Pediatric obstructive sleep apnea syndrome and anesthetic management

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Sleep-related breathing disorders require special attention in children who spend a considerable time sleeping. Obstructive sleep apnea syndrome is characterized by episodes of upper airway obstruction during sleep. Symptoms include hyperactivity, enuresis, headache, failure to thrive, and increased respiratory effort and total sleep time. The most common cause is adenotonsillar hypertrophy. Coexisting diseases are obesity, neuromuscular and craniofacial anomalies, and Down's syndrome. Early diagnosis is important to minimize neurocognitive, cardiac and developmental complications.

Polysomnography is the gold standard for diagnosis. Although the features of pediatric obstructive sleep apnea syndrome are distinctly different from that in adults, it may predispose to the adult type of the syndrome.

As therapy concerns several surgical approaches as well as conservative techniques, anesthetic management calls for particular attention. Pre- and postoperative sedation must be performed cautiously and patients must be watched closely with respect to airway obstruction and hypoventilation. Difficult intubation must always be considered.

Key words: apnea, obstructive, sleep, children, general anesthesia.

Introduction

Sleep is thought to be a vital physiological phenomenon. A newborn may sleep for almost sixteen hours a day, and thus sleep-related breathing disorders have a great impact on life quality[1,2]. Upper airway resistance, obstructive hypopnea, and obstructive sleep apnea syndromes (OSAS) are considered as sleep related disorders which may occur at any age in childhood[1-6]. Among those, OSAS is described as a disorder characterized by repeated episodes of upper airway obstruction during sleep, usually associated with snoring, restless sleep, behavioral problems and reduced blood oxygen saturation[7-11].

The aim of this article was to discuss the aspects and anesthetic management of pediatric OSAS.

History

"Damn that boy; He's gone to sleep again. Joe! Joe!" (Sundry taps on the head with a stick, and the fat boy, with some difficulty, roused from his lethargy)–Charles Dickens, The Pickwick Papers, 1837. As described by famous author Dickens in classical literature and William Osler (1892) in medical literature, sleep-related disorders have long been recognized, but were not systematically studied till the middle of the 20th century. The first series of children with obstructive sleep apnea was reported in 1976[1,2,4,5,9].

Epidemiology

Obstructive sleep apnea syndrome occurs in all ages, and concerns neonates and adolescents. During childhood, the disease can be seen in both sexes, whereas male predominance is noted in adults (Table I). It is more common in African-Americans, either due to anthropomorphic differences or socioeconomic factors. Some of the studies support familial basis[1,2,12-14]. The estimated prevalence of the disease is 1-4%, whereas some investigators have noted prevalence up to 10%[1,2,10,11,13,15-18].

Table I. Clinical Features of Obstructive Sleep Apnea in Children and Adults

Clinical features	Children	Adults
Age	Preschooler	Middle-aged
Gender	Equal	Male predominance
Most common etiology	Adenotonsillar hypertrophy	Obesity
Weight	Thin, normal, obese	Obese
Daytime sleepiness	Rare	Common
Behavior	Hyperactivity	Cognitive impairment
Polysomnographic diagnosis	Cyclic or prolonged obstruction	Cyclic obstruction
	Preserved sleep architecture	Decreased delta and REM sleep
	EEG arousal in <50% of apneas and subcortical responses without EEG arousal	EEG arousal at each apnea
	Common in REM sleep	Common in non-REM sleep
Treatment		
Surgical	T&A (Most cases)	UPPP (selected cases)
Medical	CPAP	CPAP
	Opioid antagonists	Progesterone
		Acetozolamide
		Theophylline
		Protriptyline

T&A : Tonsillectomy and adenoidectomy.
UPPP: Uvulopalatopharyngoplasty.
CPAP: Continuous positive airway pressure.
PSG : Polysomnography.
REM : Rapid eye movements.

Pathophysiology

In general, sleep apnea may result from the obstruction of the upper airway involving the nares to the larynx. Obstruction may be due to a fixed anatomic structure, such as adenotonsillar hypertrophy, or it may be due to a collapse of the patent pharynx. The airway has a tendency to collapse in the presence of negative pressure with the association of inspiration and airflow. Muscular tone of the palate, pharynx and tongue balance the patency. During sleep, with the loss of muscle tone, the airway may not remain patent; gravity may also cause soft tissue prolapse resulting in obstruction. It is thought that the etiology and features of childhood OSAS differ from that observed in adults (Table I). Nevertheless, some investigators have found common features of adult and childhood OSAS and have proposed that both entities were related. However, since children are forced to learn and adapt, the effects of disordered sleep may be more profound than in adults[1-3,9,19,20].

The rate of childhood OSAS peaks at 2-8 years of age, when the tonsils and adenoids are the largest with respect to airway size. Adenotonsillectomy (T&A) is the treatment of choice in adenotonsillar hypertrophy, since most children improve well after the operation[1,2, 9,10,13,15,21].

Some studies have supported the role of tonsils and adenoids in the etiology of OSAS[1,2]. Nevertheless, since children with OSAS do not have symptoms of obstruction when they are awake, some other structural and neuromuscular factors are assumed to be associated with the pathogenesis[1,2]. On the other hand, some of the studies have failed to show any correlation between tonsil size and severity of the disease. Finally, some children

who were cured after adenotonsillectomy had recurrence during adolescence. It thus appears that childhood OSAS is a dynamic process resulting from a combination of structural and neuromotor abnormalities[1,2,10,13,22].

Children with craniofacial anomalies show signs of OSAS in early infancy (Table II). Structural anomalies such as intrauterine interference with mandibular growth resulting in glossoptosis and potential clefting of the palate predominate as the cause of OSAS in this group of anomalies, whereas neuromuscular factors predominate in cerebral palsy[1,2,9,13]. Patients with Down syndrome also have a high incidence of OSAS due to macroglossia, obesity and hypotonia (Table II)[9]. Interestingly, it can also be seen in patients with myelomeningocele and Arnold-Chiari malformation due to compression or dysplasia of the brainstem, with a prevalence up to 20%[1,2,20,24]. Neuropathies (Charcot-Marie-Tooth disease), congenital myopathies, muscular dystrophies (Duchenne muscular dystrophy), myotonias and myasthenia gravis are the other diseases that cause central or obstructive apnea (Table II). Mitochondrial encephalopathies such as Leigh's syndrome may also cause OSAS in children (Table II)[13].

Table II. Uncommon Diseases Associated with Childhood OSAS

Craniofacial Anomalies
Pierre Robin syndrome
Treacher Collins' syndrome
Goldenhar's syndrome
Hallermann-Streiff syndrome
Brachmann-deLange syndrome
Craniosynostosis
Crouzon's-Apert's syndrome
Pfeiffer's syndrome
Neuropathies
Charcot-Marie-Tooth syndrome
Muscular dystrophy
Duchenne muscular dystrophy
Myotonias
Myasthenia gravis
Mitochondrial encephalopathies
Leigh's syndrome
Others
Prader-Willi syndrome
Down syndrome
Arnold-Chiari malformation
Meningomyelocele
Cerebral palsy
Acromegaly

Prader-Willi syndrome is another genetic disorder with hypotonia, obesity, and developmental delay, which may accompany childhood OSAS[25]. Unlike adults, children with OSAS have normal ventilatory drive either when awake or asleep. When compared with control subjects, they have abnormal responses only under specialized conditions, such as diminished responses to repeated hypercapnia when awake and reduced arousal in response to CO_2 when asleep[1,2,21].

Upper airway functions are regulated by central ventilatory drive, chemoreceptors, upper airway pressure, pulmonary mechanoreceptors and sleep state. The tendency of the upper airway to collapse is inversely related to the upper airway dilator muscle tone, which is activated by hypoxemia, hypercapnia and upper airway subatmospheric pressure. Normal children may maintain a patent upper airway increasing their neuromotor tone via increased ventilatory drive. It is assumed that compensatory mechanisms are deficient in children with OSAS, but it was shown that such children might even partially compensate for increased upper airway resistance, preventing complete collapse[1,2,20]. These children have been shown to have elevated arousal threshold in response to hypercapnia and increased upper airway resistance, and this fact is also shown by polysomnography (PSG). Unlike adults, sleep pattern is preserved in children and excessive daytime sleepiness is rare. However, subcortical arousals demonstrated by movements and autonomic discharges occur frequently, resulting in neurobehavioral and autonomic complications[1,2,13,20].

Children with OSAS have increased arousal threshold in rapid eye movement (REM) sleep. This is a protective mechanism, because REM sleep in children is an important factor facilitating growth and maturation. It is not clear whether genetic factors have an influence on ventilatory drive and anatomic features[1,2].

Diagnosis

Early diagnosis of OSAS is very important to minimize neurocognitive, cardiac, behavioral and developmental complications that occur

as a result of the syndrome[26]. Evaluation includes history, physical examination and diagnostic tests[9,27-32].

Proper diagnosis and treatment require a team approach consisting of pediatricians, ear, nose and throat surgeons, anesthetists and dentists.

History

Parents are the first to note the signs of pediatric OSAS. Snoring and apnea are the symptoms that cause great anxiety among parents, but snoring is rare. Hyperreactivity, irritability, aggressiveness, learning problems, and enuresis are the common behavioral problems[33,34]. Daytime hypersomnolence is rare, unlike in adults (Table I). Mouth breathing is a typical finding of nasal obstruction and may be associated with OSAS. Brief awakening, frequent position changing, exacerbation of obstruction in supine position and excessive sweating during sleep are the typical symptoms reported by parents[1,2,20].

Physical Examination

Failure to thrive is a significant feature of adenotonsillar hypertrophy[1,2,20]. Obesity may also relate to OSAS in children due to excessive soft tissue in the nasopharynx. Adenoid facies, rhinorrhea, and pectus excavatum are the features of nasal obstruction. The voice may also be hyponasal due to the lack of nasal resonance[9,20].

Once OSAS is suspected, the upper airway must be evaluated from the nares to the larynx. Nasal obstruction by mucosal congestion or a structural anomaly such as septum deviation may be the cause. The oropharynx should be examined for adenotonsillar hypertrophy, whereas micrognathia or retrognathia and a large posteriorly prolapsing tongue must be ruled out[1,2,9,20].

Laboratory Findings

There is an unclear relationship between growth hormone (GH) levels and OSAS. GH level is reduced in OSAS, returning to normal levels after therapy. Patients with acromegaly are at risk due to abundant soft tissue in the tongue base, hypopharynx and supraglottic pharynx (Table II)[1,2,9]. The severity of OSAS has also been found to correlate with fasting insulin levels, which may be elevated in obese children[1,2,35].

Radiologic Diagnosis

Lateral neck X-ray may be used to check airway obstruction by enlarged tonsils and adenoids. Cephalometric X-ray may reveal the nature of the obstruction and facial bone abnormalities. Chest X-ray and ECG recordings can be used to rule out right ventricular hypertrophy and cor pulmonale[9,13,36]. Magnetic resonance imaging (MRI) has shown that the most restricted part of the airway is the adenotonsillar region in children with OSAS[37].

Pulse Oximetry and Capnography

Overnight pulse oximetry and capnography may help to assess partial airway obstruction, increased physiological dead space and hypoventilation. A positive result may be a good predictor of an abnormal PSG result[8,13].

Polysomnography

A single PSG is an adequate measure and is accepted as a gold standard for diagnosis of OSAS[22,38-42]. The standardized PSG study consists of a video camera, electroencephalogram, electro-oculogram, chin electromyogram, electrocardiogram, pulse oximetry and assessment of oro-nasal airflow, and chest, abdomen and leg movements. PSG studies reveal differences between children and adult OSAS (Table I)[1,2].

Sleep Studies

Audiotaping, videotaping and pulse oximetry are used for sleep studies. Movements, snoring and oxygen saturations are noted throughout the sleeping period. The results may assess the indication for operation, the aim for postoperative respiratory failure and baseline outcome values. However, the results of these studies are inadequately investigated and there is no committee consensus[8,43].

Morbidity

Untreated OSAS may result in serious morbidity.

Neurobehavioral Morbidity

There is a significant correlation between the severity of OSAS and the aspects of cognition and behavior. It was shown that behavioral

problems and quality of life improved or reversed after therapy, whereas cognitive deficits were partially improved[1,2,33,44]. A technical study of the American Academy of Pediatrics revealed behavioral disturbances in 76%, hyperactivity in 42%, and decreased school performance in 16%. Although it is thought that daytime sleepiness is uncommon in children, the same study mentioned 84% excessive daytime sleepiness[8].

In summary, several studies generally show an almost three-fold increase in neurobehavioral morbidity[1,2,8,44].

Cardiovascular Morbidity

The association between OSAS and hypertension remains controversial. Systemic hypertension is a well-documented sequela of adult OSAS. Studies reveal diastolic rather than systolic hypertension in children. However, left ventricular hypertrophy was noted in children, indicative of systemic hypertension. In addition to the circulatory system, chronic hypercapnia and hypoxia lead to pulmonary vascular hypertension and cor pulmonale. Cardiovascular and pulmonary sequelae can be reversed by treatment[1,2,8, 11,18,44,45].

Somatic Growth

Children with OSAS confront a higher risk of failure to thrive. The underlying mechanism is not fully understood. It has been postulated that dysphagia may occur due to enlarged tonsils and adenoids or that increased respiratory effort during sleep might have increased the metabolism. As young children breathe thorough nares, nasal obstruction interferes with their feeding as well. Recently, decreased insulin growth factor-1 levels were noted in some children, indicative of hormonal mechanisms. Weight gain has been noted after the treatment[1,2,8,9,20,44].

Miscellaneous

Nocturnal enuresis has been documented in children with OSAS. A notable improvement was achieved after the treatment[8,34].

A link between sudden infant death syndrome (SIDS) and OSAS has been noted. There is a family history of SIDS in adults with OSAS. However, when supine versus a prone position has been advised, the incidence of SIDS decreased, indicating that OSAS did not play a major role in SIDS deaths[1,2,46].

Chronic hypoxia in OSAS may lead to polycythemia due to increased erythropoiesis. Similarly, hypoxia causes crises in patients with sickle cell anemia[9].

Differential Diagnosis

Upper airway resistant syndrome (UARS) is more common than OSAS in children. Mild craniofacial anomalies, enlarged adenotonsils, and family history of adult-sleep disordered breathing are some of the features of the disease. Abnormal breathing and, in contrast with OSAS, tachypnea without saturation drops are recorded during PSG. Esophageal pressure monitoring may be the only method to confirm the diagnosis[5].

The congenital central hypoventilation syndrome (CCHS) is an uncommon disease of childhood characterized by inadequate autonomic respiratory control. It belongs to the neurocristopathies with unknown etiology. There are isolated forms, whereas combined forms with pheochromocytoma, neuroblastoma, medullary carcinoma of the thyroid, carcinoid tumors and Hirshsprung's disease are also recognized[6].

In contrast to almost all types of OSAS, children with CCHS breathe better in REM sleep. Although these children have intact chemoreceptor sensitivity to acute hypoxia, they have decreased response to progressive hypoxia[1,6].

Cranial MRI may reveal the cause of central hypoventilation. Metabolic disorders should be excluded.

Treatment
Non-surgical Treatment

The mainstay of OSAS therapy in children is T&A (Table I). Most of the nonsurgical therapies were used either prior to or when T&A failed to resolve OSAS. Pharmacological approaches such as progesterone, acetozolamide, theophylline, and protriptyline are used in adults (Table I). There are reports about opioid antagonists used in pediatrics[44].

Corticosteroids

Systemic use of steroids has been found unsatisfactory due to the short duration of therapy and the need for larger doses. Intranasal

topical steroid therapy was found effective in reducing adenotonsillar size; however, it is only a palliative treatment, providing temporary relief[44].

Nasal Continuous Positive Airway Pressure Therapy

Nasal continuous positive airway pressure (nCPAP) was first described in 1984 (Table I). Later, it was proposed as an alternative to tracheotomy in craniofacial and neuromuscular diseases. nCPAP is tolerated well by most children (80%). Several mild side effects are air leaks or skin breakdown around the nares. However, hypoventilation or midface hypoplasia is also reported. CPAP mask sizes and effectiveness of O_2 pressure must be monitored every 6-12 months[1,2,44,47].

Bilevel positive airway pressure administration was also found to be an effective treatment modality. In order to optimize efficacy from CPAP therapies, parents must be motivated and educated[47-49].

Surgical Therapies
Adenotonsillectomy

Studies have shown that symptoms almost completely resolve following T&A. It appears that T&A is curative in 75-100% of children even if they are obese[1,2,7,8,44,50,51]. However, postoperative recurrence is more frequent than expected. There are reports about recurrence during puberty or several years after complete relief. Cephalometric X-ray studies reported some anatomical anomalies as an explanation for recurrence[7]. Lim[51] and coworkers point out the lack of randomized controlled studies on efficacy of T&A in the treatment. Children with OSAS are at high risk of postoperative respiratory complications due to edema, hemorrhage, increased secretions and respiratory depression due to anesthetics[1,2,9].

Uvulopalatopharyngoplasty (UPPP)

Children with OSAS usually respond well to T&A. Uvulopalatopharyngeal procedures may be necessary occasionally. UPPP has been used since 1981 for the treatment of snoring in adults. The procedure consists of several modifications involving removal of tonsils and posterior edge of the soft palate. It has been reported to be successful in relieving OSAS symptoms in children with cerebral palsy and hypotonic upper airway muscles. It has not been applied in uncomplicated pediatric patients. Caution must be taken for nasopharyngeal stenosis as a late complication[1,2,7,52].

Maxillomandibular Advancement Osteotomy (MMO)

Pediatric patients with craniofacial and skeletal anomalies such as mandibular hypoplasia are successfully treated by several surgical procedures, which are classified as Phase I and Phase II surgery. Phase I consists of nasal reconstruction, UPPP and hypopharyngeal surgery, whereas Phase II involves MMO, which is assumed to be the most successful surgical therapy of OSAS, with a medium-term success rate of 90%. In 40% of the cases, based on radiocephalometric findings, MMO may be considered as the primary form of the therapy. Malocclusions also require treatment in 23% of the cases. In this respect, dentists must be a part in the team approach[7,29-32,53].

Tracheotomy

Tracheotomy was the first choice of treatment in the past. However, while it is still a life-saving process in severe craniofacial anomalies, it has been shown that tracheotomy leads to improvement but not elimination of OSAS[7,52].

ANESTHETIC MANAGEMENT

Pediatric otolaryngology is one of the most challenging areas of surgery for the anesthesiologists. While devoting attention to the safety of the patients, anesthesiologists must also allow the surgeon optimal access in order to ensure the best possible outcome for the surgery. Recently, children under one month of age have been considered to be at higher risk due to their physiologic characteristics. Respiratory control and cardiovascular responses are immature in this age group; hypoxia may cause apnea rather than hyperventilation, whereas cardiac ouput is rate dependent. Responses to the anesthetic agents vary according to age as well. Inhalational anesthetics may cause more depression on cardiac output in preterm infants and newborns than in older children.

Although estimated anesthetic mortality varies widely, it is as low as 1 in 50,000 cases in healthy children[7,54-56].

Preoperative Assessment

Most of the routine otolaryngological operations are performed as outpatient procedures. Preoperative information includes history of previous anesthetics, current medications, recent infections, and allergic conditions. Children with preexisting diseases or with family history of inherited disease must be carefully screened by radiologic and laboratory findings. Cardiac problems, which may coexist with OSAS, should be investigated by ECHO and electrocardiography. If the anticipated blood loss may exceed 10 mL kg^{-1}, hemoglobin estimation and blood crossmatching are required. It is generally agreed that healthy children do not require investigation. The physical examination must especially concern airway anatomy including mouth, tongue, jaw and neck.

Children who are given information about surgery and anesthesia have been shown to be less anxious, nevertheless separation from their parents is a traumatic process. Some clinics allow parents to enter the operating room until induction to alleviate the separation anxiety, but some of the children still require pharmacological sedation[54-56].

Generally, sedation is avoided in outpatient otolaryngological surgery, but if needed, the choice of drugs is based on the age and specific conditions of the patients. Children younger than eight months may not need premedication, whereas children eight months to three years may not accept oral administration, in which case the rectal route is more suitable for this age group. Children older than three years tolerate oral premedication well[56].

Table III. Intraoperative Monitoring Standards

- Presence of qualified anesthesia staff
- Evaluation of
  - Oxygenation
    - Oxygen analyzer
    - Pulse oximeter
  - Ventilation
    - Auscultation of breath sound
    - Watching chest movements
    - Observation of reservoir bag
    - Capnography, end tidal CO_2
  - Circulation
    - Arterial blood pressure per 5 minutes
    - Continuous electrocardiogram
  - Body temperature

Benzodiazepines, such as midazolam or opioids, are the drugs of choice for sedation. However, studies have shown that premedication with benzodiazepines, and especially opioids, provokes apnea by decreasing the activity of the nervus genioglossus selectively, which may lead to loss of upper airway patency. Respiratory depression, apnea and desaturation less than 1% were reported with midazolam. Opioids such as fentanyl produce dose-dependent depression when given in combination with midazolam.

Anticholinergic premedication may be desirable to avoid secretions and vagal responses due to airway manipulation. Especially children under six months of age are more sensitive to anesthetics with respect to cardiac depression. As their cardiac output is heart rate-dependent, the use of anticholinergic agents such as atropine (0.02 mg kg^{-1}) or glycopyrrolate (0.01 mg kg^{-1}) is mandatory in this age group at the time of induction by intravenous (iv) route[54-56].

To avoid aspiration, an adequate period of fasting must be maintained. As prolonged period of fasting is unpleasant both for children and family and causes intravascular volume depletion as well, it is advised to take clear liquids up to 2 to 3 hours before induction[21,55-58].

Intraoperative Management

Intraoperative anesthetic complications of OSAS have not been specifically examined[44]. It is assumed that application of monitoring standards considered by the American Society of Anesthesiologists reduced overall incidence of intraoperative complications (Table III)[56].

Anesthesia can be induced by inhalation anesthetics via facemask, by injection of sedative or hypnotic agents via iv route or by intramuscular (im) injection (Table IV). Halothane had been the drug of choice for inhalational induction due to its lack of

Table IV. Induction Techniques and Drugs

Techniques	Drugs
Inhalational	Halothane
	Sevoflurane
	Nitrous oxide
Intravenous	Thiopental
	Propofol
Intramuscular	Ketamine

pungency, but an even less pungent agent, sevoflurane, is taking its place. Young children prefer inhalational induction due to fear of needle. Smart scented masks provide relief for children during the procedure. Older children and teenagers usually prefer iv induction. Eutectic mixture of local anesthetic (EMLA) cream may be beneficial for painless access of iv route if applied 45 minutes before induction. The ultra-short acting barbiturate thiopental is the most commonly used intravenous induction agent (5-6 mg kg^{-1}). Care must be taken especially in hypovolemia or poor myocardial contractility. Propofol as an alkylphenol is an alternative induction agent. The major advantages of propofol are rapid recovery with minimal nausea and vomiting, but painful injection is its major disadvantage. Care must be taken for hypotension, apnea and desaturation[56].

In the patient who does not cooperate with standard induction techniques, ketamine, a phencyclidine derivative with potent analgesic and amnestic properties, is suitable for im induction (5-10 mg kg^{-1}). It can also be given intravenously (1-3 mg kg^{-1})[56]. Ketamine may be the drug of choice for induction in hypovolemic patients because it has sympathomimetic properties and does not cause hypotension. It may also be used in patients with reactive airways, because by increasing the circulating catecholamines it causes relaxation of tracheal smooth muscle. Production of copious secretions, postoperative nausea and vomiting, and hallucinations are its main disadvantages.

Intubation, which is facilitated by muscle relaxants, follows induction. Succinylcholine, the only depolarizing relaxant, is indicated in rapid-sequence inductions and in the treatment of laryngospasm. It is the most rapid acting of all muscle relaxants. Bradycardia in repeated doses, causing hyperkalemia due to depolarization of myoneural junction, and increased intraocular and intracranial pressure are the disadvantages of the drug. It is hydrolyzed by pseudocholinesterase enzyme. Several nondepolarizing muscle relaxants with various speed of onset and duration of action may be available for intubation or maintenance, but none of them, even rocuronium, produces as rapid intubation conditions as succinylcholine (Table V)[56].

Table V. Anesthetics and Muscle Relaxants Used in Maintenance of Anesthesia

Anesthetics
Inhalational
Nitrous oxide
Halothane
Isoflurane
Sevoflurane
Desflurane
Intravenous
Morphine sulfate
Fentanyl
Remifentanyl
Propofol
Midazolam
Muscle relaxants (Nondepolarizing)
Mivacurium
Rocuronium
Atracurium
Vecuronium
Pancuronium

Recently, sevoflurane has been accepted as the drug of choice in the induction and maintenance of outpatient otolaryngologic procedures in infants for several reasons: It is less pungent than halothane and other inhalational agents, emergence is rapid, and it has less depressive effect on the cardiovascular system than halothane. Because its offset is so rapid, narcotics must be coadministered in maintenance to avoid perception of pain. Fentanyl is the most commonly used narcotic as supplement to anesthesia, but caution must be taken for bradycardia and chest wall rigidity. Propofol may be administered as intravenous infusion for the maintenance of anesthesia in combination with narcotics or nitrous oxide (Table V)[56].

Difficult airway management and intubation are the most challenging problems in otolaryngologic procedures. The infant tongue is larger in proportion to the size of the mouth than in adults. This anatomic characteristic contributes to obstruction when induction agents and muscle relaxants are given. The enlarged adenotonsils also obstruct the upper airway resulting in difficult airway management via face mask[58]. Anatomical abnormalities such as craniofacial anomalies with micrognathia, Down syndrome with obesity and large tongue result in difficult or impossible visualization of the glottis. If the glottis can be visualized, the obstruction due to cysts, adenotonsils or other anatomical abnormalities causes difficulty

in inserting the tracheal tube. Preservation of spontaneous ventilation may provide a guide to the laryngeal inlet, especially in the presence of difficult airway. Introducers and endotracheal tubes with smaller diameters must be available.

If the larynx is not visualized, laryngeal mask airway (LMA) may be introduced. Furthermore, the LMA can be used as a guide through which a tube or fiberoptic bronchoscope may be introduced. All airway management algorithms propose fiberoptic bronchoscopy as the final step in the management of difficult airway; if needed, urgent tracheotomy must be performed[54-62].

Impaired cardiac and pulmonary functions must always be considered in patients with OSAS. Care must be taken with respect to cardiovascular depression caused by anesthetics. Besides standard monitorization, invasive monitorization of arterial blood and central venous pressures may be mandatory.

Postoperative Management

Extubating the infant or child with a difficult airway should be a careful teamwork effort as in intubation[58]. Children with OSAS are prone to respiratory complications postoperatively due to upper airway edema, increased secretions, respiratory depression secondary to analgesic and anesthetic agents and postoperative pulmonary edema, with a prevalence of 16-27%[1,2,9,21,44,54-58]. Risk factors for postoperative complications are reported as age under two years, craniofacial anomalies affecting the pharynx (especially midfacial hypoplasia and retrognathia), obesity or, conversely, failure to thrive, previous upper airway trauma, severe OSAS, concomitant UPPP, and history of prematurity[1,2,44].

Close postoperative monitoring is mandatory for these patients on the first postoperative night. If upper airway induced respiratory insufficiency supervenes, CPAP or bilevel positive airway pressure may be used to avoid reintubation[44].

Postoperative nausea and vomiting (PONV) may be associated with otolaryngological procedures at rates as high as 70%, causing an unpleasant recovery and delayed discharge. The use of narcotics, nitrous oxide and halothane increase the risk of PONV, whereas propofol reduces this risk. Several antiemetics have been found efficient in controlling PONV. Metoclopramide, dehydrobenzperidol, and ondansetron are the drugs used worldwide for this purpose[56].

To assess the degree of pain, visual analogue scale may be helpful in preverbal children. Intravenous opioid administration is the mainstay of severe pain therapy. Milder degrees of pain may be handled by acetaminophen, codeine or ketorolac[54-56].

Results of PSG reveal that hospitalization is necessary only for high-risk patients with OSAS. Children younger than three years of age, and those with cerebral palsy, seizure disorders, and congenital heart disease and prematurity are considered to be at particular risk in the postoperative period[56].

Conclusion

Obstructive sleep apnea syndrome is a common cause of childhood sleep-related breathing disorders, and its pathophysiology is poorly understood. It is thought to be caused by a combination of anatomic and neuromotor dysfunction.

A teamwork effort consisting of pediatrician, ear/nose/throat surgeon, anesthetist and dentist is needed in diagnosis and treatment of the disease.

Adenotonsillectomy is the mainstay of treatment of childhood OSAS, whereas children with several anatomic anomalies, as in craniofacial syndrome, may require additional surgical approaches. Special care and knowledge are needed for anesthetic management. Difficult airway, respiratory depression and problems due to coexisting disease such as hypertension, cor pulmonale or obesity are some of the difficulties that the anesthetists may face.

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