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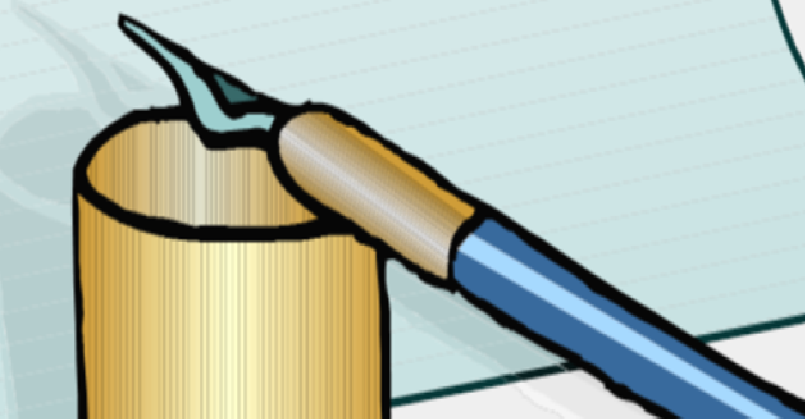

Introduction

Obstructive sleep apnea in children describes a continuum of disorders ranging in severity from primary snoring which is seen in 20% of children, to obstructive sleep apnea(OSA) which is seen in 2% of children ***(Lerman, 2009).***

The most common etiology of OSA in children is adenotonsillar hypertrophy. This becomes clinically significant in the 2-6 year age group as the adenoids and tonsils enlarge but the absolute size of the airway is still small. The tonsils may be large enough to meet in the midline "kissing tonsils" ***(Schwengel et al., 2009)***.

OSA Children have failure to thrive, central nervous system and cardiovascular complications. Failure to thrive is possibly due to increased work of breathing at night or reduced secretion of growth hormone. Central nervous system complications include memory, learning and behavioural problems ***(Wilkinson et al., 2000)***.

OSA Children may be obese and may suffer from systemic hypertension. Adenotonsillar hypertrophy may contribute in part to OSA and these children may benefit from tonsillectomy ***(Wilkinson et al., 2000)***.

Surgical and anesthetic preassessment should identify those children at high risk for complications after adeno-tonsillectomy ***(Brown et al., 2006)***.

Children with OSA should be anesthetized with care; deep anesthesia should be avoided and they are sensitive to opioids ***(Brown et al., 2006)***.

Those undergoing adenotonsillectomy should be cared in intensive care unit (ICU) postoperatively as the obstruction will not be relieved on the first postoperative night which may be due to swelling at the surgical site ***(Brown et al., 2006)*** .

Aim of the Work

Aim of the work is to diagnose children with OSA preoperatively, manage them intraoperatively and prevent postoperative complications.

PATHOPHYSIOLOGY

A. PATHOPHYSIOLOGY OF SNORING:

Snoring is one sign of number of different disorders. The sounds of snoring originate in the collapsible part of the airway. It involves the soft palate, uvula, tonsils, tonsillar pillars, base of tongue and pharyngcal muscles and mucosa. According to *Bonsignore et al.(2006)* four factors singly or in combination, contribute to snoring:

1. Incomplete tone of palatal, lingual muscles is the cause of the most child onset snoring. In deep sleep stages, such musculature fails to participate in the respiratory cycle to keep the airway open during inspiration. The dilator effect of the pharyngeal muscles and the protrusive effect of the genioglossus muscles are inadequate, so the tongue falls backward into the airway and vibrates against the flaccid soft palate, uvula, and pharyngeal tissues. This is exaggerated when the child consumed sedative hypnotics, tranquilizers, or antihistamines. Hypothyroidism also contributes to poor muscle tone, apnea, snoring and as do neurological disorders such as cerebral palsy, muscular dystrophy, and myasthenia ***(Bonsignore et al., 2006)***.

2. Space-occupying masses encroaching on the airway can contribute to snoring. In children, snoring is almost always

from enlarged tonsils and adenoids. Retro-or micrognathia produces a tongue that is large relative to the space available for it to occupy, but Down's syndrome and Acromegaly produce an absolute tongue enlargement ***(Bonsignore et al., 2006)***.

3. Excessive length of the soft palate and uvula narrows the nasopharyngeal aperture ***(Bonsignore et al., 2006)***.

4. Restriction to airflow in the nose creates increased negative pressure during inspiration which draws together the flaccid tissue in the collapsible part of the airway where they vibrate and cause snoring during an allergy attack or cold ***(Bonsignore et al., 2006).***

B. PATHOFHYSIOLOGY OF SLEEP APNEA:

Obstructive sleep apnea (OSA) is characterized by asymptomatic, recurrent episodes of complete or partial upper airway obstruction during sleep. These episodes of obstruction are associated with recurrent oxyhemoglobin desaturations and arousals from sleep. OSA associated with excessive daytime sleepliness (EDS) is commonly called Obstructive Sleep Apnea Syndrome (OSAS) ***(Mahowald, 2006).***

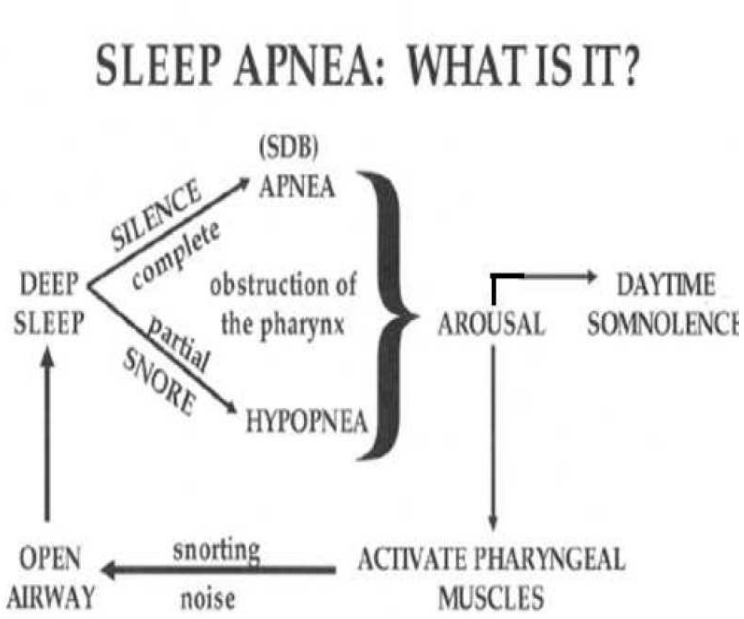

Figure (1): pathophysiology of obstructive sleep apnea ***(Mahowald, 2006)***

Incidence

Age

The prevalence of OSAS increases with age ***(Young et al., 2002)*.**

Sex

OSAS occurs in children of all ages with a prevalence rate of 2%. 20

Race

The prevalence of sleep apnea in African Americans appears to be greater than in white Americans.

Three major factors are now recognized as critical in determining whether the upper airway collapses or remain open during sleep ***(Mehra et al., 2006).***

1- The muscle activity of the dilators of pharyngeal airway.

2- The negative pressure generated during inspiration.

3- The structural anatomy of the airway.

Most patients with obstructive sleep apnea syndrome (OSAS) demonstrate upper airway obstruction at either the level of the soft palate (i.e. nasopharynx) or the level of the tongue (i.e. oropharynx) ***(Mehra et al., 2006).***

Obstruction can occur during any sleep state but often noted during rapid eye movement (REM) sleep. Nasal continuous positive airway pressure can ameliorate the situation by keeping the pressure in the upper airway positive, thus acting as a splint to maintain airway patency ***(Schwab et al., 2003).***

Upper airway neuromuscular activity- including reflex activity- decreases with sleep and this decrease may be more pronounced in patients with OSAS ***(McGinley et al., 2008).***

Reduced ventilatory motor output to upper airway muscles is believed to be the critical initiating event leading to upper airway obstruction; this effect is most pronounced in patients with an upper airway predisposed to collapse for anatomical reasons as shown in table (1) ***(McGinley et al., 2008)*.**

Table (1): Anatomical causes of obstructive sleep apnea in children ***(McGinley et al., 2008)***

	CAUSES
Nose	Choanal atresia, polyposis, dermoid cyst, tumours (gliomas) teratomas, fibrous histiocytomas, encephaloceles, foreign bodies.
Nasopharynx	Adenoidal hypertrophy, stenosis, pharyngeal flap for soft palate, tumours.
Mouth and Oropharynx	Hypertrophied tonsils, adipose tissue*, macroglossia (lingual hemangioma or lingual lymphangioma), micrognathia, tempromandibular joint ankylosis.
Larynx	Tracheal atresia, intrinsic tracheal lesions, extrinsic compression (i.e. goiter), laryngeal and tracheal webs.

(*)Obese patients with OSAS typically have increased amounts of adipose tissue in their oral and pharyngeal tissues, including the uvula, tonsils, tonsillar pillars, tongue, aryepiglottic folds, and lateral pharyngeal walls, changing the shape of the oropharynx to an ellipse with a short transverse and long anteroposterior axis**.** OSAS correlates closely with hypertrophy of adenotonsillar complex prevails as dominant etiology ***(McGinley et al., 2008)*.**

The severity of OSAS

The ASA Guideline recommends that the practitioner decides or determine the severity of OSA as mild, moderate or severe (scored as 1, 2 or 3, respectively) ***(Chung et al., 2007).***

There are only two ways to determine the severity of OSA and they are by clinical impression and polysomography ***(Chung et al., 2007).***

Assessment of severity by polysomnography is based on the apnea/hypopnea index (AHI) ***(Patil et al., 2007).***

Apnea/hypopnea index (AHI):

It is derived from the total number of episodes of apnea and hypopnea divided by the total sleep time ***(Patil et al., 2007).***

The severity of obstructive sleep apnea syndrome (OSAS) is classified and scored as follows :

- Mild =5-15 apneas per hour
- Moderate disease—AHI of 16 to 30 events per hour
- Severe disease—AHI of greater than 30 events per hour
- Scoring is 1= mild, 2= moderate, 3= severe.

The pathophysiology of snoring and obstructive sleep apnea is determined by a number of predisposing factors for OSA as shown in Table (2) ***(Loadsman and Hillman, 2001).***

Table (2): predisposing factors for OSA ***(Loadsman and Hillman, 2001)***

Condition	Examples	Contribution
Obesity, body fat Distribution	obesity, Prader-Willi syndrome	Complex and ill-defined
Race/genetics		Anatomical similarity
Age		Tissue laxity
sedatives Analgesics, anesthetics		Muscle relaxation,depresse d arousal
Nasal obstruction	Septal deviation, chronic nasal congestion	Increased pharyngeal negative pressure
Pharyngeal obstruction	Tonsillar and adenoidal hypertrophy	
Cranio-facial abnormality	Down's Pierre-Robin	macroglossia micrognathia
Laryngeal obstruction	Laryngomalacia, tracheomalacia	Laryngeal collapse
Endocrine/Metabolic	Hypothyroidism Cushing's Stroke poliomyelitis	Upper airway infiltration or myopathy, obesity
Neuromuscular disorders	Myotonic dystrophy, dysautonomia, tetraplegia	Disordered pharyngeal nemomuscular function

The clinical features and complications associated with the obstructive sleep apnea syndrome essentially result from the two main effects of the obstructive episode, which are hypoxia and the generation of high negative pressure as shown in figure (2) ***(Carlson et al., 1994)***.

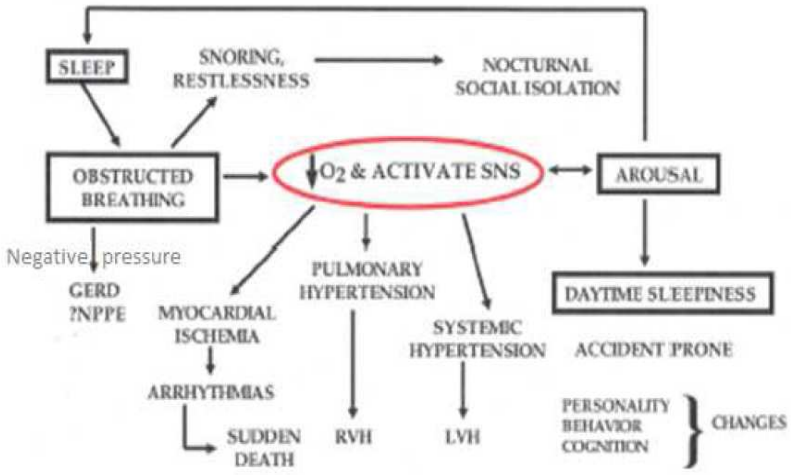

Figure (2): Clinical picture and complications of OSA ***(Carlson et al., 1994)***

I-Hypoxia

During an apneic episode there is no air flow into the lungs, thus if the episode is long enough the patient will become hypoxic and hypercapnic. The degree of hypoxia will be determined by the duration of the apneic event, lung volume (which is reduced in obesity) and any coexistent neuromuscular or cardiopulmonary disorders ***(Carlson et al., 1994)***.

Hypoxia is associated with a rise in sympathetic output and catecholamine production and the resulting peripheral vasoconstriction causes transient pulmonary and systemic hypertension ***(Carlson et al., 1994).***

Levels of many different inflammatory mediators are increased in patients with OSAS**.** Thus result in multiple health risks, particularly those of cardiovascular- and airway-related origin ***(Selmi et al., 2007).***

They include:

- Long standing pulmonary hypertension.
- Systemic hypertension.
- Ischemic heart disease.
- Metabolic syndrome.
- Bradycardia.
- Stroke.
- Polycythemia.

- ***Long standing pulmonary hypertension*** was thought to develop only in patients with coexisting chronic obstructive airway disease but it has been found in patients without any respiratory diseases**.** This should alert for possible pulmonary disease and need for postoperative vigilance ***(Chaouat et al., 1996)***.

- ***Systemic hypertension*** is observed in 50-70% of patients with OSAS ***(Peppard et al., 2000).***

In time, the hypertension becomes fixed, which in turn causes hypertrophy in one or both of the respective ventricles; the hypoxemia can cause myocardial ischemia which can cause myocardial arrhythmias and sudden unexpected death ***(Benumof, 2001).***

- ***Ischemic heart disease:*** The combination of intermittent hypoxemia and increased sympathetic discharge shifts the balance between myocardial oxygen supply and demand ***(Sabers et al., 2003).***

- ***Metabolic syndrome*** refers to clustering of a group of defined metabolic and physical abnormalities. Children with metabolic syndrome commonly have abdominal obesity, reduced levels of high-density lipoprotein (HDL), hyperinsulinemia, glucose intolerance, hypertension, and other characteristic features (endothelial dysfunction, microalbuminuria, polycystic ovary syndrome, hypoandrogenism, nonalcoholic fatty liver disease, hyperuricemia) ***(Liberopoulos et al., 2005).***

Clinical criteria for diagnosing metabolic syndrome require at least three of the following described in table (3) ***(Liberopoulos et al., 2005).***

Table (3): Clinical Criteria for Diagnosing Metabolic Syndrome ***(Liberopoulos et al., 2005)***

Criteria	Defining Value
Abdominal obesity	Waist circumference >102 cm in men and >88 cm in women
Triglycerides	>or =150 mg/dL
High-density lipoprotein Cholesterol	<40 mg/dL in men and <50 mg/dL in women
Blood pressure	>or =130/85 mm Hg
Fasting glucose	> or =110mg/dL

*Three of five criteria must be met.

- **Bradycardia:** During an obstructive episode the effort of trying to inspire against closed glottis results in increased vagal activity with subsequent bradycardia. The presence of hypoxia significantly worsens the bradycardia ***(Carlson et al., 1994).***

- **Stroke:** An AHI of greater than 5 was associated with increased risk of stroke ***(Munoz et al., 2006).***

- **Polycythemia**: associated with prolonged hypoxia ***(Carlson et al., 1994).***