Acute and chronic airway obstruction in children

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Abstract

Airway management is one of the fundamental skills of any anaesthetist. Considerable anatomical changes occur between birth and adulthood during the development of the paediatric airway. Knowledge of these changes will influence airway planning during childhood. Airway obstruction complicates airway management and any anaesthetist working with children should be able to assess the airway for the presence of obstruction and generate a differential diagnosis of cause. This article aims to summarize key anatomical features of the paediatric airway, common causes of airway obstruction in children and provide suggestions for how to manage these patients.

Keywords Airway management; children; obstruction

Royal College of Anaesthetists CPD Matrix: 1C01, 2A01, 2D02

Learning objectives

After reading this article, you should be able to:

- describe the differences between a paediatric and adult airway
- recognize the most common causes of airway obstruction in children
- propose a management plan for a child with airway obstruction

Anatomical changes of the paediatric airway

At birth, the head is proportionally larger than the body with a prominent occiput that causes a tendency toward neck flexion.[1] Neonates and young infants are obligate nasal breathers and any reduction in airway diameter will impede airflow. A relatively large tongue can obstruct the airway and this is exacerbated by decreased muscle tone during anaesthesia.[1]

The glottic opening is the narrowest part of the airway in both children and adults, but the airway at the level of the fixed cricoid cartilage ring is particularly vulnerable to subglottic oedema and subsequent stenosis.[2]

The epiglottis also develops with age. In adults it is broad, whereas in infancy it is narrower and softer, and in neonates it is often relatively longer and U-shaped in appearance (Figure 1). At laryngoscopy, lifting the epiglottis using a straight blade to provide a view of the vocal cords may be useful.[1]

At birth, the cricoid cartilage lies at the lower border of the fourth cervical vertebra compared to the sixth cervical vertebra in adults.[1] In infancy, the more superior location of the larynx can make visualization of the laryngeal inlet more difficult during laryngoscopy, however as the structures are loosely embedded, they can be externally manipulated into a better position.

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Causes of airway obstruction (Table 1)

Craniofacial anomalies

Children with craniofacial conditions will be mostly managed in tertiary level paediatric centres. However, they may present to district general hospitals in emergency situations. These conditions include cleft palate, craniofacial dystosis, mandibulofacial dystosis, hemifacial macrosomia and Down syndrome. All will require additional thought when planning a suitable airway management strategy.

Cleft lip and palate is the most common craniofacial disorder affecting 1 in 750 live births.[3] Airway obstruction can occur if the tongue falls into the cleft obstructing the nasal airway. This can easily be overcome using an oropharyngeal airway. Pierre Robin syndrome is associated with cleft palate and is characterized by micrognathia, glossoptosis and airway obstruction.[4] The presence of sleep apnoea in these children might predict difficulty maintaining mask ventilation, and intubation should be expected to be difficult due to micrognathia.

Children with craniofacial dysostosis have a degree of midface hypoplasia with the mandible appearing relatively prognathic. The palate is high arched with small nasal passages resulting in a degree of choanal stenosis. This is a feature of Pfeiffer, Apert and Crouzon syndromes. Airway management may be complicated by difficulty maintaining a seal with a face mask due to the small midface. Closure of the mouth can cause the tongue to occlude the airway due to the smaller oral cavity, worsened by choanal stenosis. Intubation is usually straightforward unless neck mobility is limited.[3]

Treacher Collins syndrome (mandibulofacial dystosis) is a rare disorder affecting 1 in 50,000 live births.[4] It is characterized by maxillary, zygomatic and mandibular hypoplasia.[3] This, combined with a small oral aperture, temporomandibular joint (TMJ) abnormalities and a high arched or cleft palate can cause difficulties with laryngoscopy. Both mask ventilation and tracheal intubation are likely to be difficult. The airway worsens with age and previous intubation success does not guarantee this to be repeated. This contrasts to Pierre Robin syndrome when intubation becomes easier with age.[4]

Goldenhar syndrome is characterized by hemifacial microsomia (mandibular hypoplasia, auricular abnormalities with overlying soft tissue loss and facial nerve weakness) with macrostomia and vertebral bone abnormalities.[3] If bilateral, the condition can easily be confused with Pierre Robin syndrome. Children with Goldenhar syndrome may also have fused or hemivertebrae causing a limitation in neck movement. Intubation under directed vision should be expected to be difficult and will remain so after reconstruction of the jaw and temporomandibular joint due to contractures.[3]

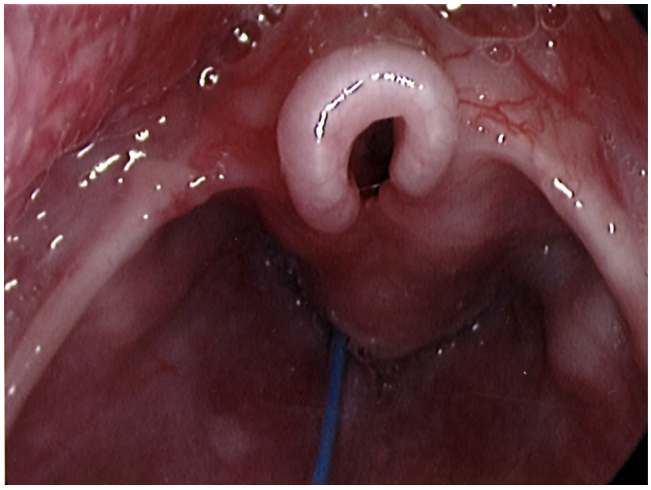

Figure 1 U-shaped epiglottis in a neonate, note that this image was taken with a suspension laryngoscope in place for ENT surgery, commonly the long neonatal epiglottis points further backwards obscuring much of the larynx.

Causes of acute and chronic airway obstruction

Site		Acute	Chronic
Nose			Choanal atresia Congenital stenosis of piriform aperture
Oropharynx		Peritonsillar abscess Retropharyngeal abscess Diphtheria	Micrognathia Macroglossia Tonsillar hypertrophy
Larynx	Supraglottis	Epiglottitis	Laryngomalacia Laryngeal web
	Glottis	Laryngospasm	Vocal cord palsy
	Subglottis	Croup	Subglottic stenosis Subglottic haemangioma
Trachea		Bacterial tracheitis	Tracheomalacia Mediastinal mass
Bronchi		Foreign body Asthma Bronchiolitis	Bronchomalacia Vascular ring

Table 1

The mucopolysaccharidoses are a group of genetic disorders characterized by a lysosomal enzyme deficiency leading to the accumulation of mucopolysaccharides throughout the body. This causes progressive airway obstruction due to an enlarged tongue and blockage of the nasal passages. Bone marrow transplantation is now used to reverse mucopolysaccharide deposition and subsequently airway obstruction.[3]

Trisomy 21 is the most common chromosomal abnormality in children, and anaesthetists in both tertiary and district general hospitals working with children will manage this condition. These patients may experience airway obstruction due to a narrowed nasopharynx, a relatively large protuberant tongue and the presence of a cleft lip or palate. The larynx and cricoid ring are smaller with an increased risk of subglottic stenosis and so a smaller tracheal tube size is usually appropriate. There is also a high incidence of atlanto-axial instability and neck movement should be minimized to prevent injury with particular caution being given to prevent hyperflexion.[3]

Nasopharynx

Neonates are obligate nasal breathers and therefore any nasal obstruction can cause airway obstruction and subsequent respiratory distress. Choanal atresia is the most common cause of nasal obstruction; other causes include congenital stenosis of the pyriform aperture or nasal masses (encephaloceles, gliomas, dermoid cyst or teratomas).

Choanal atresia: (Figure 2) occurs in 1 in 8000 live births and is the most common congenital nasal anomaly.[5] It is defined by the anatomical closure of the posterior choanae in the nasal cavity due to a failure in embryonic development. Choanal atresia is a component of CHARGE syndrome (coloboma of the eye, heart malformations, choanal atresia, growth retardation, genital or urinary defects and ear defects) and two-thirds of patients will have an associated defect. It can be membranous or bony in nature but is usually mixed.[5] When bilateral, choanal atresia will present in early life with respiratory distress, apnoea or cyanosis and difficulty feeding. The inability to pass a small suction catheter distally through both nares is suggestive of choanal atresia however flexible nasal endoscopy and computed tomography (CT) will confirm the diagnosis.[5] Management involves surgical correction.

Oropharynx

Tonsillitis and peritonsillar abscess: Tonsillar hypertrophy is a common condition with tonsillectomy being a routine procedure for anaesthetists working with children. Children undergoing adenotonsillectomy should be assessed for the presence of obstructive sleep apnoea (OSA) preoperatively to stratify their postoperative risk. These children are at higher risk of supraglottic obstruction, breath holding and oxygen desaturation during anaesthesia. They require more time for inhalational induction and emergence from anaesthesia. The use of long-acting opioids is controversial due to concerns of postoperative respiratory depression and this needs to be balanced against analgesic requirements. Patients with severe OSA should be considered for high-dependency care following adenotonsillectomy because surgery will not immediately normalise their breathing pattern. Tonsillitis will rarely cause airway obstruction but it can develop into a peritonsillar abscess. This is characterized by severe unilateral throat pain and occasionally, trismus. Tonsillectomy during episodes of acute inflammation is controversial due to the concerns of haemorrhage, but surgery may be necessary if an abscess has developed. An abscess will be evident by asymmetrical tonsillar swelling and deviation of the uvula away from the affected side.[6]

Retropharyngeal abscess: The retropharyngeal space is a potential space extending from the skull base to the posterior mediastinum on either side of the midline. Lymph nodes in this space can become inflamed due to a respiratory tract infection or

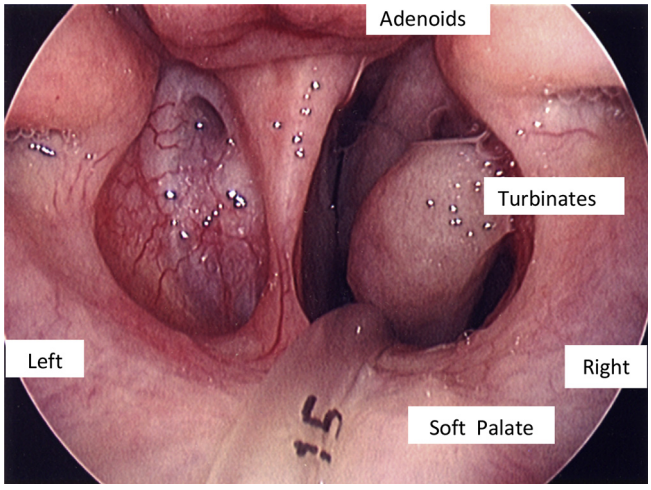

Figure 2 Unilateral left choanal atresia. This picture is taken from the back of the nasopharynx looking upwards and forward. The palate is being held out of the way by a suction catheter in the right nostril.

rarely, laryngoscopy or dental procedures[7] with subsequent retropharyngeal abscess formation. Children may present with symptoms of pharyngitis that progresses to high fever, neck swelling and stiffness, drooling and torticollis. Stridor is a rare finding.[5] As with a peritonsillar abscess, trismus may be present due to spasm of the medial pterygoid muscle, complicating subsequent airway management.[7] Stridor and respiratory distress are found in 5% of cases.[6] Surgical drainage of the abscess with antibiotics is required. Antibiotic therapy should target gram positive bacteria and anaerobes prior to the results of cultures.

Larynx

Laryngomalacia is the most common cause of airway obstruction at the level of the larynx. Other causes include vocal cord palsy or paralysis, subglottic haemangiomas, laryngeal clefts, webs or cysts.

Laryngomalacia: is the most common cause of stridor in the newborn.[5] Stridor is caused by turbulent airflow through the area of obstruction and is more noticeable when supine or during feeding, agitation or crying. Symptoms begin within the first 4–6 weeks of life and typically resolve by 24 months.[5] Diagnosis is confirmed by flexible endoscopy with visualization of supraglottic tissue collapsing into the airway and causing obstruction during inspiration (Figure 3). Characteristic abnormalities include elongation and lateral extension of the epiglottis, redundant bulky arytenoids and shortening of the aryepiglottic folds.[5] There are varied combinations of abnormalities in each individual patient resulting in a wide spectrum of disease. Most infants will have mild symptoms and are managed conservatively with the expectation that symptoms improve with growth. Positive pressure ventilation can be helpful in more symptomatic children and surgery is reserved for those who are failing to thrive, as evidenced by falling growth centiles.[5] Helium has low viscosity and will improve flow through an orifice. However a change from FiO_2 of 1 to FiO_2 of 0.3 in helium only increases flow by 50% so it is not always beneficial. In a longer procedure a patient who is easy to oxygenate may still accumulate CO_2 and helium may be useful.

Vocal cord paralysis: is the absence of movements of the vocal folds due to dysfunction of the motor nerve supply to the larynx.[5] It may be unilateral or bilateral. Bilateral vocal cord paralysis is usually idiopathic although it may be associated with central nervous system disorders such as cerebral palsy, hydrocephalus or spina bifida.[8] There may also be concomitant airway disease such as laryngomalacia, tracheobronchomalacia or subglottic stenosis. Bilateral vocal cord paralysis presents as an airway emergency with a high-pitched inspiratory stridor at rest that worsens with agitation.[5] Immediate intervention may be required. Unilateral vocal cord paralysis is generally well tolerated and may go unnoticed. Unlike bilateral vocal cord paralysis where a near normal voice is present, unilateral vocal cord paralysis can cause dysphonia with a hoarse cry when agitated.[5] Aspiration is a risk in both scenarios. When the clinical condition allows, diagnosis can be made via flexible laryngoscopy. Progressive airway obstruction requires intubation followed by tracheotomy. This is maintained long term awaiting spontaneous recovery. If this fails to occur then vocal cord lateralization procedures, arytenoidectomy and laser cordotomy can be considered to aid decannulation.[8]

Laryngospasm: The paediatric population have more pronounced laryngeal reflexes compared to adults and laryngospasm is not uncommon perioperatively. Complete laryngospasm is defined as complete closure of the larynx following an external stimulus. This is in contrast to partial laryngospasm that describes strong approximation of the vocal cords.[2] With complete laryngospasm, there will be chest movement without ventilation, whereas with partial laryngospasm, stridor may be heard due to the passage of air through a small space at the posterior commissure.[2] The management of laryngospasm is based on increasing the inspired oxygen concentration, providing positive airway pressure ventilation, removal of the triggering stimulus, deepening anaesthesia with propofol, or an inhalational agent if gas exchange is possible, and early progression to neuromuscular blockade if initial steps fail.

Anaphylaxis

Anaphylaxis can occur at any time and may be severe and life threatening. It can cause acute airway obstruction due to oedema of the retropharynx and larynx.[6] Treatment relies on prompt recognition with an ABCDE assessment, removal of the precipitant and treatment with adrenaline. The treatment algorithm developed by the UK Resuscitation Council (https://www.resus.org.uk/anaphylaxis/) should be followed.

Acute upper airway infections

Classically, three upper airway infections are described as causes of acute airway obstruction in children. Epiglottitis, croup and bacterial tracheitis can be difficult to distinguish from each other however slight differences in the clinical presentation may make one condition more likely.

Epiglottitis: is a severe, life-threatening infection characterized by oedema of the anterior surface of the epiglottis and supraglottic structures leading to rapid onset airway obstruction.[7] It was traditionally caused by *Haemophilus influenzae* type B, however after the introduction of the childhood vaccination

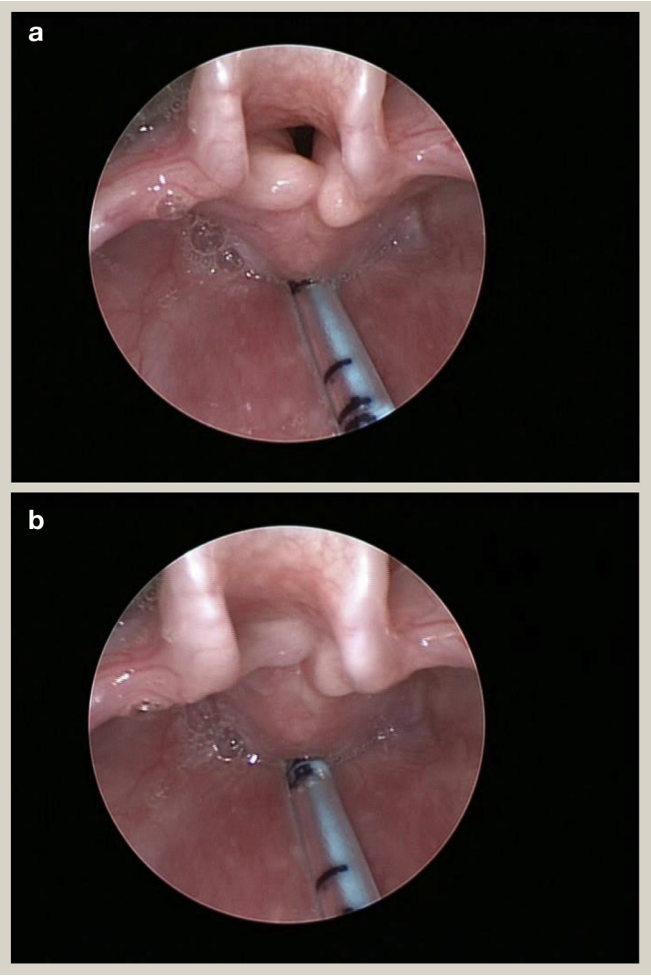

Figure 3 (**a**) Infant with laryngomalacia – exhalation. The airway is patent but the arytenoids are big & floppy with prominent cuneiform cartilages. (**b**) The same patient – now in inhalation, the arytenoids have been sucked into the larynx nearly obliterating the airway. Stridor was clearly audible.

programme in 1990, the incidence has fallen. Vaccine failure or other *Haemophilus* or *Streptococcus* species are responsible for new presentations.[7] These patients tend to be older and present with atypical symptoms.

Classically, children presented with a short history of respiratory distress, drooling, stridor and high fever.[7] An expiratory snore was common. They appeared toxic and naturally adopted a 'tripod' position in an attempt to maximize their airway patency.[7]

Management includes maintaining a calm environment until senior anaesthetic and ear, nose and throat (ENT) teams are present. Respiratory arrest due to complete airway obstruction can occur unprovoked or if intervention is attempted, and therefore the child should be undisturbed until their airway can be secured. Early administration of intravenous antibiotics is important however any attempt at venous cannulation should be delayed until after intubation as it may worsen respiratory distress.[7]

Early intubation after presentation is preferable due to the risk of complete obstruction of the laryngeal inlet. During intubation pressure on the thorax to release an air bubble may help identify the glottic opening to aid tracheal tube placement.[6] Diagnosis will be made during laryngoscopy and the causative organism can be identified following blood and laryngeal cultures.

Croup: or viral laryngotracheobronchitis is an acute clinical syndrome characterized by fever, hoarseness, barking cough and inspiratory stridor due to inflammatory airway wall oedema in the subglottic region. It typically affects children from 6 months to 4 years with a peak incidence at 2 years of age.[9] It is most commonly caused by parainfluenza virus type 1–3 but respiratory syncytial virus, influenza A or B, adenovirus or rhinovirus can also be causative.[7] (Figure 4).

There is a wide spectrum of disease severity and therefore clinical management must be guided by the progression of signs and symptoms such as stridor, work of breathing, hypoxia or apathy. Unlike epiglottitis, children can lie flat and are able to manage their oral secretions.[7]

If the clinical condition allows, management will be initially supportive with the use of steroids and nebulized adrenaline. Dexamethasone and nebulized budesonide are effective for all severities of croup and provide a reduction in symptom severity, the duration of hospital stay and readmission rate.[7] There is no optimal route of administration.[9] Nebulized adrenaline will produce a transient benefit by reducing subglottic oedema within 30 minutes after administration. Children will require continued monitoring and frequent assessment with the expectation of symptoms returning.[7] Intubation may be required in the most severe cases with a smaller tracheal tube size than usual. Extubation should be successfully achieved after 2–3 days.[9]

Bacterial tracheitis: (also known as pseudomembranous croup or bacterial laryngotracheobronchitis) is characterized by an invasive, exudative bacterial infection of the trachea. It is most commonly caused by *Staphylococcus aureus* but *Streptococcus* species, *Moraxella catarrhalis* and *Haemophilus influenza* may feature. It affects children aged 6 months to 8 years. Compared to croup, children with bacterial tracheitis are likely to be older and are less likely to respond to nebulized adrenaline. They are also more likely to develop respiratory failure compared to children with either croup or epiglottitis.[6]

Children tend to present with symptoms of an upper respiratory tract infection with the clinical picture progressing to a rapid onset of high fever, stridor and respiratory distress. Like croup, they can manage their oral secretions.[6]

Timely intubation prior to complete upper airway obstruction and antibiotic therapy covering *Staphylococcus* and other Gram positive organisms is recommended. Diagnosis can be made by endoscopy when subglottic oedema will be seen with the formation of pseudomembranous exudates. Removal of these can significantly improve symptoms. If the patient is too unstable to tolerate bronchoscopy, repeated use of suction catheters can produce similar results. Samples should be sent for culture with results used to target antibiotic therapy.

Subglottis

Congenital subglottic stenosis: is defined as a tracheal diameter of less than 4 mm at the cricoid in a term neonate.[8] It is the most common laryngeal anomaly that requires a tracheostomy in

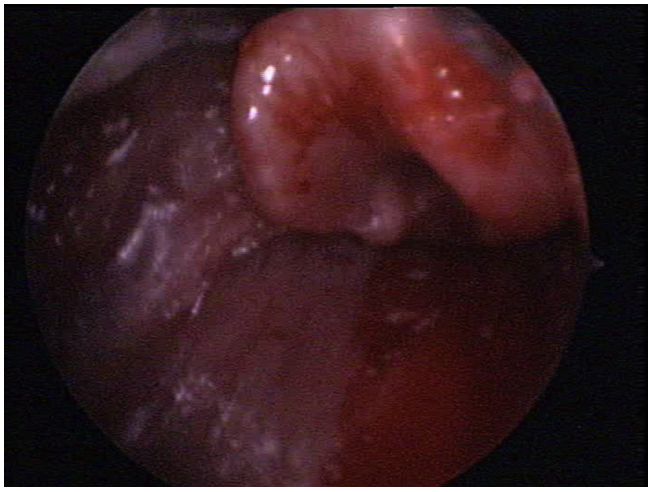

Figure 4 A swollen red epiglottis, this patient had croup/tracheitis.

neonates. It is a continuum of embryonic failures including laryngeal atresia, stenosis and webs and results from the incomplete recanalization of the laryngotracheal tube during the third trimester.[4] There are two types. The membranous form is milder and more common, resulting from submucosal hypertrophy with excess connective tissue (Figure 5). The cartilaginous type results from an abnormal shape of the cricoid cartilage.[8] The neonate most typically demonstrates a biphasic stridor with or without increased work of breathing.[5] Plain radiographs may demonstrate a narrowing at the level of the subglottis. Diagnosis is made after rigid bronchoscopy. Most cases will resolve spontaneously as the child grows although those with more severe disease will require tracheostomy. Laryngotracheoplasty may be needed to allow decannulation.[8]

Post extubation stridor and acquired subglottic stenosis: Post-extubation stridor is due to superficial injury of laryngeal tissue after prolonged ventilation with an endotracheal tube. This may be more likely if infection is present or if the child has oesophageal reflux. Nebulized adrenaline can be used to reduce subglottic oedema if present. Dexamethasone can be used prior to, and after extubation for 48 hours to reduce the incidence of stridor although the evidence for this is limited.[9] Resolution is usually spontaneous.

Severe injury causing ulceration and the formation of granulation tissue can lead to the development of acquired subglottic stenosis (Figure 6). Its incidence is reduced due to significant efforts to improve safety, reduce the duration of intubation and the monitoring of cuff pressures. A smaller tracheal tube size may be needed if intubation is required. If the obstruction is over 70% of the lumen, surgery or a tracheostomy may be needed.[9]

Trachea and bronchi

Tracheomalacia: is the most common congenital anomaly of the trachea. It is caused by an absence, deficiency or malformation of tracheal cartilage making the airway more vulnerable to collapse during respiration. The main bronchi can also be involved, or be affected in isolation. It is associated with prematurity and numerous congenital diseases and can be secondary to prolonged ventilation, tracheostomy or extrinsic compression of the trachea.[5]

Patients with tracheomalacia will likely have chronic symptoms of dyspnoea, stridor and feeding difficulties starting after the first few weeks of life, but these may worsen during episodes of lower respiratory tract infection or agitation. Positive pressure ventilation, either invasively or non-invasively can help to relieve the obstruction.[5]

Mediastinal masses: Mediastinal masses can cause life-threatening airway obstruction due to tracheobronchial compression. They can be due to congenital, neoplastic or infectious causes. Patients may complain of shortness of breath on exertion or with changes in position. A chest X-ray is useful but computed tomography provides essential information about the degree and site of airway obstruction.[10] (Figure 7).

General anaesthesia can precipitate complete airway obstruction in symptomatic patients due to a decrease in inspiratory and bronchial muscle tone and reduction in lung volume making the airway more susceptible to extrinsic compression.

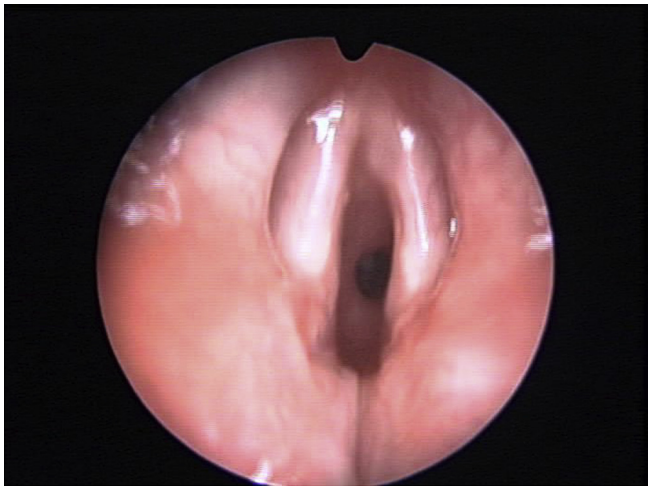

Figure 5 Congenital subglottic stenosis, this patient had trisomy 21 and had never been intubated.

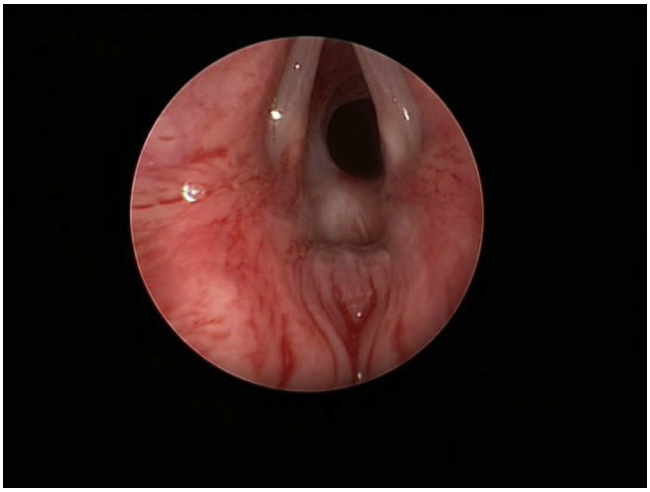

Figure 6 Acquired subglottic stenosis from prolonged endotracheal intubation in a neonate. Normal vocal cords with distal circumferential scarring, more pronounced on the posterior trachea, is normal for this aetiology.

Figure 7 Large cystic hygroma involving the neck and mediastinum. The internal jugular vein runs through the cysts. At MLB the trachea was significantly narrowed and deviated.

Airway obstruction can be relieved by passing the endotracheal tube distal to the obstruction. Altering the position of the patient to a lateral or prone position may assist with ventilation.[10] Due to the risks of general anaesthesia, tissue sampling under local anaesthesia should be considered when possible.

Foreign body: Accidental inhalation of a foreign body remains a life-threatening event for children. It most commonly occurs in children under 3 years old. A history of a witnessed choking event with symptoms including cough, dyspnoea, wheeze or stridor with no other signs of illness is highly suggestive of an inhaled foreign body. Children may also have delayed presentation with recurrent pneumonia, empyema, persistent cough or wheeze. The majority of foreign bodies will lodge in the bronchial tree with the remainder at the level of the larynx or trachea (3% and 13% respectively).[6] The right side is more commonly affected than the left.

Acutely, a choking child should be managed according to advanced paediatric life support guidelines. Subsequently, a child with a suspected inhaled foreign body will be a managed based on their clinical picture and the suspected position of the object in the respiratory tract. A foreign body in the trachea can cause complete airway obstruction whereas this is less likely if it lodges more distally.

A chest X-ray may be useful to detect the position of the foreign body if emphysema or air trapping (Figure 8) is present and will also exclude complications such as pneumonia or pneumothorax. Most inhaled foreign bodies will be organic in nature and subsequently difficult to outline; a normal appearing X-ray does not exclude the presence of a foreign body.

Management of these children will vary depending on the degree of airway obstruction and the location of the foreign body. Inhalational induction was traditionally preferred to maintain spontaneous respiration. Historically, oxygen in helium (heliox) was used to reduce turbulent airflow across the obstruction and improve oxygenation but its use is not supported by evidence. When anaesthetized, direct laryngoscopy may reveal the object and forceps should be immediately available for its removal. If unsuccessful and after the airway is topically anaesthetized, a rigid bronchoscope will be used with grasping forceps to remove the foreign body.

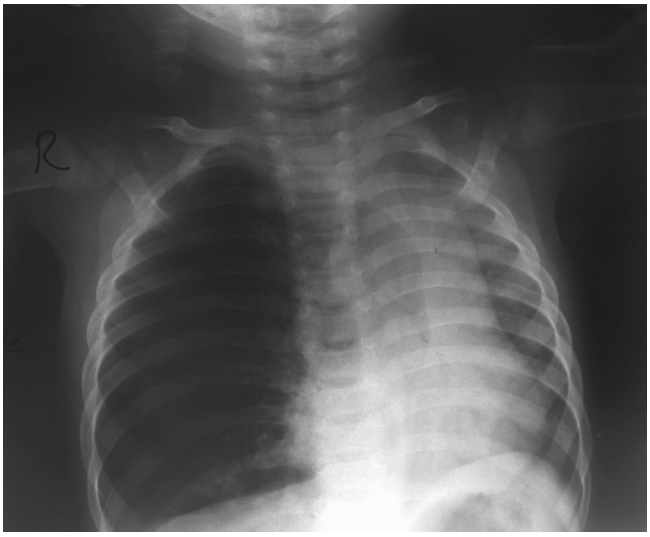

Figure 8 A 2-year old with right-sided air trapping as a consequence of the ball valve effect due to a foreign body.

Thermal injuries and burns: Laryngeal burns, caustic ingestion or smoke inhalation can cause life-threatening upper airway obstruction. Any child with a scald injury to their face should be suspected of having aspirated hot liquid and be assessed for laryngeal oedema.[9] Nebulized adrenaline may be beneficial in these cases as a method of temporarily reducing swelling prior to more conclusive management.

Anaesthesia

Management of patients with airway obstruction should follow a structured ABCDE approach as described by the Advanced Paediatric Life Support group.[7] Oxygen should be administered if tolerated by the child. Problems with airway patency will be suspected immediately if stridor or stertorous breathing is present. Stertor is a grunting or snoring sound heard during inspiration and can be caused by adenotonsillar disease.[5] Stridor can be inspiratory when the airway is obstructed at the level of the vocal cords or higher, expiratory with tracheal obstruction, such as in tracheomalacia, or biphasic such as in croup or subglottic stenosis. During quiet observation, the patient's respiratory pattern and rate should be noted along with their position and any obvious difficulty swallowing. Likewise, with a look, listen and feel approach, the assessment of breathing will provide clues to on-going abnormalities. Evidence of increased work of breathing will be demonstrated by suprasternal, intercostal and subcostal recession, tracheal tug and use of accessory muscles. Head bobbing and nasal flaring may be seen in neonates and infants. Asymmetrical breath sounds or the presence of wheeze can direct attention to lower airway pathology. The child's haemodynamic state, presence of fever and level of consciousness should be considered.

History may assist with diagnosis of the cause of airway obstruction. Sudden onset of symptoms may suggest anaphylaxis or an inhaled foreign body whereas a more insidious onset may suggest an infective process.[7] Any noted change in voice or hoarseness suggests pathology at a laryngeal or supraglottic level.[7] Signs of respiratory distress may not be evident at rest, however a history of abnormal breathing when agitated or during feeding might suggest pathology.

Many children will be managed supportively initially. Severe or worsening airway obstruction will require tracheal intubation. Appropriate anaesthetic assistance and senior personnel should be present. A clear step-wise plan should be developed for each patient, including when to proceed to a surgical airway. The Difficult Airway Society has produced guidance with the support of the Association of Paediatric Anaesthetists to assist with this decision making process. This is available at https://www.das.uk.com/guidelines/paediatric-difficult-airway-guidelines. The use of a supraglottic airway should be considered as an aid to resuscitation in infants greater than 2 kg and older than 34 weeks' gestation, especially in those where mask ventilation is difficult or intubation is unsuccessful.

Prior to induction, simple measures should be undertaken to improve positioning. A shoulder roll can be a useful aid to reduce excess neck flexion caused by the relatively large occiput in infants and can also help stabilize the head. A head ring may help or hinder the process. Equipment to relieve gastric distension should be immediately available as this could impair gas exchange if left unchecked. Prophylactic use of antimuscarinic drugs may be considered if secretions are predicted to be a problem.

The choice of induction technique will need to be considered on an individual basis depending on the clinical scenario and the skill base of the operator. There is no clear consensus whether an intravenous or inhalational induction is superior.[7] Inhalational induction means intravenous access need not be sited before the child is anaesthetized and avoids the distress this might cause. It also confers the benefit of maintaining airway tone and allowing depth of anaesthesia to be increased at a rate dependant on the degree of airway obstruction. With the slow onset of anaesthesia in cases of airway obstruction, there is less chance of the patient becoming apnoeic. There are clear benefits of maintaining spontaneous ventilation until the airway is secured although the required patient cooperation isn't always achievable in paediatric practice. Alternatively, in the circumstance of reduced airflow, inhalational induction can be particularly slow and most anaesthetists are more familiar with intravenous induction. Onset of anaesthesia can be controlled more predictably and the depth of anaesthesia will be more reliably guaranteed. Controlled doses of anaesthetic agent can ensure spontaneous respiration continues if there are concerns that the situation may develop into a can't intubate, can't ventilate scenario.[6] In some situations, high-flow nasal oxygen should be considered to prolong the time to desaturation during apnoea at induction. This may provide valuable time when ventilation or intubation is difficult. Ultimately the approach to anaesthetic induction should be based on what the anaesthetist is most comfortable with. In children where laryngeal or trachea patency is questioned, a smaller tracheal tube size may be required. Immediately available senior ENT support may be essential, when passing a rigid bronchoscope may be an option if intubation fails.[2] ◆

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