

Obstructive sleep apnea syndrome in children and anesthesia

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Obstruction of the upper airway during sleep occurs frequently in children. The incidence of sleep disordered breathing, which includes habitual snoring, ranges from 3.2-12%, and the incidence of true obstructive sleep apnea (OSA) has been reported between 0.7 and 10.3%.¹⁻³ Some investigators think that despite the increased recognition of these disorders, they still may be under diagnosed.⁴ The consequences of not treating these children are significant.⁵ OSA and airway insufficiency during sleep are associated with complications ranging from daytime somnolence and behavioral disturbances to pulmonary hypertension and cor pulmonale. Children with sleep disordered breathing present commonly for general anesthesia, both for operations related to the airway, and for unrelated operations. OSA and other abnormalities of breathing during sleep may alter the optimal management of the anesthetic and of postoperative care in both situations, so it is important for the anesthesiologist to understand the pathophysiology of these syndromes.

Definitions

Obstructive sleep apnea syndrome: a disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction that disrupts normal ventilation during sleep and normal sleep patterns.³

Primary snoring: episodes of upper airway obstruction that result in increased airway resistance, but are not accompanied by apneic periods of greater than 10 seconds duration, frequent arousals from sleep or abnormalities in gas exchange.

A polysomnogram must be obtained to confirm the diagnosis of OSA, and to distinguish it from less serious sleep disordered breathing and primary snoring.^{1,3,6} These may not be easy to differentiate on the basis of a history of noisy breathing alone, and documentation of lack of airflow, oxygen desaturation, and paradoxical chest wall motion are necessary. Oximetry alone is not a sensitive discriminator; although the positive predictive value of cyclic periods of desaturation was high, the negative predictive value was low, so negative results did not rule out OSA.⁷ Clinical scoring schemes are useful to screen children with a history of noisy breathing and suggestive symptoms to determine who should undergo further evaluation, and can discern the severe cases from the normal children, but are unable to sort out the vast group in the middle with moderate symptoms.⁸

The polysomnogram consists of measurements of:

- airflow (capnometry or thermistor)
- chest wall motion (respiratory inductive plethysmography or other methods)
- oxygenation (continuous pulse oximetry)
- sleep staging (EEG, electromyography)
- ECG

In some cases, where gastroesophageal reflux is part of the differential diagnosis, esophageal pH is added.

Etiologies

The most common cause of OSA in children is *adenotonsillar hypertrophy*. In fact, OSA has replaced chronic or recurrent tonsillitis as the most common reason for performing tonsillectomy and adenoidectomy.⁹ Most otolaryngologists consider significant (as opposed to mild) OSA to be a relative contraindication to outpatient management of adenotonsillectomy, especially in children less than 3

years of age, although in one small study the postoperative complications in these younger children were not related to obstructive events.¹⁰ Those investigators suggested that the severity of OSA, rather than patient age, may be a more predictive factor, but this is in conflict with other reports, which recommend that age less than 3 years should be considered an independent discriminator.^{11,12} In a group of 134 children selected for outpatient tonsillectomy, 83% of whom carried the diagnosis and indication for surgery of OSA, 11 (8.2%) were admitted for inpatient observation after experiencing respiratory problems in the post-anesthetic care unit.¹³ These patients as a group were significantly younger than those discharged home (average 4 vs. 6.3 years). Preoperative evaluation and assessment of OSA was not described.

Children with OSA may have abnormal ventilatory responses to both hypoxia and hypercarbia due to the chronic exposure to hypoxic and hypercarbic conditions during sleep.^{14,15} These responses can take up to several weeks to revert to normal following resolution of the obstruction. There are concerns, therefore, about the ability to maintain adequate ventilation and oxygenation following the exposure to general anesthetics and to opioids given for postoperative analgesia. A study of 15 otherwise normal children, ages 1 to 18, with mild OSA used preoperative and postoperative pneumograms to assess respiratory status on the night after adenotonsillectomy. Nine of these children received a halothane-based anesthetic, and six received a fentanyl-based technique. The number of obstructive events decreased and the nadir of oxygen saturation improved from 78 to 92%. The authors concluded that in cases of mild OSA without other underlying disorders, intensive postoperative monitoring is not necessary.¹⁶ Since the only criterion that appears to be accurate in the diagnosis and stratification of severity in OSA is polysomnography and the history or pulse oximetry alone are neither specific nor sensitive enough, this test may be an important discriminator for who needs inpatient vs outpatient management following adenotonsillectomy.^{1,3,6} In clinical practice, however, polysomnography is often not performed in preoperative evaluation, and clinical criteria of airway obstruction is frequently used as a clinical indicator. There is little in the literature to support this practice, but it is very prevalent.^{17,18}

Children with neurological disorders have an increased incidence of OSA.¹⁹ Those with cerebral palsy, especially those with bulbar symptoms, often have poor hypopharyngeal muscle tone. While the airway may be stable during the awake state, during sleep there is inadequate neural input to maintain the patency of the upper airway, and collapse ensues. In severe cases, tracheostomy may be required, but many children with this condition, depending upon their anatomy and the dynamic state of the airway, may benefit from adenotonsillectomy or palatal or pharyngeal procedures which improve supraglottic airway patency.¹⁹

Craniofacial syndromes with anatomic abnormalities of the upper airway account for a small but important group of infants and children with OSA. While there are many syndromes that contribute to this population, among the most common and most severe are Pierre-Robin anomalad, Treacher Collins syndrome, and many other syndromes that prominently feature retrognathia or a hypoplastic mandible. Some of these children may require tracheostomy to provide a patent airway during sleep.

Children with Trisomy 21 (Down syndrome) commonly experience OSA for multiple reasons. Their globally diminished muscle tone also affects the muscles of the supraglottic upper airway, leading to collapse during sleep. Anatomically, these patients have a small oropharynx and hypopharynx with a large protruding tongue (macroglossia). Finally, the incidence of adenotonsillar hypertrophy is increased. The overall incidence of OSA in these patients is very high, approaching 70%, and the incidence of complications such as cor pulmonale is also increased compared with the general population.^{20,21}

Management

As with any underlying disorder, information gleaned from the history is of great value in formulating the perioperative plan. A description of symptoms related to OSA, their severity, and provocative and palliative factors should be sought from the patient's parents. Particular attention should be paid to the effects of position during sleep, as this may aid in optimal patient positioning during induction and emergence of anesthesia. Interventions during sleep, such as supplemental oxygen, BiPAP, special positioning aids, etc., should be noted. One should review any preoperative studies that may have been obtained. Polysomnogram results are of particular use in determining the severity of the obstruction and its consequences. The patient with a high risk of cor pulmonale should have an EKG, and possibly an echocardiogram, obtained.

The use of premedication or pre-induction sedation is somewhat controversial, although it need not be. Premedication can be used as long as the dose and drug chosen are appropriate, and the patient's degree of OSA is not severe. In the majority of cases, doses can be decreased to prevent oversedation but still achieve anxiolysis and cooperation. Long acting drugs, or drugs with long elimination half-lives, such as barbiturates or chloral hydrate, should be avoided. A recent study of healthy children showed that as little as 0.25 mg/kg of oral midazolam was as efficacious as larger doses at achieving both anxiolysis and amnesia, without oversedation or delayed emergence.²² In children with severe OSA, particularly with shorter operations, one must be careful to avoid having residual preoperative sedation affecting respiratory drive in the postoperative period, when other sedating agents such as opioid analgesics may be needed.

Induction of anesthesia should proceed in the same manner as any patient with a potentially compromised or difficult airway. We most commonly use an inhalation induction technique, since the maintenance of spontaneous ventilation may be advantageous. Nevertheless, there are arguments to be made that an intravenous induction with propofol may offer some benefit in that the airway may then be secured in a more rapid fashion. One should anticipate that at least partial obstruction may occur as soon as consciousness is lost, and that the alterations in supraglottic airway tone produced by volatile anesthetics may contribute further to airway obstruction. Early assistance (not control) of ventilation and application of CPAP usually helps stent open the upper airway. If aeration is diminished, inhalation induction may be prolonged. If one is unsure of the ability to visualize the airway, it is best to avoid neuromuscular blockade, which risks converting a difficult airway to a lost one! The choice of an endotracheal tube versus an LMA or PLA (perilaryngeal airway) is more dependent on the operation than the degree of upper airway obstruction. Any airway device that effectively bypasses the obstruction should be as efficacious as another in this regard.

Maintenance of anesthesia can be accomplished by many techniques, but one must have a clear understanding of what the state of the airway will be at the end of the operation. For example, in the case of adenotonsillectomy, the physical obstruction of the tonsils and adenoids will be removed during the operation, but postoperative edema, particularly in the adenoidal bed, may still produce partial obstruction. In the child with Trisomy 21, the obstructing problem of the large tongue and small mouth will persist, so that child may be expected to have more problems at emergence. In cases where it is necessary or advantageous to have a child with minimal or no residual anesthetic effects in place at emergence, TIVA with propofol and remifentanyl offers a great advantage, with far less risk of emergence delirium. The new short acting volatile anesthetics desflurane and sevoflurane do not appear to be quite as good in this regard, nor do they predictably result in an emergence free from coughing. For the patient with OSA who has undergone an operation that is not related to the airway, the maintenance technique should reflect the plan for emergence (see below), and should ensure that residual sedation be minimized.

Emergence from anesthesia (whether to extubate the patient deep or awake) is largely dependent on the assessment of the airway during induction and on an assessment of how that airway was altered during surgery. As described above, the patient with clinically significant OSA is also likely to have abnormalities in the ventilatory response to both hypoxia and hypercarbia that will persist even after surgery to relieve airway obstruction. For this reason, a sedated patient in the PACU has some risk of central hypoventilation, especially when these responses are further blunted by the administration of opioid analgesics. This is not to say that opioids are contraindicated for postoperative analgesia, or that deep extubation is contraindicated. One must, however, be careful and judicious in the application of these techniques in the patient with OSA. Measured, deliberate titration of opioid to effect with close observation and monitoring is necessary. Administering small frequent doses of short acting opioids such as fentanyl until an optimal analgesic level is achieved is safer than giving one or two larger bolus doses of longer acting drugs. The use of non-opioid analgesics, either as primary agents when possible, or as adjunctive agents, adds to safety as well by reducing opioid requirements. For non-airway surgery, regional blockade offers similar increased margins of safety. Ketorolac, because of its effect on platelet function, is contraindicated in tonsillectomy and adenoidectomy.

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