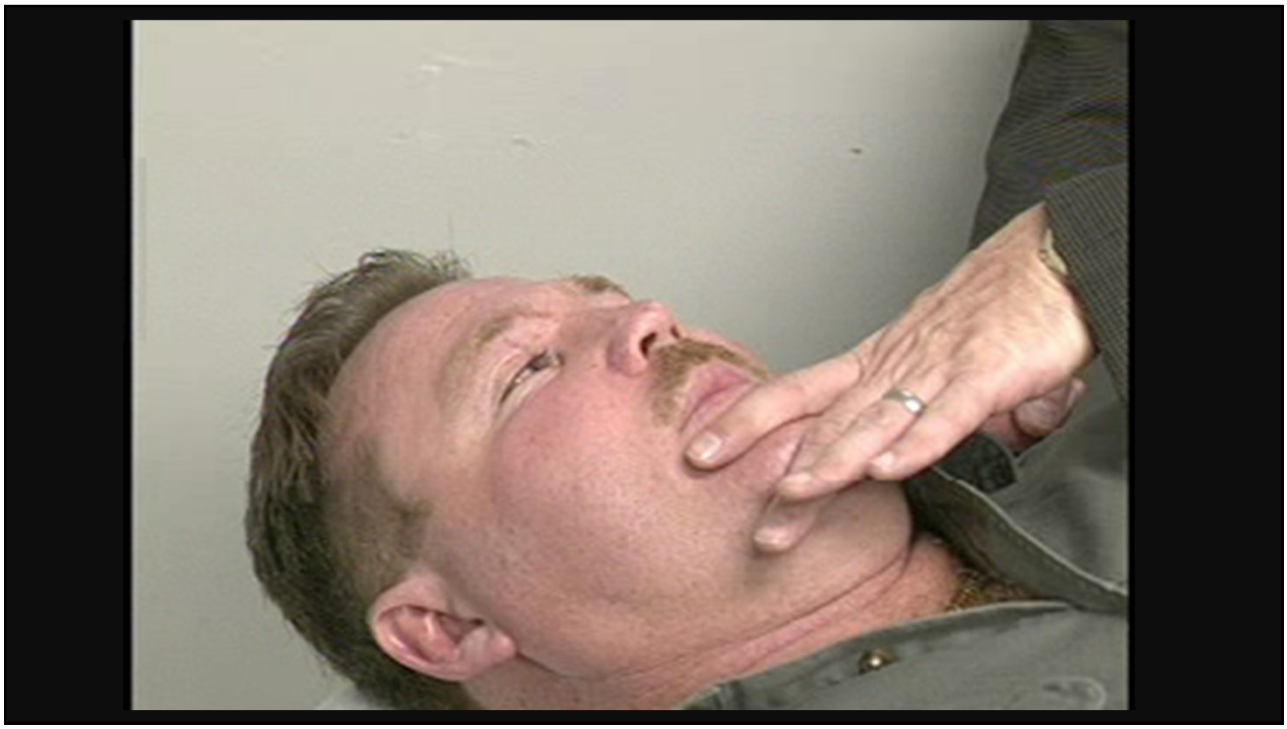

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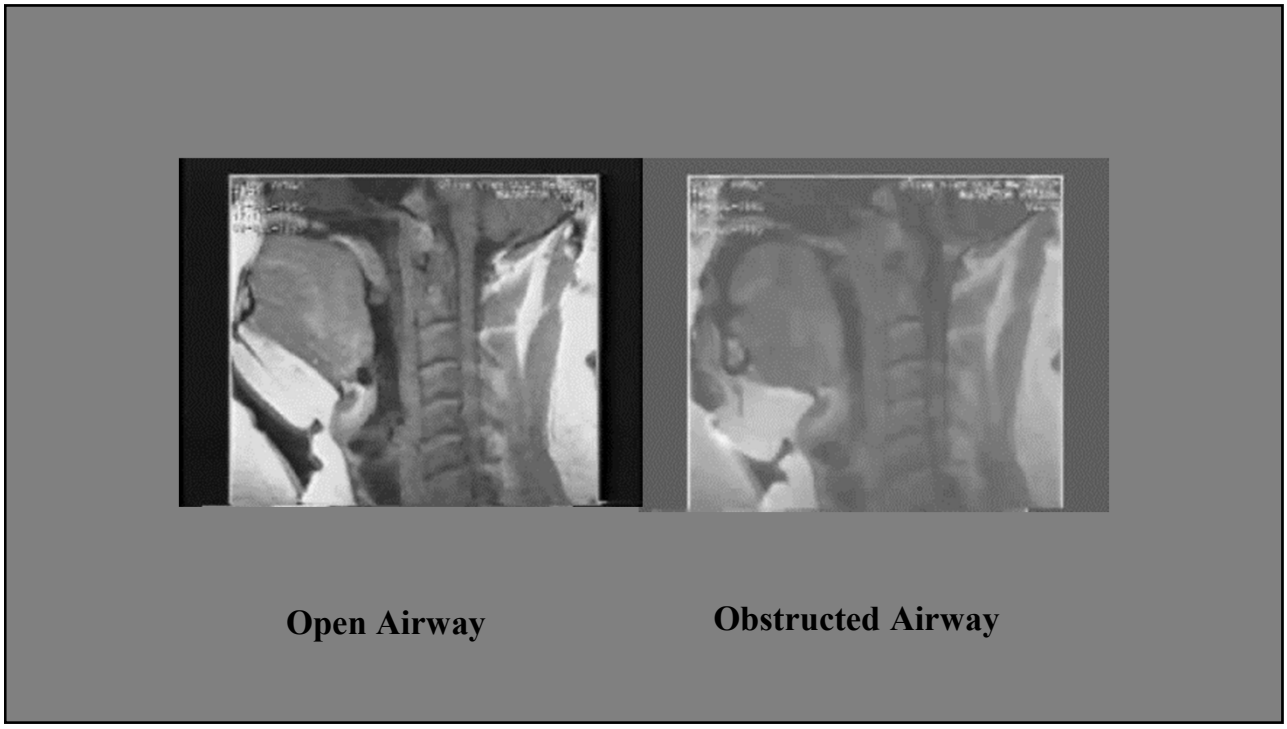
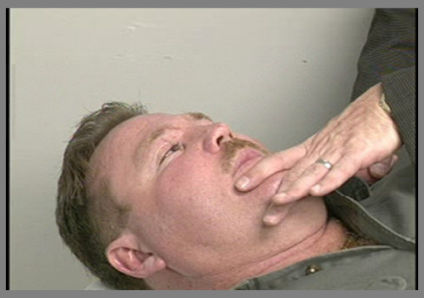


80

Examination of the Upper Airway in a Dynamic Fashion
Chin-Press / Chin-Press Tongue-Curl Maneuver



Simmons, J.H., Mann, C.A., Gulliminault, C. **"The Chin Press/Tongue Curl Maneuver as Part of the Physical Exam on Patients Suspected of Sleep Related Obstructive Respirations."** Somnologie (1997) 1 (Suppl. 2), 1-56.

Simmons, J.H., Mann, C., Lebby, R. **The Chin Press Maneuver: A Method of Evaluating the Upper Airway During the Physical Exams,** Sleep Research Vol 26, 1997.

Simmons J., Mann C., Zuberi N., Lebby R., and Shellock F., Abstract - **"Evaluating the Upper Airway During the Physical Exam with the Chin Press Maneuver".** Sleep Research, 1996 Vol. 24 p. 269.

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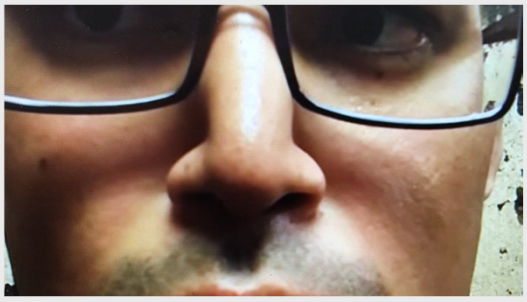
Examination of the Nasal Airway

- Collapse of the Nasal Bridge from the negative pressure



- Less than 10% of OSA patients resolve with Tx of the nasal anatomy alone
- Awakening with Nasal Congestion is a sign of obstructive respirations during sleep

82

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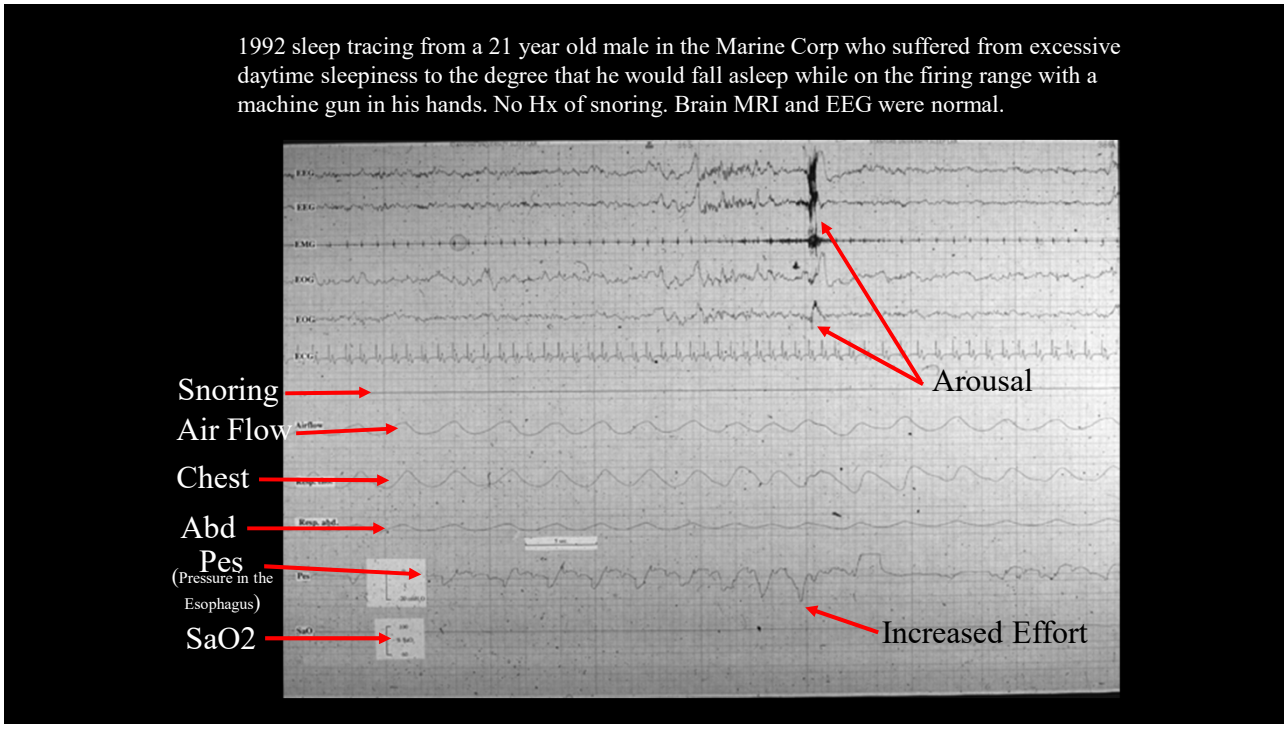
1992 sleep tracing from a 21 year old male in the Marine Corp who suffered from excessive daytime sleepiness to the degree that he would fall asleep while on the firing range with a machine gun in his hands. No Hx of snoring. Brain MRI and EEG were normal.



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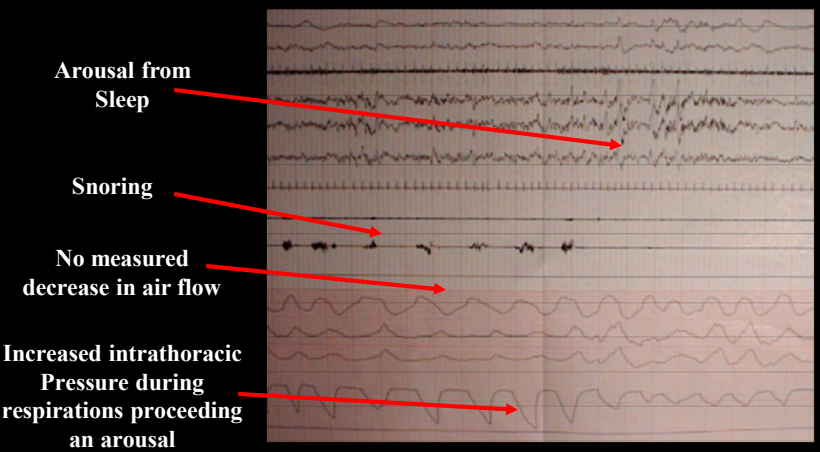
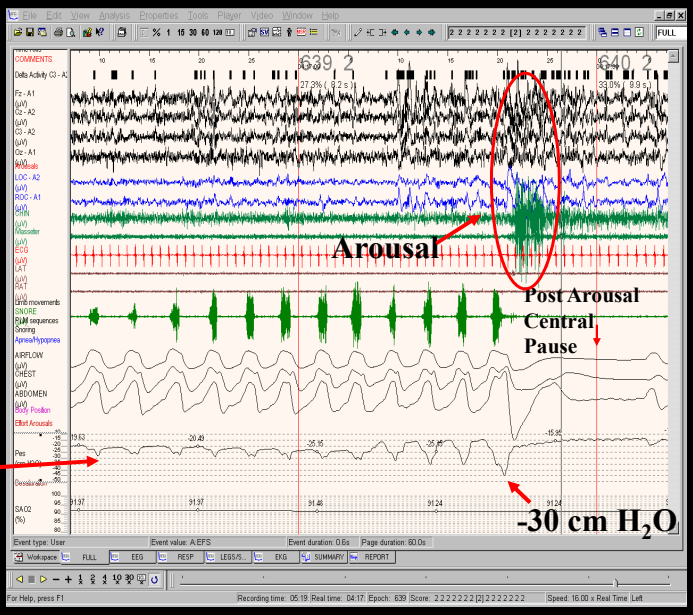
Snoring Arousal Consistent with the Upper Airway Resistance Syndrome



85

RERA = Respiraotory Effort Related Arousal



86

Sleep, 15:S13–S16

From Obstructive Sleep Apnea Syndrome to Upper Airway Resistance Syndrome: Consistency of Daytime Sleepiness

Christian Guilleminault, Riccardo Stoohs, Alex Clerk, Jerald Simmons and Michael Labanowski

Stanford Sleep Research Center, Palo Alto, California, U.S.A.

Summary: Some patients with excessive daytime sleepiness who do not present the features of obstructive sleep apnea syndrome (OSAS) present a sleep fragmentation due to transient alpha EEG arousals lasting between three and 14 seconds. These transient EEG arousals are related to an abnormal amount of breathing effort, indicated by peak inspiratory esophageal pressure (Pes) nadir. In the studied population, these increased efforts were associated with snoring. Usage of nasal CPAP, titrated on Pes nadir values, for several weeks eliminated subjective daytime sleepiness and improved Multiple Sleep Latency Test scores from baseline evaluations. Patients suspected of CNS hypersomnia should be asked about continuous snoring, and their clinical evaluation should include a good review of maxillo-mandibular and upper airway anatomy.

Sleep, 1992; Vol 15, No 6, Supp. pp S13-S16.

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Silent Upper Airway Resistance Syndrome*

Prevalence in a Mixed Military Population

David A. Kristo, MD, FCCP; Christopher J. Lettieri, MD; Teotimo Andrada, MS; Yvonne Taylor, DrPH; and Arn H. Eliasson, MD, FCCP

Study objectives: **The upper airway resistance syndrome (UARS) is a recently described form of sleep-disordered breathing in which transient increases in upper airway resistance result in repetitive EEG arousals. UARS is not associated with apnea or diminished airflow, although snoring and excessive daytime somnolence (EDS) are common. This report describes a subset of patients with UARS diagnosed by polysomnography who do not manifest snoring, which we define as *silent upper airway resistance syndrome* (SUARS).**

Design: **A retrospective review of all polysomnographies performed at our sleep disorders center during 2000.**

Setting: **Sleep disorders center of a large, academic, military hospital.**

Patients: **Our center serves military personnel, military retirees, and their dependent families.**

Interventions: **Esophageal manometry during polysomnography was routinely performed on patients with hypersomnolence (Epworth sleepiness scale > 10) who demonstrated a total arousal index ≥ 10/h and a respiratory disturbance index of < 5/h on prior polysomnography. UARS was definitely diagnosed in patients who demonstrated repetitive increased upper airway resistance (IUAR) associated with brief EEG arousals followed by normalization of esophageal pressure (Pes). IUAR was defined by a pattern of crescendo negative inspiratory Pes of ≤ − 12 cm H_2O.**

Results: **During calendar year 2000, we performed 724 polysomnographies in 527 patients. Obstructive sleep apnea was diagnosed in 383 patients (72.6%), and 44 patients (8.4%) were found to have UARS. In four patients with UARS (0.8% of total and 9.1% of UARS), snoring was not reported by history or observed during polysomnography, and SUARS was ultimately diagnosed.**

Conclusions: **UARS may occur in the absence of clinically significant snoring and may be an occult cause of EDS. We report a prevalence of SUARS of 9% among UARS patients and nearly 1% of all patients studied for hypersomnolence by polysomnography.**

(CHEST 2005; 127:1654–1657)

Key words: esophageal manometry; excessive daytime sleepiness; hypersomnolence; increased upper airway resistance; obstructive sleep apnea; sleep-disordered breathing; upper airway resistance syndrome

Abbreviations: BMI = body mass index; EDS = excessive daytime sleepiness; ESS = Epworth sleepiness scale; OSAS = obstructive sleep apnea syndrome; Pes = esophageal pressure; RDI = respiratory disturbance index; RERA = respiratory effort-related arousal; SUARS = silent upper airway resistance syndrome; TAI = total arousal index; UARS = upper airway resistance syndrome

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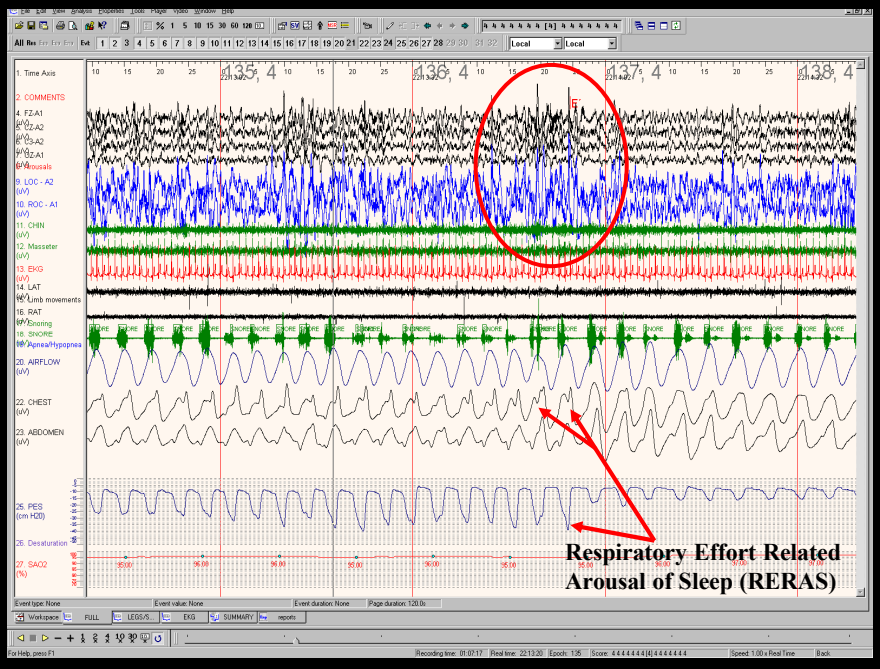


90

Types of Respiratory Disturbances During Sleep

- Obstructive Apnea
- Central Apneas
- Obstructive Hypopneas
- Central Hypopneas
- Respiratory Effort Related Arousals (RERA) (snore arousals)

Apnea Hypopnea Index (AHI) = Apneas + Hypopneas per hour sleep

Respiratory Disturbance Index (RDI) = Apneas + Hypopneas + RERAs per hour sleep

91

Hypopnea Scoring Rules implemented by the AASM in 2013

Two different scoring rules currently exist, causing confusion.

Rule 1A (Recommended rule)
Score a respiratory event as a hypopnea if ALL of the following criteria are met:

a. The peak signal excursions drop by ≥30% of pre-event baseline
b. The duration of the ≥30% drop in signal excursion is ≥10 seconds.
c. There is a ≥3% oxygen desaturation from pre-event baseline or the event is associated with an arousal. Note: No SaO2 desaturation required.

Rule 1B (Acceptable but not the recommended rule) Unfortunately many sleep facilities use this rule. Medicare only recognizes this rule.

Score a respiratory event as a hypopnea if
a. The peak signal excursions drop by ≥30% of pre-event baseline
b. The duration of the ≥30% drop in signal excursion is ≥10 seconds.
c. There is a ≥ 4% oxygen desaturation from pre-event baseline Note: No mention of arousals. Arousals are not part of 1B Hypopneas.

92

Pes catheter - Mylar feeding tube - typically a 6 French

93

Pes Placement

94

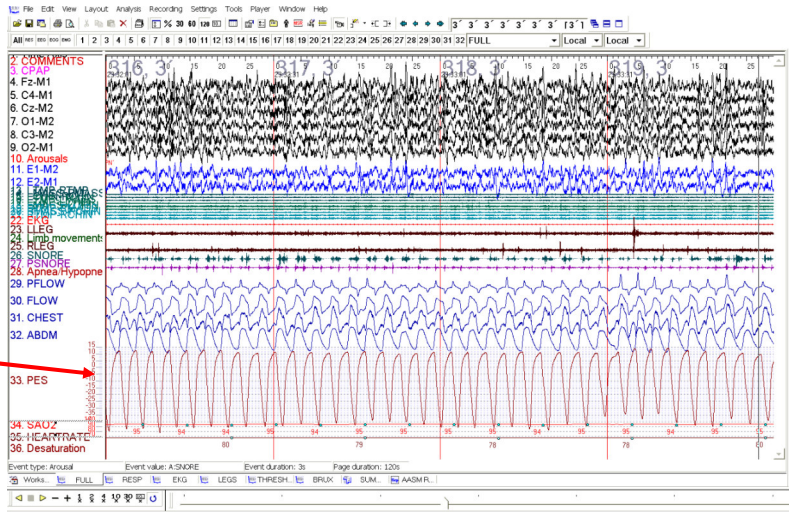
Patients with the Upper Airway Resistance Syndrome do not usually present with physical features typical of severe Obstructive Sleep Apnea Patients. This patient had a previous diagnosis of Chronic Fatigue Syndrome and was found to have the UARS

95

Prolonged periods of high intra-thoracic pressure in a patients with intermittent A-fib

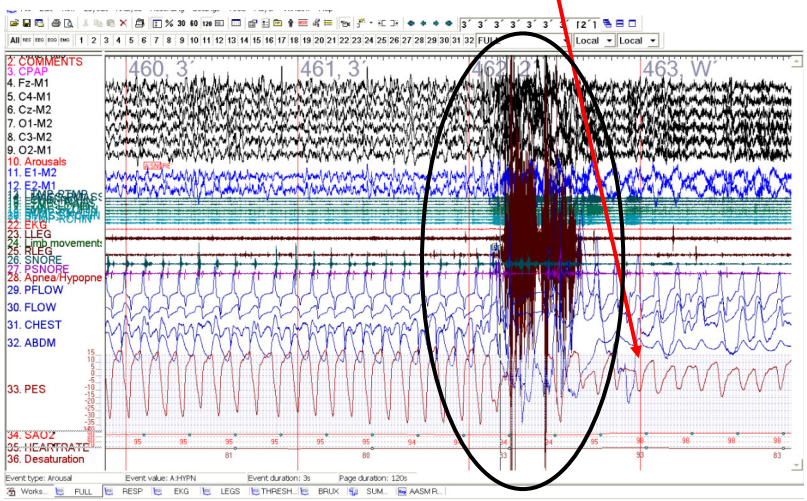
Greater Than -50 cm H2O With no pauses in breathing.

SaO2 is normal



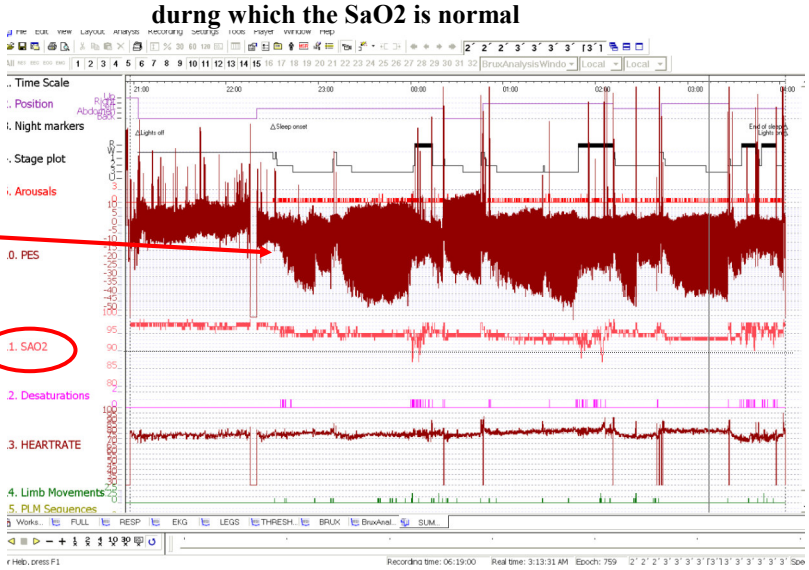
96

Obstructive breathing improves with the arousal



97

Hypnogram showing long periods of increased respiratory effort during sleep durng which the SaO2 is normal



Large Pes Pulls

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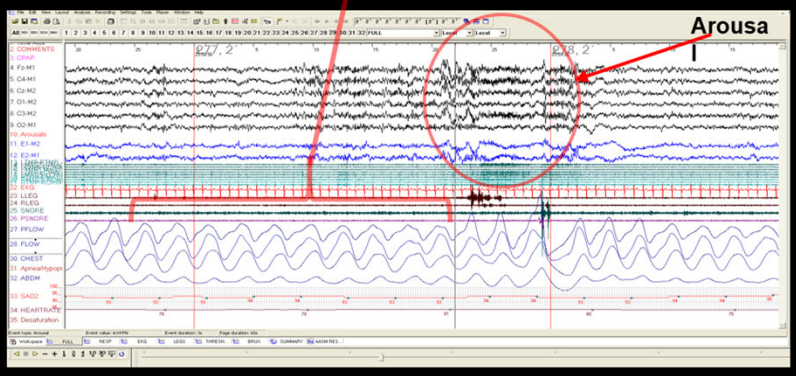
The Full Spectrum of Obstructive Breathing During Sleep.

Snoring **Gasping** **Pauses in breathing**

Normal Breathing → Respiratory Effort Related Arousals of Sleep (RERAS) → Hypopneas with normal SaO2 (Hypopnea Rule 1A) → Hypopneas with SaO2 desaturations (Hypopnea Rule 1B) → **Complete Obstructive Apnea**

99

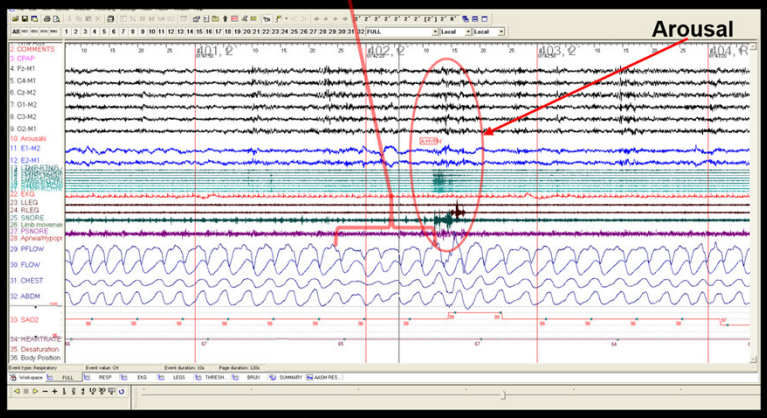
Hypopnea noted by >10 sec decrease in air flow leading to an arousal but SaO2 maintained at **93%-94%**



1 minute display screen

100

Hypopnea noted by a decrease in air flow for more than 10 sec ending with an arousal with the SaO2 maintained between **98% to 99%**



2 minute display screen

101

Clinical Case

Middle aged female with a Hx of morning headaches, morning gastric distress, sleep bruxism, excessive daytime sleepiness, morning nasal congestion.

Had a NPSG study done at a sleep center

102

RESPIRATORY ANALYSIS:

Nicole GoldFarb demonstrated minimal evidence of obstructive sleep apnea, with an Apnea Hypopnea Index of 0.0 events per hour overall. Oxygen saturation nadir was normal at 95%.

No snoring was noted.

BODY POSITION ANALYSIS:

Position	Total Duration (min)	Sleep Dur. (min)	REM (%)	CA	OA	MA	HYP	RERAs	RDI	AHI
L	77.7	73.5	58.6	0	0	0	0	0	0.0	0.0
S	307.5	238.6	9.8	0	0	0	0	0	0.0	0.0
R	62.8	49.4	0.0	0	0	0	0	0	0.0	0.0

Position	Duration (min)	Sleep Dur. (min)	REM (%)	CA (#)	OA (#)	MA (#)	HYP (#)	RERA (#)	RDI (#/h)	AHI (#/h)
Supine	307.5	238.6	9.8	0	0	0	0	0	0.0	0.0
Non-Supine	140.50	122.90	60.26	0.00	0.00	0.00	0.00	0	0.00	0.00

RESPIRATORY EVENT INDEX SUMMARY (TOTAL SLEEP TIME):

	REM #/h (REM)	NREM #/h (NREM)	TST #/h (sleep)
AHI*	0.0	0.0	0.0
RDI**	0.0	0.0	0.0

*AHI = # apneas + # hypopneas/TST **RDI= #apneas + #hypopneas + # of RERAS which cause arousal/TST

AROUSAL SUMMARY:

	AHI arousals	RERA arousals	RDI arousal	PLM arousals	Snore arousals	Spont. arousals	Total
Arousal(s) Total (#	0	0	0	5	0	53	58
Arousal Index (#/h	0.0	0.0	0.0	0.8	0.0	8.8	9.6

[redacted]
Diplomate, Am. Bd. of Sleep Medicine
Performance Sleep Centers

Nicole Goldfarb 4

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Recording Code: NG091019-1854_AUSIPatient Name: Goldfarb, Nicole
Recording Date: 9/10/2019 Birth date: 1/24/1979 MedRec.:

Respiratory Effort Related Arousal

The change in the flow signal could be from movement triggered by the arousal.

Nasal Pressure signal does not clearly show flow limitation.

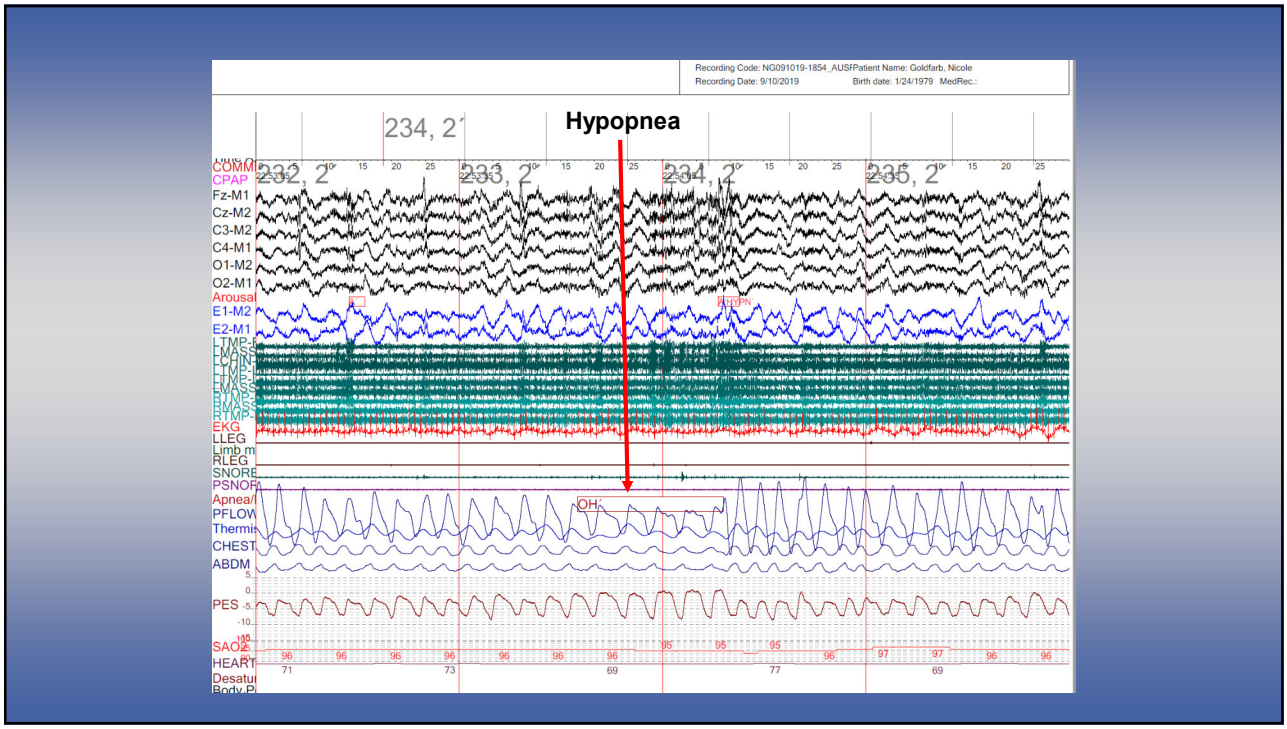
Increased Pes

Normal Pes

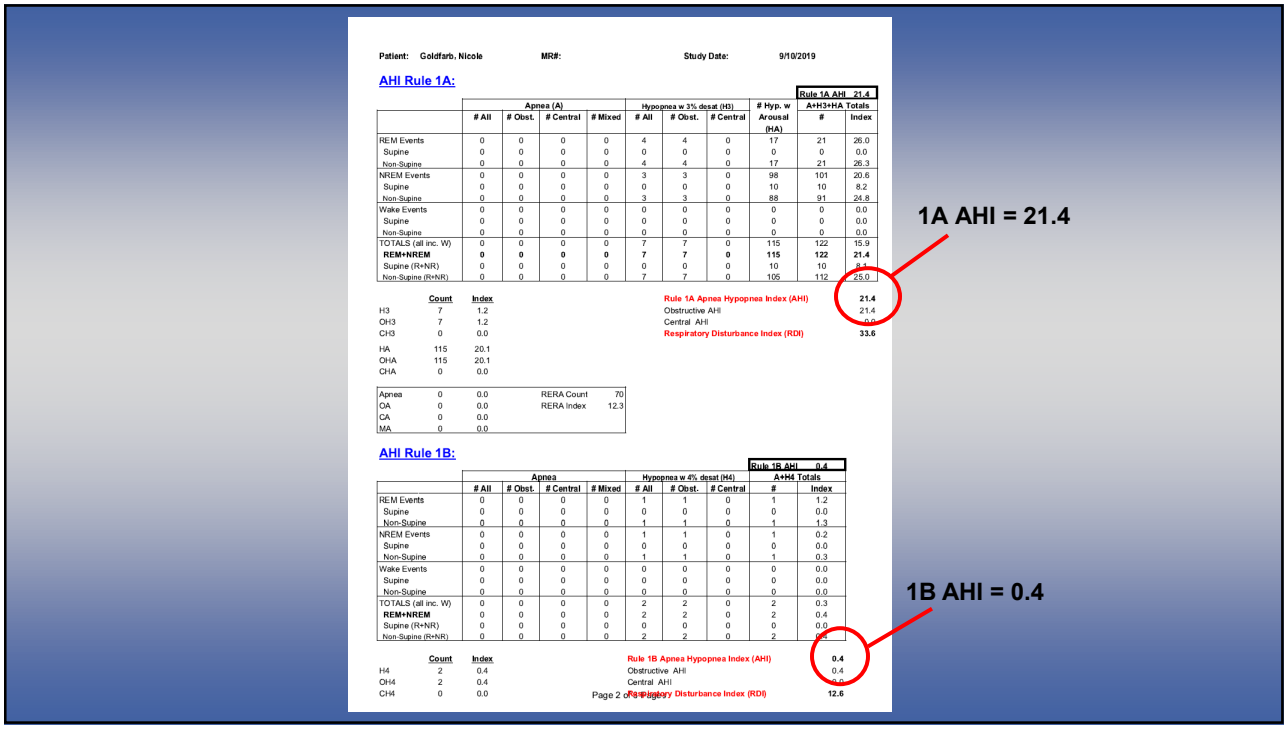
COMM
CPAP
Fz-M1
Cz-M2
C3-M2
C4-M1
O1-M2
O2-M1
Arousal
E1-M2
E2-M1
LTMP-
LMAS
LCHIN
LTMP-
LTMP-
LMAS
RTMP-
RMAS
RTMP-
EKG
LLEG
Limb M
RLEG
SNORE
PSNOR
Apnea/
PFLOW
Thermis
CHEST
ABDM
PES
SAO2
HEART
Desatu
Respire
Body P

RERA

104



105



Patient: Goldfarb, Nicole MR#: Study Date: 9/10/2019

AHI Rule 1A:

Rule 1A AHI 21.4

	Apnea (A)				Hypopnea w 3% desat (H3)			# Hyp. w Arousal (HA)	A+H3+HA Totals	
	# All	# Obst.	# Central	# Mixed	# All	# Obst.	# Central		#	Index
REM Events	0	0	0	0	4	4	0	17	21	26.0
Supine	0	0	0	0	0	0	0	0	0	0.0
Non-Supine	0	0	0	0	4	4	0	17	21	26.3
NREM Events	0	0	0	0	3	3	0	98	101	20.6
Supine	0	0	0	0	0	0	0	10	10	8.2
Non-Supine	0	0	0	0	3	3	0	88	91	24.8
Wake Events	0	0	0	0	0	0	0	0	0	0.0
Supine	0	0	0	0	0	0	0	0	0	0.0
Non-Supine	0	0	0	0	0	0	0	0	0	0.0
TOTALS (all inc. W)	0	0	0	0	7	7	0	115	122	15.9
REM+NREM	**0**	**0**	**0**	**0**	**7**	**7**	**0**	**115**	**122**	**21.4**
Supine (R+NR)	0	0	0	0	0	0	0	10	10	8.1
Non-Supine (R+NR)	0	0	0	0	7	7	0	105	112	25.0

	Count	Index
H3	7	1.2
OH3	7	1.2
CH3	0	0.0
HA	115	20.1
OHA	115	20.1
CHA	0	0.0

Apnea	0	0.0	RERA Count 70
OA	0	0.0	RERA Index 12.3
CA	0	0.0	
MA	0	0.0	

Rule 1A Apnea Hypopnea Index (AHI) 21.4
Obstructive AHI 21.4
Central AHI 0.0
Respiratory Disturbance Index (RDI) 33.6

1A AHI = 21.4

AHI Rule 1B:

Rule 1B AHI 0.4

	Apnea				Hypopnea w 4% desat (H4)			A+H4 Totals	
	# All	# Obst.	# Central	# Mixed	# All	# Obst.	# Central	#	Index
REM Events	0	0	0	0	1	1	0	1	1.2
Supine	0	0	0	0	0	0	0	0	0.0
Non-Supine	0	0	0	0	1	1	0	1	1.3
NREM Events	0	0	0	0	1	1	0	1	0.2
Supine	0	0	0	0	0	0	0	0	0.0
Non-Supine	0	0	0	0	1	1	0	1	0.3
Wake Events	0	0	0	0	0	0	0	0	0.0
Supine	0	0	0	0	0	0	0	0	0.0
Non-Supine	0	0	0	0	0	0	0	0	0.0
TOTALS (all inc. W)	0	0	0	0	2	2	0	2	0.3
REM+NREM	0	0	0	0	2	2	0	2	0.4
Supine (R+NR)	0	0	0	0	0	0	0	0	0.0
Non-Supine (R+NR)	0	0	0	0	2	2	0	2	0.5

	Count	Index
H4	2	0.4
OH4	2	0.4
CH4	0	0.0

Rule 1B Apnea Hypopnea Index (AHI) 0.4
Obstructive AHI 0.4
Central AHI 0.0
Respiratory Disturbance Index (RDI) 12.6

1B AHI = 0.4

Page 2 of Report

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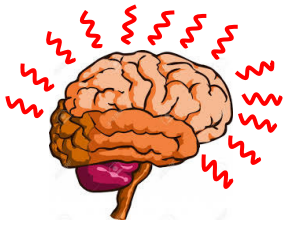
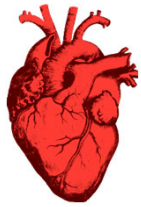
The Full Spectrum of Obstructive Breathing During Sleep.

Snoring Gasping Pauses in breathing

< - Bruxing / Clenching During Sleep - >

Normal Breathing → Respiratory Effort Related Arousals of Sleep (RERAS) → Hypopneas with normal SaO2 (Hypopnea Rule 1A) → Hypopneas with SaO2 desaturations (Hypopnea Rule 1B) → **Complete Obstructive Apnea**





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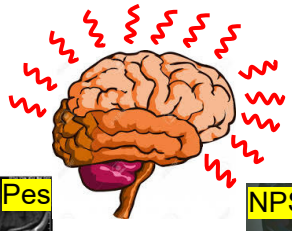
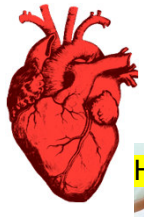
The Full Spectrum of Obstructive Breathing During Sleep.

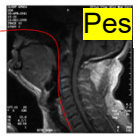
Snoring Gasping Pauses in breathing

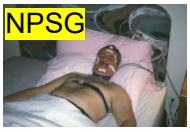
< - Bruxing / Clenching During Sleep - >

Normal Breathing → Respiratory Effort Related Arousals of Sleep (RERAS) → Hypopneas with normal SaO2 (Hypopnea Rule 1A) → Hypopneas with SaO2 desaturations (Hypopnea Rule 1B) → **Complete Obstructive Apnea**









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Home Sleep Apnea Testing (HSAT) Devices

(These are NOT equivalent to in-lab sleep studies)

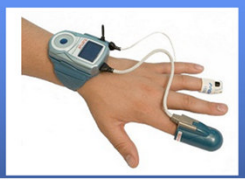
These are only able to reliably identify Moderate to Severe OSA

Apnea Link

Ares

Nasal Flow
Oximtry
Pulse Rate
Snoring
Head position

WatchPAT



- PAT— Peripheral Arterial Tone, which is a physiological signal that changes with autonomic nervous system changes, which can change by respiratory disturbances during sleep.
- Oximetry
- Body Position and Actigraphy (the measurement of body movement while sleeping)
- Heart Rate
- Snoring—loud snoring is a major indicator of sleep apnea.

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CSMA

ResMed

Apnea Link

ApneaLink - Report of

Treating physician

Patient data
First name:
Last Name:
Street:
City, ST, Zip:
Phone:

Recording
Date: 4/16/2022
Start: 1:00 AM .
End: 8:27 AM .
Duration: 7 h 26 min

AHI*

Normal range | Suspected pathological breathing disorder

Result (74)

ODI = 66 / hr

Analysis (Flow evaluation period: 7 h 26 min / SpO2 evaluation period: 7 h 16 min)

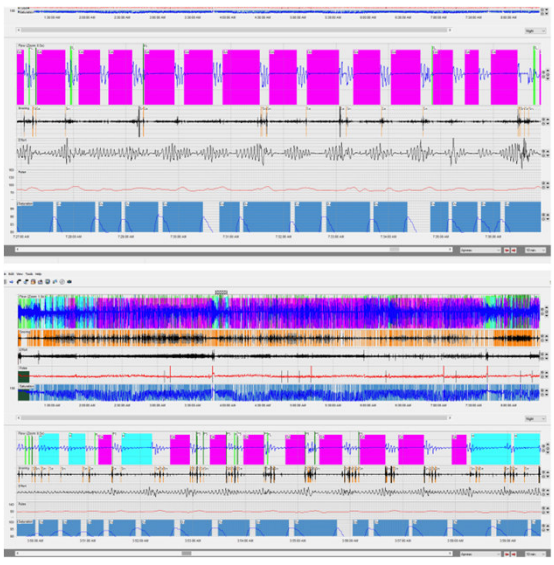
Indices		Normal
AHI*:	73.6	< 5 / h
RI*:	76.2	< 5
Apnea index:	61.9	< 5 / h
UAI:	0	
OAI:	61.9	
CAI:	0	
MAI:	0	
Hypopnea index:	11.7	< 5 / h
% Flow lim. Br. without Sn (FL):	11	< Approx. 60
% Flow lim. Br. with Sn (FS):	11	< Approx. 40
ODI Oxygen Desaturation Index*	66.1	< 5 / h
Average saturation:	83	94% - 98%
Lowest desaturation:	46	-
Lowest saturation:	46	90% - 98%
Baseline Saturation:	95	%
Minimum pulse:	71	> 40 bpm
Maximum pulse:	113	< 90 bpm
Average pulse:	86	bpm
Proportion of probable CS epochs:	0	0%

Analysis status: Edited manually

Result	
Average breaths per minute [bpm]:	5.05
Breaths:	2249
Apneas:	460
Unclassified apneas:	0 (0%)
Obstructive apneas:	460 (100%)
Central apneas:	0 (0%)
Mixed apneas:	0 (0%)
Hypopneas:	87
Flow lim. Br. without Sn (FL):	252
Flow lim. Br. with Sn (FS):	319
Snoring events:	2006
No. of desaturations:	480
Saturation <= 90% :	311 min (71%)
Saturation <= 85% :	235 min (54%)
Saturation <= 80% :	162 min (37%)
Saturation <= 89% :	294 min (67%)
Saturation <= 88% :	278 min (64%)

Great example of when a HSAT result is without question showing OSA

Severe OSA with one event per minute



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Home Sleep Testing Devices

(These are NOT equivalent to in-lab sleep studies)

These are only able to reliably identify Moderate to Severe OSA events

Apnea Link

Heart Rate
Oximetry
Air Flow

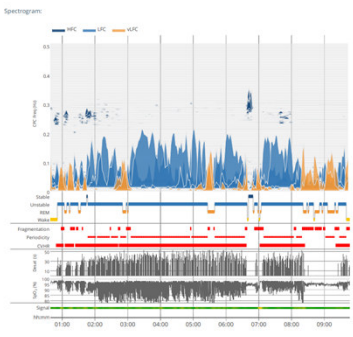
Ring Oximeter with Cardiopulmonary Coupling analysis

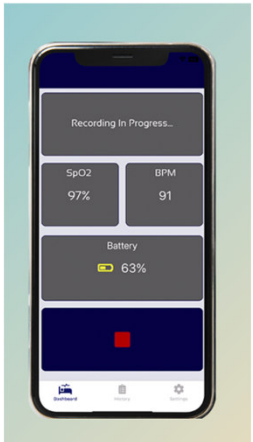
111

Home Sleep Apnea Testing

HSAT can rule in the diagnosis of OSA but can not Rule it out

It can be as easy as wearing an oximeter ring at night with special analysis (CardioPulmonary Coupling)





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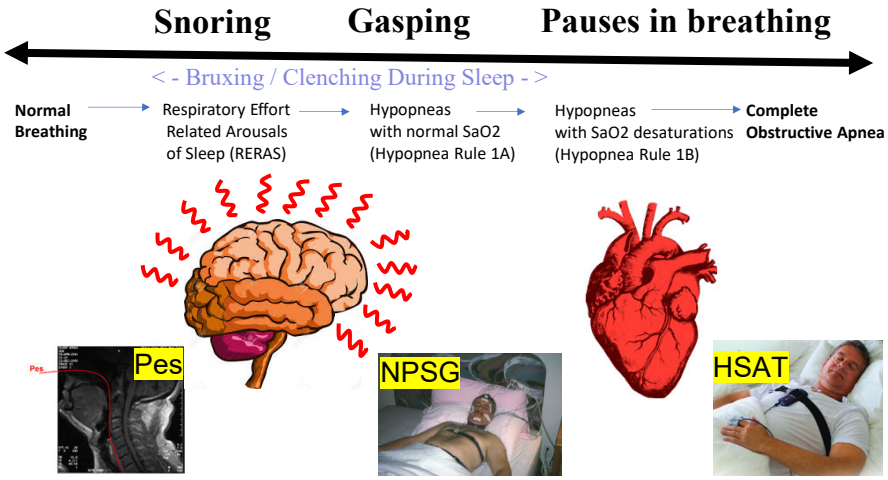
The Full Spectrum of Obstructive Breathing During Sleep.



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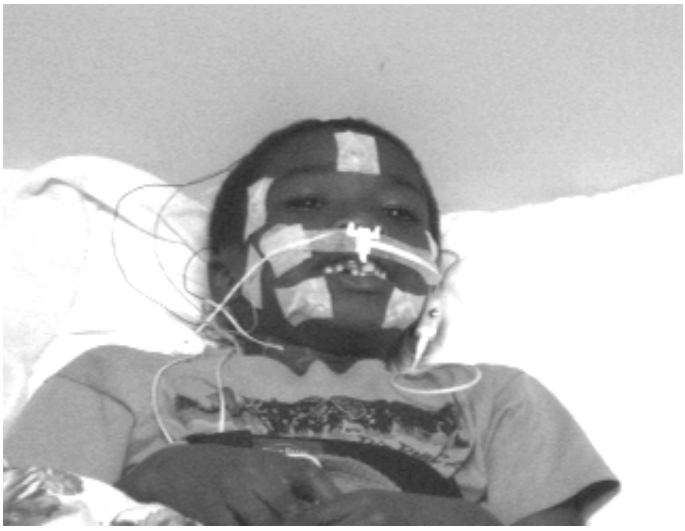
Pediatric Sleep Patient with Pes

Look at his teeth



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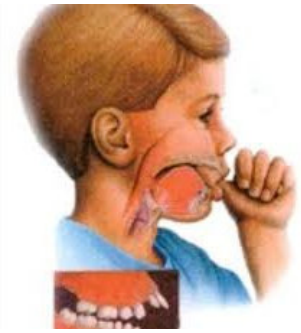
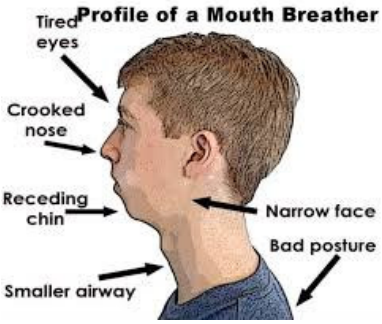
We will cover clues that suggest a child or adolescent may have sleep related breathing problems.
The goal is for early detection





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ADA Children's Airway Initiative

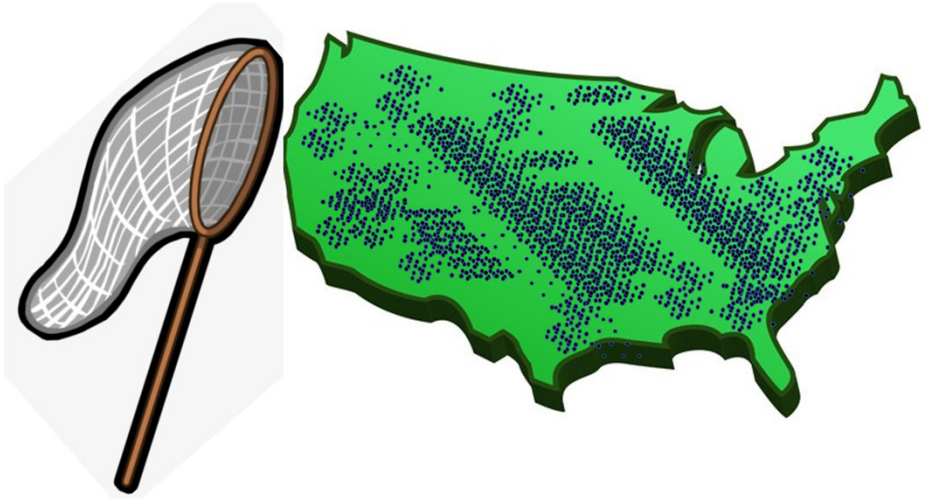
Children's Airway Screener Taskforce

CAST is primarily a dental initiative working to enhance the diagnosis of OSA in children. However, CAST consists of clinicians with a variety professional backgrounds, including several physicians. It MUST be recognized that the CAST position is that the diagnosis of OSA must be established by a physician and treatment by a dentist should be in collaboration with physician.

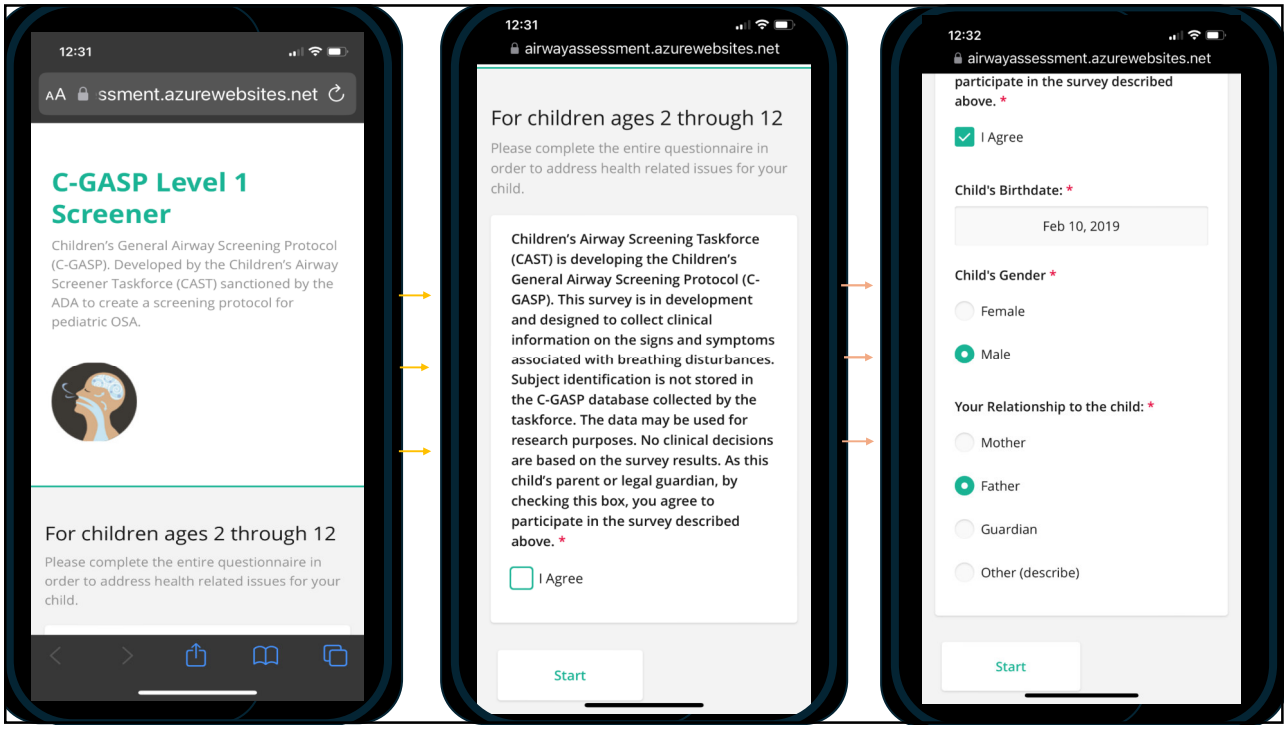
116

We need to a protocol that would act as a large net

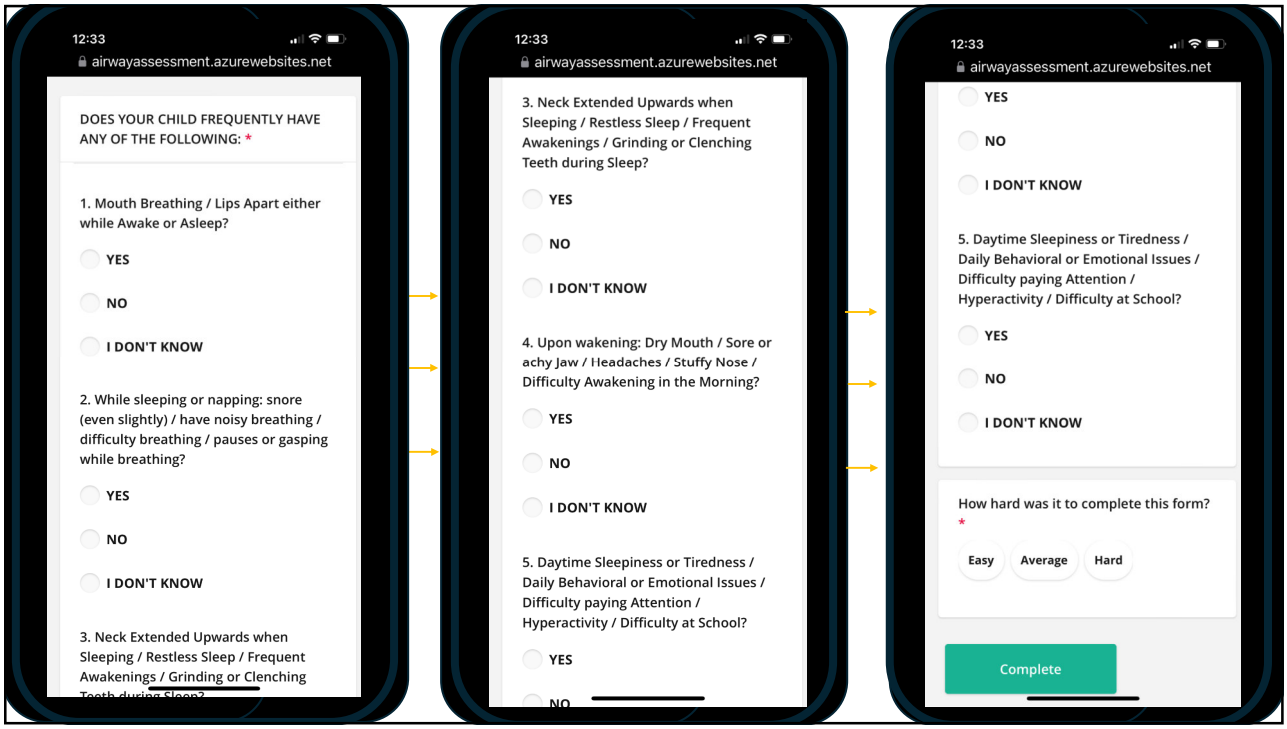
to catch all kids that are at risk of OSA.



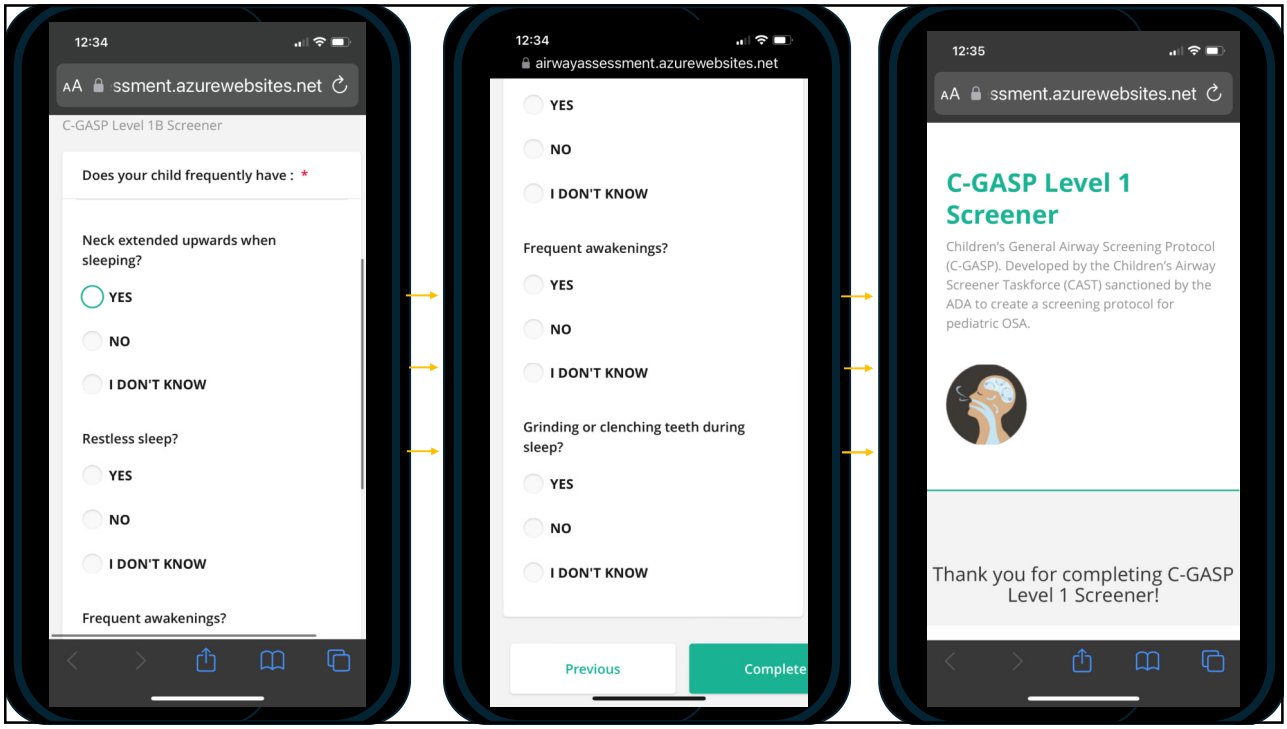
117



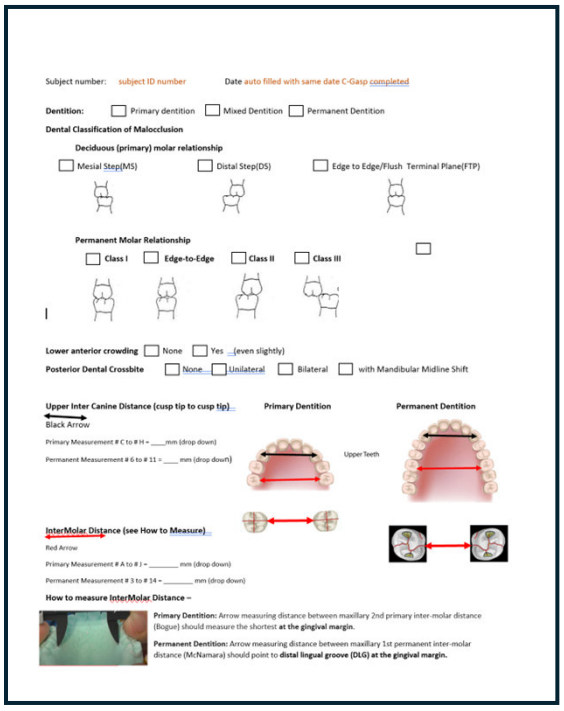
118


12:33
airwayassessment.azurewebsites.net
DOES YOUR CHILD FREQUENTLY HAVE ANY OF THE FOLLOWING: *
1. Mouth Breathing / Lips Apart either while Awake or Asleep?
YES
NO
I DON'T KNOW
2. While sleeping or napping: snore (even slightly) / have noisy breathing / difficulty breathing / pauses or gasping while breathing?
YES
NO
I DON'T KNOW
3. Neck Extended Upwards when Sleeping / Restless Sleep / Frequent Awakenings / Grinding or Clenching Teeth during Sleep?
YES
NO
I DON'T KNOW
4. Upon wakening: Dry Mouth / Sore or achy Jaw / Headaches / Stuffy Nose / Difficulty Awakening in the Morning?
YES
NO
I DON'T KNOW
5. Daytime Sleepiness or Tiredness / Daily Behavioral or Emotional Issues / Difficulty paying Attention / Hyperactivity / Difficulty at School?
YES
NO
I DON'T KNOW
How hard was it to complete this form? *
Easy
Average
Hard
Complete

119


12:34
ssment.azurewebsites.net
C-GASP Level 1B Screener
Does your child frequently have : *
Neck extended upwards when sleeping?
YES
NO
I DON'T KNOW
Restless sleep?
YES
NO
I DON'T KNOW
Frequent awakenings?
12:34
airwayassessment.azurewebsites.net
YES
NO
I DON'T KNOW
Frequent awakenings?
YES
NO
I DON'T KNOW
Grinding or clenching teeth during sleep?
YES
NO
I DON'T KNOW
Previous
Complete
12:35
ssment.azurewebsites.net
C-GASP Level 1 Screener
Children's General Airway Screening Protocol (C-GASP). Developed by the Children's Airway Screener Taskforce (CAST) sanctioned by the ADA to create a screening protocol for pediatric OSA.
Thank you for completing C-GASP Level 1 Screener!

120


Subject number: subject ID number Date auto filled with same date C-Gasp completed

Dentition: Primary dentition Mixed Dentition Permanent Dentition

Dental Classification of Malocclusion

Deciduous (primary) molar relationship

Mesial Step(MS) Distal Step(DS) Edge to Edge/Flush Terminal Plane(FTP)

Permanent Molar Relationship

Class I Edge-to-Edge Class II Class III

Lower anterior crowding None Yes (even slightly)

Posterior Dental Crossbite None Unilateral Bilateral with Mandibular Midline Shift

Upper Inter Canine Distance (cusp tip to cusp tip)

Black Arrow

Primary Measurement # C to # H = ____mm (drop down)

Permanent Measurement # 6 to # 11 = ____ mm (drop down)

Primary Dentition

Permanent Dentition

Upper Teeth

InterMolar Distance (see How to Measure)

Red Arrow

Primary Measurement # A to # J = _____ mm (drop down)

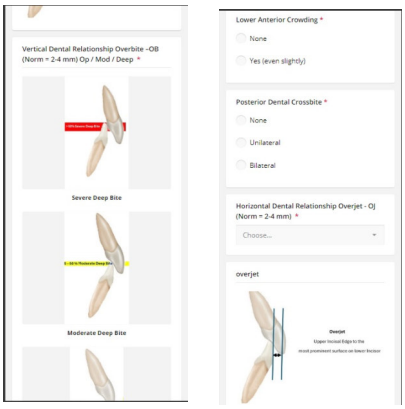
Permanent Measurement # 3 to # 14 = _____ mm (drop down)

How to measure InterMolar Distance –

Primary Dentition: Arrow measuring distance between maxillary 2nd primary inter-molar distance (Bogue) should measure the shortest at the gingival margin.

Permanent Dentition: Arrow measuring distance between maxillary 1st permanent inter-molar distance (McNamara) should point to distal lingual groove (DLG) at the gingival margin.

Anatomic associations:



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How to participate in the C-GASP pilot research project.

(To participate you must administer to ALL children 2-12 yrs old while participating. You can end at any time. 100% voluntary)

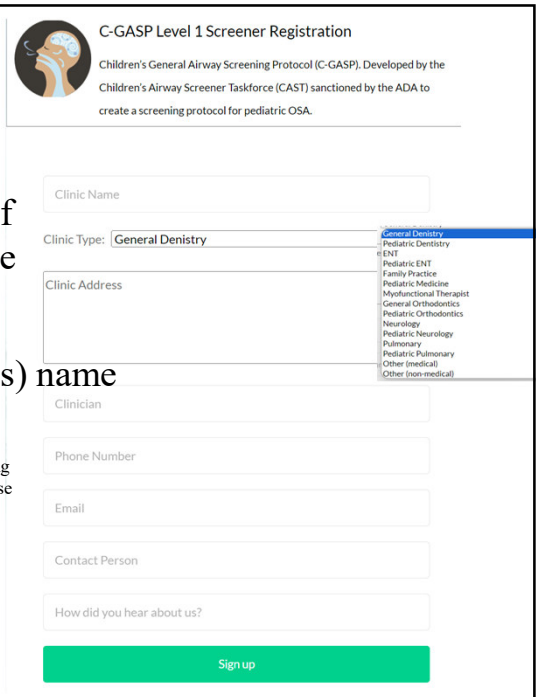
Select the type of practice you have

Your (Clinician's) name

Cell phone and email and name of the person initially coordinating the activity within the office (these can be changed later)

Go to this QR code link

https://airwayassessment.azurewebsites.net/register



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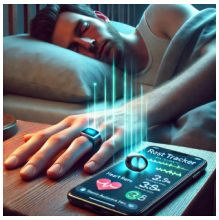
Nightly Remote Patient Monitoring of Sleep



Patients Home

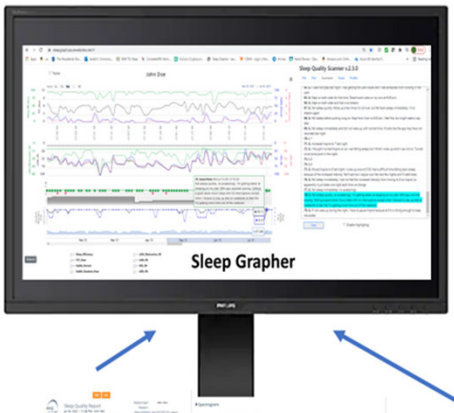
Cloud computation

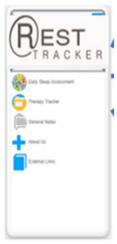
Medical Office

Doctors Review / Medical Records

123

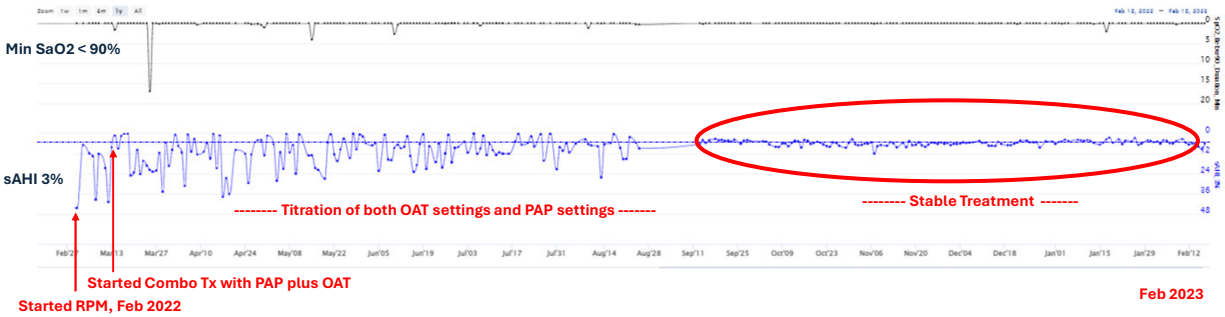
REST Sleep Grapher
Remote Patient
Monitoring Program





124

R.V. is a 67 y/o male with severe OSA with a AHI 3% = 44 / hr, SaO2 min 90%, not adequately treated with oral appliance therapy alone so now on Combination CPAP and Oral Appliance Therapy



Note the night-to-night variability in the patients OSA.

125

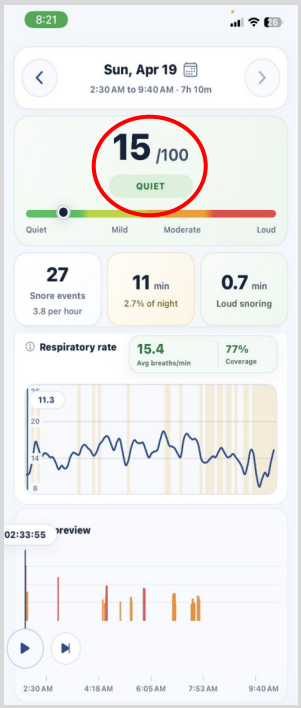
Snoring App To Monitor Trends In A Patients Snoring

126

Monitoring Snoring

Patient with complaint of snoring
Monitoring snoring with a phone app
Track interventions
Uses a nasal dilator intermittently

The improvement in snoring from use of the dilator is evident in the results from the two nights



Without Dilator

With Dilator

127

The Future Landscape of Medication Treatment of OSA

Weight Reduction
GLP-1 /GIP
(Tirzepatide)

Upper Airway Motor Tone (Genioglossus & pharyngeal dilators)

AD109
Combination of:
Atomoxetine ↑ NEP
Oxybutynin mACh
↓

AVE0118
K^+ Channel Blocker

Dronabinol (THC)
↑CB1/CB2 receptors

Loop Gain Stabilization
Acetazolamide
Inhibits carbonic anhydrase
↑ CO2 / ↑ PH – stimulate resp

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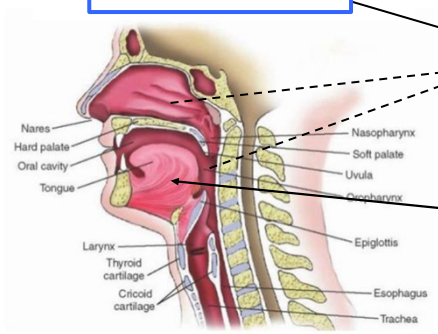
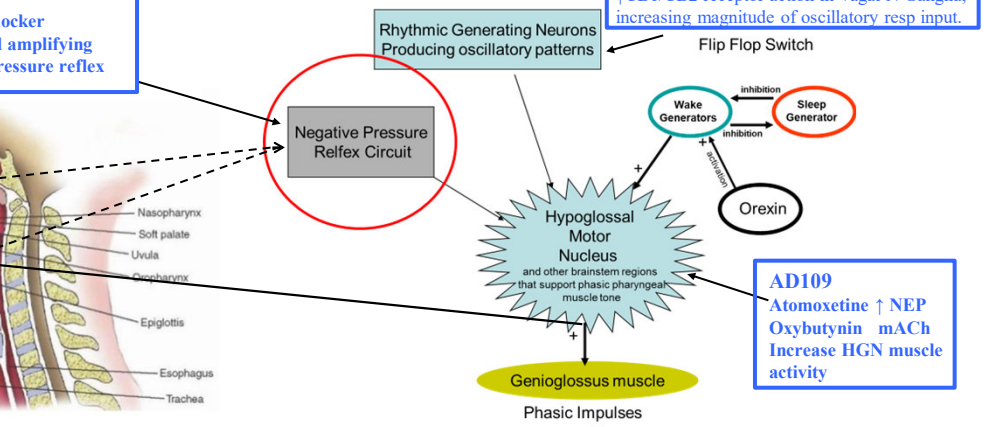
Two Types of Medications Enhancing Upper Airway Muscle Activity For Tx of OSA

AVE0118
K^+ Channel Blocker sensitizing and amplifying the negative pressure reflex

Dronabinol (THC)
↑CB1/CB2 receptor action in Vagal N Ganglia, increasing magnitude of oscillatory resp input.





AD109
Atomoxetine ↑ NEP
Oxybutynin mACh
Increase HGN muscle activity

129

Loop Gain Stabilization

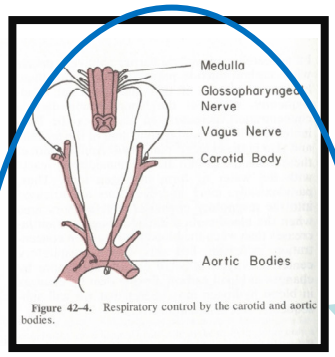
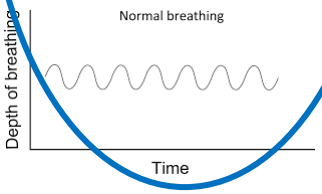
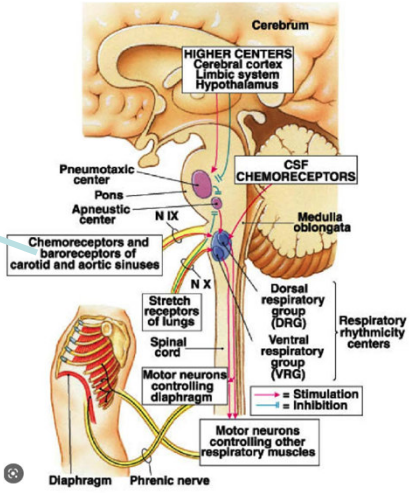


Figure 42–4. Respiratory control by the carotid and aortic bodies.

The Main, Normal Feedback Loop, based on CO2 levels in the blood from sensors in the Aortic and Carotid Bodies





Acetazolamide
Inhibits carbonic anhydrase
↑ CO2 / ↑ PH – stimulates Respiration and the stabilizes instabilities from increased Loop Gain

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IHL-42X

In Phase 2 trials

Combination of **acetazolamide and dronabinol** designed to treat Obstructive Sleep Apnea (OSA) by:

Reducing upper airway collapsibility (Dronabinol)

Enhancing ventilatory stability (Acetazolamide)

Studies have demonstrated 35%–40% reduction in OSA severity in studies

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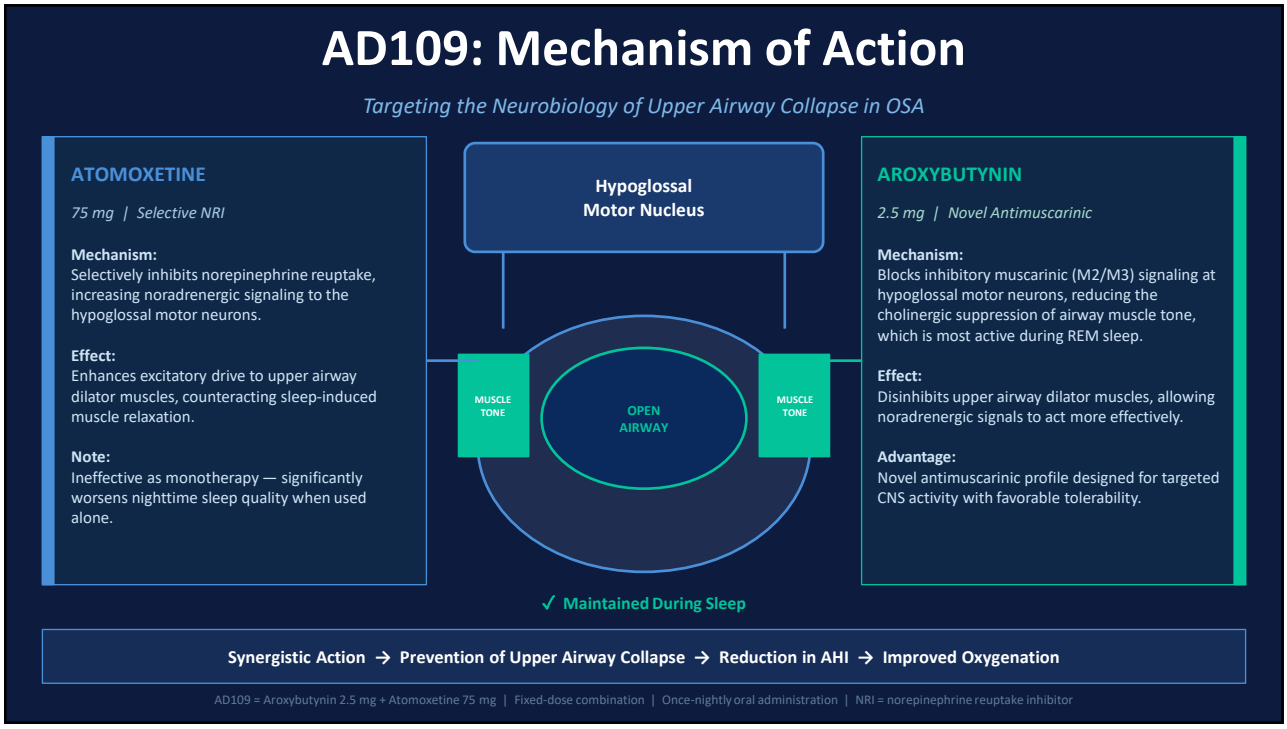

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AD109 (Apnimed)

Oral pill aroxybutynin (2.5 mg) and atomoxetine (75 mg) taken at bedtime.
Amatomoxetine increases noradrenergic drive to hypoglossal motor neurons, while aroxybutynin reduces inhibitory muscarinic signaling at the same neurons — prevents the pharyngeal muscle atonia that underlies airway collapse in OSA.

Pivotal Studies for FDA Submission

SynAIRgy (NCT05813275) —
- 6-month trial enrolling 646 adult, 73 centers
- mild (34.4%), moderate (42.4%), and severe (23.2%) OSA.

- Achieved AHI reduction of 55.6%
- 51.2% experienced a reduction in OSA disease severity
- 22.3% achieved complete disease control (AHI < 5 events/hour).
- No serious adverse events

LunAIRo (NCT05811247) —
- 12-month randomized, double-blind, placebo-controlled trial
- 660 adult, 64 centers, 46% females
- mild (37%), moderate (33%), and severe (30%).

- AHI reduction of 46.8%.
- The reduction in AHI remained significant after 51 weeks
- About 23% of participants achieved complete disease control (AHI < 5)

FDA Submission Status

AD109 granted Fast Track Designation in 2022
Apnimed expected New Drug Application submission date to the FDA in Q2 2026.

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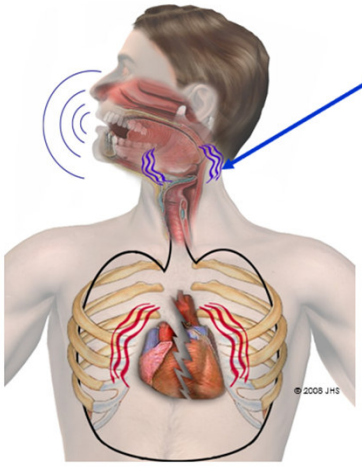
Don't Let *Snoring* Break Your Heart!™



Snoring & problems with sleep are associated with:

- **O**bstructive **S**leep **A**pnea (**OSA**)
- **E**xcessive **D**aytime **S**leepiness (**EDS**)
- Morning Headaches / Jaw pain / TMJ Disorder
- Poor concentration and ADD / ADHD
- Fibromyalgia/ Daytime Fatigue / Irritability
- Chronic Fatigue Syndrome
- High blood pressure / Heart Disease
- Acid Reflux / Type II Diabetes
- Increased risk of heart attacks and stroke
- Ruined relationships
- Bedroom Stress

Snoring **can be a sign of OSA. Untreated OSA can even lead to death!**

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Official Learner+ Champion | SEC 2026 Sleep Education Consortium

Sleep Education Consortium (SEC) partners with Learner+, a clinician-centric reflective learning platform that rewards CME/CE credits to busy clinicians anytime and anywhere learning happens. Learn more about how you can **reflect to unlock credits** below. View CME Credit Info

REFLECT NOW

https://champions.learnerplus/sec/

OSA is not dependent on low oxygen levels in the blood

What inspired you to reflect?

Pick the context and a clinically relevant concept or phrase that inspired you to reflect.

Reflective Learning Moment

OSA is not dependent on low oxygen levels in the blood

Step 1 of 4

Next

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That's all for now

Thank You

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