Obstructive sleep apnea (OSA) in children is one of the most common problems encountered by the otolaryngologist. It was described frequently in adults but was not clearly defined as of true medical significance in children until 1976. Since then, rapid advances in technology and increasing recognition have propelled pediatric sleep apnea into both fame and notoriety.

Snoring is the hallmark of sleep disordered breathing. It occurs in up to 27% of school-aged children and peaks at 2-8 years. This is coincident with the peak in size and degree of immunologic activity of the tonsils. The reported prevalence of sleep apnea in this age group is 2-3%. Snoring again increases in children 15 years and above with nearly half the males and a third of the females snoring habitually. Characteristics of pubertal children with OSA closely mimic adult patterns and are usually addressed as such.

Airway collapse in OSA is dictated by many factors. Anatomic obstruction caused by adenotonsillar hypertrophy is the most readily recognizable etiology. Certain craniofacial characteristics also result in a smaller airway. Moreover, functional pharyngeal muscle tone varies in response to sleep state, pressure-flow airway mechanics and respiratory drive to determine the cross sectional area of the upper airway. In children with primary snoring, narrowing occurs at the level of the soft palate. In those with OSA, collapse is at the level of the tonsils and adenoids. Interestingly several researches have failed to demonstrate significant correlation between adenotonsillar size and OSA. This discrepancy is now being attributed primarily but not solely to the increased incidence of childhood obesity. Upper airway, neck, chest and abdominal fat deposition give rise to upper airway narrowing, increased mass loading, decreased chest and diaphragmatic excursions. These result in an obstructive as well as restrictive pattern of respiratory compromise. Although obese children may have concomitant adenotonsillar hypertrophy, addressing this exclusively rarely leads to resolution of OSA.

The consequences of untreated childhood OSA encompass a broad range of morbidities including behavioral disturbances and learning deficits, cardiovascular disease, metabolic disturbances, somatic growth compromise, decreased quality of life and psychiatric illness. Mouth breathing is a clinical presentation worthy of special mention. It has been known that adenoid hypertrophy in chronic mouth breathing leads to “adenoid facies.” This is characterized by an incompetent lip seal, narrow upper dental arch, increased anterior face height, steep mandibular plane angle, and a retrognathic mandible. Craniofacial development progresses rapidly and retains its plasticity until early puberty (12 or 13 years). Thereafter, growth slows down as the adult face begins to set. If mouth breathing is left
untreated by this age the probability that the child will eventually develop adult-pattern OSA is greater. Fortunately, not all children develop these complications. Environmental exposure and genetic susceptibility certainly play a role in making a child more vulnerable to the effects of OSA. On the other hand, some of these morbidities may not be completely reversible despite treatment. Therefore, timely and appropriate management of OSA is crucial in ensuring conditions for optimal development.

Tonsillectomy and adenoidectomy remain the primary mode of treatment for childhood OSA. Fear of rheumatic fever and its complications traditionally prompted immediate removal of the tonsils and adenoids. In 1978, OSA was not documented as an indication for tonsillectomy. An increasing trend was demonstrated such that in 1986 19% of cases were due to OSA. By 2003, upper airway obstruction was the reason for surgery in 96% of tonsillectomies performed in children less than 36 months over a period of 2 years in a tertiary center.

A meta-analysis of 14 studies reporting polysomnographic outcomes of tonsillectomy and adenoidectomy showed a summary success rate of 82.9%. Significant improvement in quality of life based on validated questionnaires measuring sleep disturbance, physical symptoms, emotional symptoms, hyperactivity and daytime functioning without supporting polysomnographic results have also been reported. However, this still leaves a number of children with residual disease. Readily identifiable risk factors for surgical failure are untreated nasal obstruction, maxillomandibular deficiency, obesity and a high respiratory index. Further treatment using medications, additional surgery or positive airway pressure therapy is usually necessary for this group of patients.

The first 24 hours after surgery is probably the most critical time for developing complications. Patients have deeper sleep due to chronic poor sleep quality and sedation or may be placed in supine position. OSA as an operative diagnosis automatically increases the risk of the patient. Other factors are low weight, obesity especially those with associated co-morbidities (hypertension, asthma and type II diabetes), age less than 3 years and those with severe pulmonary hypertension. Identified problems are supraglottic obstruction, breath holding, desaturation on induction and emergence. In children less than 6 years 6.4% experienced morbidities which were primarily respiratory. More than half of these children (57.7%) had desaturations necessitating use of an artificial airway via nasopharyngeal airway or endotracheal intubation and 18% had significant chest findings on radiograph particularly atelectasis, infiltrates and pulmonary edema.

Children less than 3 years old have nearly a 2-fold increased risk for respiratory complications.

Risk-assessment prior to surgery is essential in achieving a safe perioperative outcome. Close coordination with other concerned physicians particularly pediatric subspecialists and anesthesiologists is fundamental. The hospital wherein the procedure will be conducted should have provisions for thorough intra- and post-operative monitoring. The decision to admit to the ICU after surgery is dictated by severity of illness, the presence of co-morbidities and young age.

Pediatric obstructive sleep apnea is an entity in evolution. Heterogenous patient profiles especially in the face of rising obesity, changing syndrome definitions and polysomnographic parameters, innovations in treatment and even legal issues will continue to challenge every otolaryngologist. Notwithstanding, otolaryngology should remain in the foreground in treating pediatric OSA. Despite attendant risks and limitations, pediatric sleep surgery in the hands of the informed otolaryngologist is still the most useful tool in helping children recover from sleep disordered breathing.
REFERENCES


