The Pediatric Airway: Anatomy, Challenges, and Solutions

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Abstract

Sedation may alter laryngeal anatomy, function, and respiratory mechanics; therefore, it is essential that the practitioner has a thorough understanding of the pediatric airway. Physical examination reveals the general condition of a patient and the degree of the airway compromise. During sedation, adequate oxygenation and ventilation must be maintained despite a relative decrease in rate and depth of respiration. Conditions that interfere with the integrity of the laryngeal inlet or upper larynx may impair effective ventilation as a result of partial or complete airway obstruction. Sleep-disordered breathing (SDB) is a spectrum of disorders ranging from primary snoring to obstructive sleep apnea syndrome (OSAS). When sedation without a secured airway is planned it is imperative that the level of consciousness, adequacy of ventilation, and oxygenation be continuously monitored and the risk of apnea be evaluated. When a child is sedated, the best prevention is to insure that the position provides the best anatomic orientation for airway patency.

Keywords

Sleep-disordered breathing (SDB) • Obstructive sleep apnea (OSA) • Airway obstruction • Upper respiratory infection (URI) • Pharyngeal anatomy • Laryngomalacia • Anterior mediastinal mass • Obstructive sleep apnea syndrome (OSAS) • Laryngeal mask airway (LMA) • Pediatric advanced life support (PALS) • American Heart Association (AHA)

One of the most important aspects of planning sedation is consideration of the airway of each individual patient. Sedation may alter laryngeal anatomy, function, and respiratory mechanics; therefore, it is essential that the practitioner has a thorough understanding of the pediatric airway.

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Anatomy of the Pediatric Airway

Airway compromise in the infant or child may result from abnormalities in the nasal cavities, nasopharynx, oral cavity, pharynx, and neck. The airway is comprised of the larynx, trachea, bronchi, and alveoli. The trachea in the infant is smaller than that of the adult and since the function of the trachea is passive during respiration, anatomic differences in the infant and adult trachea are not as apparent as they are in the larynx [1]. The infant larynx is not a miniature version of the adult larynx and there are essential differences between these two organs. The differences are related to size, location, and configuration, and must be considered since the primary function of the larynx is to protect the lower airway and regulate airflow during respiration by controlling the resistance

K.P. Mason (ed.), *Pediatric Sedation Outside of the Operating Room: A Multispecialty International Collaboration*,
DOI 10.1007/978-1-4939-1390-9_7,

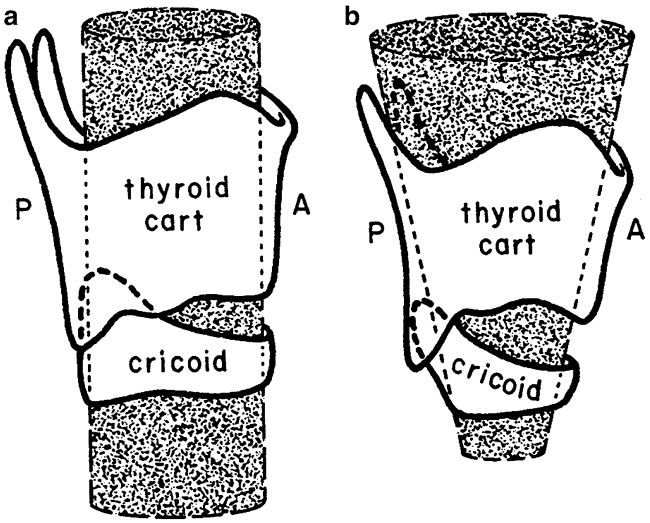

Fig. 7.1 Configuration of (**a**) the adult larynx and (**b**) infant larynx (Reprinted with permission from Wheeler M, Coté CJ, Todres D. The Pediatric Airway. Chapter 5. In: Coté CJ, Todres ID, Goudsouzian NG, Ryan JF (editors). A Practice of Anesthesia for Infants and Children, 3rd edition. Philadelphia, PA: W. B. Saunders Company. 2001)

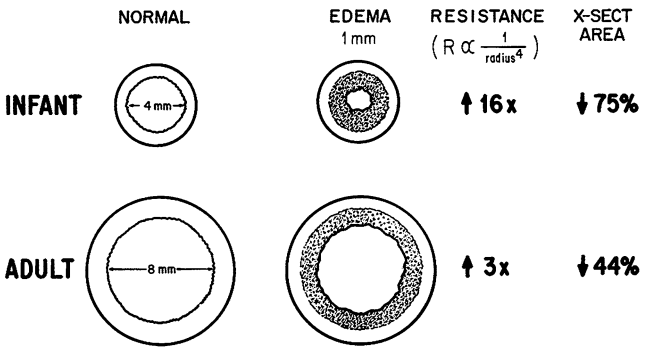

Fig. 7.2 Relative effect of circumferential edema on the infant and adult airway (Reprinted with permission from Wheeler M, Coté CJ, Todres D. The Pediatric Airway. Chapter 5. In: Coté CJ, Todres ID, Goudsouzian NG, Ryan JF (editors). A Practice of Anesthesia for Infants and Children, 3rd edition. Philadelphia, PA: W. B. Saunders Company. 2001)

during inspiration and exhalation. The cricoid ring is the narrowest portion of the infant larynx. Although this has recently been questioned, there are insufficient data to refute the validity of this anatomic finding [2]. In the infant and child, the cricoid cartilage is a non-expansile complete ring whereas, this cartilage is open at the posterior aspect in the adult patients [3, 4] (Fig. 7.1). In the adult patient the vocal cords are the narrowest part of the airway, providing the cylindrical shape of the adult larynx in contrast to the cone shape of the pediatric larynx. This is an important distinction to make since the resistance to airflow is inversely proportional to the fourth power of the radius ($R = 1/\text{radius}^4$ [5]). One centimeter of circumferential edema in the infant larynx will decrease the cross-sectional area by 75 % and increase the resistance by 16-fold as compared to the same one centimeter of edema in the adult larynx, which will result in a decrease in the cross-sectional area of only 44 % and threefold increase in resistance (Fig. 7.2). This becomes relevant when sedating a child with either a history of prolonged intubation in which the tracheal lumen may be narrowed, or a child with a recent upper respiratory infection or croup, which also may result in a circumferentially narrow airway (Figs. 7.3 and 7.4).

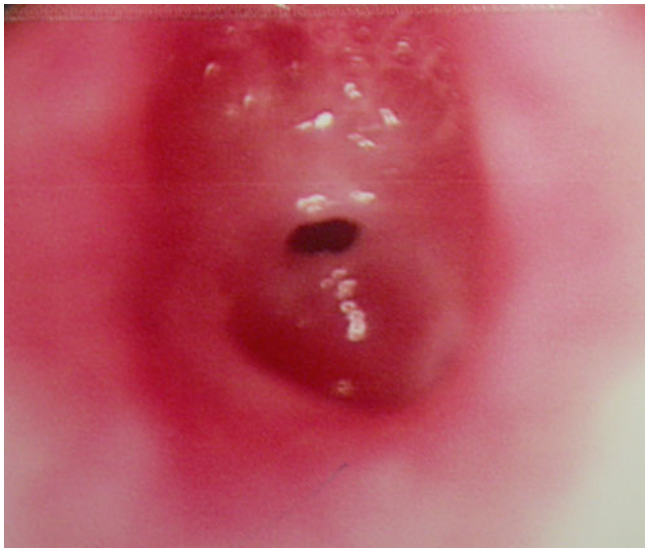

Fig. 7.3 Child with post-intubation subglottic stenosis (Photo courtesy of Reza Rahbar, DMD, MD, Children's Hospital Boston)

The larynx of the infant and young child is higher than in the adult patient. The adult larynx is located at C6–7, whereas it is at C4 in the infant and descends to the adult location as growth occurs during childhood. The cephalad location of the infant larynx makes oral ventilation difficult, and as a result the infant is an obligate nasal breather for the first year of life [5]. The epiglottis projects vertically in the adult, but posteriorly in the infant. The infant epiglottis is also narrower and omega shaped, which makes it more prone to obstructing the laryngeal inlet [6] (Fig. 7.5). In the setting of nasal congestion, effective ventilation may be compromised in the unaltered state and worsened after sedation.

The tongue of the infant is larger in relation to the oral cavity than that of the older child and adult. In neonates, the tongue is more anterior than the larynx so that the epiglottis can contact the soft palate and allow respirations and sucking simultaneously. This does, however, predispose the infant to airway obstruction more readily than the older child. At birth, the base of the tongue resides in the oral cavity and gradually descends with the larynx to a more caudad position

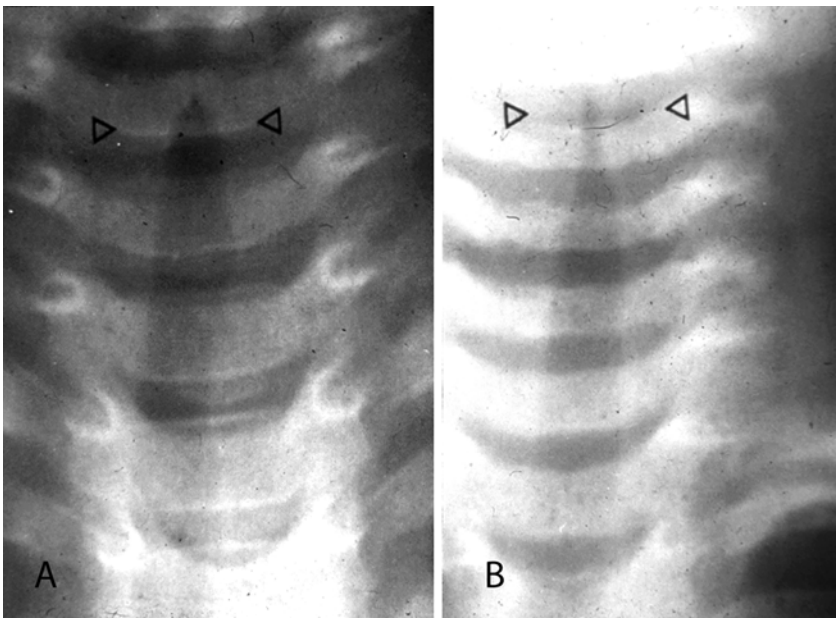

Fig. 7.4 Plain X-ray of the airway of a child with (**a**) severe croup and (**b**) mild croup. Note the subglottic narrowing and appearance of the characteristic "Chrysler Building" sign (Photo courtesy of Reza Rahbar, DMD, MD, Children's Hospital Boston)

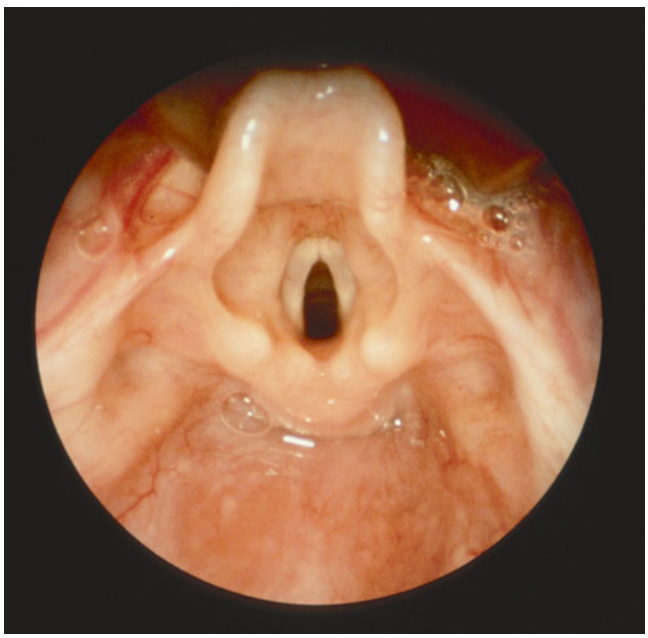

Fig. 7.5 Normal infant larynx. Note the omega-shaped epiglottis (Photo courtesy of Reza Rahbar, DMD, MD, Children's Hospital Boston)

by the fourth year of life. The ratio of soft tissue to bony structures is higher in the infant and thus predisposes this group of patients to a greater risk of mechanical oropharyngeal obstruction. The combination of small nares, large tongue, small mandible, excess soft tissue, and short neck also increases the infant's susceptibility to airway obstruction [7]. The ribs of the infant and small child are more horizontal in orientation than those of the older child and adult, and more flexible, which therefore predisposes the child to ventilatory compromise. As previously noted, since the metabolic rate and oxygen consumption of infants is double than that of the adult and the functional residual capacity is smaller, the rapidity of desaturation in the infant and child is much greater. For this reason optimal surveillance of the airway and respiratory mechanics is essential if hypoxia is to be avoided [8].

Normal spontaneous breathing is accomplished by minimal work, and obstruction of either the upper or lower airway will result in increased work of breathing. To avoid this it is essential that airway obstruction and compromises in ventilation be recognized and corrected early. Infants and children may rapidly progress from normal breathing to obstruction, and compromised respiration to respiratory distress, and eventual cardiac arrest. Since oxygen consumption is higher in infants, decreases in oxygen delivery will result in more rapid compromise than is observed in older patient populations. The presence of apnea leading to inadequate alveolar ventilation may rapidly progress to hypoxemia, hypercarbia, and eventual tissue hypoxia.

Assessment of the Pediatric Airway for Sedation

Physical examination reveals the general condition of a patient and the degree of the airway compromise. Laboratory examination may include assessment of hemoglobin, a chest

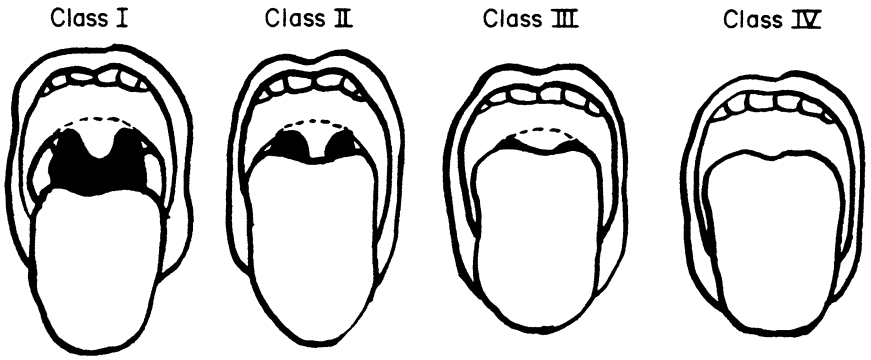

Fig. 7.6 Mallampati classification of pharyngeal structures (Reprinted with permission from Samsoon GL, Young JR. Difficult tracheal intubation: A retrospective study. Anaesthesia. 1987 May;42(5):487–490)

radiograph, and barium swallow, which can aid in identifying lesions that may be compressing the trachea. Other radiologic examinations such as magnetic resonance imaging (MRI) and computed tomography (CT) scan may be indicated in isolated instances but are not routinely ordered.

The physical examination of the airway in children begins with simple observation, since approaching an anxious child may cause inconsolable crying and distortion of the physical examination. Observation of the general appearance, noting color of the skin and the presence of pallor, cyanosis, rash, jaundice, unusual markings, birthmarks, and scars from previous operations should be documented.

The degree of mouth opening should be noted and full examination of the oropharyngeal area should be completed. The distance from the temporomandibular joint to the angle of the ramus is helpful in the assessment of the adequacy of the mouth opening. The distance between the angle of the ramus and the mentum is a good predictor of the ability of the mandibular bony structure to accommodate the oropharyngeal soft tissue. The presence of loose teeth should be documented. Special attention should be paid to the condition of the soft and hard palates, the dentition, and the size of the tongue. The relation of the tongue to the other oropharyngeal structures should be noted. For instance, a large thick tongue may pose minimal increased risk for airway obstruction in a child with an otherwise normal oropharynx but may cause severe risk in the child with a narrow oropharynx or a high arched palate (as may be present in children with craniofacial abnormalities and syndromes) where the tongue occupies a greater proportion of the bony structure volume. The amount of the posterior pharynx that can be visualized is important and correlates with the difficulty of intubation, and in sedated patients would correlate with the potential for airway obstruction. The Mallampati classification (Class I–IV) is based on the structures visualized with maximal mouth opening and tongue protrusion in the sitting position (Fig. 7.6) [9, 10]. The soft palate, fauces, uvula, and pillars are visualized in patients with a Class I airway. The soft palate, fauces, and portion of the uvula but no pillars are visualized in Class II. The soft palate and base of uvula are visualized in Class III and only the hard palate is visualized in Class IV [10]. Tonsil size should be evaluated since the tonsils of pediatric patients are frequently enlarged and may be the source of upper airway obstruction. A standardized system for evaluation of tonsils exists and is based on the percentage of pharyngeal area that is occupied by hypertrophied tonsils. Class 0 tonsils are completely limited to the tonsillar fossa. Class +1 tonsils take up less than 25 %, Class +2 tonsils take up between 25 and 50 %, and Class +3 tonsils take up 50–75 % of the pharyngeal area. Class +4 tonsils take up greater than 75 % of the oropharynx and are commonly referred to as "kissing tonsils" [11] (Fig. 7.7). Tonsillar hyperplasia may increase the risk of airway obstruction in the sedated patient when the tonsils occupy the oropharyngeal space outside of the tonsillar fossa as in Class +3 and Class +4 anatomy. Conversely, lesser degrees of hyperplasia as seen in Class +1 and Class +2 may result in airway obstruction in the sedated patient with craniofacial abnormalities such as Down syndrome.

Abnormal facies might be an indication of a syndrome or constellation of congenital abnormalities. One congenital anomaly often is associated with others. The neck should be examined primarily to determine if the trachea is midline and to evaluate tracheal length and soft tissue volume. In the child with a short neck and abundant soft tissue, the potential for oropharyngeal airway obstruction is greater.

The rate, depth, and quality of respirations should be evaluated. The pattern of breathing should be noted as well as the rate and depth of respiration. Use of accessory muscles may indicate an increased work of breathing due to an effort to overcome upper or lower airway obstruction. Nasal or upper respiratory obstruction is indicated by noisy or labored breathing. The color, viscosity, and quantity of nasal discharge should be documented. If the child is coughing, the origin of the cough (upper versus lower airway) and the quality (dry or wet) can be evaluated even before auscultation of the lungs. The presence of wheezing, audible stridor, or retractions should be noted. The airway should be evaluated for ease of intubation in the case of urgent intervention.

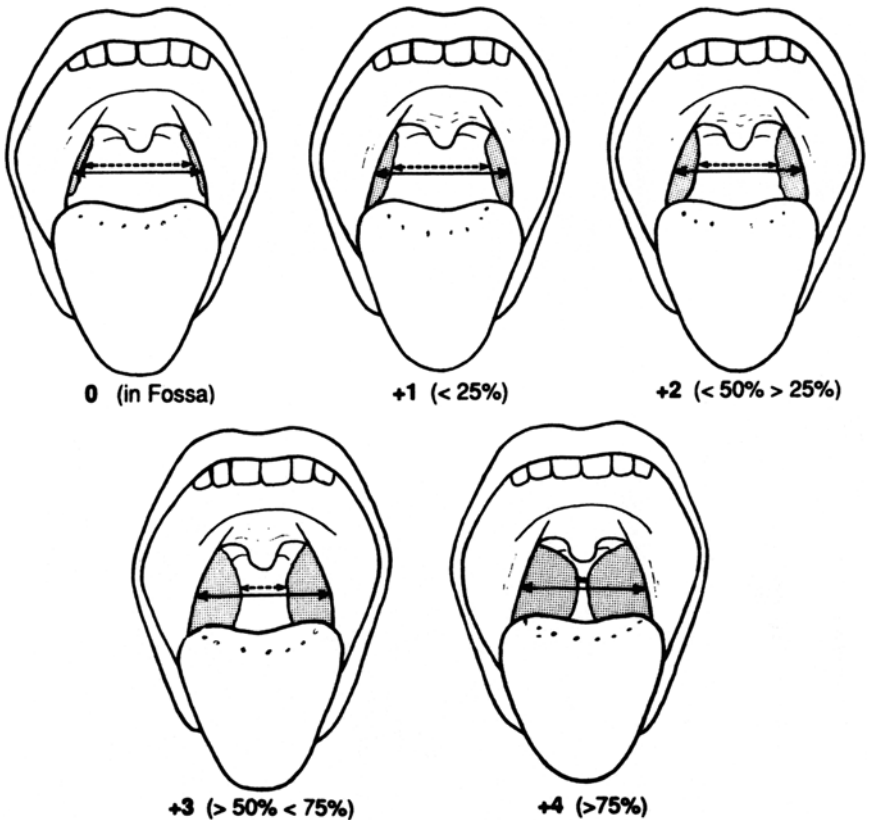

Fig. 7.7 Classification of tonsillar hypertrophy (Reprinted with permission from Brodsky L. Modern assessment of tonsils and adenoids. Pediatr Clin North Am. 1989;36: 1551–1569. WB Saunders)

If the child will not open his or her mouth, a manual estimation of the thyrohyoid distance should be made. Children with micrognathia, as in Pierre Robin syndrome or Goldenhar syndrome, may be especially difficult to intubate, especially in an unanticipated situation.

Risk Factors for Airway Compromise or Depression

During sedation, adequate oxygenation and ventilation must be maintained despite a relative decrease in rate and depth of respiration. Any condition that causes airway compromise should be thoroughly evaluated prior to administration of sedation agents to determine if alteration in respiratory parameters will result in impaired ventilation.

During normal breathing the flow of air is laminar. As previously mentioned the resistance is inversely proportional to the fourth power of the radius. Increased airway resistance occurs when the diameter of an airway is decreased under constant pressure. The radius of an airway may be decreased by circumferential edema, external compression, mucous, secretions, or bronchoconstriction. The work of breathing increases in patients with upper or lower airway disease. Increased airway resistance, decreased lung compliance, and altered central control of respiration will all affect the adequacy of respiration.

Adequacy of respiration may be based on respiratory rate, respiratory effort, tidal volume, chest auscultation, and pulse oximetry. The normal respiratory rate in infants under 1 year of age is up to 30 breaths per minute. The respiratory rate declines to 20 breaths per minute by age 8 years and equals the adult rate of 16–17 breaths per minute by age 18. Alterations in the respiratory rate can indicate underlying comorbidity such as fever, pain, acidosis, and sepsis in tachypneic patients and impending cardiovascular collapse in the bradypneic patient. Increased respiratory effort as recognized by nasal flaring, chest retractions, and uncoordinated chest excursions should alert the clinician that an increased work of breathing may increase if excessive sedation is administered.

Noisy breathing due to obstructed airflow is known as *stridor*. Inspiratory stridor results from upper airway obstruction; expiratory stridor results from lower airway obstruction; and biphasic stridor is present with midtracheal lesions. The evaluation of a patient with stridor begins with a thorough history. The age of onset suggests a cause since laryngotracheomalacia and vocal cord paralysis are usually present at or shortly after birth, whereas cysts or mass lesions develop later in life. Information indicating positions that make the

stridor better or worse should be obtained, and placing a patient in a position that allows gravity to aid in reducing obstruction can be of benefit during anesthetic induction.

Patients at risk for airway compromise may have either anatomic or physiologic abnormalities that may predispose them. Anatomic abnormalities may cause the oropharyngeal or tracheobronchial airway to be compromised and ventilation to be impaired by small changes in position. The anatomic imbalance between the upper airway soft tissue volume and the craniofacial size contributes to pharyngeal airway obstruction. Pharyngeal size is determined by the soft tissue volume inside the bony enclosure of the mandible. The magnitude of pharyngeal muscle contraction is controlled by neural mechanisms and the interaction between the anatomical balance and neural mechanisms, which are suppressed in sedated patients, determines pharyngeal airway size and patient ability to maintain a patent airway. An anatomic imbalance between the upper airway soft tissue volume and craniofacial size will result in obstruction. Anatomic imbalance may be compensated for by enhanced neural mechanisms that regulate pharyngeal dilator muscles in patients during wakefulness. When neural mechanisms are suppressed during sleep or sedation, relaxation of pharyngeal dilator muscles occurs and the pharyngeal airway severely narrows [12]. Small changes in function in the setting of normal anatomy may similarly cause inadequate oxygenation. Increasing the distance between the mentum and cervical column will transiently relieve the obstruction. This is achieved by positioning the patient in the sniffing position. Similarly, the sitting position displaces excessive soft tissue outside the bony enclosure through the submandibular space.

Laryngomalacia is the most common cause of stridor in infants and is usually benign and self-limited. It occurs during inspiration and is most often due to a long epiglottis that prolapses posteriorly and prominent arytenoid cartilages with redundant aryepiglottic folds that fall into the glottis and obstruct the glottic opening during inspiration (Fig. 7.8). There is little obstruction during exhalation since the supraglottic structures are pushed out of the way during expiration. Intermittent low-pitched inspiratory stridor is the hallmark symptom, which appears during the first 2 weeks of life. Symptoms peak at 6 months of age when they are at their worst, then gradually resolve. Although most children are symptom-free by 18–24 months, the stridor can persist for years. The definitive diagnosis is obtained by direct laryngoscopy and rigid or flexible bronchoscopy. Preliminary examination is usually carried out in the surgeon's office. A small, flexible fiberoptic bronchoscope is inserted through the nares into the oropharynx, and the movement of the vocal cords is observed [13]. Other etiologies include foreign body aspiration, infection such as croup or laryngotracheobronchitis, edema, or mass lesions such as cyst or tumor.

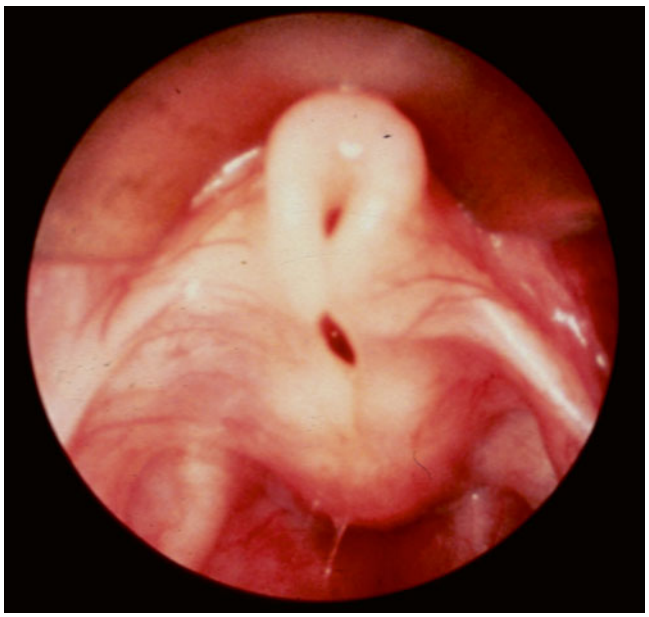

Fig. 7.8 Larynx of an infant with laryngomalacia (Photo courtesy of Reza Rahbar, DMD, MD, Children's Hospital Boston)

Grunting is a low-pitched sound that results when a patient exhales against a closed glottis and is heard on exhalation. Infants and children often grunt to keep the small airways and alveoli open in an attempt to optimize ventilation and oxygenation. The presence of grunting may be a sign of severe respiratory distress and impending respiratory failure. Underlying causes include pneumonia, acute respiratory distress syndrome, pulmonary edema, congestive heart failure, and abdominal splinting.

Wheezing during inspiration or exhalation, or both, indicates intrathoracic obstruction of small airways. It may be a result of intrinsic reactive airways, bronchospasm, or foreign body aspiration. Hypoxemia that is present in the wheezing patient may worsen during administration of sedation.

One of the most challenging decisions in caring for children is establishing criteria for cancelation of a procedure in the presence of an upper or lower respiratory infection. Children presenting with symptoms of uncomplicated upper respiratory infection who are afebrile, with clear secretions, and appear otherwise healthy should be able to safely undergo sedation. A history of nocturnal dry cough, wheezing during exercise, and wheezing more than three times in the recent 12 months, or a history of present or past eczema may be associated with an increased risk for bronchospasm, desaturation, or airway obstruction [14]. Nasal congestion, purulent sputum production, and a history of reactive airway disease are predictors of adverse respiratory events, and children with these advanced symptoms of upper and potential lower respiratory disease should not undergo sedation [15].

There are many syndromes that have anatomic components related to the airway. A large tongue is associated with Down, Hunter, Hurler, and Beckwith-Wiedemann syndromes. Congenital hypothyroidism and Pompe disease are also associated with a large tongue. Patients with Pierre Robin, Treacher Collins, and Goldenhar syndromes, as well as children with congenital hemifacial microsomia, have micrognathia, high arched palate, and a potential to have early airway obstruction when sedated. Children with tonsillar hypertrophy are at risk for mechanical airway obstruction due to large tonsils occupying a greater portion of the oropharyngeal airway than normal-sized tonsils.

Former premature infants are at risk for untoward respiratory events during sedation. There is a more gradual slope of the CO_2 response curve in the preterm infant, which predisposes this group of patients to apnea. All neonates exhibit periodic breathing, which is manifested as interrupted ventilation by self-corrected short periods of apnea without desaturation or bradycardia [16]. This tendency diminishes by 45 weeks postconceptual age. Apnea of prematurity and postanesthetic apnea are predominantly central in origin, with about 10 % due to mechanical obstruction. The response to airway obstruction with apnea is common in infants with periodic breathing and decreases with increasing postnatal age. In the sedated neonate and former premature infant, benign periodic breathing may evolve into frank apnea, which must be managed by stimulation or assisted ventilation. To detect postanesthetic or post-sedation apneic events, it is suggested that infants whose age is under 56 weeks postconception be monitored for 24 h after the procedure [17].

Conditions that interfere with the integrity of the laryngeal inlet or upper larynx may impair effective ventilation as a result of partial or complete airway obstruction. Upper respiratory infections cause increased secretions, which may occlude the larynx in addition to the inflammatory response that can compromise the internal diameter of the laryngeal inlet. Laryngotracheobronchitis or croup also decreases the internal laryngeal diameter and produces the same clinical outcome. The incidence of epiglottitis has decreased dramatically in the past decade but may still be encountered. These patients have not only inflammation of the epiglottis but edema of the surrounding structures, which severely restricts the size of the larynx and encroaches on the area for ventilation to occur.

Patients who have sustained airway trauma or thermal injury should be considered in this category as well. Children who have experienced prolonged intubation may have decreased laryngeal inlet diameter as a result of fibrosis from congenital or acquired subglottic stenosis (Figs. 7.9 and 7.10). Any agent that will decrease the pharyngeal muscle tone and rate and depth of respiration in this setting should be given with extreme caution and warrants vigilance.

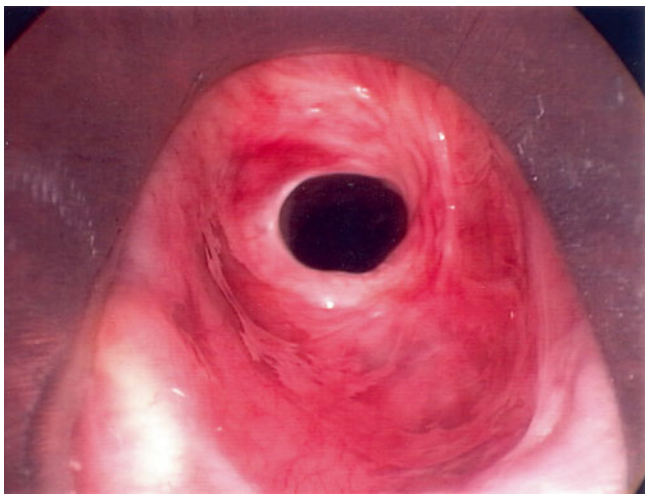

Fig. 7.9 Larynx of an infant with congenital subglottic stenosis (Photo courtesy of Reza Rahbar, DMD, MD, Children's Hospital Boston)

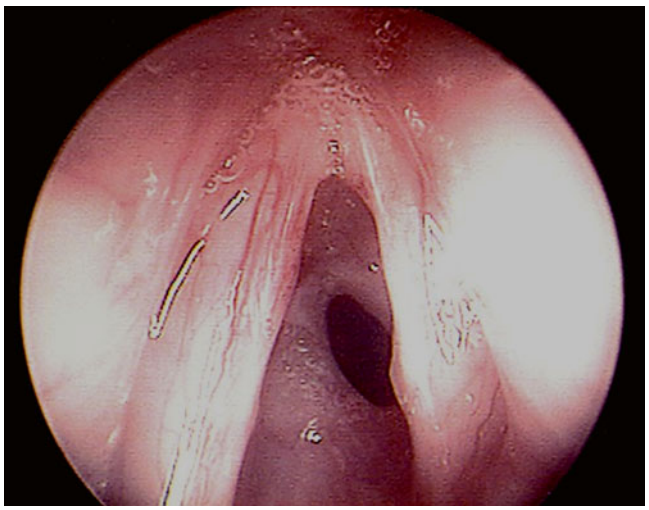

Fig. 7.10 Larynx of an infant with acquired post-intubation subglottic stenosis. (Photo courtesy of Reza Rahbar, DMD, MD, Children's Hospital Boston)

Other conditions that restrict the laryngeal inlet are subglottic stenosis, laryngeal cysts, and papillomatosis.

There is a similar concern for narrowing and compromise of the larynx from external factors. Goiter or other tumors of the neck that are extrinsic to the larynx may cause compression and functional restriction to ventilation. Children with arthrogryposis or congenital abnormalities in which the neck is fused may have difficulty with positioning and subsequent ventilation when airway function is depressed during sedation.

Children with an anterior mediastinal mass are at significant risk for airway compromise during sedation due to compression of the intrathoracic larynx (Figs. 7.11 and 7.12).

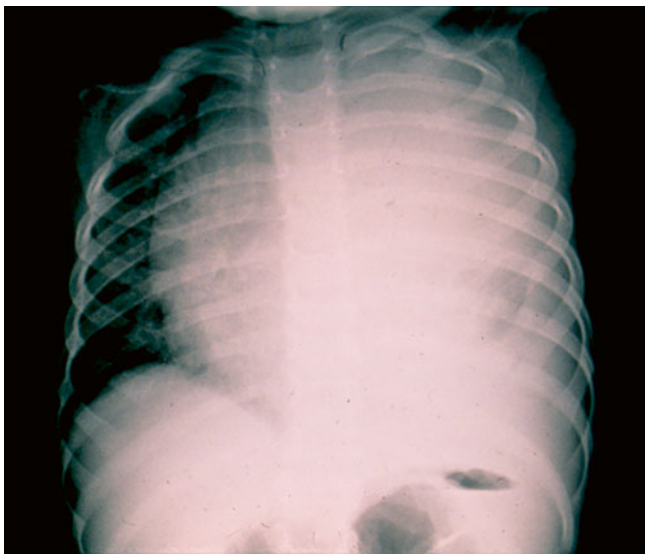

Fig. 7.11 A 20-month-old male with a large anterior mediastinal mass

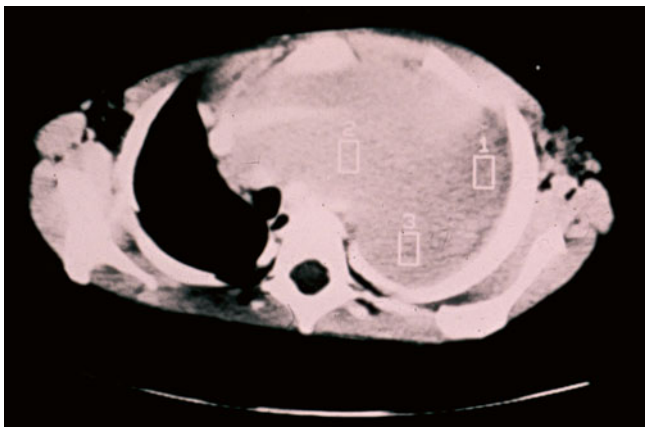

Fig. 7.12 CT scan illustrating a large anterior mediastinal mass compressing the lung and causing tracheal deviation

Although lymphomas constitute the largest group of masses that arise in the anterior mediastinum, other masses that may present in this location include teratomas, cystic hygromas, thymomas, hemangiomas, sarcomas, desmoid tumors, pericardial cysts, and diaphragmatic hernias of the Morgagni type.

To understand the pathophysiology of the anterior mediastinum, it is important to be familiar with the anatomy. The mediastinum is defined as the extrapleural space in the thorax that is bounded anteriorly by the sternum, posteriorly by the thoracic vertebrae, superiorly by the thoracic inlet, and inferiorly by the diaphragm. Structures contained within the mediastinum that may undergo compression from an enlarging mass are the trachea and the main stem bronchi, superior vena cava, aortic arch, main pulmonary artery, and a portion of the heart itself.

Patients with anterior mediastinal masses may present with varied signs and symptoms referable to both the cardiovascular and respiratory systems and are directly related to the location and size of the mass, as well as the degree of compression of surrounding structures. The most commonly observed respiratory symptom is cough, especially in the supine position, which results from anterior compression of the trachea. Infants younger than 2 years of age are more likely to experience wheezing as a sign of tracheal compression, whereas children older than 2 years of age usually present with malaise, cough, fever, and neck mass. Other respiratory findings in patients of all ages include tachypnea, dyspnea, stridor, retractions, decreased breath sounds, and cyanosis on crying, all of which should alert the practitioner to some degree of airway compromise that may worsen when positive intrathoracic pressure is generated.

Cardiovascular symptoms result from compression of the aortic and pulmonary vessels, as well as the right atrium and right ventricle. This can lead to both hypotension secondary to inadequate cardiac filling and restricted pulmonary blood flow resulting in poor oxygenation despite adequate ventilation. Findings referable to the cardiovascular system include fatigue, headache, hypotension or pallor in the supine position, a feeling of light-headedness, superior vena cava syndrome (facial edema, cyanosis, jugular venous distension), and the appearance of a new murmur, especially in the area of the pulmonary valve. It is essential that the practitioner search for these signs and symptoms when interviewing and examining patients with mediastinal masses in an attempt to ascertain the degree of respiratory and cardiovascular compromise present. Patients with minimal symptoms can have catastrophic events if subtle indicators are overlooked. Improvement of these physiologic changes is often quickly achieved by moving the patient into a sitting or left lateral position.

Sleep-Disordered Breathing

Sleep-disordered breathing (SDB) is a spectrum of disorders ranging from primary snoring to obstructive sleep apnea syndrome (OSAS). The mildest form of SDB is primary snoring, which is noisy breathing without clinical manifestations and occurs in 20 % of normal children [18]. Although SDB affects 10 % of the population, only 1–4 % will progress to OSAS. OSAS is characterized by periodic, partial, or complete obstruction of the upper airway during sleep. Airway obstruction is characterized by an anatomic imbalance between the upper airway soft tissue volume and craniofacial size. Suppression of pharyngeal dilator muscles during sleep and anesthesia occurs in the patient with obstructive sleep apnea, as opposed to patients who are just noisy breathers or have mild to moderate snoring.

Repetitive arousal from sleep to restore airway patency is a common feature as are episodic sleep-associated oxygen desaturation, hypercarbia, and cardiac dysfunction as a result

of airway obstruction. Individuals who experience obstruction during sleep may have snoring loud enough to be heard through closed doors or observed pauses in breathing during sleep. They may awaken from sleep with a choking sensation. Parents report restless sleep in affected children and frequent somnolence or fatigue while awake despite adequate sleep hours. These children fall asleep easily in nonstimulating environments and are difficult to arouse at their usual awakening time. Type 1 OSAS is characterized by lymphoid hyperplasia without obesity, whereas type 2 OSAS patients are obese with minimal lymphoid hyperplasia. Approximately, 10 % of OSAS is present in preschool and school-aged children and is thought to decline after 9 years of age [4].

Obesity changes craniofacial anthropometric characteristics, therefore body mass index of 95 % for age or greater is a predisposing physical characteristic that increases the risk of developing OSAS. Children with craniofacial abnormalities including a small maxilla and mandible, large tongue for given mandibular size, and thick neck have a similar increased risk. Many of these children have syndromes that are associated with additional comorbidities. Anatomic nasal obstruction and Class IV touching tonsils reduce oropharyngeal cross-sectional area, which constitutes an additional risk. Pharyngeal size is determined by the soft tissue volume inside the bony enclosure of the mandible, and an anatomic imbalance between the upper airway soft tissue volume and craniofacial size will result in obstruction.

The magnitude of pharyngeal muscle contraction is controlled by neural mechanisms and the interaction between the anatomical balance and neural mechanisms determines pharyngeal airway size. Increased neural mechanisms can compensate for the anatomical imbalance in obstructive sleep apnea patients during wakefulness. When the neural mechanisms controlling pharyngeal dilator muscles are suppressed during sleep or anesthesia (as is present in non-OSAS patients), the pharyngeal airway severely narrows because of the anatomical imbalance. There is additional decrease in ventilator response and impairment of the arousal response. Craniofacial morphology may influence the severity of obstruction in boys more than girls [19]. Increasing bony enclosure size will provide relief of airway obstruction. This is only accomplished surgically by mandibular advancement. Increasing the distance between the mentum and cervical column by positioning will transiently relieve the obstruction as long as the sniffing position is maintained. Similarly, the sitting position displaces excessive soft tissue outside the bony enclosure through the submandibular space.

The long-term effects of OSAS are not limited to the airway. These children have other systemic comorbidities. Increased body mass index and obesity may lead to increased cognitive vulnerability as illustrated by the increased frequency of hyperactivity and increased levels of C-reactive protein. The duration of OSA has no relation to reversibility of neurobehavioral impairment since many believe that episodic hypoxia alters the neurochemical substrate of the prefrontal cortex causing neuronal cell loss. Metabolic syndrome consists of insulin resistance, dyslipidemia, and hypertension. It is felt that OSAS is a risk factor for metabolic syndrome in obese children but not in nonobese patients. Cardiovascular and hemodynamic comorbidities are more common in OSAS patients. These consist of altered regulation of blood pressure as well as alterations in sympathetic activity and reactivity. Also present are endothelial dysfunction and initiation and propagation of inflammatory response facilitated by increases in levels of C-reactive protein [20, 21]. Systemic inflammation using interleukins as a marker is a component of OSAS in both obese and nonobese children and is reversed after tonsillectomy. Systemic hypertension, changes in left ventricular geometry, and intermittent hypoxia leading to pulmonary artery hypertension are well-described comorbidities present in patients with OSAS.

The mainstay of OSAS management is surgical removal of tonsils and adenoids, which carries an 85 % success rate in resolving OSAS. Recurrence may occur in children with craniofacial abnormalities and in others, and if surgical intervention does not resolve the problem, continuous positive airway pressure (CPAP) at night is the next treatment modality. Many of these children, however, may present for imaging or require sedation prior to removal of the tonsils or adenoids.

For patients undergoing sedation, the preoperative evaluation begins with the history. (Refer to Chap. 4.) Questions to ask parents include the presence of difficulty breathing during sleep, snoring, gasping, retractions, apnea during sleep, sweating during sleep, restless sleep or behavioral problems, and/or somnolence during the day [22, 23]. A positive finding of any of the aforementioned characteristics should alert the practitioner to the possibility of some degree of OSAS [24]. Specific attention should be paid to the frequency of tonsillar infection, recent upper respiratory infections, SDB, and cardiovascular abnormalities. The physical exam should include observation of audible respiration, mouth breathing, nasal quality of speech, chest retractions, long facies, retrognathic mandible, and inspection of tonsillar size. Auscultation should be specifically directed to detect wheezing and stridor. Polysomnography (PSG), otherwise known as the sleep study, is the gold standard for diagnosis of OSAS. A sleep study is suggested to direct the postoperative or postprocedural disposition. It is essential in patients with comorbidities and high-risk features such as morbid obesity, craniofacial abnormalities, neuromuscular disorders, cor pulmonale, systemic hypertension, difficulty breathing during sleep, growth impairment due to chronic obstructed breathing, and a history of severe prematurity [25]. Obesity changes craniofacial anthropometric characteristics and a body mass index of 95 % for age or greater is a

risk factor for OSA, which should be quantified by PSG. Craniofacial abnormalities that specifically include small maxilla and mandible, large tongue for given mandible size, and thick neck similarly should be evaluated by sleep study. Despite this, most patients do not have this examination prior to surgery. It is expensive, time consuming, and unavailable in some medical centers. The nadir of oxygen saturation and respiratory disturbance index (RDI), which is the number of apneic episodes per hour, are measured during PSG. Apnea is defined as decreases in airflow greater than 90 % for two breaths or more. Hypopnea is defined as decreases in airflow greater than 50 % coupled with 3 % decrease in oxygen saturation or electroencephalogram (EEG) arousal. An RDI of two or more is necessary for the diagnosis of OSAS. Mild OSAS is defined as RDI of 5–10 events, moderate 10–20 events, and severe 20–30 events. The STOP-BANG questionnaire has been in use in the adult population since 2009 to predict the presence of OSA in the absence of a sleep study [24]. It is comprised of eight questions designed to predict moderate to severe OSA. Although it has good predictive value for alerting practitioners to adults with OSA, it is not a good predictor of sedation-related adverse events (SRAE) in children [26]. Although the presence of OSA does not seem to be a risk factor for hypoxia in adults undergoing moderate sedation, this has not been demonstrated in the pediatric population [27].

When sedation without a secured airway is planned, it is imperative that the level of consciousness, adequacy of ventilation, and oxygenation be continuously monitored and the risk of apnea be evaluated. Patients exposed to recurrent hypoxia exhibit an altered response to narcotics, which is manifested by decreases in minute ventilation, respiratory frequency, and tidal volume. It is therefore suggested that no sedative premedication be administered to OSAS patients prior to a general anesthetic and narcotics be administered in incremental doses, beginning with one-half the recommended dose, until adequacy of ventilation and respiration is determined. Patients with OSAS who are given the same dose of narcotic as non-OSAS patients have a very high risk of serious respiratory compromise [28, 29]. Similarly, patients should not be discharged until fully awake and breathing at a baseline rate and depth. The supraglottic obstruction secondary to decreased muscle tone may contribute to desaturation. Children who have increased severity of OSAS, low weight, and age under 3 years exhibit a higher rate of complications [30]. They are more likely to require supplemental oxygen, the use of an oral airway, and require assisted ventilation. Slow return of upper airway tone may lead to desaturation and laryngospasm on emergence, especially in those patients who are known to have an RDI greater than 30.

There is no agreement on the specific criteria that preselect an elective OSAS patient for admission and monitoring postprocedure [31].

Inclusive characteristics may include the following: PSG-proven OSAS with RDI >40, RDI >20 plus either desaturation <70 % or age less than 3 years, or weight <3 % for age. Children with craniofacial syndromes or neuromuscular disease are included as children with complex or cyanotic cardiac disease. Additional indications include morbid obesity, known cor pulmonary and pulmonary hypertension and preexisting asthma or other unrelated respiratory comorbidities.

Identification and Treatment of Airway-Related Adverse Events

The best way to minimize airway and respiratory compromise is to optimize the situation and prevent it. When a child is sedated, the best prevention is to ensure that the position provides the best anatomic orientation for airway patency. The patient should be in the supine position with the head in a sniffing position and shoulders slightly elevated. This requires that the protrusion of the occiput is balanced by slight shoulder elevation to prevent neck flexion and airway compromise (Fig. 7.13). Supplemental oxygen should be administered by nasal prongs, mask, or blow-by to keep oxygen saturation above 95 %.

If, despite proper positioning, the airway becomes obstructed and ventilation is compromised, an oropharyngeal or nasopharyngeal airway may be placed. Both of these devices improve ventilation by maximizing the space for gas entry between the tongue and posterior pharynx. The appropriate size must be chosen to prevent worsening of the obstruction or irritation of the larynx resulting in laryngospasm (Fig. 7.14). The appropriate oropharyngeal airway size may be determined by measuring the distance between the lips and the angle of the mandible. If the airway is too large, the tip may rest on the epiglottis and cause laryngeal irritation and spasm. If the airway is too small it may compress the tongue and cause it to move posteriorly, thus causing worsening of the oropharyngeal obstruction. The proper nasopharyngeal size may be estimated by measuring the distance between the nares and the angle of the mandible. Extreme caution must be used when placing a nasopharyngeal airway in a toddler or young child due to the presence of hypertrophied adenoid tissue, which can bleed profusely when dislodged [32]. If airway patency is not restored with repositioning of the head and shoulders despite the use of an artificial airway, the jaw thrust may be useful. This maneuver increases the distance between the base of the tongue and the vocal cords and helps to provide the maximum area for air exchange. In addition, positioning the patient on his/her side with the mouth opened may also relieve obstruction.

If it is determined that ventilation must be assisted to maintain oxygenation, then bag-mask ventilation may be instituted. The laryngeal mask airway (LMA) may also be a

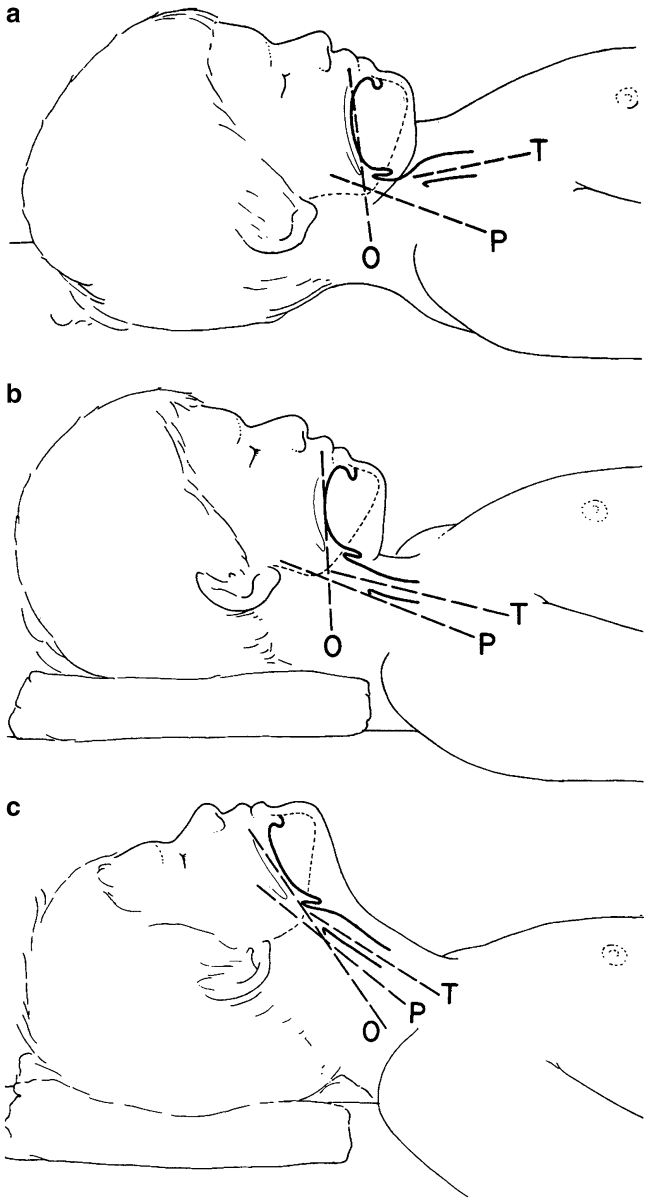

Fig. 7.13 Alignment of oral, pharyngeal, and tracheal axis variation with head position (Reprinted with permission from Wheeler M, Coté CJ, Todres D. The Pediatric Airway. Chapter 5. In: Coté CJ, Todres ID, Goudsouzian NG, Ryan JF (editors). A Practice of Anesthesia for Infants and Children, 3rd edition. Philadelphia, PA: W. B. Saunders Company. 2001)

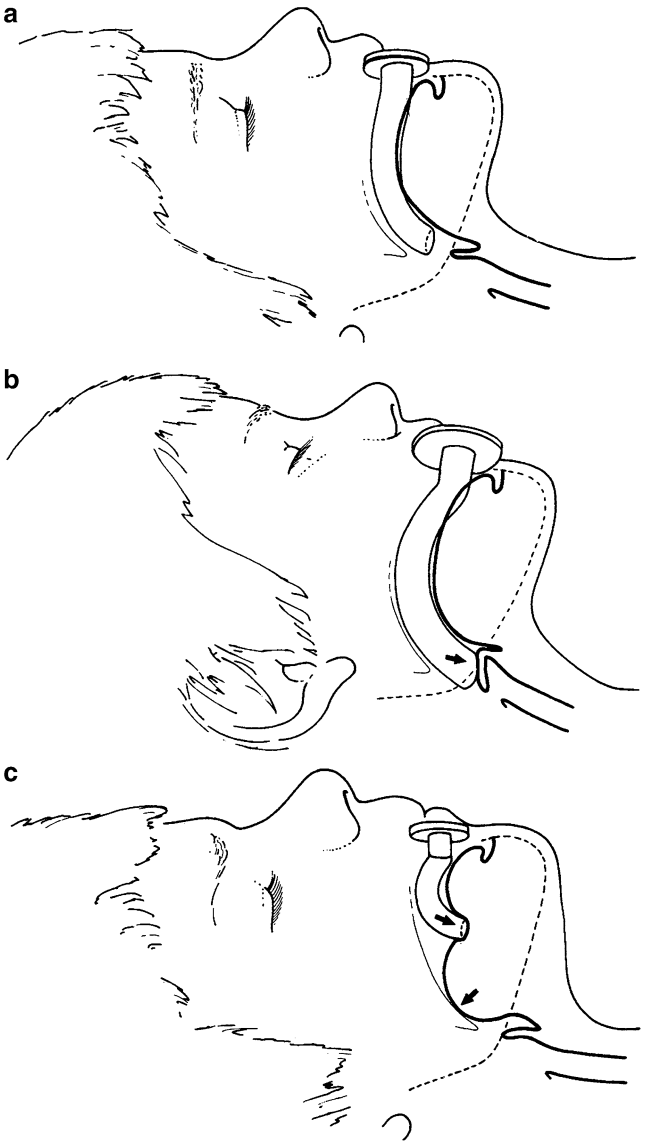

Fig. 7.14 Effects of different sizes of oropharyngeal airway placement (Reprinted with permission from Wheeler M, Coté CJ, Todres D. The Pediatric Airway. Chapter 5. In: Coté CJ, Todres ID, Goudsouzian NG, Ryan JF (editors). A Practice of Anesthesia for Infants and Children, 3rd edition. Philadelphia, PA: W. B. Saunders Company. 2001)

useful adjunct if the patient has progressed beyond spontaneous ventilation and requires assisted or controlled ventilation. The LMA is an appropriate intermediate step to maintain an airway that does not require endotracheal intubation and is a part of the Pediatric Advanced Life Support (PALS) algorithms of the American Heart Association.[1] The LMA is inserted without the need to visualize the vocal cords and forms an airtight seal around the glottis rather than plugging the pharynx. This positioning provides both a patent path for gas entry during positive pressure ventilation and simultaneously prevents the supralaryngeal structures from encroaching on the glottis. The vocal cords move freely during respiration and are not manipulated, thus avoiding a potent stimulus for laryngospasm. The ideal patient position for insertion is the supine sniffing position, but it can be inserted in the neutral position as well. In infants and young children, the epiglottis is prominent and may provide a mechanical barrier to successful placement. To overcome this, it is recommended that the LMA be

[1] http://www.heart.org/HEARTORG/

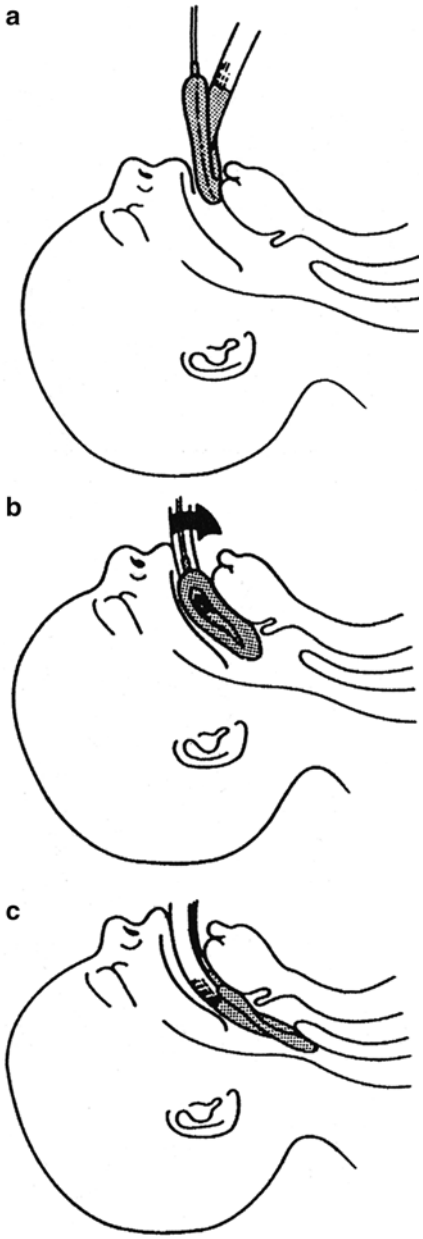

Fig. 7.15 Technique of laryngeal mask insertion in infants and children (Reprinted with permission from Haynes SR, Morton NS. The laryngeal mask airway: A review of its use in paediatric anaesthesia. Paediatr Anaesth. 1993;3:65. Blackwell Publishing)

place with the vented side facing the palate and advanced while turning in an attempt to flick the epiglottis out of the way (Fig. 7.15) [33]. Assisted spontaneous ventilation may be carried out in this manner. If undisturbed, the LMA provokes very little stimulus and can be left in place until the patient's protective reflexes have returned and respirations resume spontaneously. If ventilation cannot be achieved, endotracheal intubation with controlled ventilation may have to be instituted.

Conclusion

Sedation of children for diagnostic or therapeutic procedures is often an alternative to general anesthesia due to the common belief that it carries less risk and requires fewer resources. Although this is not a completely erroneous perspective, sedation is not without risks. A thorough understanding of the pediatric airway anatomy at each developmental stage is essential as well as the physiologic consequences that occur when consciousness is altered. Appropriate monitoring must be utilized and personnel who are knowledgeable with regard to the potential adverse events and skills to treat them must be immediately available. When these conditions are met, sedation of infants and children is a reasonable and safe practice.

Case Studies

Case 1: Obstructive Sleep Apnea

A 5-year-old boy with osteomyelitis Class 4 tonsillar hyperplasia presents to the interventional radiology suite for insertion of a peripherally inserted central catheter (PICC) for antibiotic administration. Attempts at PICC insertion were unsuccessful due to patient movement and difficulty in locating an appropriate vessel. The mother reports that the child is otherwise healthy, except that he seems to choke when he is asleep and sometimes awakens startled in the middle of the night. He is overweight for his age and has some difficulty concentrating and sitting still in school. His physical exam reveals him to be a moderately overweight boy with a short neck and nasal breathing. His oropharyngeal examination is positive for Class 4 kissing tonsils, which occupy greater that 75 % of the oropharyngeal volume and a Mallampati Class 3 classification for intubation. He is taking no medications and has not had a sleep study.

The considerations for this child would be appropriateness for sedation, choice of monitoring required, and postprocedural disposition. This is a child in whom a sleep study would be desirable, but in the absence of this information it may be assumed that he is at risk for OSAS based on his weight, short neck, and large tonsils. He may undergo sedation, but is at risk for airway obstruction and desaturation, thus he must be monitored in the presence of a practitioner who has airway management skills should this occur. Monitors should include electrocardiograph (EKG), pulse oximeter, capnography, and blood pressure measurements. Supplemental oxygen should be administered by nasal

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cannula. Some head-up position should be maintained as much as possible to facilitate diaphragmatic excursion. Agents that maintain spontaneous respirations and do not produce significant respiratory depression should be considered. Due to the probability of OSAS, this patient should be admitted to the hospital overnight for observation. The inclusion criteria for overnight admission include obesity, Class 4 tonsils, as well as a history consistent with significant SDB and probable OSAS. Alternatively, if the child underwent tonsillectomy and adenoidectomy in advance of sedation, the radiologic study could be scheduled 2–3 weeks postoperatively. Waiting this amount of time ensures that the hypopharynx would be well healed. In this case, if a repeat sleep study was repeated and improved, the postsedation admission might be eliminated; however, in the absence of a repeat sleep study, the overnight postsedation admission still is required.

Case 2: Anterior Mediastinal Mass

An otherwise healthy 14-year-old male presented to his pediatrician with a history of new-onset cough and difficulty sleeping. The only significant findings on physical exam were shortness of breath when lying down, some jugular venous distention in the supine position, and a single enlarged cervical lymph node. Breath sounds were diminished bilaterally but more on the left side. The child was sent to the hospital for a chest X-ray and a large anterior mediastinal mass was noted. An MRI for further classification was requested.

Patients with an anterior mediastinal mass may present with varied signs and symptoms referable to both the cardiovascular and respiratory systems. Symptoms are directly related to the location and size of the mass, as well as the degree of compression of surrounding structures. The most commonly observed respiratory symptom is cough, especially in the supine position, which results from anterior compression of the trachea by a mass located in the anterior mediastinum. Infants younger than 2 years of age are more likely to experience wheezing as a sign of tracheal compression, whereas children older than 2 years of age usually present with malaise, cough, fever, and a neck mass. Other respiratory findings in patients of all ages include tachypnea, dyspnea, stridor, retractions, decreased breath sounds, and cyanosis on crying, all of which should alert the anesthesiologist to some degree of airway compromise that may worsen when positive intrathoracic pressure is generated.

Cardiovascular symptoms result from compression of the aortic and pulmonary vessels, as well as the right atrium and right ventricle. This can lead to both hypotension secondary to inadequate cardiac filling and restricted pulmonary blood flow, resulting in poor oxygenation despite adequate ventilation. Findings referable to the cardiovascular system include fatigue, headache, hypotension or pallor in the supine position, a feeling of light-headedness, superior vena cava syndrome (facial edema, cyanosis, jugular venous distension), and the appearance of a new murmur, especially in the area of the pulmonary valve. It is essential that the clinician search for these signs and symptoms when interviewing and examining patients with mediastinal masses in an attempt to ascertain the degree of respiratory and cardiovascular compromise present. Patients with minimal symptoms can have catastrophic events when sedated if subtle indicators are overlooked.

Sedation is best accomplished with the child in the semi-Fowler or full sitting position, since the supine position leads to decreased expansion of the rib cage and cephalad displacement of the diaphragm. Patients who are asymptomatic while awake may exhibit airway obstruction during sedation in the supine position, which is explained by a reduction in the dimensions of the thorax that limits the available space for the trachea relative to the tumor. The increase in central blood volume that accompanies the supine position can also lead to increased tumor volume and size, thus contributing to the potential for airway obstruction. The patient should breathe spontaneously and a small dose of sedative agents may be administered as the patient is lowered into position. Agents that suppress respirations should be avoided. The adequacy of ventilation and blood pressure should be checked at frequent intervals until the optimum surgical position has been achieved. If at any time a decrease in blood pressure occurs and causes an inability to oxygenate despite adequate ventilation or if an inability to provide adequate ventilation is encountered, the patient should be returned to the upright or lateral position. This will generally relieve airway obstruction caused by the tumor mass.

Case 3: The Child with a "Cold"

A 4-year-old child presents for sedation for a brain MRI. He was born at 36 weeks gestation and his mother had an uncomplicated delivery. He was slightly

(continued)

hypotonic at birth and has not reached his expected milestones. He walks but still exhibits some weakness in both extremities. His pediatrician is concerned and wishes to make certain that there is no intracranial lesion or other central nervous system pathology. He has a history of reactive airway disease and uses a bronchodilator, but has not had to use it in the recent 6 months.

His mother reports that he had a "cold" 10 days ago but is "fine" now. His symptoms initially included fever to 101 ° F, purulent nasal discharge, and cough. He is now sneezing and has an occasional cough and clear runny nose, especially in the morning, and has been afebrile for 5 days. He has not been given any acetaminophen in 5 days. His lungs are clear on auscultation and he does not have wheezing, rales, or rhonchi.

When considering whether or not to proceed with the requested study in this patient, the first step is to determine the urgency of the procedure and if the result will change therapy or inform a diagnosis. Each case is unique and must be determined on the individual risk/benefit basis. Acute symptoms of an upper respiratory infection (URI) should be differentiated from the same symptoms demonstrated in noninfectious chronic conditions. For instance, sneezing and clear runny nose are present in allergic rhinitis, which does not carry the same risk for the patient as a URI. Identification of a mild URI, severe URI, or lower respiratory infection must be made since the implications are different with regard to risk and potential cancelation. Mild URI consists of minimal cough, no fever, clear runny nose, sneezing, a nontoxic appearance, normal activity level, and clear lung fields on auscultation. A severe URI is accompanied by symptoms of malaise, fever greater than 38.3 °C, sneezing, productive cough, toxic appearance, and upper airway congestion. A child with a lower respiratory infection usually has a severe productive cough with purulent sputum, wheezing, fever, rales, rhonchi, toxic appearance, and tachypnea with or without respiratory distress.

Children under the age of 5 years usually experience 4–6 URIs each year, especially during the winter season, and the inflammatory response and increased reactivity of the lower airway may persist for up to 6 weeks after a viral infection. If a child's procedure is canceled for 6 weeks, he/she is usually into the next URI, so the most prudent recommendation is to wait until the acute symptoms have resolved and then reschedule 2 weeks after that. Children with known URI may experience an increase in respiratory events when intubated or the airway is instrumented; however, there is no increase in laryngospasm or bronchospasm when there is a natural airway. There is, however, a significant risk of oxygen desaturation and hypoxemia even with the use of supplemental oxygen.

Considerations for proceeding in this child include the following: This is not an urgent procedure so the procedure may be rescheduled. This child has a resolving URI and demonstrates only mild symptoms without lower respiratory involvement. The reactive airway disease is not an active problem and not a cause of increased risk. Since there still is a risk of hypoxemia and increased oxygen requirement if the study proceeds, he should have full monitoring and supplemental oxygen administered. He should also demonstrate that he can lie flat without coughing prior to the start of the case. If he cannot, and the case is postponed, the end point should be no coughing and reschedule 2 weeks after all symptoms have resolved.

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