Childhood Obstructive Sleep Apnea

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SLEEP DISORDERED BREATHING

Spectrum Of SDB includes

- Primary snoring
- Upper airway resistance syndrome (UARS)
- Obstructive hypoventilation (hypopneas)
- Sleep apnea

- Represents increasing severity of upper airway obstruction

SLEEP APNEA

- Apneas represent the complete cessation of airflow through the nose and mouth for a duration that is determined by age-appropriate norms

Of three types :

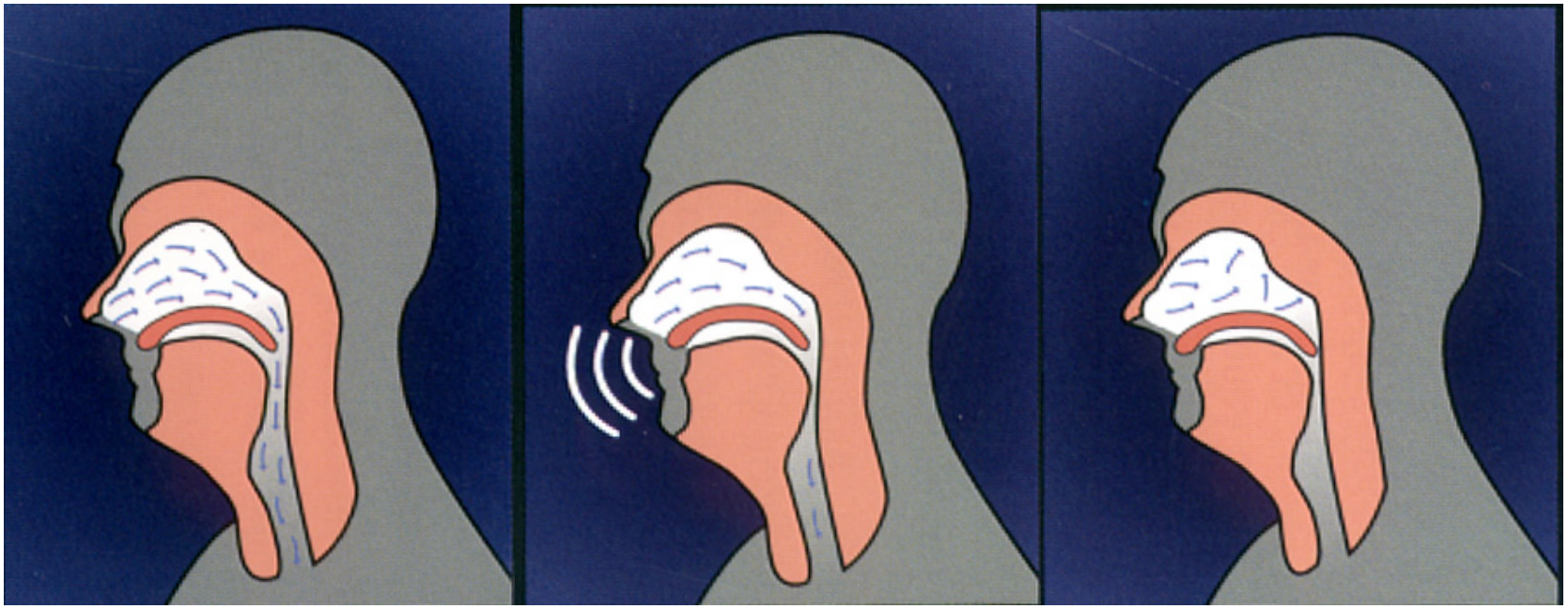
- Central Sleep Apneas
- Obstructive Sleep Apneas
- Mixed Apneas

WHAT IS OSA?

- Sleep disorder characterized by recurrent episodes of narrowing or collapse of pharyngeal airway during sleep despite ongoing breathing efforts.

- These often lead to
  - Acute derangements in blood gas disturbances
  - Surges of sympathetic activation
  - Periodic arousal from sleep (fragmented sleep)

WHAT IS OSA?



Normally during sleep, the muscles which control the tongue and soft palate hold the airway open

If these muscles relax, the airway narrows, causing snoring and breathing difficulties

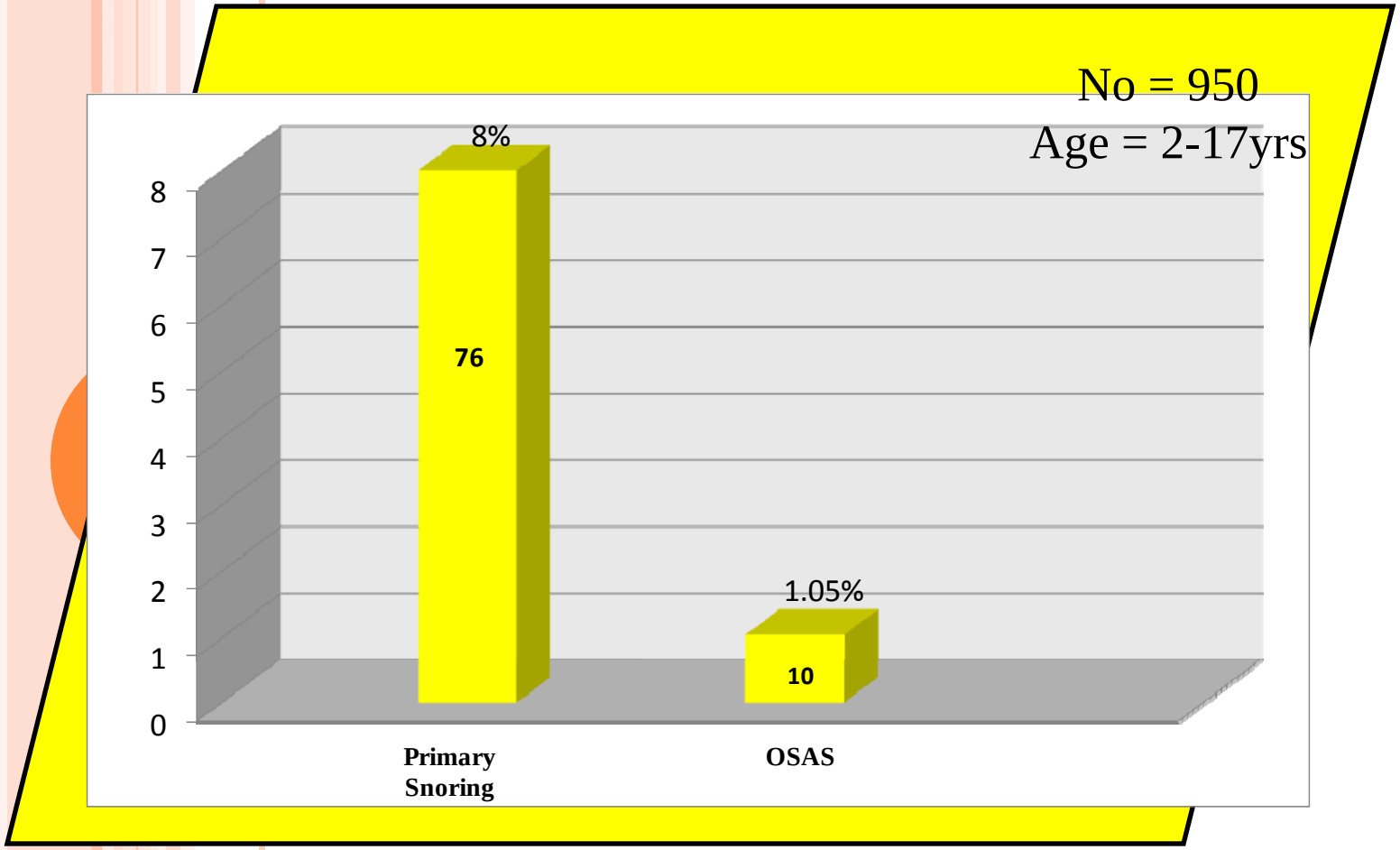
If these muscles relax too much or if Obstruction is present, the airway can become completely blocked, preventing breathing

SLEEP DISORDERED BREATHING : EPIDEMIOLOGY

- Incidence varies from 1-3% in children of preschool age in most western studies.
- In Indian studies among urban children, incidence was about 5% of all sleep disorders

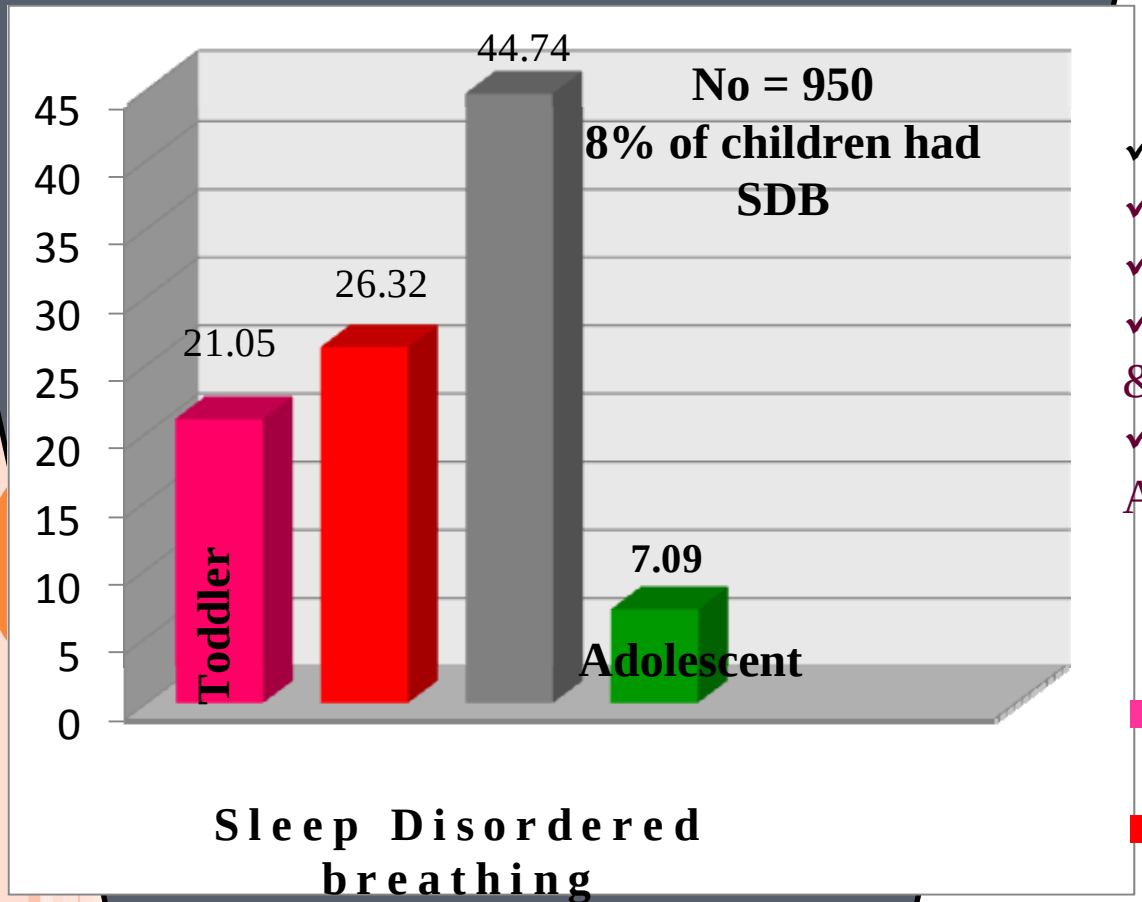
  Suri JC et al. Indian J Sleep Med 2008;3:0973-340X
  Bharti et al. Indian Pediatrics 2006; 43:35-38.
- More common in African-American and Asian children due to anatomic features of upper airway

Prevalence Of Snoring / OSAS



H Paramesh. Pankaj Sharma OSAS and DNB thesis Study 2010

Sleep Disorder Breathing (SDB) & Causes



- ✓Adenoid Hypertrophy = 50%
- ✓Allergic Rhinitis = 58%
- ✓ Asthma= 35%
- ✓Adenoid Hypertrophy = 7.9% & Asthma
- ✓Adenoid Hypertrophy, = 5.2% AR & Asthma

- Toddler
- Pre school
- School going
- Adolescent

DESCRIPTION OF OSA EVENT

- Decreased alveolar ventilation
- Decreased alveolar PO2 ; increased alveolar PCO2
- Decreased arterial PO2 ; increased arterial PCO2
- Stimulation of arterial chemoreceptor's; central chemoreceptor's
- Arousal/ partial awakening

WHY OBSTRUCTION OCCURS DURING SLEEP

- Supine position
- Control of breathing during REM sleep :
  - Lack of “wakefulness” drive
  - Decreased tone of pharyngeal muscles
  - Depressed reflexes, including pharyngeal dilator
  - Decreased tone of intercostals and accessory muscles (less effect on diaphragm)
  - Depression of minute volume
  - Depression of response to hypoxia

ANATOMIC FACTORS THAT PREDISPOSE TO OSA

1.Nasal :

- Anterior nasal stenosis
- Choanal stenosis/atresia
- Deviated nasal septum
- Seasonal/perennial rhinitis
- Nasal polyps/foreign body/hematoma

2.Nasopharyngeal/Oropharyngeal :

- Adenotonsillar hypertrophy
- Macroglossia
- Cleft palate/velopharyngeal flap repair

3.Craniofacial :

- Micrognathia/retrognathia
- Midface hypoplasia (e.g. down syndrome)
- Mandibular hypoplasia (e.g. Pierre Robin Syndrome)

FUNCTIONAL FACTORS THAT PREDISPOSE TO OSA

1.REM SLEEP RELATED PHARYNGEAL HYPOTONIA

2.ABNORMAL NEURAL CONTROL :

- ✓ Generalised Hypotonia (E.g. Down Syndrome)
- ✓ Global CNS Injury (E.g. Birth Asphyxia)
- ✓ Brainstem Dysfunction (E.g. Chiari Malformation)

3.DRUGS : sedatives/anesthetics/narcotics

4.OTHERS :

- ✓ **Obesity**
- ✓ Autonomic Dysfunction
- ✓ Excessive Oral Secretions

ATOPY & OSA

- In a large number of studies in adults with atopy, there is higher prevalence of snoring.
- A significant association between asthma and snoring [this effect is independent of upper respiratory tract symptoms (e.g. rhinitis), cigarette smoking, and race] has been found in children.
- Atopy is now recognized as an independent risk factor for snoring.

RISK FACTORS

- Male sex
- African American race
- Recurrent otitis media
- Asthma & persistent wheezing
- Tobacco smoke exposure and maternal smoking
- Past history of prematurity

OBESITY AND OSA

- The classic presentation of children with OSA as underweight children with adenotonsillar hypertrophy is being substantially replaced by young patients who are either overweight or obese.

- For every 1 kg/m2 increment in BMI beyond the mean BMI for age and gender, the risk of OSA increased by 12%.

- In obese, upper airway narrowing results from fatty infiltration of upper airway structures promoting pharyngeal collapsibility.

Gozal D *et al.* *Sleep* 29, A74 (2006).

- Obesity reduces the intrathoracic volume and diaphragmatic descent during inspiration, particularly in the supine position, resulting in lower oxygen reserves and increased work of breathing during sleep.

- Obesity is associated with peripheral and central leptin (an adipocyte-derived hormone) resistance, which in turn leads to relatively ineffective elevation of circulating leptin levels. Thus, reduced bioavailability of leptin resulting in altered ventilatory responses may also play a role in the interaction between obesity and OSA.

SYMPTOMATOLOGY

OSA manifestations can be categorized into following:

1. Sleep related symptoms
2. Daytime symptoms
3. Neurobehavioural consequences

SLEEP RELATED SYMPTOMS

- Snoring *(many children who snore do not have OSA, but very few children with OSA do not snore)*
- Breathing pauses
- Choking or gasping arousals *(may result in nocturnal awakenings, but are more likely to cause fragmented sleep)*
- Restless sleep
- Nocturnal diaphoresis
- Enuresis
- Unusual sleeping positions

(keep their neck hyper extended)

DAYTIME SYMPTOMS

- Morning headaches *(due to cerebral vasodilatation because of CO_2 retention)*
- Excessive daytime sleepiness (EDS) *(evaluated by Epworth sleepiness score)*
- Dry mouth
- Chronic mouth breathing
- Poor appetite and failure to thrive

NEUROBEHAVIORAL SYMPTOMS

- Deficits in attention*(Most children with SDB do not present with daytime sleepiness and are more likely to be hyperactive or inattentive, often being diagnosed with ADHD.)*
- Memory deficits
- Mood disturbance
- Subjective sleepiness
- Poor school performance

SIGNS OF OSA

Typically no respiratory abnormality while awake,however in longstanding cases patient may develop following:

- Arterial blood gases while awake may show metabolic alkalosis *(due to CO_2 retention).*
- Systemic hypertension *(Due to repeated increases in sympathetic tone resulting in vascular remodeling)*
- Pulmonary hypertension (Right Axis Deviation on ECG)
- Polycythemia
- Cor pulmonale
- Bradycardia during apneic event & tachycardia after airflow restoration

CRITERIA FOR DIAGNOSIS OF OSA :APNEA HYPOPNEA INDEX (AHI)

Total number of episodes of apneas and hypopneas averaged per hour of sleep is regarded as apnea/hypopnea index

- AHI < 5 » no OSA
- AHI 5-15 » mild OSA
- AHI 15-30 » moderate OSA
- AHI > 30 » severe OSA

DIAGNOSIS OF OSA

The following triad of symptoms is highly suggestive of OSA in children :

- Snoring
- Nocturnal breathing difficulties
- Witnessed respiratory pauses

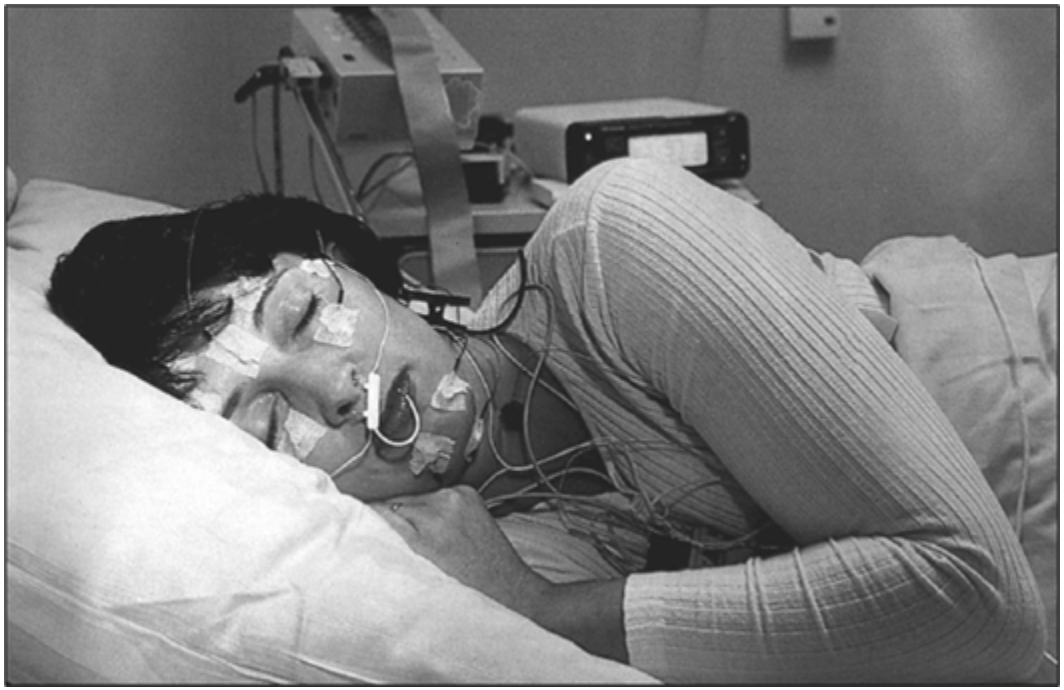
OVERNIGHT OXIMETRY

- Measures oxygen saturation & provides pulse rate data.
- Falls and rises in oxygen saturation are regarded as oxygen ‘dips’.
- Oxygen desaturation index (ODI) - number of times oxygen saturation falls by 4%
- ODI > 15 per hour » suggestive of OSA

POLYSOMNOGRAPHY

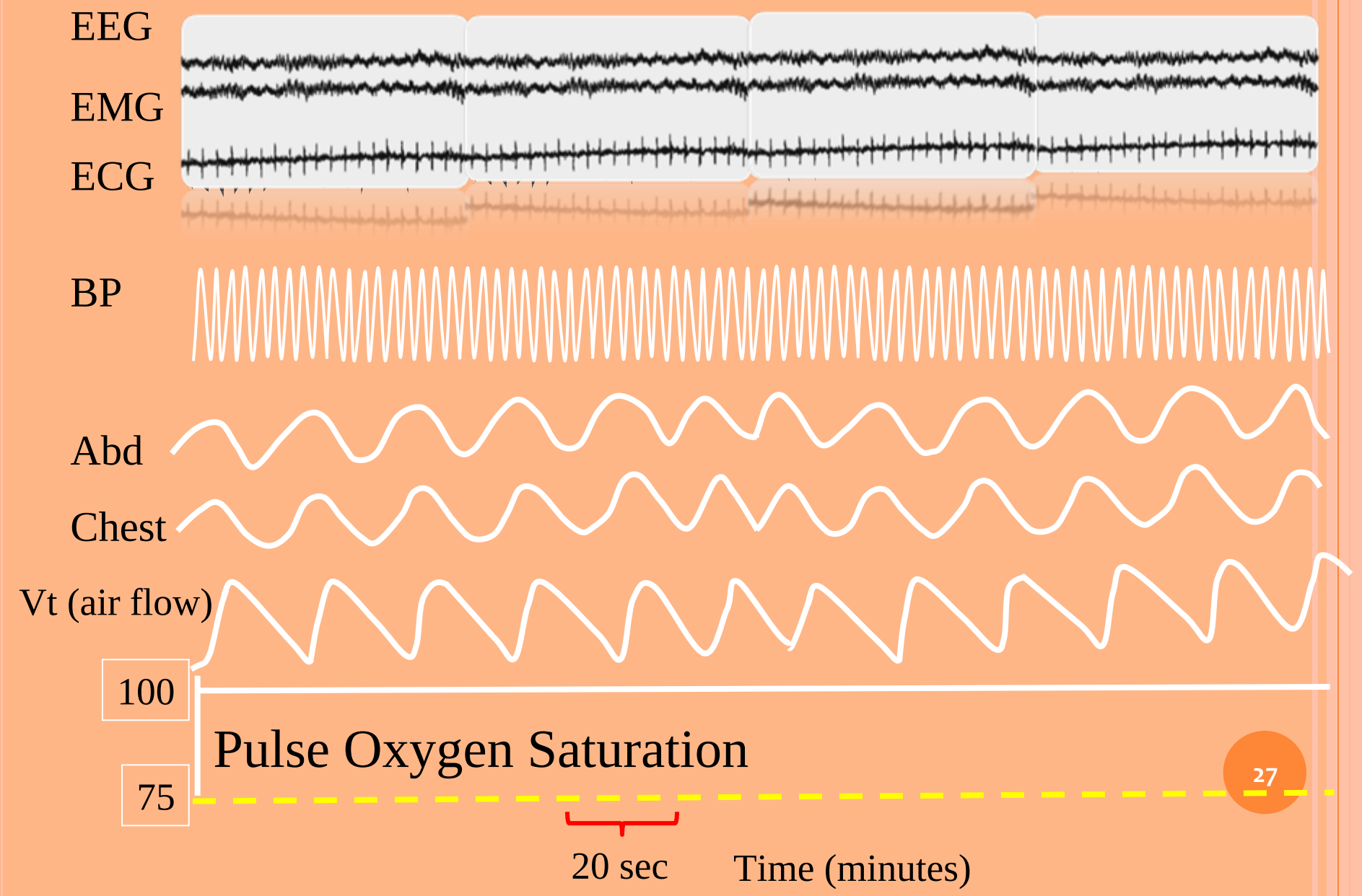
Variables that may be determined include:

- EEG and EOG *(for sleep state); EMG*
- Airflow at nose or mouth *(thermistor, pneumotachograph)*
- End-tidal CO_2
- Chest and abdominal motion *(impedance plethysmography)*
- ECG
- Blood pressure
- Pulse oximetry
- Esophageal pressure *(intrapleural pressure)*
- Autonomic nervous system activity *(finger tonometer)*

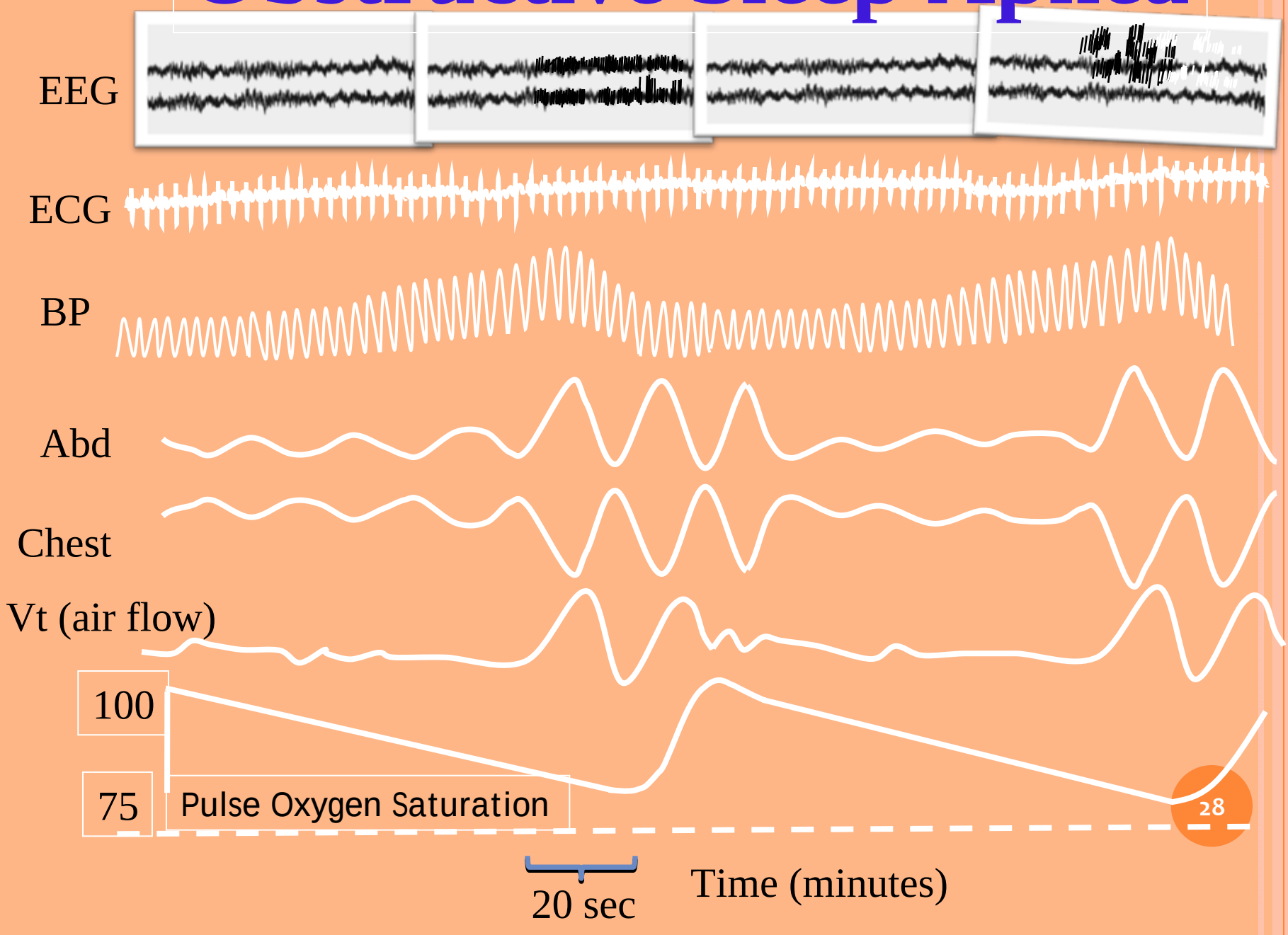


www.tmjsleepcenter.com

Normal Polysomnograph



Obstructive Sleep Apnea



OTHER INVESTIGATIONS

- Lateral neck radiograph

(To evaluate the size of the adenoids)

- Complete blood count *(To detect polycythemia suggestive Of chronic hypoxemia)*
- ECG & ECHO *(For evidence of Cor Pulmonale Or Right Ventricular Hypertrophy)*

PHARMACOLOGIC MANAGEMENT

- Topical nasal steroids (*for treatment of nasal obstruction to reduce snoring)*
- Steroids & antibiotics (*in acute management of infected pharyngeal tissues that have compromised upper airway patency*)
- Nasal decongestants (*for treatment of allergic rhinitis*)

SURGICAL TREATMENT

- In majority of cases of pediatric OSA, adenotonsillectomy is the first line of treatment

- Reported cure rates post adenotosillectomy range from 75-100% in normal healthy children

OTHER TREATMENT MODALITIES

1.Lifestyle modifications:

- Positional therapy (attaching a firm object, such as a tennis ball, to the back of a sleep garment to prevent the child from sleeping in supine position)
- Weight loss

2.Oral appliances : Continuous Positive Airway Pressure (CPAP)

3.Surgical:

- Uvulopalatopharyngoplasty
- Tracheostomy
- Mandibular distraction

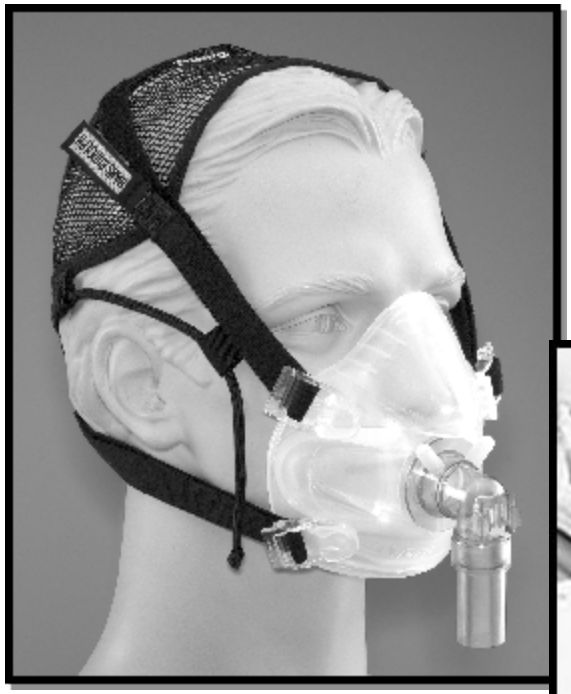
CPAP (NASAL CPAP / BIPAP)

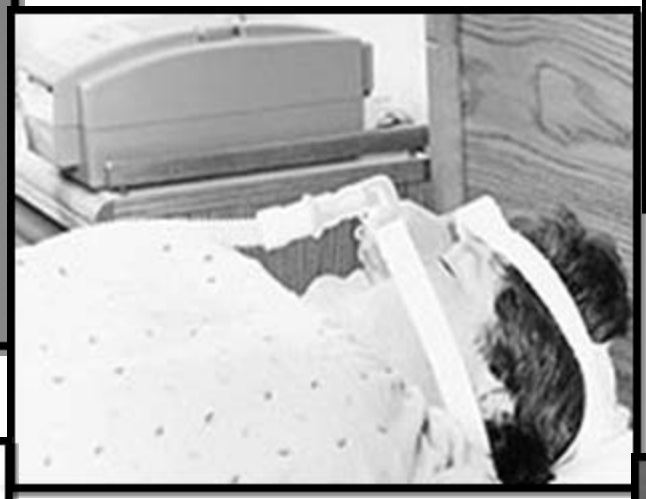
- Most common treatment in adults
- Can be used successfully in children and adolescents

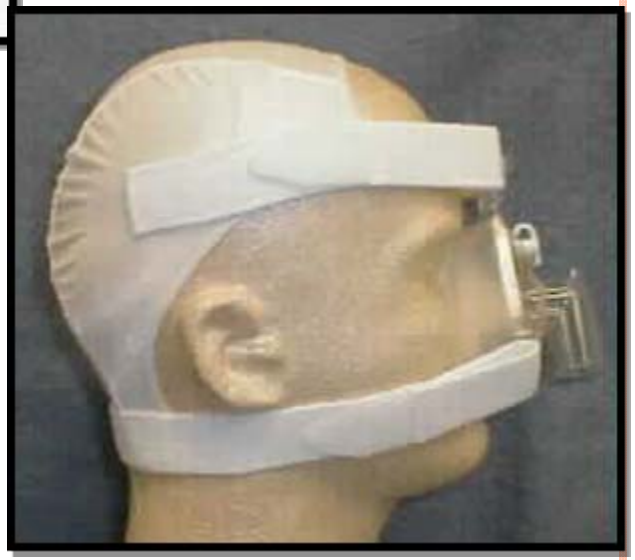
Indications:

- ✓ *Removal of tonsils/adenoids not indicated*
- ✓ *Residual disease following adenotonsillectomy*
- ✓ *Major risk factors not amenable to treatment with surgery (like obesity, hypotonia)*

- CPAP delivers humidified, warmed air through an interface (mask/nasal pillows) that, under pressure, effectively "splints" the upper airway open

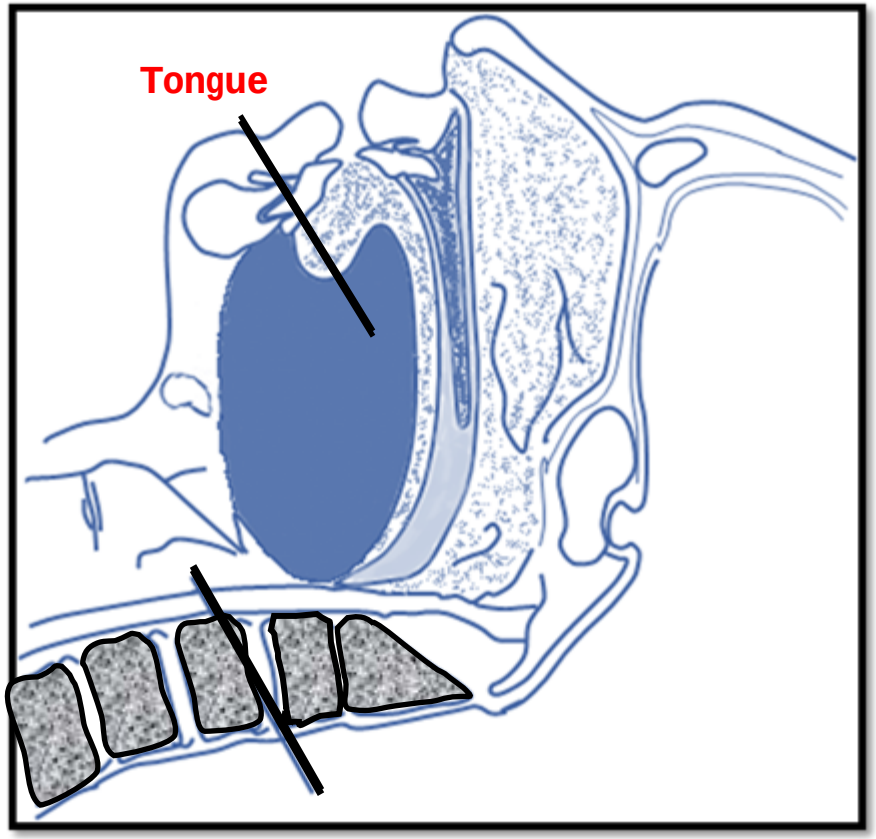






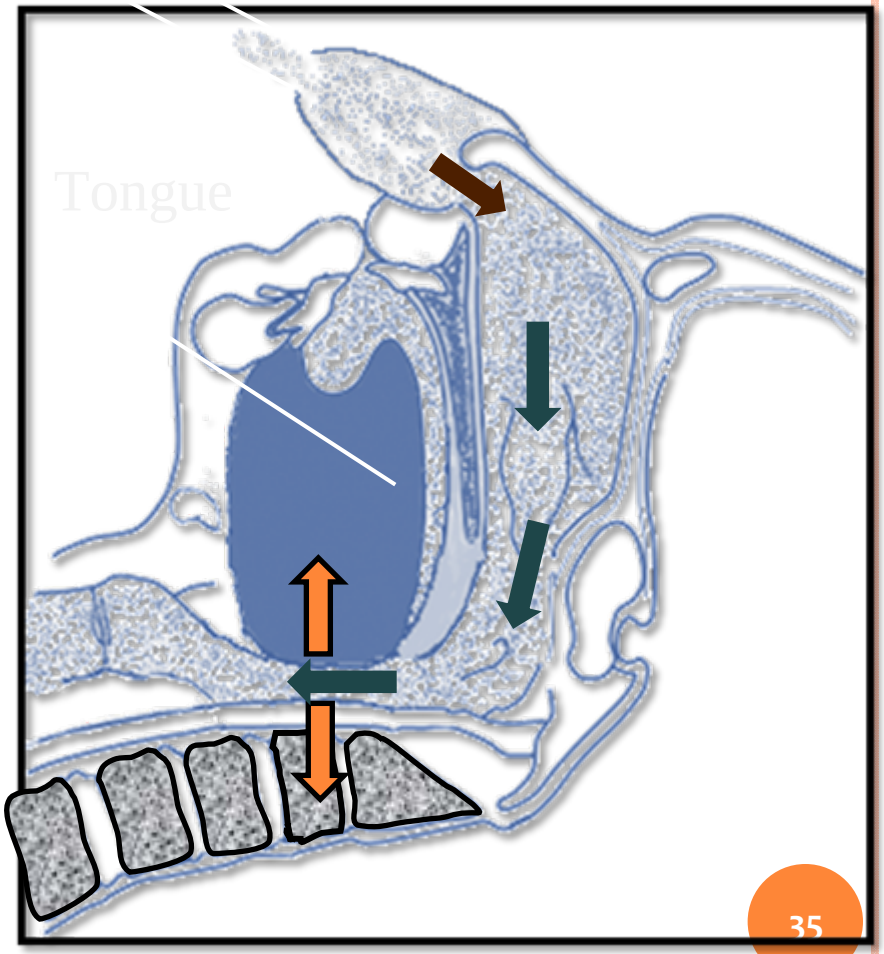
Obstructive Sleep Apnea

Sites Of Obstruction During Sleep Apnea



Laryngopharynx

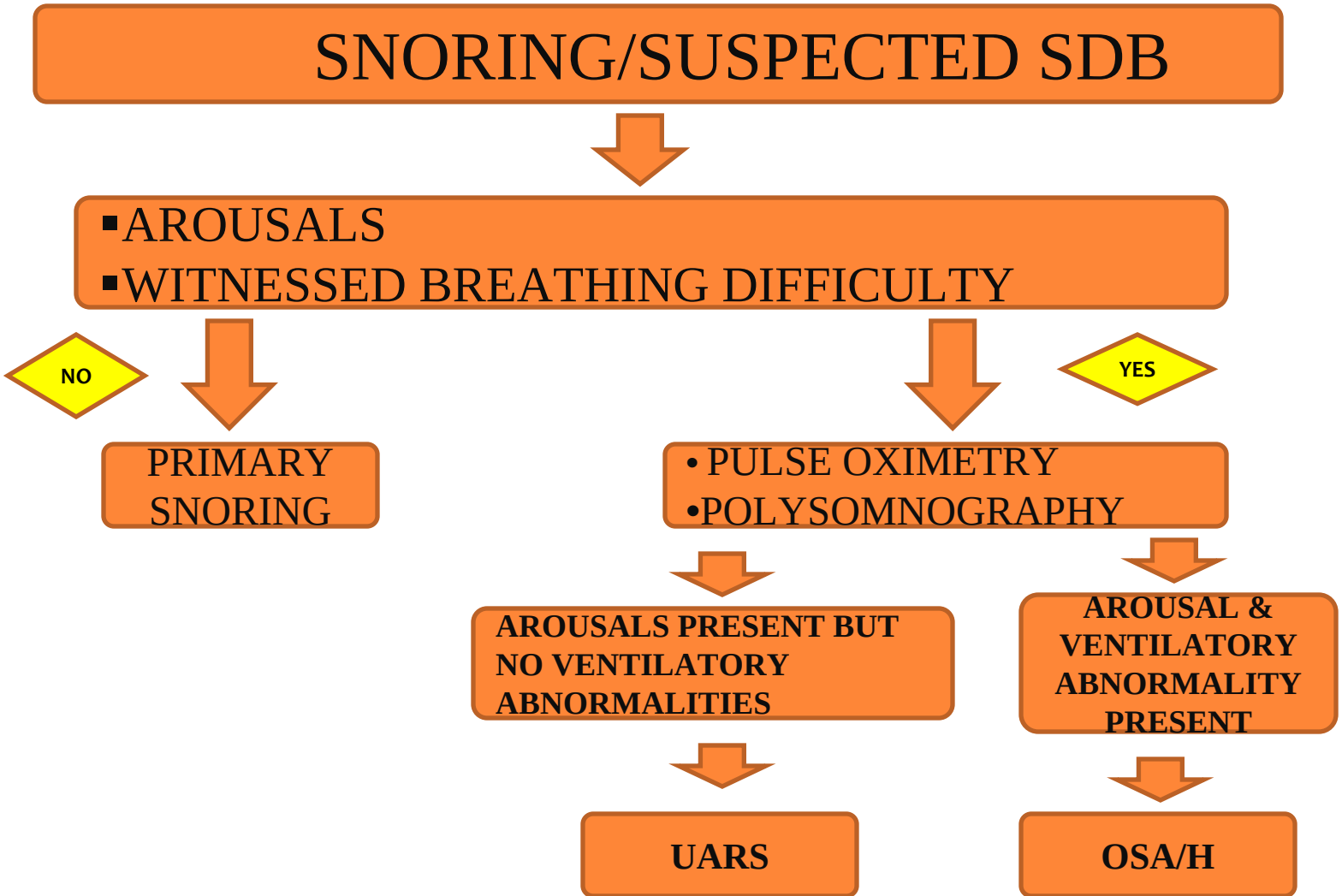
With CPAP



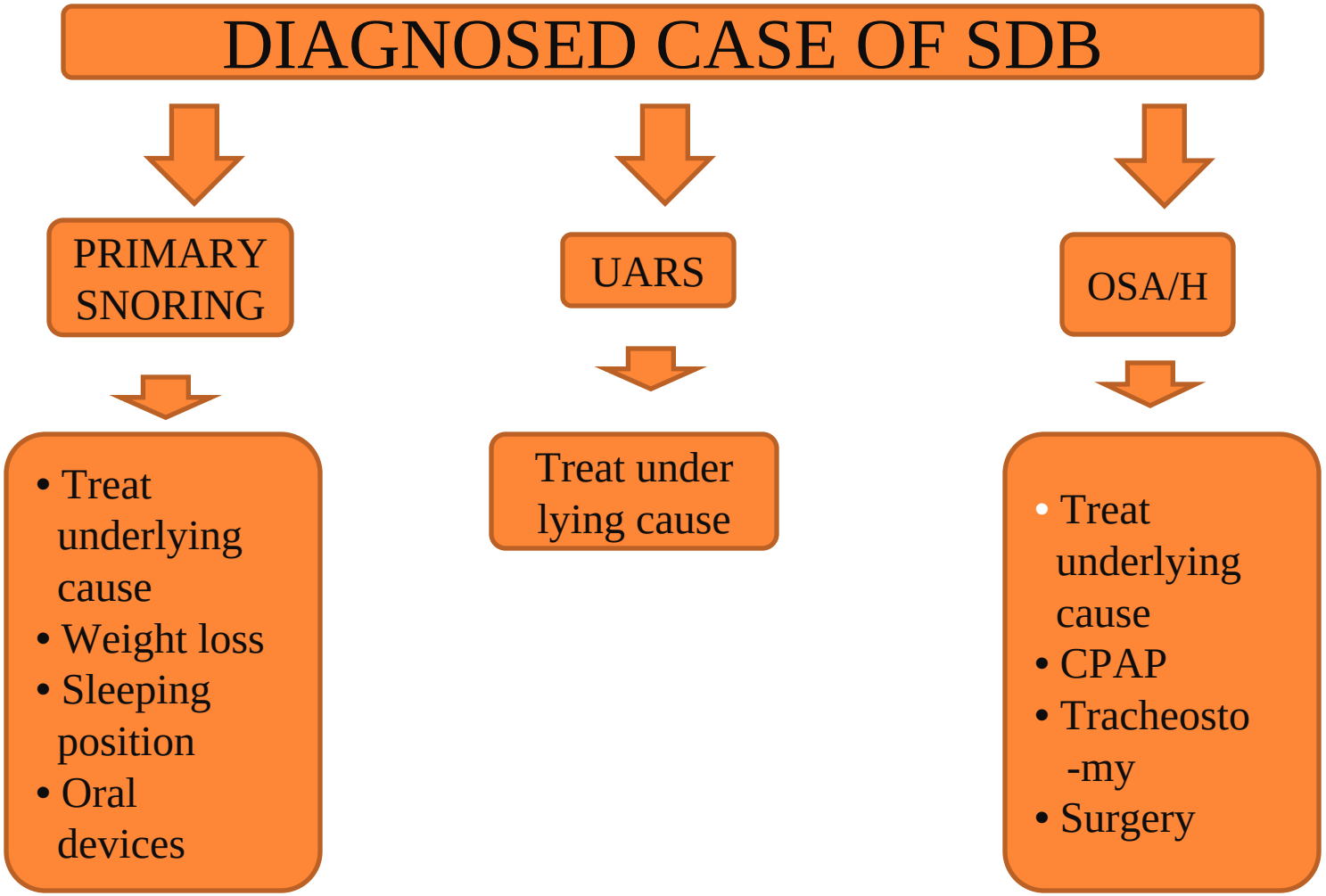
TRACHEOSTOMY

- Treatment of choice if severe upper airway obstruction is present in both wakefulness & sleep *(particularly when vocal cord dysfunction, impaired swallowing, or absent laryngeal protective reflexes exist)*
- May be necessary for severe OSA complicated by cor pulmonale when CPAP is unsuccessful or not tolerated
- Alternative is mandibular distraction osteogenesis/maxillomandibular reconstruction surgery

DIAGNOSTIC APPROACH TO A CASE OF SNORING



MANAGEMENT APPROACH TO A CASE OF SDB

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