# Sleep Disorders Associated with Upper Airway Obstruction in Children

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Acute upper airway obstruction is a well-recognized, easily diagnosed medical emergency. Chronic persistent or intermittent upper airway obstruction, however, may not be obvious on routine physical examination of the awake child, but its consequences may be just as severe. Obstruction may increase in the supine position or be further accentuated by sleep. Sleep disorders with associated ventilatory disturbance are not uncommon in these children and may lead to a wide variety of physical and behavioral disorders. Some children progress to florid heart failure and eventual death if the diagnosis is delayed. With the aid of modern technology, we are now able to recognize the process at its earlier stages, but we are just beginning to realize the great impact of this problem.

## Etiology and Pathophysiology

In children the most common cause of chronic airway obstruction and sleep-related obstructive phenomena is hypertrophy of the nasopharyngeal lymphoid tissue, specifically the adenoids and tonsils. However, obstruction at any site along the upper airway may cause similar disturbances, as seen with glottic web, vocal cord paralysis, nasal septal deviation, and the Pierre-Robin syndrome. Cinefluoroscopy during sleep in children with obstructive sleep apnea shows that the soft palate falls backward against the posterior pharynx, the tongue moves posteriorly, and the lateral walls of the hypopharynx approximate medially during inspiration. The child gasps in an effort to overcome the obstruction, arousing partially, with loud snoring marking the resumption of air flow. In children without a history of sleep-related problems, the hypopharynx remains patent during sleep. The fact that not all children with large tonsils and/or adenoids exhibit sleep disturbances suggests that central nervous system control of respiration and muscle tone may play a large part in this problem.

## Laboratory Findings in Children With Sleep Apnea

With the increased awareness of the importance and diverse effects of sleep disorders, special laboratories have been established to monitor electrographically physiologic variables during sleep, a process called *polysomnography*. The specific approach varies somewhat between laboratories but often includes continuous monitoring of the EEG, EKG, respiratory effort by chest and/or abdominal strain gauges, or an endoesophageal pressure transducer), nasal airflow, extraocular movements, chin muscle tone, and arterial oxygen saturation. Audiovisual monitors may also be included. With this information the occurrence, duration, and type of apneic episodes can be noted as well as any accompanying changes in heart rate or rhythm, and arterial oxygen saturation. The amount of time spent inthe various sleep stages can also be quantitated, and movements and behaviours during sleep may be noted. These studies are important in diagnosing the nature of the sleep disorder (such as central or obstructive apnea) and the severity of the problem. Using polysomnography, Guilleminault has defined the respiratory irregularities during sleep. A *respiratory pause* is defined as

cessation of airflow for less than 10 seconds; an *apneic episode* is a cessation of airflow for more than 10 seconds. A *sleep apnea syndrome* is defined as 30 episodes or more of apnea during a seven-hour sleep period. These definitions, however, vary with different investigators, and the boundary between normality and abnormality is not yet well defined.

Apnea is of several types, depending on its etiology. *Central apnea* is the cessation of breathing owing to the failure of the respiratory center to initiate a respiration. This is recorded by abscence of chest movement or chane in endoesophageal pressure, and the lack of nasal and oral airflow. *Obstructive apnea* is unsuccessful airflow despite a respiratory effort, as seen by increased chest wall or abdominal movements and increased negative endoesophageal pressure. *Mixed apnea* is a combination of these two types, being the absence of respiratory efforts followed by unsuccessful respiratory efforts. In another study by Guilleminault eight children diagnosed as having a sleep apnea syndrome were monitored, and a range of 78 to 816 apneic periods was noted during one nocturnal sleep period. Obstructive apnea was noted in 83 per cent of these episodes, with the remainder equally divided between central and mixed episodes. Eliaschar reported a series of 14 children with symptoms of moderate to severe upper airway obstruction. On polysomnographic monitoring, five patients had only central had central, obstructive, and mixed episodes. However, even "normal" children were found to have some apneic periods. Seven children without upper airway obstruction had an average of 21 central apneic episodes per night. Carskadon et al looked at 22 normal children in which an average of 18 central respiratory pauses per night were found, half of these being less than 10 seconds in duration. In the eight children with predominantly obstructive apnea described by Guilleminault, each apneic episode was accompanied by oxygen desaturation, at times down to 40 mm Hg. The oxygen saturation value, as determined by ear oximetry, depended mainly on the length of the apneic period, and was generally lowest following obstructive apnea, as compared with the centraland mixed types. Tilkian suggests that this phenomenon may be caused not only by hypoventilation but by ventilation-perfusion mismatch in the lungs during respiratory obstruction.

To assess more accurately the degree of hypoventilation experienced by children with a history of sleep apnea, arterial blood gas measurement has been employed. In some severely affected children increased $pCO_2$ and decreased $pO_2$ may be found even in blood samples from the awake child. In many children, however, an indwelling arterial catheter must be inserted in order to compare blood gas values in the awake child often having normal values with those values when the child is sleeping and presumably experiencing the greatest obstruction.

Hemodynamic changes caused by airway obstruction are also seen. Radiographs of the chest may reveal cardiomegaly and there may be changes of right-sided hypertrophy on an electrocardiogram. Cardiac catheterization data are mostly limited to those children who have presented with cardiac complications, usually frank congestive heart failure. Features of cor pulmonale are found, such as righ heart enlargement with elevated right-sided pressures and pulmonary hypertension. In some, evidence of left ventricular dysfunction is also apparent. When relief of the airway obstruction is obtained during catheterization, the abnormal values are seen to normalize. Abnormalities of systemic blood pressure are also seen in patients with airway obstruction. Guilleminault reported that systemic hypertension occurred in five of the

eight patients in his series. Blood pressure returned to normal following relief of the airway obstruction. In adults with predominantly obstructive sleep apnea, the normal pattern of decreased systemic arterial pressure during sleep did not occur and cyclic elevations in systemic arterial pressures began with the apneic episode. This was attributed to hypoxemia triggering sympathetic discharge and peripheral vasoconstriction. Disturbances in cardiac rate and rhythm have also been noted. Guilleminault reported marked sinus arrhythmias associated with sleep apneic episodes. Massumi describes a child with bradycardia in association with obstructive episodes.

## Clinical Picture of Children With Sleep Apnea

Several behavioral aberrations have been noted in patients with sleep apnea. Excessive daytime sleepiness is quite often the presenting complaint, and may range from lethargy and increased napping to frank "sleep attacks" resembling narcoleptic episodes. However, in contrast to narcoleptic patients, these patints do not feel refreshed or rested after these sleep periods. During these daytime sleep attacks, apneic episodes may be observed. These sleep attacks may be secondary to both disordered nocturnal sleep and to CNS effects of cardiorespiratory dysfunction. Paradoxically, these children may appear hyperactive, as they move around trying to avoid falling asleep. The effects on school performance and mental functioning may be marked, with the children variously diagnosed as hyperactive, learning disabled, and in some cases mentally retarded. It is not known how much of a role the repetitive hypoxemia plays.

Nighttime behavious is also affected. In Guilleminault's series of eight cases of sleep apnea syndrome, three children fought going to sleep in their own rooms, and two of the children reported hypnagogic hallucinations. When children with the sleep apnea syndrome do fall asleep, loud snoring is reported nearly universally. Rather than lying supine, some of these children may require several pillows or may even prefer to sleep in a sitting position. Depending on the severity of the obstruction, sleep, as monitored by EEG, becomes more disturbed, with microarousals at the end of apneic episodes just prior to resumption of respiration. Parents have described their children's sleep as very restless, with abnormally increased body movement, sometimes accompanied by somniloquism and somnambulance. Enuresis occurring in previously night-dry children has also been reported to occur in patients with sleep apnea. Guilleminault reported the recurrence of enuresis in seven of the eight children in that series. It was noted that these enuretic episodes occurred all during the night, in contrast to the occurrrence of primary enuresis mainly in the first third of the night (during slow-wave sleep).

The sleep apnea syndrome has been associated with both overweight and underweight conditions, and occurs in children of normal weight. In the series of eight children reported by Guilleminault et al five were underweight and two were overweight. Daytime somnolence was present in both groups. Stool et al described three children with recent weight gain and obesity, airway obstruction, and somnolence. These children experienced some degree of airway obstruction prior to their weight gain, which in turn seemed to increase their symptoms. After surgical relief of the obstruciton, two children lost weight; the third was lost to follow-up. Menashe also reported one child who became obese during the illness but lost weight postoperatively.

## Diagnosis

History and physical examination are the first steps in evaluating the degree and cause of chronic airway obstruction leading to sleep disorders. A detailed description of the child's sleeping habits must be obtained, noting excessive daytime sleepiness, sound or restless sleep, snoring, respiratory pauses or apneic periods, associated cyanosis and diaphoresis, position during sleep, nightmares, enuresis, and peculiar behaviors. Other associated symptoms include morning headaches, weight changes, dysphagia, behavior and mood changes, and declining school performance.

The physical examination of the awake patient may provide helpful clues to the source of the obstruction. Mouthbreathing, with or without noisy respirations at rest, may be noted. The nose must be examined for conditions and lesions which may be contributing to the obstruction, such as signs of allergy, infectious rhinorrhea, polyps, and anatomic problems such as a deviated septum. Direct or indirect visualization of the adenoidal mass may be possible in some children. Examination of the throat includes noting tonsillar size in relation to the size of the oropharynx. Speech qualities should also be noted, especially hyponasal speech suggesting nasal obstruction, and a "hot potato" voice suggesting a large amount of tissue in the oropharynx. The chest and heart should be auscultated for signs of cor pulmonale, including rales, a prominent $P_2$ heart sound, and a gallop rhythm. Palpation of the abdomen for an enlarged liver or spleen may aid in this diagnosis as will edema of the extremities. Measurement of blood pressure is also a necessary part of the examination.

In addition to routine physical examination, patients should be examined while asleep or at least supine and relaxed. If the child is subject to excessive sleepiness, he or she may unwittingly demonstrate this problem during the course of the history and physical examination.

Radiographic studies may also be helpful. A cephalometric lateral neck radiograph can demonstrate the relationship between the size of the adenoid mass and the space in which it is countained. A small amount of radiopaque contrast material can be instilled into the nose to further demonstrate blockage of the nasal choanae. Cinefluoroscopy has been advocated as a noninvasive technique to demonstrate occlusion of the pharyngeal and hypopharyngeal airway during sleep. A chest radiograph is important in looking for evidence of cor pulmonale.

Polysomnographic monitoring is useful in documenting sleep disturbances and their relationship to airway obstruction. In the absence of a formal sleep laboratory, individual components of the study may be approximated. Respiratory and cardiac monitors may be used; audio and video tapes of the sleeping and awake patient may be compared. Arterial blood gas determinations employing an indwelling arterial catheter may be obtained. However, these are momentary values, and arterial catheters are not without their risks and complications. Noninvasive methods of continuous measuring of $PaO_2$, $PaCO_2$ and oxygen saturation employing cutaneously applied oxygen and carbon dioxide electrodes and ear-oximeters are currently being developed and used to obtain these values. Cardiac catheterization is performed when indicated. Kravath et al have advocated the placement of a nasopharyngeal tube in patients with suspected airway obstruction, and comparison of

polygraphic studies and blood gas values before and after tube placement as a method of diagnosis.

## Treatment

In most children, symptoms of the sleep apnea syndrome are relieved by elimination of the upper airway obstruction, usually by adenoidectomy with or without tonsillectomy. In some cases, previously adenoidectomized children will display the syndrome secondary to tonsillar hypertrophy alone. In some children, however, adenoidectomy and tonsillectomy are not sufficient, suggesting a greater CNS role in failure to maintain upper airway patency, and improvement is noted only after an airway is established by use of a nasopharyngeal tube or tracheotomy. A tracheotomy valve may be used, which can be closed during the day and opened only at night. In well selected cases tracheotomy essentially reverses all aspects of the sleep apnea syndrome. In children with other sites of airway obstruction, treatment must be aimed at the specific problem.

## Summary

Improved case identification of children with upper airway obstruction during sleep should result if physicians are aware of such signs and symptoms as excessive daytime sleepiness, loud snoring, restless sleep, recurrent nocturnal enuresis, systemic and pulmonary hypertension, undergrowth or obesity, and cor pulmonale. Furthermore, partial airway obstruction during wakefulness may be a risk factor for the development of sleep apneas or hypopneas. In suspected cases, polysomnography is a useful method for confirming and quantitating the type (central, obstructive, or mixed) and extent of ventilatory disturbance during sleep and its functional significance (such as arterial oxyhemoglobin desaturation or cardiac arrhythmia). Other methods may be employed to yield similar data.

There seemto be at least two groups of children reported in the literature, those in whom there is a specific surgically correctable lesion (such as adenotonsillar hypertrophy) versus those who eventually need tracheotomy because of collapse of upper airway musculature during sleep. In the latter group of children, it is necessary to hypothesize an additional defect in the CNS regulation of respiration during sleep.

Further research is necessary to define the boundary between normal and abnormal breathing during sleep, and to understand more thoroughly the effects of intermittent hypoventilation on daytime functioning.