

Chapter 5

The Paediatric Airway: Normal and Abnormal

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Abstract Management of paediatric airway is a great challenge especially for the non-paediatric anaesthesiologist. The children's airway is different from adults and any mishandling of it can lead to airway obstruction and hypoxia. The first part of the chapter aims at providing a basic understanding of anatomy and physiology in paediatric airway, followed by a basic airway evaluation. The second half of this chapter provides simple principles for management of the abnormal paediatric airway.

Keywords Paediatric airways · Anatomy · Physiology · Management · Abnormal airway

Introduction

Managing paediatric airway poses a great challenge to many physicians as the anatomy and physiology of a child's airway is considerably different from an adult [1, 2]. Congenital abnormalities and acquired diseases can complicate the management of the child's airway further. Hence, the objective of this chapter is to enhance the understanding of the normal anatomy and physiology of a child and its clinical implications. The management of basic and abnormal airway in children is also described in this chapter. This will hopefully help the attending physician to manage the children's airway safely.

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Anatomy of the Airway

Airway is defined as the passage whereby oxygen reaches a person's lungs from the environment and for the carbon dioxide to be removed from the lungs back into the environment. This passage extends from the nose or mouth, joining the pharynx, larynx, glottis, trachea, bronchus and bronchioles in the lungs [3].

The anatomy of a child's airway differs from an adult in many ways. Firstly, the size of the airway is smaller, hence making it slightly more difficult to manage. Infants have smaller mouth and larger tongue as compared to adult. They have short and floppy epiglottis with vocal cord that is anterior. The vocal cord is located higher at the level C3 and C4 as compared to C5 and C6 in adult. These characteristics can cause the process of laryngoscopy and intubating a child difficult. A straight blade like Miller's blade would probably be more suitable to intubate a neonate than a curve Macintosh blade. We should lift up the floppy epiglottis in children with a straight blade like Miller's in order to visualize the vocal cord better. Due to the anterior vocal cord, positioning the child's head in the neutral position is the optimal way to view the vocal cord at laryngoscopy. In fact, extreme neck extension can actually obstruct the airway.

In children, the trachea is narrow, short and funnel-shaped with the narrowest part at the level of cricoid cartilage. On the other hand, adult's trachea is cylindrical and the narrowest part at the vocal cord region (Figs. 5.1 and 5.2). Therefore, choosing the right size of endotracheal tube for intubation is important as the narrowest point of the trachea in children is at the subglottic area. A tight fitting endotracheal tube at the vocal cord can cause tracheal oedema, which can lead to laryngeal damage, tracheal damage and subglottic stenosis after extubation.

Children have smaller trachea in comparison to adult. The average diameter of an infant's trachea is 3–4 mm as compared to the average diameter of adult's

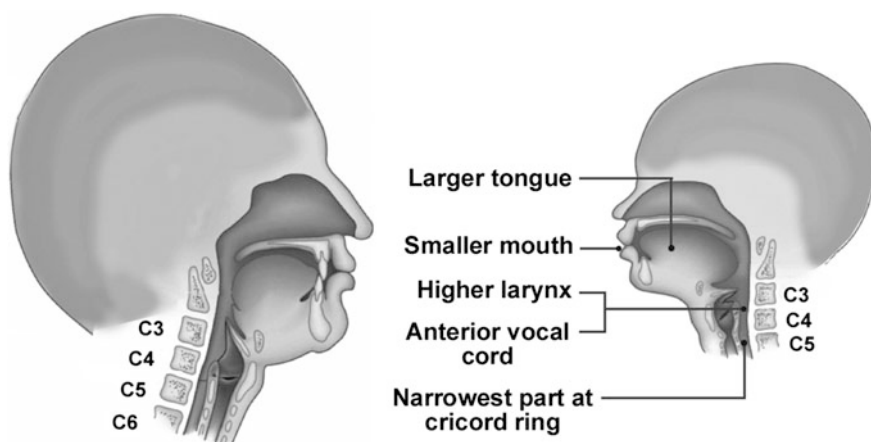


Fig. 5.1 Differences of paediatric and adult airway

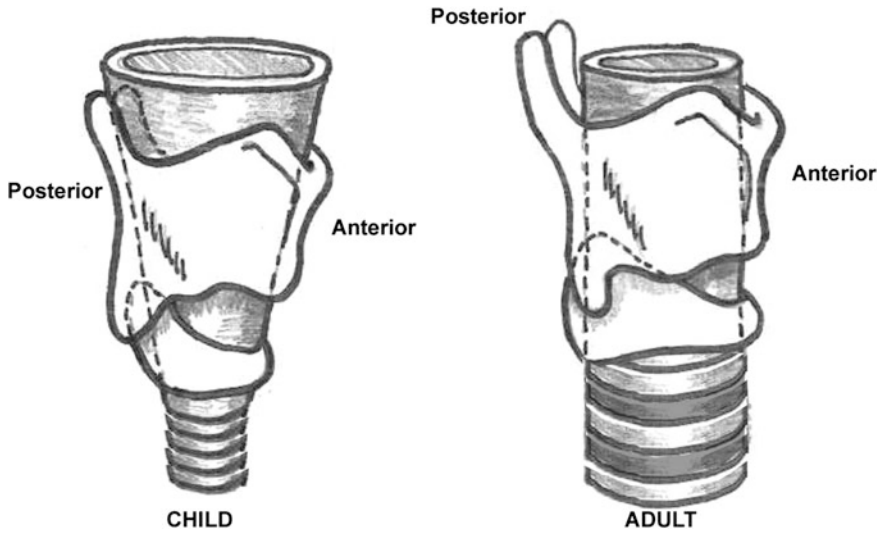


Fig. 5.2 Comparison of larynx of a child and an adult

trachea is 18 to 20 mm [4, 5]. Hence the ratio of the diameter of a neonate's trachea to adult trachea is about 1:5. The small radius of the trachea in infant has a great impact on the airway resistance. A small change in the airway radius will

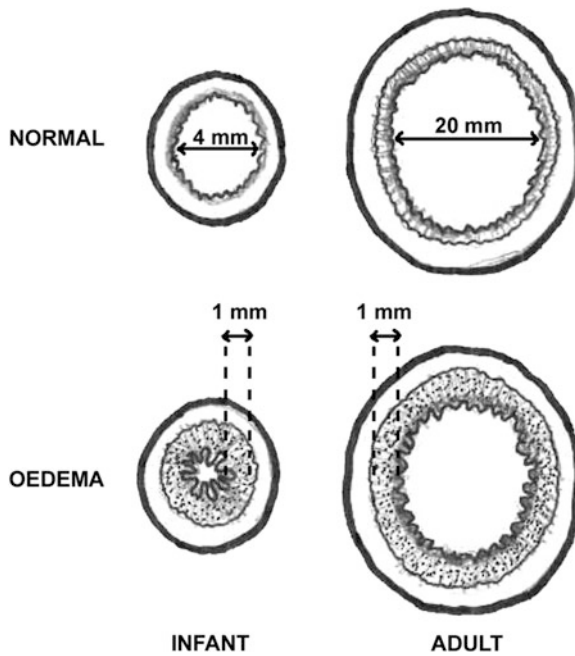


Fig. 5.3 The effect of oedema on the airway resistance of an infant versus an adult

increase the resistance of the airway by the fourth power as shown by the Poiseuille equation and illustrated in Fig. 5.3.

$$R = 8nl/\pi r^4$$

(R = resistance, l = length of the airway and r = radius of the airway
n = viscosity of the gas)

Therefore, smaller airways cause them to be more susceptible to obstruction due to oedema.

Poiseuille's law: if the radius of the airway is halved, the resistance will increase by 16x.

Neonate has immature laryngeal, tracheal and bronchial structures. These structures are poorly cartilaged, hence are soft, very compliant and are easily compressed. This can lead to dynamic airway compression especially at the thoracic inlet where the pressure gradient between the atmosphere and the intra tracheal pressure is the greatest. These cartilages are fully developed at the age of 10–12 years [6].

Physiology of the Airway

Newborn infants are obligatory nasal breathers as they are unable to coordinate the movement of their laryngeal structures with respiratory effort. Most of them will be able to coordinate this movement by 3–5 months of age [7]. Their narrow nasal passages can be easily blocked. In fact, the major cause of the airway resistance in infant originates from the nasal passages. As an obligate nasal breather, this can lead to upper airway obstruction in infant [8]. This can be due to congenital causes such as bilateral choanal atresia, acquired such as secretions during upper respiratory tract infection or iatrogenic such as insertion of nasogastric tube.

Infant's ribs are more cartilaginous than adult's ribs. Therefore, their chest wall is more compliance when compared to adults. On the other hand, they have low lung compliance due to the presence of thick-walled alveolar precursors and decreased amounts of elastin. As a consequence, the infant's alveolar are more prone to collapse, resulting in lower resting lung volume. To overcome these problems, infants try to maintain a larger resting lung volumes with a several mechanism such as premature cessation of the expiratory phase (braking) at the laryngeal and diaphragmatic levels and stabilization of the chest wall with increased intercostal tone during exhalation [9]. The lung compliance improves with increasing age of a child [10].

The closing volumes of very young children encroach the functional residual capacity. Closing volume is the lung volume at which terminal airways begin to collapse. Large closing volumes increase dead-space ventilation and can lead to atelectasis and shunting. Children also have lesser number of alveoli in comparison to adult. They only have approximately 10 % of adults' alveoli. The alveoli

clusters only develop to adult level over the first 8 years of life. Most importantly, children have higher oxygen consumption when compared to adult. The oxygen consumption of a child is 6 ml/kg/min as compared to adult, 3 ml/kg/min. In order to optimize work of breathing to meet the high oxygen demand, infants breathe at higher respiratory rate rather than a bigger tidal volume. As a result of high oxygen consumption and limited lung reserves, neonates are at higher risk of hypoxia.

Infants depend primarily on diaphragmatic breathing, as their ribs are horizontal preventing the 'bucket handle' action movements like in adult's breathing. This limits the increase in infants' tidal volume. Therefore, their minute ventilation is rate dependant. A normal respiratory rate for a neonate is around 30–50/min. In addition, infants have less of type 1 oxidative muscle fibers in the diaphragm and intercostals muscle that allows repetitive motion without fatigue. Hence, a sustained increase in respiratory rate in infant will lead to muscle fatigue and apnea. The number of this muscle increases in children when they grow over the first year of life.

The control of breathing in a neonate is different compared to adult. Hypoxia causes the neonate to increase the ventilation transiently, before depressing the ventilation. The initial increase in ventilation to hypoxia is due to the stimulation of the peripheral chemoreceptors, mainly the carotid body. The cause of depressed ventilation to hypoxia in the later part in the neonate is not fully understood, but this could be due to the immature control of respiration located at the medullary respiratory centers [11]. In premature babies this response is exaggerated. The response to hypercarbia is the same as in adults, but is more rapid because of a lower resting carbon dioxide level. The ventilatory response of neonate to hypoxia is affected by temperature, level of arousal and maturity of the neonate.

Anaesthesia affects the control of breathing in neonates in many ways. The respiratory depressant and sedative effects of the anaesthetic drugs and exposure to cold ambient temperature in the operating theatre can blunt the neonate's ventilatory response to hypoxia. Hence, the anaesthetist should use drugs that have better respiratory and sedative recovery profile. It is also important to keep the patient in normothermia perioperatively with active warming devices.

Evaluation of Paediatric Airway

In managing a child's airway, it is utmost important to evaluate and assess the child systematically. A careful medical history, physical examination and a review of relevant investigation should be performed before any decision of intubating the airway is taken. A history should include, history of systemic illness that predisposes a patient to airway problem such as Asthma, history of recent upper respiratory tract infection (URTI) and any previous records of anaesthesia and intubation. In clinical examination, apart from examining the airway, we should also examine the cardiovascular and respiratory system thoroughly. This is because, any problems in these systems can predispose patient to hypoxia during airway manipulation. We should also look for any obvious features of difficult airway such as craniofacial

abnormality and related syndromes. Certain congenital syndromes are associated with difficult airway. This will be discussed in the next section.

Child's Airway and Clinical Implications

A neonate with distended acute abdomen is at risk of developing hypoxia. The distended abdomen will lead to splinting of the diaphragm that limits the depth of diaphragmatic movements. The splinting of the diaphragm will also cause alveolar collapsed in the neonate. A combination of a sick neonate with a high oxygen demand, poor lung reserves and limited ventilation rate will render the neonate to desaturate very rapidly.

The Abnormal Paediatric Airway

The paediatric airway is vulnerable to obstruction because of its anatomy, size, and susceptibility to injury and disease. This risk is increased if the airway is abnormal. A heterogenous group of pathological conditions affects the airway. Management depends on the specific disorder. Interventions by clinicians with special skills can adequately manage airway emergencies (obstruction and respiratory failure), ideally in the Operation Theatre (OT) [12].

This chapter does not address the management of the abnormal or obstructed airway inside the OT, that is, the tools and strategies for intubation and intra-operative management.

To understand the individual characteristics of the entire spectrum of abnormal airway disorders, doctors are encouraged to read further. Recognition and management of some specific lesions are highlighted only.

Recognition

Routine evaluation of the airway requires a thorough history and physical examination. Any of the following features from history and physical assessment should alert the clinician to potential problems and further work-up:

A. History

- Snoring, noisy breathing
- Chronic cough, frequent respiratory tract infections
- Sudden onset coughing, choking or foreign body aspiration
- Feeding problems with respiratory distress
- Hoarse voice, stridor, cyanosis

- Previous anaesthetic problems (difficult intubation, extubation or difficult mask ventilation)
- History of congenital syndrome

B. Physical Examination

- Respiratory rate, baseline oxygen saturation
- Retractions of suprasternal, intercostal, subcostal muscles
- Facial expression, nasal flaring, mouth breathing, drooling, colour of mucous membrane
- Mouth opening, size of mandible, location of trachea
- Masses involving oropharynx, face, neck
- Stridor (inspiratory/expiratory)
- Global appearance (congenital disorders)

Additional investigations, apart from a detailed history and physical examination, help in diagnosis and decision making. Radiographs of the upper airway may locate the aetiology and site of airway obstruction. Extra information may be obtained from MRI and CT scan. Biomedical imaging is best undertaken in the presence of skilled personnel with appropriate facilities for airway management. Endotracheal intubation takes priority over radiographic diagnosis when respiratory failure is imminent. Blood gases are useful but performing an arterial puncture on an agitated child may aggravate respiratory distress.

General Management of Airway

A patent airway has to be ensured at all times, preventing hypoxia, respiratory acidosis, aspiration and asphyxia. In the early stage before deterioration sets in, non-invasive airway management for respiratory distress in a sick child with abnormal airway may be managed by oxygen supplementation using nasal prongs, positioning prone or lateral and CPAP devices. Removal of secretions is important and tube feeding may be initiated.

A child with stridor alerts every clinician. Stridor is the sound caused by abnormal airflow during breathing [13]. The cause of stridor can be located in extrathoracic (nose, pharynx, larynx, trachea) or intrathoracic airway (tracheo-bronchial tree). Stridor may be acute or chronic and may be congenital or acquired. It is a sign and not a diagnosis. The paediatrician has to determine the severity of respiratory compromise and the need for immediate intervention. Referral to an ENT surgeon for an upper and lower airway endoscopy depends on whether a significant lesion is suspected. Collaboration with colleagues from related disciplines for follow-up and subsequent management is often required.

Flexible bronchoscopy is a technique to evaluate, among its many other uses, the functional disorders of the airway. It is relevant particularly to predict the

development of subglottic stenosis and tracheomalacia in neonates who receive prolonged ventilation and are oxygen dependent.

A broad classification of the abnormal paediatric airway that focuses on aetiology is: congenital conditions, inflammatory disorders and foreign body in the airway/airway trauma.

Congenital Conditions

Varying degrees of chronic airway obstruction manifest in this group of children. In some, the airway is not the only issue but there are problems with the heart, central nervous system and other body organs. Sleep-disordered breathing syndromes can lead to adverse complications in the cardiovascular system (pulmonary hypertension), neurocognitive function and growth. These syndromes occur more frequently in patients with craniofacial disorders where early recognition and surgical corrections are team-based and highly specialized [14].

Some of the syndromes associated with abnormal airways include the following:

- Congenital neck masses (dermoid cyst, cystic hygroma, lymphangioma, neurofibroma)
- Congenital anomalies of respiratory tract (chonal atresia, tracheomalacia, laryngomalacia, tracheal stenosis, laryngeal web, vascular ring)
- Congenital syndromes (Pierre Robin, Down's syndrome, Goldenhar, Cruzon, Achondroplasia)
- Metabolic disorders (mucopolysaccharidosis)

The biggest challenges for acute airway management in congenital airway disorders are anatomical features associated with hypoplasia of the mandible, hypoplasia of the midface or associated with a large tongue (macroglossia). A multidisciplinary approach is generally needed with referrals to several subspecialties including ENT and Anaesthesiology.

An example of congenital condition is Pierre Robin Syndromes (Fig. 5.4). Pierre Robin syndrome represents an abnormal developmental process associated with a cluster of typical clinical features:

- micrognathia, retrognathia
- cleft palate
- glossoptosis (implying a relatively large tongue with risk for upper airway obstruction)

The baby has inspiratory and expiratory airway distress. Obstruction leads to hypoxia, pulmonary hypertension, cor pulmonale or failure to thrive (secondary to difficulty in swallowing and aspiration). Despite the risk for breathing difficulties, the goal of treatment is to optimize growth and adequate nutrition. General



Fig. 5.4 Pierre Robin syndrome

measures such as placing the infant in the prone or lateral position may prevent the tongue falling backwards. Nasogastric or gastrostomy tube feedings may be considered. Respiratory distress may be managed without an operation (either by prone positioning, short term intubation, or placement of a nasopharyngeal airway). Gastroesophageal reflux seems to be more prevalent in these infants. The reflux of acidic contents in the posterior pharynx and upper airway can worsen airway obstruction. Treatment options include upright positioning, small and frequent feedings and pharmacotherapy (such as proton pump inhibitors).

When conservative treatment fails, emergency surgical treatment by an artificial ankyloglossia or glossopexy may prevent recurrent glossoptosis. The technique of tongue fixation below the mandible is a surgical option. The placement of a button helps to prevent the stitches cutting through (Fig. 5.5).



Fig. 5.5 Tongue fixation below the mandible



Fig. 5.6 Laryngoscopy view of swollen epiglottitis

Inflammatory Disorders

Inflammatory disorders of the airway in children usually result in progressive airway obstruction as seen in epiglottitis, croup and diphtheria.

Acute epiglottitis is most common between ages 2–8 years old but can occur at any age [15]. It is an airway emergency and potentially life threatening. Epiglottitis is, in reality, supraglottitis because the inflammatory process frequently involves other structures such as the aryepiglottic folds, the arytenoids and the entire supraglottic area (Fig. 5.6). The most important cause of infection is *Haemophilus influenzae* (Type B) for which there is a specific vaccine. Some cases are due to *Streptococcus pneumoniae* and other bacteria. Epiglottitis presents acutely with high fever, anxiety, stridor, drooling and dysphonia. The child looks toxic and prefers to sit and lean forward to avoid the pain caused by the epiglottis touching the posterior pharyngeal wall. Rapid diagnosis and airway intervention are necessary because sudden airway obstruction can occur due to rapid progression of the swelling. Lateral soft tissue radiograph may reveal the classic “thumb sign” of the swollen epiglottis. CT imaging can differentiate other conditions that have similar clinical presentation (such as peritonsillar abscess or retropharyngeal abscess).

If the child deteriorates in the emergency department, rapid intubation has to be performed, bearing in mind the difficult airway and the dangers of laryngospasm and total airway obstruction. If condition of the child permits, joint evaluation and treatment by trained anaesthesiologist and otolaryngologist can ensure optimal management of the airway in a controlled manner with facilities including rigid bronchoscopy, cricothyrotomy and tracheostomy. After the airway is secure,

antibiotic therapy is the key to resolution of the infection. Second or third-generation cephalosporin, either alone or in combination with penicillin or ampicillin for streptococcal cover, is suitable.

Foreign Body in the Airway/Airway Trauma

These are acute airway disorders associated airway instability and sudden airway obstruction. Examples are airway burns, accidents with faciomaxillary and oropharyngeal injuries, cervical fracture and dislocation, inhalational injury and post-intubation croup. Rapid assessment is essential. Resuscitative measures are prioritized to airway and circulation in a collapsed child. Urgent biomedical imaging may not be possible.

Foreign body in the airway is not an uncommon problem (Fig. 5.7). A high index of suspicion may be the only clue for a foreign body (FB) in the airway. A history of choking is pertinent. Food items (peanut, bean) are common in infants and toddlers while non-food objects (safety pin, coin) are aspirated in older children. Significant obstruction is indicated by agitation, cyanosis, a weak or absent cry, retractions and stridor. The urgency to establish a patent airway depends on the degree of obstruction and functional impairment. A chest radiograph can confirm the presence and the location of radiolucent objects in the respiratory tract. Retrieval of the FB is the only definitive treatment [16].

Complete airway obstruction may occur on arrival at the emergency department. If this happens, the FB may be located at the larynx or it is in the trachea. In this crisis, the FB is big enough and located strategically to cut-off airflow completely. Direct laryngoscopy can remove the FB above the glottis but if the FB is in the trachea, the FB is purposely pushed into one bronchus with an endotracheal tube. In this partially obstructed condition of the airway, there is some time to transport the patient to the OT for a more controlled bronchoscopic removal of the FB.

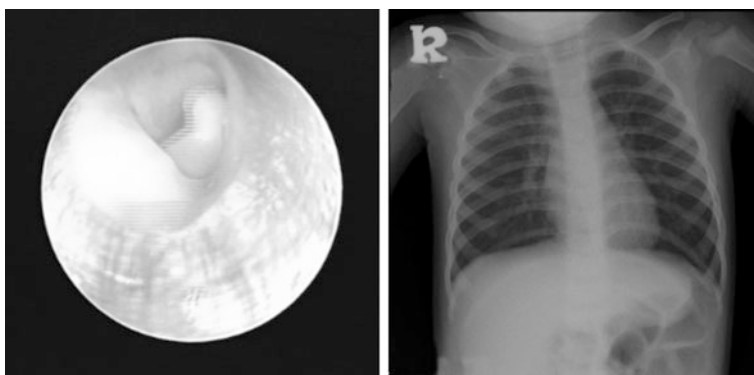


Fig. 5.7 Radio opaque bean in right bronchus

Conclusion

Management of paediatric airway is a great challenge especially for the non-paediatric anaesthesiologist. The children's airway is different from adults and any mishandling of it can lead to airway obstruction and hypoxia. The child with an abnormal airway requires a thorough evaluation and referral to a centre with paediatric expertise in all subspecialties is optimal for integral care.

References

1. Eckenhoff J (1951) Some anatomic considerations of the infant larynx influencing endotracheal anesthesia. *Anesthesiology* 12:401–410
2. Coté CJ, Ryan JF, Todres ID, Groudsouzian NG (eds) (1993) *A practice of anesthesia for infants and children*, 2nd edn. WB Saunders, Philadelphia
3. Chan YK, Ng KP (2012) *Physiological basis of acute care*. Elsevier, Singapore
4. McNiece WL, Dierdorf SF (2004) The pediatric airway. *Semin Pediatr Surg* 13:152–165
5. Dickison AE (1987) The normal and abnormal pediatric upper airway: recognition and management of obstruction. *Clin Chest Med* 8:583–596
6. Wheeler M, Coté CJ, Todres ID (2009) The pediatric airway. In: Coté CJ, Lerman J, Todres ID (eds) *A practice of anesthesia for infants and children*, 4th edn. Saunders Elsevier, Philadelphia, pp 238–243
7. Cote CJ (2012) The difficult paediatric airway. *South Afr J Anaesth Analg* 18(5):230–239
8. Wheeler DS, Wong HR, Shanley TP (2009) *Resuscitation and stabilization of the critically ill child*, 4th edn. Springer, London, pp 1–30
9. Holzman RS, Mancuso TJ, Polaner DM (2008) *Pediatric anaesthesia*, 1st edn. Lippincott Williams & Wilkins, Philadelphia, pp 3–16
10. Kovarik WD (2004) Paediatric and neonatal intensive care, critical care medicine. In: Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL (eds) *Miller's anaesthesia*, 6th edn. Churchill Livingstone, New York
11. Martin RJ, Abu-Shaweesh JM (2005) Control of breathing and neonatal apnea. *Biol Neonate* 87:288–295
12. Myer CM, Cotton RT (1995) *The pediatric airway: an interdisciplinary approach*. Lippincott Williams & Wilkins, Philadelphia, PA
13. Leung AKC, Cho H (1999) Diagnosis of stridor in children. *Am Fam Physician* 60(8):2289–2296
14. Sculerati N, Gottlieb MD, Zimble MS, Chibbaro PD, McCarthy JG (1998) Airway management in children with major craniofacial anomalies. *Laryngoscope* 108(12):1806–1812
15. Abdallah Claude (2012) Acute epiglottitis: trends, diagnosis and management. *Saudi J Anaesth* 6(3):279–281
16. Lin CH, Chen AC, Tsai JD, Wei SH, Hsueh KC, Lin WC (2007) Endoscopic removal of foreign bodies in children. *Kaohsiung J Med Sci* 23(9):447–452