Snoring and Pediatric OSA

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October 29, 2018




Snoring

* **Snoring:** a respiratory sound generated in the upper airway during sleep that occurs **WITHOUT** episodes of apnea or hypoventilation **AND** that does not cause symptoms of daytime sleepiness or insomnia

* **Snoring** can be described as: a snorting, raspy, or grunting sound while breathing during sleep, typically more so during inspiration than expiration

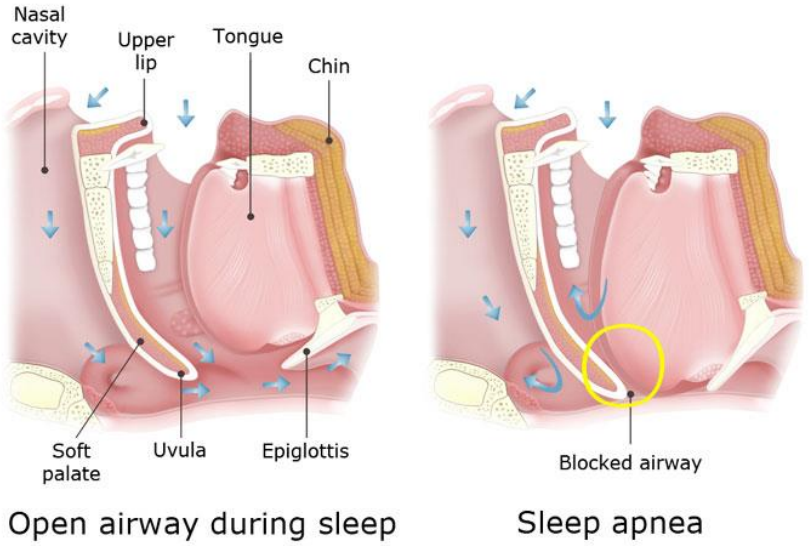
Snoring

* Frequency of intermittent snoring in children ranges from 10%->20%->35%
* Frequency of OSA in preschool and school-age children is: 1-4% (Gozal et al.)
  * Peak age 2-8 years old
  * OSA in American middle-age males=28%, females 9%
* Frequency of sleep-disordered breathing in children is: 6-27% (Gozal et al.)

Snoring Sites/Etiology

Anatomically from flutter/vibration of the:

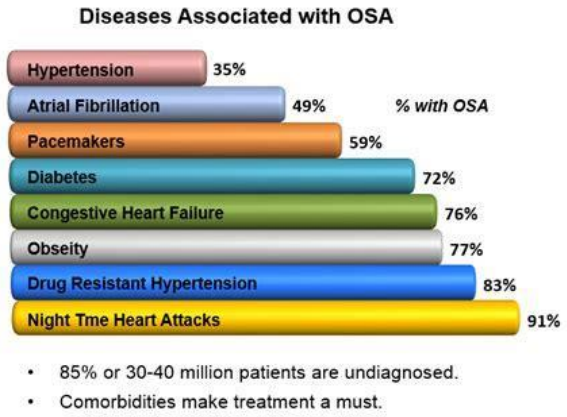
* Uvula
* Soft palate
* Pharyngeal walls/mucosa



* **Caused** by vibrating tissues within the nasal or pharyngeal airways. The snoring vibrations are **caused** by turbulent airflow through narrowed airways

Snoring Sequelae (Adults)

* Carotid artery disease/atherosclerosis
* Systemic Inflammation (CRP)
* Myocardial Infarction
* Cerebral vascular accident/ischemic stroke
* Systemic hypertension, CHF, atrial fibrillation
* Pulmonary hypertension



Snoring Sequelae (Children)

* Learning/Poor School Performance
* Lower Intelligence/IQ
* Executive Memory (hypoxemic changes to frontal lobe area)
* Attention/Concentration: ADHD-behavior (Hyperactivity/Impulsivity/Inattentive, Disruptive), apathetic/lazy
* Behavioral problems: anxiety, depression, anger/aggression, low self-esteem, poor school performance
* Social impact: sleepovers, summer camps

Snoring Sequelae (Children)

* Growth: slow growth and development
* Obesity: insulin resistance, excessive daytime fatigue, decreased physical activity, DM2→ metabolic syndrome (insulin resistance, dyslipidemia, HTN, obesity)(4.2% of 12-19 year old children)
* Nocturnal Enuresis (NEW-onset)

Habitual Snoring

Habitual snoring is often defined:

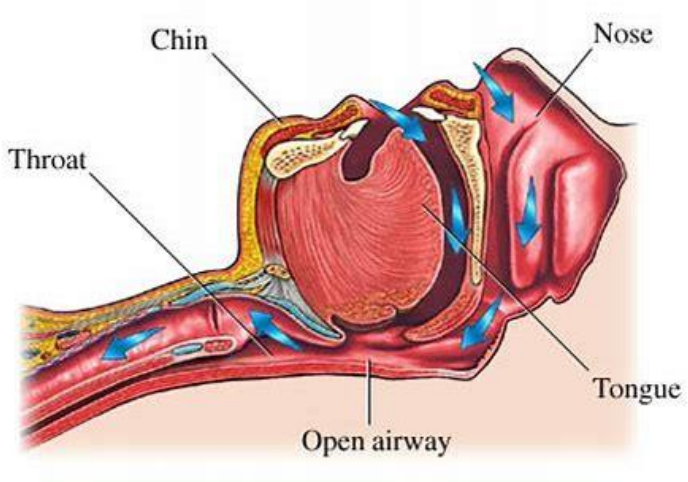
- >3 nights/week
- Estimated to occur in 10-12% of children

Habitual snoring involves 5% of 2-4 month old infants as well as 6-12 month infants

- Can involve 1-9% of infants and toddlers (2-24 months of age) and 7-10% of pre-pubertal children
- Can decrease to 3-5% of 9-14 year old children/adolescents

Snoring Associations

* 1) allergic rhinitis, eczema, asthma (inflammation, common airway)
* 2) recurrent URIs
* 3) parental snoring
* 4) passive tobacco smoke exposure (dose-dependent relationship with maternal smoking and pediatric snoring), worse during 1st year of life
* 5) obesity



Urschitz et al. Risk factors for sleep-related hypoxia in primary school children, Pediatric Pulmonology, v42 n9 (September 2007): 805-812

Primary Snoring

* **Primary snoring** previously termed "benign snoring" with no suspected detriment or associations

* Diagnosed based on polysomnography
  * AHI <1, O2 nadir >90-92%, CO2 <49-52 torr (no gas exchange abnormalities), normal sleep architecture

Primary Snoring

* Now known to be associated with obesity, hypertension, anxiety/depression, decreased neurocognitive/school performance (attention, IQ, memory) (Blunden et al.)

* Associated with increased hyperactive and inattentive behavior, poor school performance in mathematics, spelling, science, and increased daytime tiredness (Brockmann et al.)

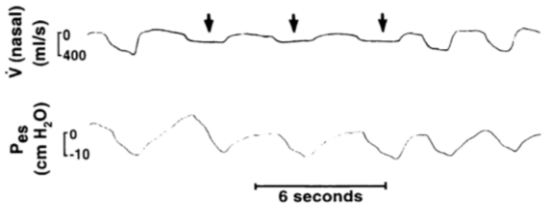
* PS associated with micro-arousals (<3 sec), possible sleep fragmentation, respiratory arousals, decreased % REM sleep, and subthreshold intermittent hypoxia/O2 desats compared to non-snoring children

Primary Snoring

* Increased risk of developing functional somatic symptoms in childhood from habitual snoring and obesity
* ADHD generally affects 3-7% of children. In children with PS or OSA, ADHD reported 2-3 times more common
* All effects not related solely to respiratory events but **sleep quality**
* Children with **fragmented sleep** had lower scores of language/cognitive skills, fine motor skills, social/ADLs skills, and general development
* **Uninterrupted sleep may be more important than sleep duration for the development of the various mental abilities in healthy preschool children**

Upper Airway Resistance Syndrome (UARS)

* Pediatric patients more likely normal weight than OSA patients
* Marked by increased pharyngeal collapsibility(Pcrit) intermediate between normal subjects and patients with mild-moderate OSA



V=Airflow

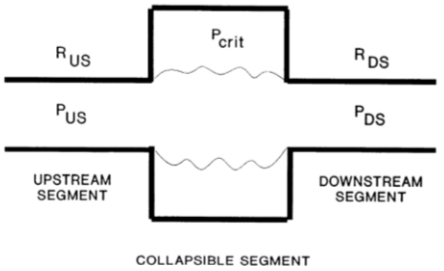
Pes=Esophageal Pressure Transducer

* PSG during sleep shows inspiratory flow limitation, increased respiratory effort, and arousal with usage of nasal cannula/pressure transducer/thermocouple system or esophageal pressure transducer
* Abnormal breathing during sleep alternating with burst of tachypnea without saturation drops
* Esophageal pressure monitoring may be the only way to confirm a suspected diagnosis
* Patients may have more insomnia and fatigue than OSA patients, women>men

Gold et al. Upper Airway Collapsibility During Sleep in Upper Airway Resistance Syndrome, Chest, v121 n5 (May 2002): 1531-1540

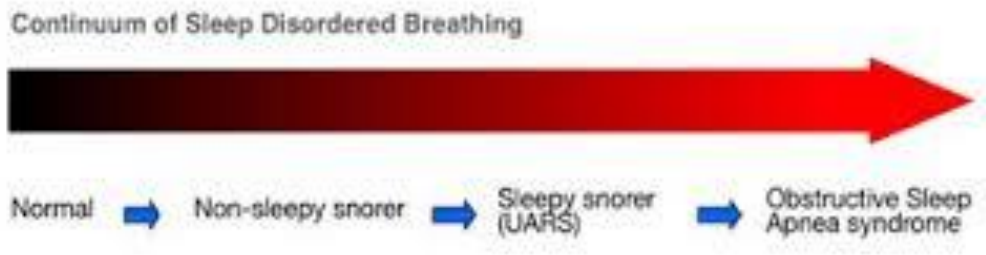
Upper Airway Collapsibility

* Pcrit is an objective measurement of upper airway collapsibility
* Pcrit reflects upper airway neuromuscular control as well as structural factors (upper airway dilators)
* Follows from the Starling Resistor Model



* Pcrit is more negative in healthy children/adults, and becomes more positive (approaching zero or +values) in patients with primary snoring, upper airway resistance, and OSA
  * Pcrit =+1.0±3.4 cm H20 mild OSA children
  * Pcrit =-19.5.0±9.2 cm H20 Primary Snoring children

Spectrum of Snoring, SDB, OSA



OSA Mechanism

* Repetitive pharyngeal collapse during sleep, which leads to markedly reduced or absent airflow, followed by oxyhemoglobin desaturation, persistent inspiratory efforts against an occluded airway and termination by arousal from sleep

In children OSA frequently results from :

* 1) adenotonsillar hypertrophy (peak age 2-8 years old)
* 2) craniofacial abnormalities (upper airway size??)
* 3) obesity

* Typically healthy children have better hypoxic/hypercapnic ventilatory responses and neuromuscular tone than adults; and thus lower Pcrits

OSA Signs/Symptoms

Night-Time S/S

* Snoring
* Noisy Breathing
* Snorting
* Paradoxical Chest/ Abdominal Movement
* Retractions
* Witnessed Apnea
* Difficulty Breathing
* Cyanosis
* Sweating
* Restless Sleep

Daytime S/S

* Mouth Breathing
* Difficulty Waking Up
* Moodiness
* Nasal Obstruction
* Daytime Sleepiness
* Hyperactivity
* Cognitive Problems

End-Stage OSA Sequelae in Children

* Pulmonary Hypertension/Cor Pulmonale
* Arrhythmias in 30%-50% in adult patients with OSA and increase with the number of apneic episodes and severity of the associated hypoxemia
  * atrial premature extra-systoles
  * ventricular premature extra-systoles
  * non-sustained ventricular tachycardia
  * sinus arrest
  * second-degree atrioventricular conduction block
* sleep apnea occurs in ~ 50 percent of adults with heart failure or atrial fibrillation which is the most common sustained abnormal heart rhythm
* Congestive Heart Failure
* Polycythemia
* Death

Pediatric Obesity

* Obesity rates among children and adolescents have almost tripled in the past 25 years
* Between 2009 and 2010, the prevalence of obesity in US children and adolescents was 16.9% -> 25%
* Type-2 diabetes mellitus, hypertension, metabolic syndrome, heart disease and PCOS, dyslipidemia, atherosclerosis, ischemic heart disease, NASH/steatohepatitis

Pediatric Obesity

* 4-5 fold increased risk of sleep-disordered breathing in children ages, 2-18 years who are obese
* For every 1 kg/m^2, in BMI beyond mean BMI for age/gender, the risk of OSA is increased by 12% (Gozal et al.)
* Gozal found that 45-55% of children referred for PSG for suspected sleep-disordered breathing were obese

Obesity and OSA

* Fatty-infiltration of upper airway structures (tongue) and pharyngeal muscles, surrounding parapharyngeal space
  * Neck circumference >17 in. males, 16 in. females
  * Reduces cross-sectional area of the pharyngeal airway and increases the collapsibility of upper airway
* Subcutaneous deposits of fat in the anterior neck and other upper airway structures exert collapsing forces promoting increased pharyngeal collapsibility

* Increased fat in abdominal and chest wall increases global respiratory load, reduces intrathoracic volume and diaphragm excursion--->decreases lung volumes and O2 reserve while increasing work of breathing

Pediatric Obesity Hypoventilation Syndrome (Pickwickian Syndrome)

1837 Charles Darwin, "Pickwick Papers"

* Obesity + awake arterial hypoventilation/hypercapnia ($PaCO_2$>45 mm HG), BMI>30 kg/m^2
* Symptoms: daytime somnolence, fatigue, morning headache
* Presentation: polycythemia, hypoxemia, cyanosis, cardiomegaly, CHF
* Presents as cardiorespiratory distress and alveolar hypoventilation
  * Decreased tidal volumes and expiratory reserve
* OHS children have a decreased ventilatory drive to increasing hypercapnia or hypoxemia (low response to chemoreceptor stimulation) compared to OSA children, +chronic respiratory acidosis

Pediatric Obesity Hypoventilation Syndrome (Pickwickian Syndrome)

* A possible role of leptin-resistance with this syndrome
* Some reversibility of blunted hypercapnia responsiveness following T&A is seen in some children suggesting a mechanical effect of obesity on the respiratory system
* Ultimately BiPAP and intensive weight loss may be required after T&A

Obesity and Surgery

* Obese children with OSA have a 23-27% risk for postoperative cardiac/respiratory complication
  * ? Hospital admission after T&A?
  * At NCH, BMI >98% for age/gender, gets admission postop

* Cure: AHI <5 or AHI <1. When obese, cures after T&A may reach only 50% of children

ROHHAD

* ROHHAD: Rapid Obesity Hypothalamic Dysfunction, Hypoventilation, and Autonomic Dysfunction
  * ~80 reported pts in literature (2018)
  * Similar to Prader-Willi Syndrome
* Rare, auto-immune condition presenting with encephalopathy, seizures, diabetes insipidus, hypoventilation, visual hallucinations, hypothalamic dysregulation
  * 40% pts have associated neural crest tumors (ganglioneuroblastoma, ganglioneuroma)
  * Pituitary/endocrine abnormalities (hypo/hypernatremia, hyperprolactinemia, central hypothyroidism, GH/ACTH deficiency)
  * Constipation, sweating problems, reduced pain sensation, light-non-responsive pupils
  * Hyperphagia, Rapid weight gain after 2-4 years old, hypoventilation is a late finding
* Many patients succumb to respiratory failure and sudden death/cardiac arrest from alveolar hypoventilation

Snoring vs. OSA

* *Carroll, et al. Inability of Clinical History to Distinguish Primary Snoring From Obstructive Sleep Apnea Syndrome in Children, Chest. Sept 1995, Vol. 108, No. 3*

No differences between PS and OSA patients with respect to:

* age
* gender
* race
* failure to thrive
* obesity
* h/o EDS
* snoring history (apneas, shaking child to awaken, loudness, frequency)
* history of cyanosis during sleep
* daytime symptoms except for mouth breathing

* **Study showed that PS in children cannot be reliably distinguished from OSAS by clinical history alone**

OSA Diagnosis by PSG

PEDIATRIC

* AHI <1—primary snoring
* AHI 1.5-5—mild OSA
  * AI>1
* AHI 5-10—moderate OSA
  * AI>5
* AHI 10+—severe OSA
* AHI 30+—very severe OSA
* (Mitchell et al.)

ADULT

AHI <5---primary snoring

AHI/RDI 5-15**—mild OSA

AHI/RDI 15-30—moderate OSA

AHI/RDI>30—severe OSA

Children in Whom A PSG Should be Performed

* Down's Syndrome, myelomeningocele, Prader-Willi Syndrome
* Mucopolysaccharidosis
* Craniofacial Abnormalities
* Obesity
* Sickle-Cell Disease
* Neuromuscular Disease (cerebral palsy, Duchenne's MD)
* Metabolic Diseases

PSGs typically only obtained in only 10-15% of all children prior to surgery nationally

PSG Recommendations

* Children <age 3 years old
* Significant co-morbidities
* Morbid obesity
* Discordant exam and parent/patient-reported history
* Teenagers (adult body phenotype)??
* Surgical Treatment Failure or Postop PSG Verification

Mild OSA (AHI 1-5)

* No consensus on treatment

Treatment Options

* Watchful waiting/expectant observation
  * (Probably more ideal in older age, small tonsils/adenoids, non-obese patients)
* Adenotonsillectomy
* Weight loss
* Medical Therapy ******

Symptomatic vs. Asymptomatic Patient??

Parent Choice?
Shared Decision-Making

Li et al., Mild OSA Surveillance

* Li et al. (Thorax, 2010)

* Study of 45 pts, 6-13 year old Chinese children with mild OSA

* No child had surgery over the follow-up interval of 2 years

* 13/45 pts (29%) had worsening of mild OSA over 2 years

By multi-variate linear regression, **children with worsening OSA were:**

* 1) younger than 3 years old, tonsil/adenoid growth over study
* 2) male
* 3) had 3-4+ tonsils
* 4) larger waist circumference/obesity
* 5) greater prevalence of habitual snoring (snoring >3 nights/week)

Li et al, Natural history and predictors for progression of mild childhood obstructive sleep apnea, Thorax, v65 n1 (201001): 27

CHAT Study

CHAT=Childhood AdenoTonsillectomy Study, 2013

* Marcus et al., NEJM, multi-institutional study, mild OSA
* Randomized controlled study, 464 children (5-9 years of age) with PSG-proven mild OSA, randomized to early T&A vs. watchful waiting
* Variables: cognitive, behavioral, and health outcomes were assessed at baseline and at 7 months
* Secondary Outcome=normalization of PSG findings was observed in 79% children in the early-adenotonsillectomy group vs. 46% children in the watchful-waiting group
* Children who DID NOT have normalization of mild OSA in the watchful waiting group were male, obese, and African-American

Marcus et al. A randomized trial of adenotonsillectomy for childhood sleep apnea. The New England journal of medicine, 2013 Jun 20; 368(25): 2366-76

Medical Therapy, Mild OSA

* *Leukotriene Modifier Therapy for Mild Sleep-Disordered Breathing in Children, 2005, American J. Respiratory Critical Care Med.*
* Goldbart, Goldman, Veling, Gozal

Receptors in germinal center of the Tonsil/Adenoid tissue
No long-term tolerance

* 24 children (ages 2-10 y.), AHI 1-5, 4 months of montelukast vs. 20 control children
* PSG, adenoid size (lateral neck x-ray) checked pre/post study
* Montelukast induced significant adenoid reduction and improved PSG parameters (AHI 1.2->0.8, oAHI 3->2)

Medical Therapy, Mild OSA

Anti-inflammatory Therapy Outcomes for Mild OSA in Children, Chest 2014

* Kheirandish-Gozal, Bhattacharjee, Bandla, Gozal

* Retrospective review, 2007-2012, children 2-14 years old, mild OSA

* Treatment: intranasal corticosteroid and montelukast for 752 children

* 62% had normalization of mild OSA with therapy upon repeat PSG, while 17.1% pts had no improvement or worsened OSA
  * Non-responding pts were older and obese
  * 12.3% ultimately had a T&A
  * 61/752 (8.1%) pts were non-adherent to therapy

Medical Therapy, Mild OSA

Intranasal corticosteroids for mild childhood obstructive sleep apnea – a randomized, placebo-controlled study

* Chan, Au, Lam, Lee, Wing, Li, Sleep Medicine, 2014
* Randomized, double-blinded, placebo-controlled trial of intranasal mometasone furoate (MF) versus placebo in children aged 6 to 18 years with mild OSA
* 50 children, Hong Kong, 2006-2008, 4 month period
* OAHI and ODI improved significantly in the MF group only
* Proportion of children having habitual snoring also reduced from 75% to 54.5%

Chan et al. Intranasal corticosteroids for mild childhood obstructive sleep apnea–a randomized, placebo-controlled study, Sleep Medicine 16 (3), Mar 2015, 358-363.

How Effective is Adenotonsillectomy for OSA or Sleep-Disordered Breathing?

* T&A is successful in 27.2% to 82.9% of patents

* In uncomplicated healthy children with T&A hypertrophy, the general success rate may range from 66.3% - 82.9% success after adenotonsillectomy if cure defined by AHI <1

Higher rates of failure s/p T&A in patients with:

* 1) Down's Syndrome (~50% cure, cure declines with increasing BMI)
* 2) age <3 years old
* 3) morbid obesity, older age patients/teenagers
* 4) craniofacial abnormalities
* 5) neuromuscular disorders
* 6) severe OSA preoperatively

Allergic Rhinitis Impact on postop T&A patients

* *The Impact of Allergic Rhinitis on Symptom Improvement in Pediatric Patients After Adenotonsillectomy, Jun-Lee, Jun Yang, Mo 2018, Clin Exp Oto*
* *131 Allergic rhinitis Pts (mean age 6), 119 Control Pts (mean age 6), s/p T&A*
* *Allergic rhinitis pts showed less improvement in snoring, mouth breathing, nasal obstruction, and rhinorrhea than control pts*
* AR group showed significantly less improvement than control group in snoring, mouth breathing, nasal obstruction, and rhinorrhea (all $P<0.05$).
* Multivariate analysis showed that preoperative mouth breathing and snoring were dependent on tonsil grade and postoperative symptoms were mainly dependent on presence of AR

Adenotonsillectomy Risks

* Perioperative complication risks (desaturations, respiratory complications/decompensation) is 8.8%
  * Pulmonary edema (POPE)
  * New sustained O2 requirement in PACU (to Floor or upon discharge)
* Unplanned re-admission rate can range from 5.0-8.0%
* Postoperative hemorrhage 1-5%, 5-10% depending on surgical indication
  * Return to OR in 0.8-1.8% of children for operative cautery
* Mortality rate is 1:10,000 to [1:50,000]

Adenotonsillectomy Benefits

* Improved asthma control (Association of Adenotonsillectomy with Asthma Outcomes in Children: A Longitudinal Database Analysis), PLoS Med, 2014
  * Airway inflammation concept, "Unified airway hypothesis"

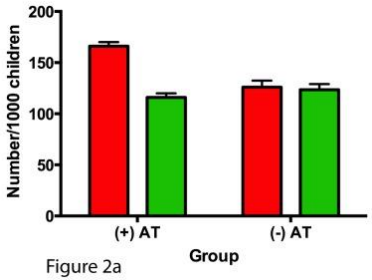


Figure 2a

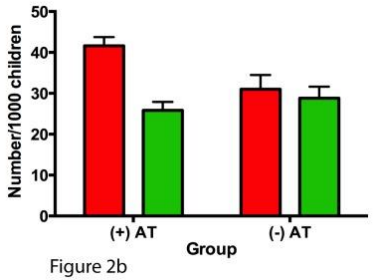


Figure 2b

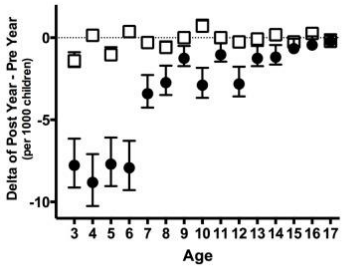


Figure 2c

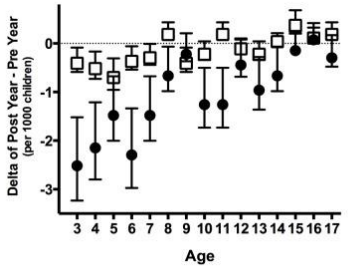


Figure 2d

Postoperative Respiratory Complications

* Age <3 years old
* High Preoperative AHI
* O2 Nadir <70-80%
* Concurrent medical co-morbidities (obesity, CP)
* Craniofacial abnormalities

Risks/Detriments of T&A

* **Association of Long-Term Risk of Respiratory, Allergic, and Infectious Diseases With Removal of Adenoids and Tonsils in Childhood**
* Sean G. Byars, PhD[1,2]; Stephen C. Stearns, PhD[3]; Jacobus J. Boomsma, PhD[2]

* Followed 1,189,061 Danish children from 1979-1999-2009
  * **Pts:** 17,460 Adenoidectomies, 11,830 Tonsillectomies, 31,377 Adenotonsillectomies

* T&A associated with 2-3 times risks of diseases of upper respiratory tract, 17% increased risk of upper airway infection (bronchitis/rhinitis)

* Slight increase in auto-immune disease, Sweden (Karolinska Institute), separate study

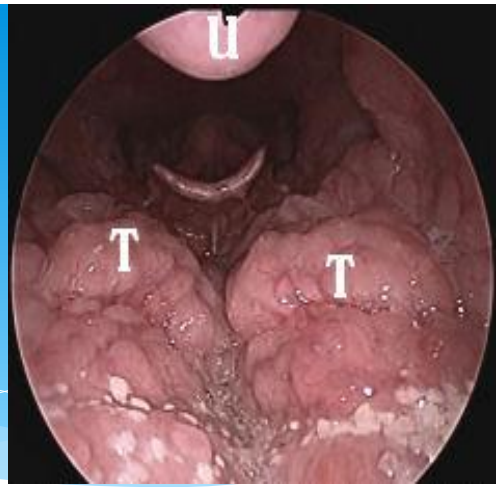
Moderate-Severe OSA

* Adenotonsillectomy +/- Adjuvant Therapy
* CPAP
* Weight Loss
* Medical Therapy
* Generally, do not need repeat PSG in healthy (normal weight) children with preop AHI ≤10 (Mild/Moderate) with large tonsils/adenoids

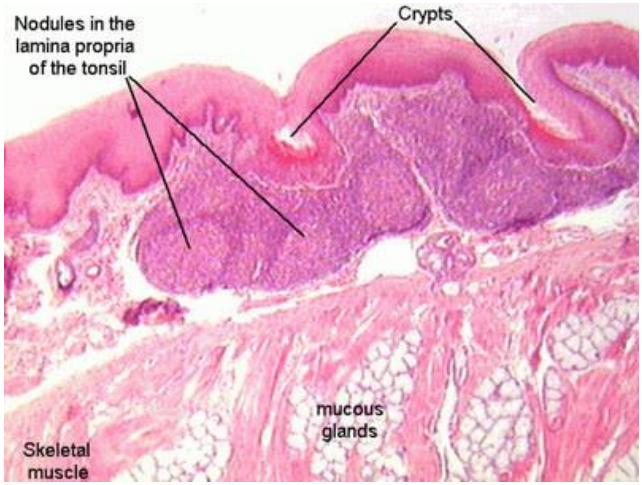
Other Treatments (Beyond T&A)

* Septoplasty +/- Inferior turbinate Coblation/SMR
* Uvulopalatoplasty/Expansion Sphincter Pharyngoplasty
* Lingual Tonsillectomy (35% of patients will have compensatory growth after T&A)
* Tongue Base Surgery (midline partial glossectomy—Down's Syndrome, Obese, Teenagers, CP)
* Craniofacial Surgery
* Tracheostomy
* Mandibular Distraction
* Rapid Maxillary Expansion
* Weight loss (weight usually increases after T&A, OSA at night can be physical)
* CPAP

Lingual Tonsils



* Epithelium—non-keratinized stratified squamous Ep.
* Contain about 30-100 exophytic follicles, located within tongue base, and composed of shallow crypts
* Lingual tonsils surrounded by a thin capsule of connective tissue
* Innervation—tonsillar branch of CN IX



* Thin reticular surface pattern of tonsils when not hypertrophic

Lingual Tonsils

Boundaries

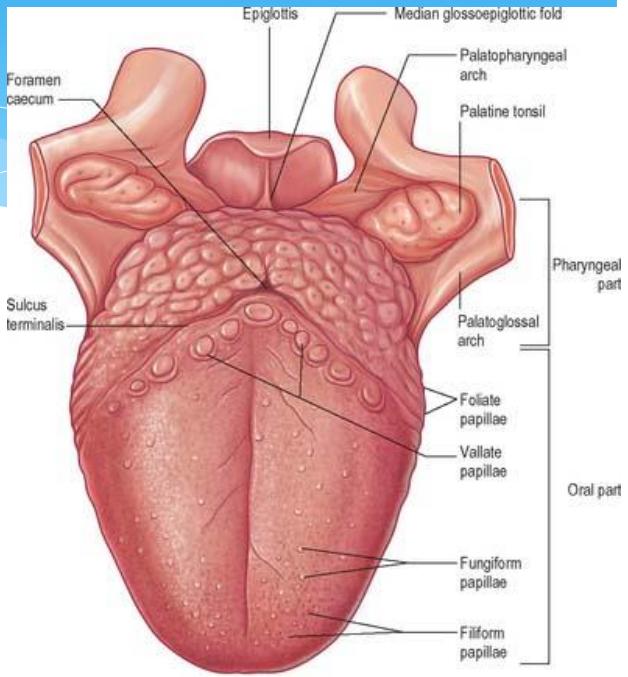
--Base of tongue musculature

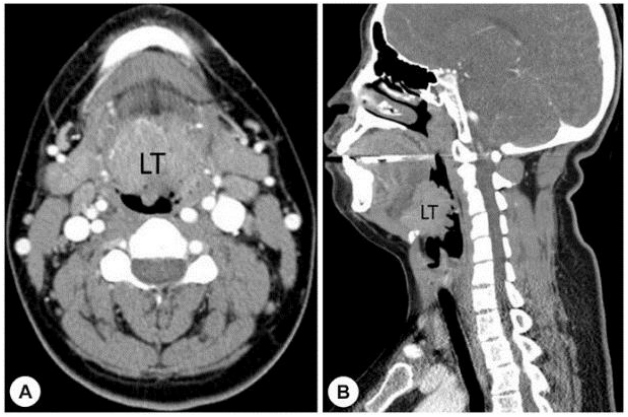
--Superior: foramen cecum/circumvallate papilla

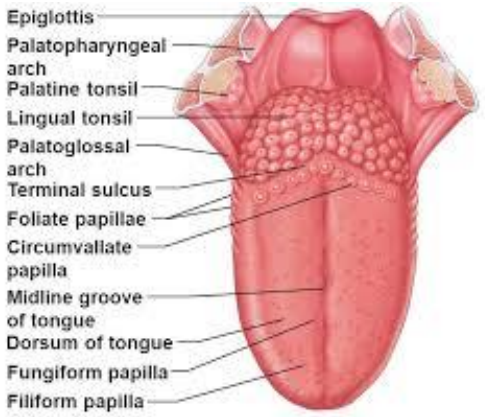
--Posterior: vallecula/epiglottis

--Inferior: vallecula

--Laterally: palatine tonsil



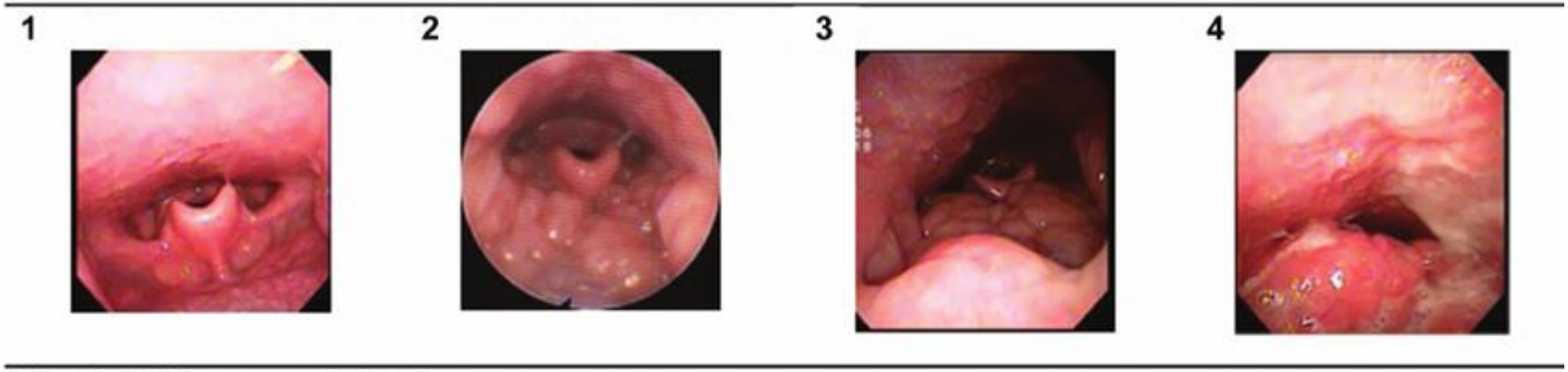




Lingual Tonsil Hypertrophy

* Grading System

(A Pediatric Grading Scale for Lingual Tonsil Hypertrophy) Norman Friedman, Jeremy Prager, Amanda Ruiz, Eric Kezirian, April 2017, Arch Oto Rhino Laryngol

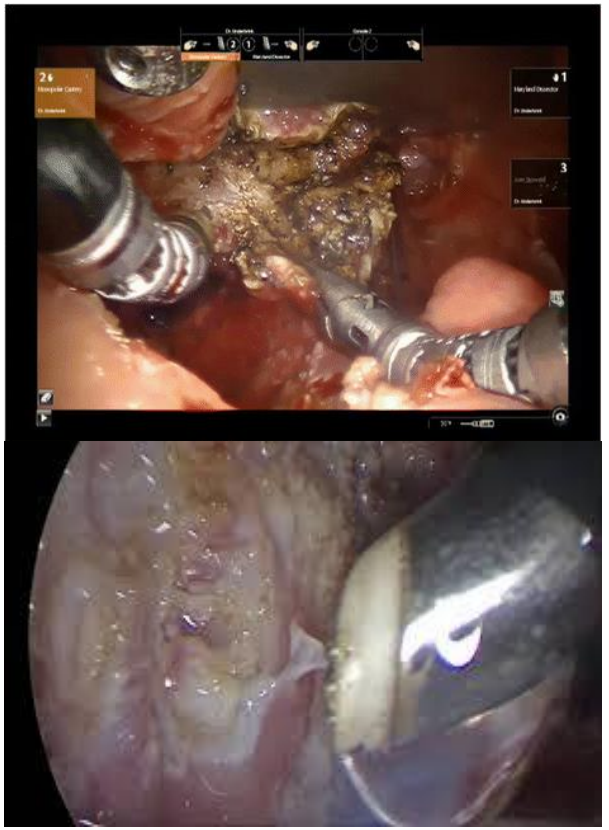


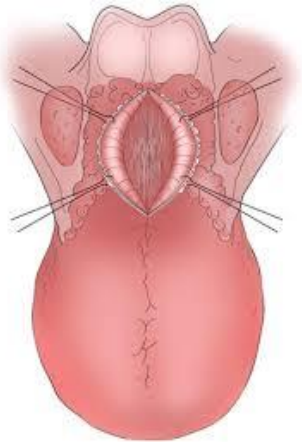
Grade 1. None to minimal
Grade 2. Mild: < 50% filling vallecula
Grade 3. Moderate: > 50% effacement of vallecula
Grade 4. Severe: Unable to visualize epiglottis

Lingual Tonsillectomy

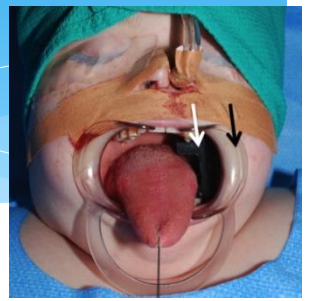
Historical Methods of Resection

* 1) Co2 Laser
* 2) Diathermy
* 3) Suction Monopolar Bovie (+90-100°C)
* 4) Microdebrider
* 5) Coblation (40-70°C)
* 6) DaVinci Robot/TORS
* 7) Cold Knife/Sharp Dissection



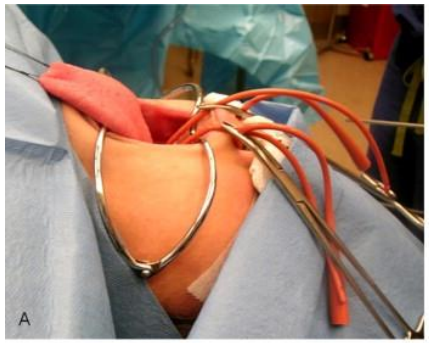


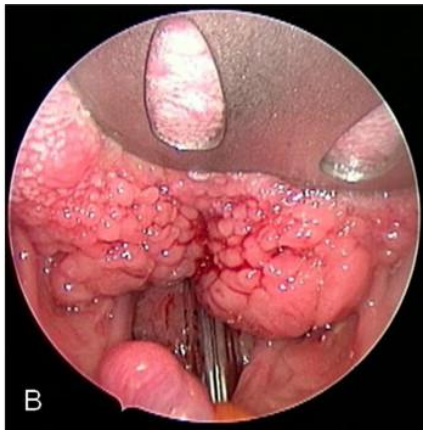
Lingual Tonsillectomy



Common Exposures for LT (Direct or Angled View ??)

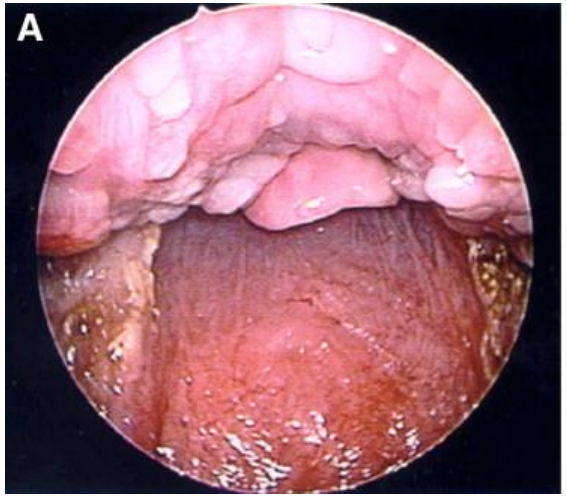
* 1) Lindholm or Parsons laryngoscope, Crowe-Davis
* 2) Tongue retraction suture(s), blue-smiley lip retractors, bite block vs. Molt retractor, Jennings Mouthgag, red rubber retractor, 70 degree scope, sweetheart retractors

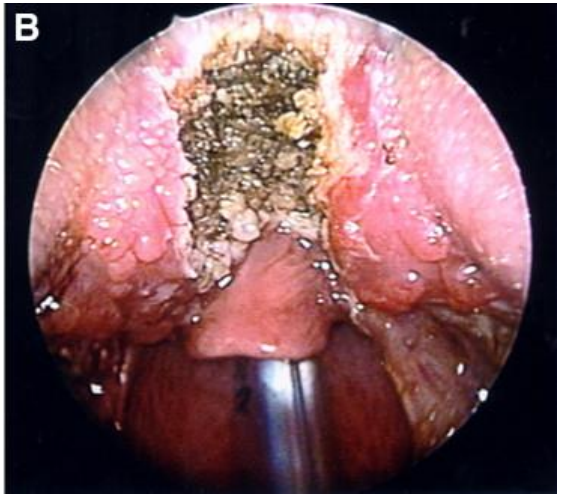




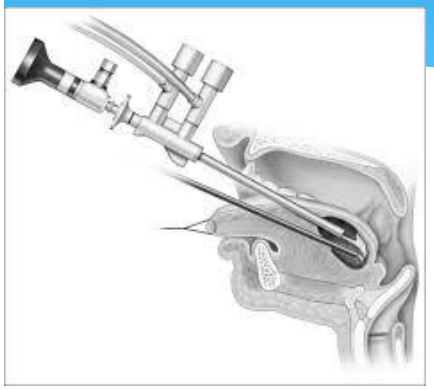
Lingual Tonsillectomy

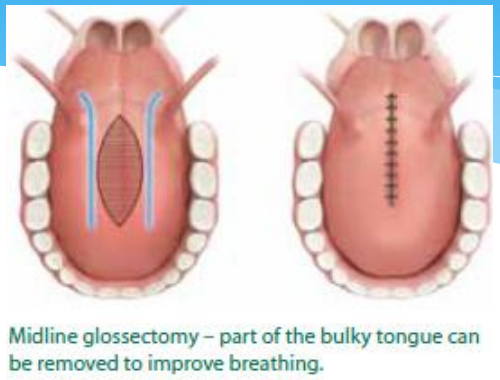
* Surgical objectives for lingual tonsillectomy?? Increase retroglottic space by how much?? ~1 cm staying within the central boundaries of the epiglottis
* Recovery?? 7-14 days, usually less painful than tonsillectomy as frequently don't completely expose BOT musculature—(~PITA)
* Bleeding risk?? 1-5% (Univ. Cincinnati data)
* Recurrence ?? Yes, but low (<5%)



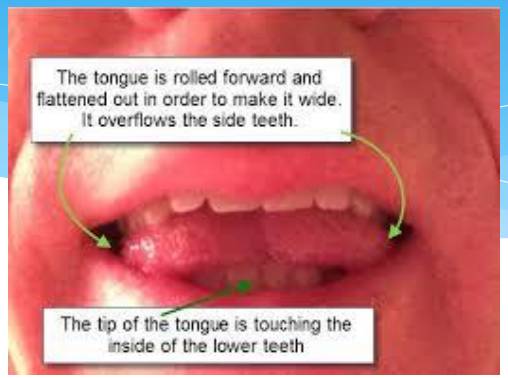


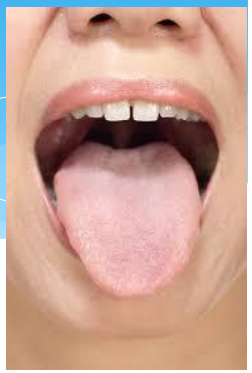
Midline Partial Glossectomy





Midline glossectomy – part of the bulky tongue can be removed to improve breathing.



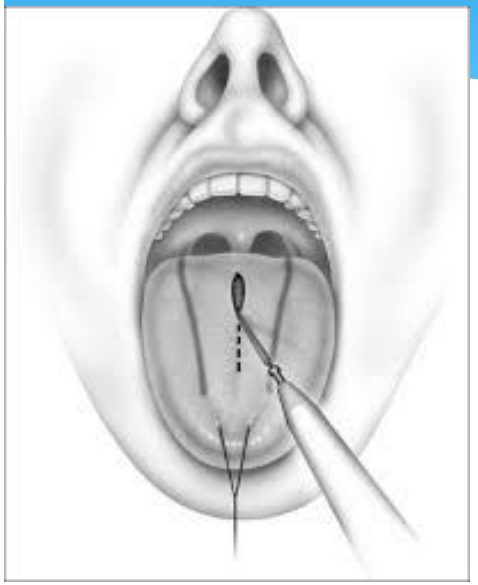


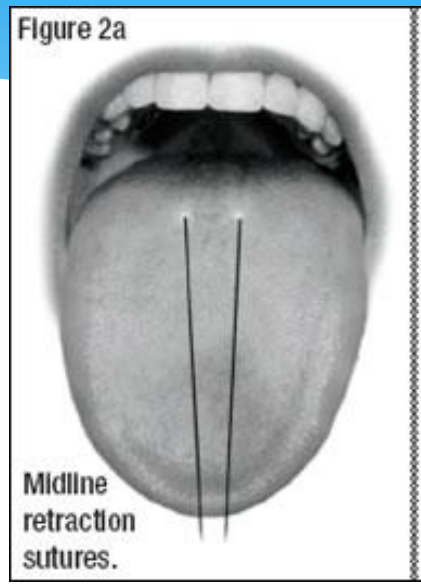
* SMILE (Submucosal intra-lingual excision)--endoscopically
* Open midline partial glossectomy/excision

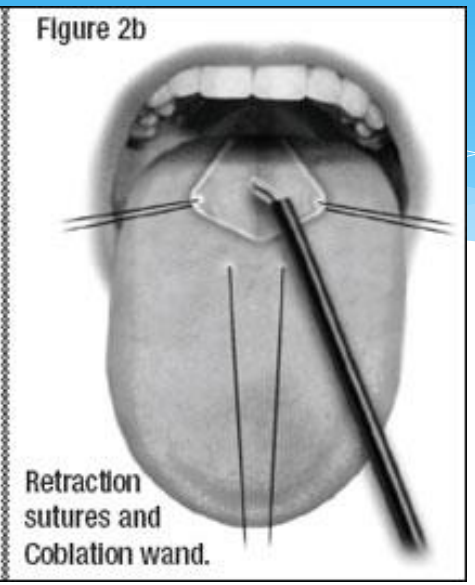
Who are Candidates (Macroglossia, Pseudo-macroglossia)

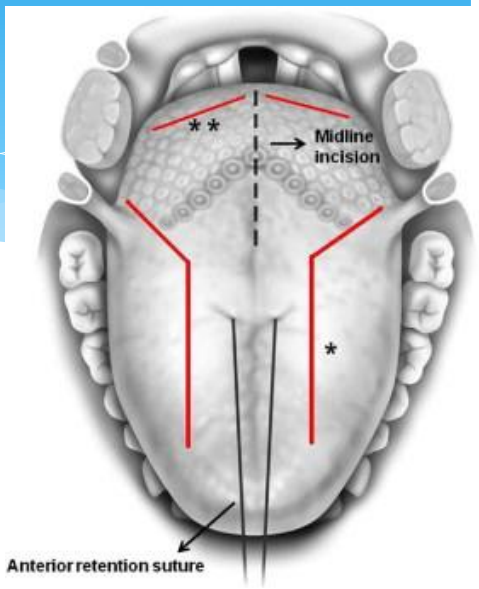
* 1) Down Syndrome
* 2) Non-syndromic Obese children with Lingual tonsil hypertrophy
* 3) Syndromic children (mucopolysaccharidoses children)

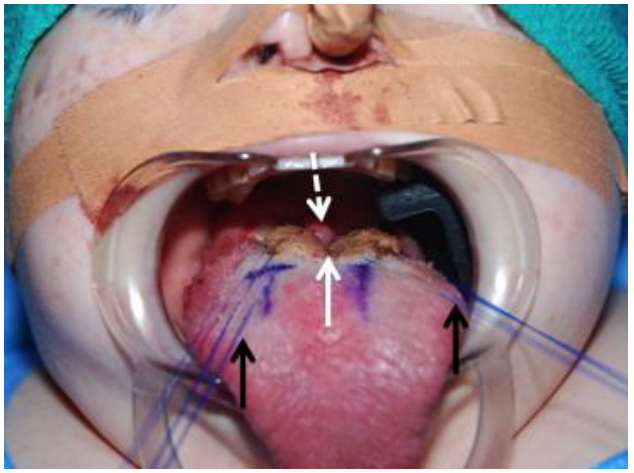
Midline Partial Glossectomy











Tongue Surgery for Pediatric OSA

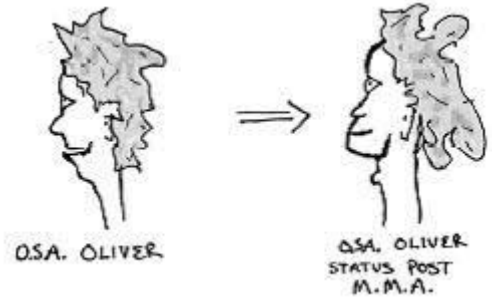
Tongue surgeries for pediatric obstructive sleep apnea: a systematic review and meta-analysis

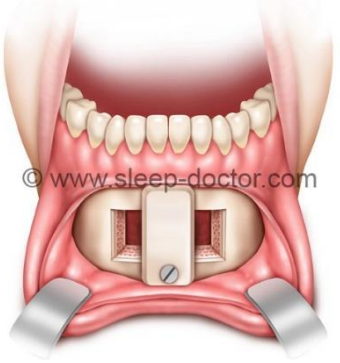
* To evaluate the international literature for studies reporting outcomes for obstructive sleep apnea (OSA) in children undergoing isolated tongue surgeries.
* Two authors searched from inception through November 14, 2016 in four databases including PubMed/MEDLINE. 351 studies were screened. Eleven studies (116 children) met criteria.
* Most children were syndromic and had craniofacial disorders, co-morbidities, or other serious medical issues. Surgeries included base-of-tongue (BOT) reduction (*n* = 114), tongue suspension (*n* = 1), and hypoglossal nerve stimulation (*n* = 1).
* The pre- and post-BOT reduction surgeries decreased apnea-hypopnea index (AHI) from a mean (*M*) and standard deviation (SD) of 16.9 ± 12.2/h to 8.7 ± 10.6/h (48.5% reduction) in 114 patients. Random effects modeling (109 patients) demonstrated a standardized mean difference for AHI of −0.78 (large magnitude of effect) [95% CI −1.06, −0.51], *p* value <0.00001.
* For BOT surgery in 53 non-syndromic children, the AHI decreased 59.2% from 14.0 ± 11.4 to 5.7 ± 6.7/h, while in 55 syndromic children, the AHI decreased 40.0% from 20.5 ± 19.1 to 12.3 ± 18.2/h. BOT reduction improved lowest oxygen saturation from *M* ± SD of 84.7 ± 7.4-87.9 ± 6.5% in 113 patients. Hypoglossal nerve stimulation and tongue-base suspension are limited to case reports.
* Most children undergoing tongue surgeries in the literature were syndromic and had craniofacial disorders, co-morbidities, or other serious medical issues. Children with a body mass index <25 kg/m^2 and non-syndromic children have had the most improvement in AHI. The specific type of surgery must be tailored to the patient. Patients with co-morbidities should undergo treatment in centers that are equipped to provide appropriate perioperative care.

Tongue Advancement/Suspension

* Genioglossus advancement not usually performed in children as mandible is still growing and difficult access to lower mandibular body without injuring tooth roots

* Dental appliances not usually utilized in children as the mandible is still growing and could affect bite (open bite/cross bite) and affect adult tooth orientation

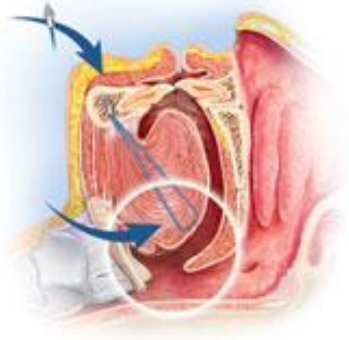


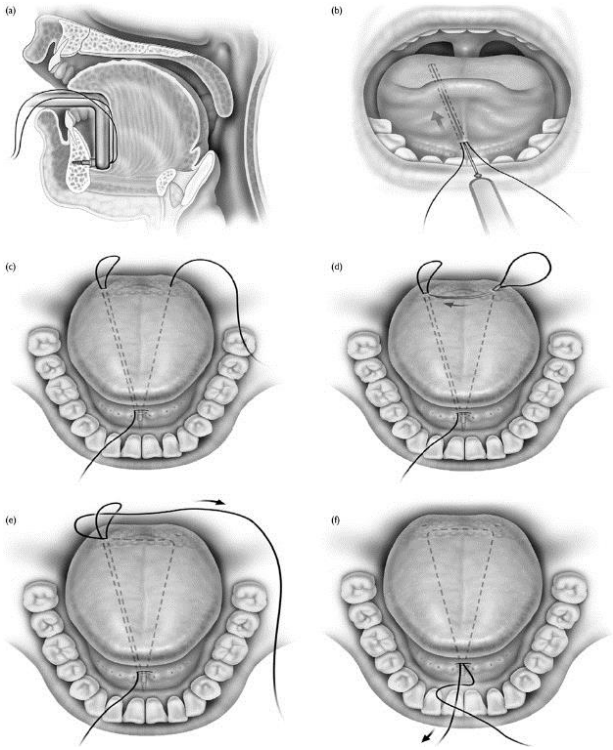


Repose vs. AIRvance vs. Encore

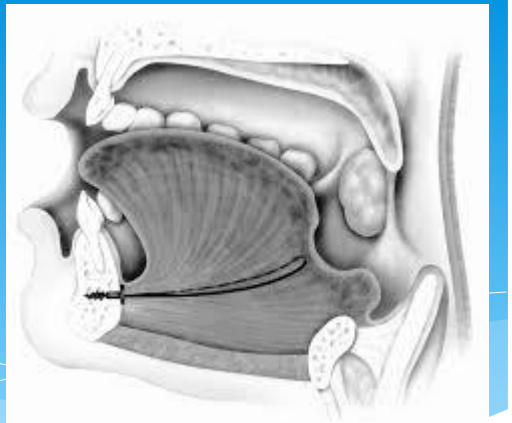
AIRvance (Medtronic)

* Reversible
* Goal to stabilize genioglossus muscle
* Use of bone-anchoring screw connected to sutures that are passed through the body of the tongue to tongue base to make a “hammock”
* Approach to mandible intra-orally vs. externally



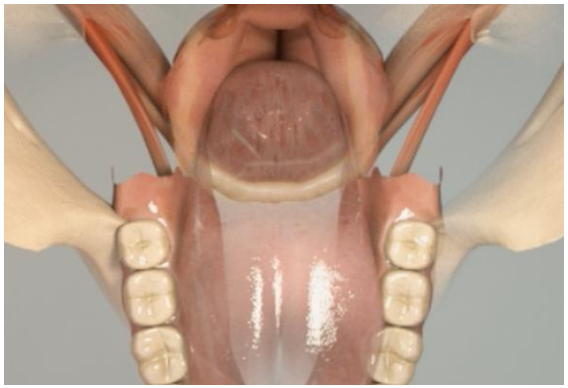


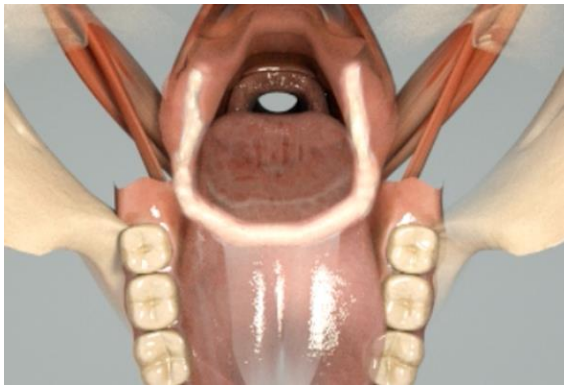
AIRvance/Repose

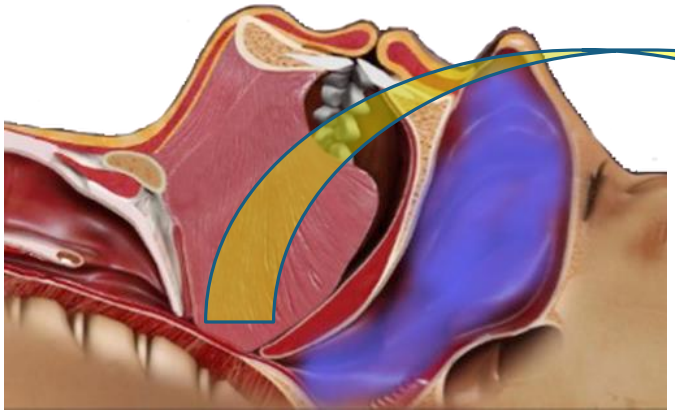


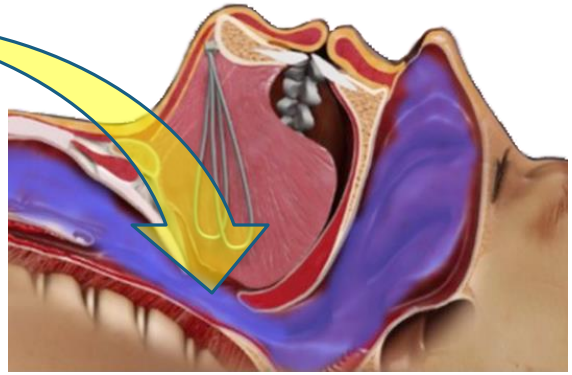
* Repose– 1 cm lateral to midline, 1 cm inferior to foramen cecum (**so as to enter *base of tongue instead of mobile tongue and not injure lingual artery***)
* Pulls BOT anteriorly and improves retroglossal space
* Complications/Morbidity: dysarthria, dysphagia, pain, abscess, infection, suture breaks, patient noncompliance, FOM swelling, sialoadenitis

Siesta Medical Encore/AirLift (2012)





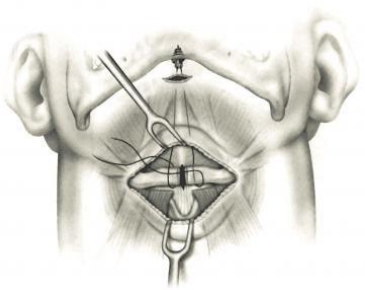




Siesta Medical Encore/AirLift

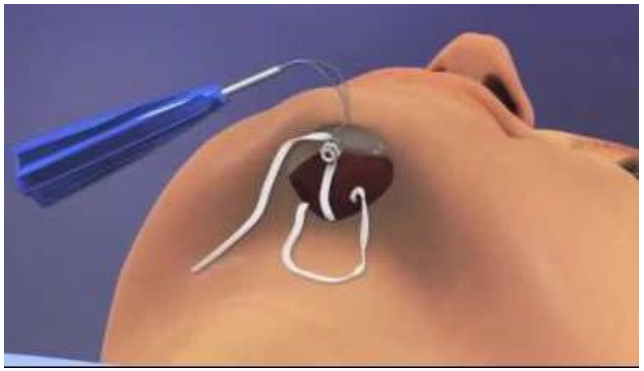
Pros

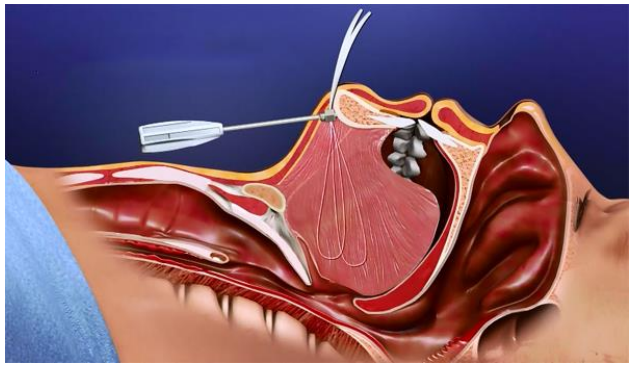
* Reversible
* Adjustable, knotless straight-line connections between suture anchor/hyoid bone
* For tongue advancement, may use multiple sutures in the BOT
* Hyoid/Tongue Suspension included in same single package

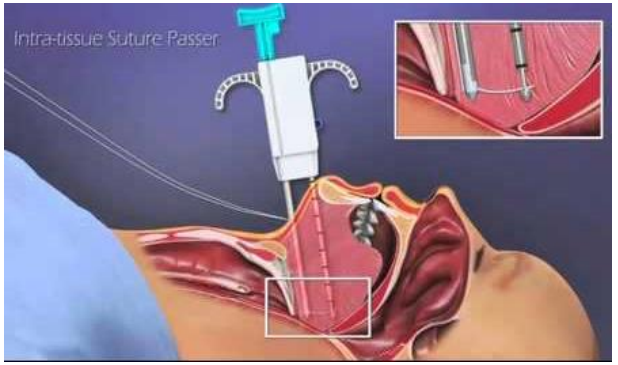


Cons

* Tongue suture not usually covered by Medicaid currently.
  * Reportedly covered by most private insurances and CMS







Hypoglossal Nerve Stimulator

* Current limited early trials in children
* Not yet FDA approved, especially not for Medicaid pts