# Acute and chronic airway obstruction in children

Gary M Doherty


## Abstract

Airway obstruction is more common in children than in adults. This is because of subtle anatomical differences in the childhood airway and an increased propensity to infection. Effects of obstruction manifest more quickly in children because of a smaller airway diameter, reduced physiological reserve and easily fatigued respiratory muscles. The anaesthetist may encounter airway obstruction in children both outside and within the operating theatre. Problems can be either anticipated or unexpected. The anaesthetist must be able to recognize risk factors for airway obstruction such as a history of respiratory symptoms, including sleep-disordered breathing, and high-risk groups, such as ex-preterm infants. An understanding of the pathophysiology of airway obstruction can help in the recognition, diagnosis and appropriate management of airway obstruction. The pathophysiology of airway obstruction is intimately linked with the anatomy and mechanics of the upper airway and the tracheobronchial tree. The pathophysiology of airway obstruction is reviewed and this knowledge applied to problems occurring inside and outside the operating theatre, including both anticipated and unexpected problems.


**Keywords** Anaesthesia; child; lower airway obstruction; upper airway obstruction

## Learning objectives

After reading this article, you should be able to:

- explain the mechanics behind wheeze and stridor
- recognize the common conditions causing airway obstruction in children
- react appropriately to airway obstruction occurring in the operating theatre

## Mechanics of airway obstruction

Upper and lower airway obstruction (Figure 1) present differently because of the different pressures inside and outside the thoracic cavity during the breathing cycle. There is a natural anatomic division between the upper and lower airway at the level of the glottis (or vocal cords). Physiologically, however, the upper airways are those lying above the thoracic inlet (nose, nasopharynx, larynx and upper trachea) and the lower airways are those lying below the thoracic inlet (lower trachea and bronchi). The thorax is separated from the neck at the thoracic inlet formed by the first thoracic vertebra, the first pair of ribs and the manubrium sterni. The airways are termed extrathoracic if they lie above the thoracic inlet and intrathoracic if they lie below.

In inspiration, a negative pressure is generated within the thoracic cavity as the thorax expands by the downward excursion of the diaphragm and the upward and outward excursion of the ribs. Air is drawn into the airways and lungs until the pressure in the lungs rises to atmospheric pressure and airflow (and, therefore, inspiration) ceases. In inspiration the negative pressure generated in the thoracic cavity is transmitted throughout the airways. The structures surrounding the extrathoracic airways in the neck are at atmospheric pressure. This pressure gradient across the extrathoracic airways causes compression in inspiration and produces stridor when collapsible or partially obstructed extrathoracic airways are present (as seen in laryngomalacia and croup).

During expiration, the elastic recoil of the lungs and chest wall generate a positive pressure on the lungs, which drives air from the

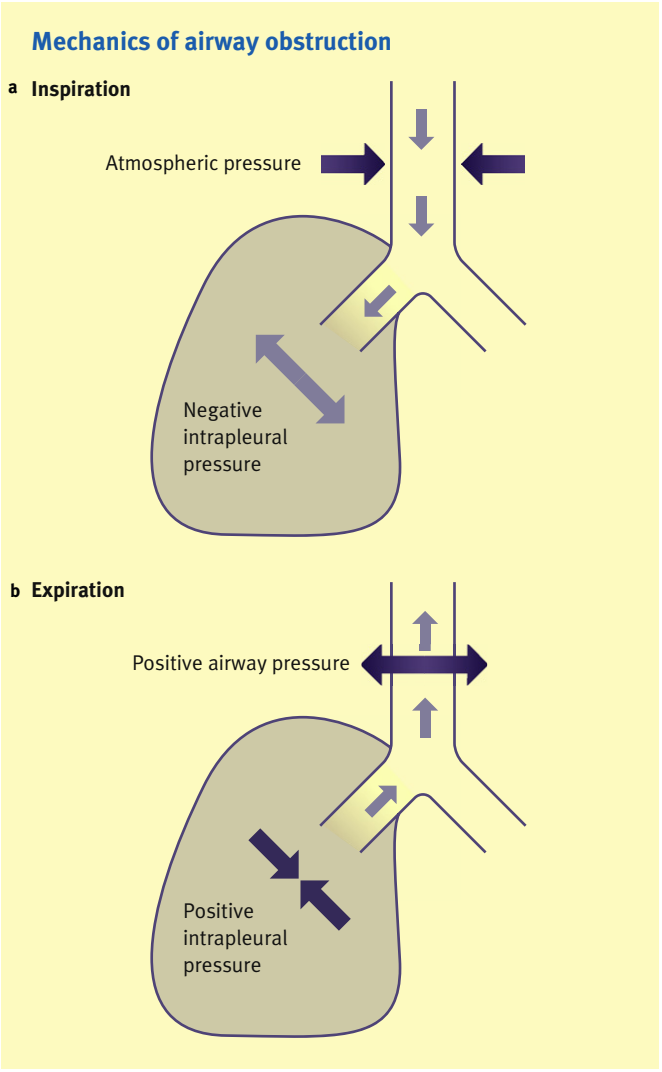


**Figure 1** Diagram demonstrating the compressive effect of the pressure gradient across extrathoracic airways during inspiration (**a**) and across intrathoracic airways during expiration (**b**).

***Gary M Doherty*** *MB BSc PhD MRCPCH is a Consultant in Paediatric Respiratory Medicine at the Royal Belfast Hospital for Sick Children, Belfast, UK.* *Conflicts of interest: none declared.*

lungs through the airways. The pressure within the airways is also influenced by the speed of the air flowing through the airways. Bernoulli's principle states that the pressure exerted by the gas is inversely related to the velocity of the gas flow. The total cross-sectional area of airways is reduced as we move from the many small distal airways to the fewer large central airways. The air therefore flows faster in the larger bronchi. As the speed of airflow increases, the pressure falls. In expiration, the positive intrathoracic driving pressure can cause compression and consequently wheeze when collapsible or partially obstructed intrathoracic airways are present (as seen in bronchomalacia and asthma). The distending pressure in the airways drops to less than the positive intrathoracic pressure because of the effect of the fast-flowing air.

In fixed airway obstruction such as subglottic stenosis, the cause and effect are clear: there is a narrow airway with subsequent limitation of airflow. However, even this process can be variable. The limitation may become apparent only if increased demands are put on the respiratory system such as during exercise, or the narrowing may become critical only if there is additional swelling such as during a respiratory tract infection. Other causes of airway obstruction can be more dynamic. In laryngomalacia or (upper tracheomalacia), the calibre of the airway at rest or during expiration is normal. It is only during inspiration that the soft airway collapses under a pressure gradient.

## Airway obstruction occurring outside the operating theatre

The anaesthetist is often the first person called in an airway emergency. Emergencies tend to differ by age:

- Neonatal — congenital airway abnormalities.
- Young child — airway obstruction is often upper airway and can be caused by infections or by foreign bodies.
- Older children — airways obstruction often occurs in the lower airways such as that seen in asthma or cystic fibrosis. 'Difficult airways' are common in trauma, either because of injury to the face, neck or chest, or because of the need for cervical immobilization.

Securing an effective airway in the emergency management of airway obstruction requires skill and a structured approach. Both an experienced anaesthetist and an ear, nose and throat (ENT) surgeon may be needed. Maintaining spontaneous ventilation is vital in airway obstruction and is a useful practice in most children with respiratory compromise. Even laboured respiratory efforts by the child are usually more effective than ventilation with bag and mask following abolition of the child's breathing with muscle relaxants. Gradual induction of anaesthesia with oxygen and an inhalational agent is preferable, with the child allowed to adopt the most comfortable position, even sitting on a parent's lap. Intravenous access is usually attempted only after gaseous induction as pain or fear may precipitate total airway obstruction in the child. Muscle relaxants should be used only if necessary to aid intubation and only once the anaesthetist has confirmed that bag-and-mask ventilation is consistently possible.[1]

## Recognizing airway obstruction in the spontaneously breathing child

In the spontaneously breathing child, extrathoracic airway obstruction produces inspiratory or biphasic stridor, which is usually monophonic. Increased effort is seen with accessory muscle use. Tracheal tug, sternal, subcostal and intercostal recession are exaggerated because of the high negative intrapleural pressures generated by the child in an attempt to overcome the obstruction. This is obvious in younger children with compliant, cartilaginous thoracic walls when even moderate upper airway obstruction can produce quite dramatic indrawing. The child will often spontaneously adopt a position which maximizes the airway diameter and the mechanical efficiency of breathing, hence the 'tripod position'. The practitioner changes this position at their peril. Changing the child's position can precipitate complete airway obstruction and collapse and the anaesthetist should avoid this until they are prepared to take control of a difficult airway and the child's breathing if necessary.

In intrathoracic (lower) airway obstruction, wheeze is usually audible. Wheeze generated from multiple points of narrowing (as in bronchospasm) is a noise characterized by the layering of many slightly different high-frequency notes producing a musical or polyphonic tone. Wheeze generated from a single fixed obstruction is similarly fixed producing a monophonic wheeze. There is usually hyperinflation of the chest and this may be asymmetrical if one lung is affected more than the other (such as with a foreign body). The signs of increased work of breathing are similar to upper airway obstruction (accessory muscle use, recession and tracheal tug). However, hyperinflation of the chest often makes these less apparent. There may be prolongation of the expiratory phase, and active expiration with use of the abdominal muscles.

Inspiratory wheeze is less prevalent in bronchospasm because a combination of hyperinflation and a negative intrathoracic pressure is believed to splint open the airways. However, in severe obstruction of either the extrathoracic or intrathoracic airways, biphasic stridor or wheeze can be heard and this is an ominous sign. When airflow diminishes because of almost complete obstruction, stridor or wheeze can disappear: this is usually a sign of impending collapse.

## Typical pathology encountered outside the operating theatre

Some of the causes of acute and chronic airway obstruction are outlined in Table 1.

### Nose and nasopharynx

Neonatal airway emergencies are usually due to congenital lesions particularly choanal atresia, Pierre Robin sequence or cystic hygroma. Bilateral choanal atresia usually presents within hours of birth with breathing difficulties, apnoea or cyanotic episodes often associated with feeding. Unilateral choanal atresia can present later in childhood with persistent unilateral rhinorrhoea.

### Larynx and upper trachea

Congenital thoracic malformations may produce compression of the lower thoracic airways but this is unusual. Occasionally these will have been anticipated because of antenatal scans. Management plans should be in place for problems anticipated antenatally and the delivery is often by elective caesarean section with the appropriate personnel in attendance. EXIT procedures are occasionally used (EX utero InTrapartum procedures) where the placental circulation is maintained after the baby is delivered until an airway is secured.

**Some examples of conditions causing airways obstruction**

| Site | | | Acute | Chronic |
|---|---|---|---|---|
| Upper | Nose | | | Choanal atresia |
| | | | | Congenital stenosis of piriform aperture |
| | Nasopharynx | | Peritonsillar abscess | Adenotonsillar hypertrophy |
| | | | Diphtheria | |
| | | | Retropharyngeal abscess | |
| | Larynx | Supraglottic | Epiglottitis | Laryngomalacia |
| | | | | Laryngeal web |
| | | Glottic | Laryngospasm | Vocal cord palsy (congenital or acquired) |
| | | | Angioneurotic oedema | Infantile haemangioma[a] |
| | | | | Laryngeal papillomatosis |
| | | Subglottic | Croup | Subglottic stenosis (congenital or acquired) |
| | | | Subglottic haemangioma | |
| Lower | Trachea | | Bacterial tracheitis | Tracheomalacia |
| | | | Inhalational burns | Vascular compression |
| | | | | Tracheal stenosis |
| | | | | Mediastinal mass |
| | Bronchi | | Asthma | Bronchomalacia |
| | | | Foreign body[a] | Bronchiectasis (including cystic fibrosis) |
| | | | Bronchiolitis | Obliterative bronchiolitis |

[a] Masses and foreign bodies can occur or lodge anywhere in the respiratory tract.

**Table 1**

Problems at the level of the glottis are less frequent. Vocal cord palsy can be congenital or acquired. Bilateral congenital vocal cord palsy can produce severe obstruction requiring immediate intervention. In infants, haemangiomas can produce stridor and obstruction, as can any cystic lesion. Cystic hygromas can massively infiltrate the soft tissues of the head, neck and chest and produce a very difficult-to-manage airway. Cystic hygromas, like haemangiomas, may progress over the first few months of life.

Laryngomalacia is the commonest laryngeal cause of upper airway obstruction in young children and is characterized by inspiratory stridor of variable degree developing usually days to weeks after birth and improving as the child grows.[2] In laryngomalacia the negative airway pressure normally generated during inspiration drags the epiglottis or aryepiglottic folds into the laryngeal inlet obstructing the airway. The problem is usually less marked in quiet respiration than when the child is agitated or distressed. It may be eased if positive airway pressure is applied. Obstruction at this level is overcome following intubation.

Clinicians working with children are familiar with the triad of common infectious upper airway obstruction syndromes: epiglottitis, croup (acute laryngotracheobronchitis) and bacterial tracheitis.[3] These conditions can be indistinguishable clinically, although classically there are certain features which make one or other condition more likely. The child with epiglottitis is described as fearful, with drooling, a quiet stridor, a fixed position (often the 'tripod' position) and a toxic appearance in keeping with a serious bacterial infection. The child with croup often seems systemically well, although they may have a high fever, and will have a stereotypical barking or seal-like cough.[4] Bacterial tracheitis can be difficult to distinguish from croup. Like the child with epiglottitis, the child with bacterial tracheitis may appear toxic, but the cough is usually less harsh than in croup as the larynx may be less involved and there are often more tracheal secretions.

### Bronchospasm

Asthma remains the commonest chronic illness in childhood and the commonest cause of lower airway obstruction. Children can have hyper-reactive airways without satisfying the other criteria of classic atopic asthma. Pre-school children can have severe and recurrent wheeze in response to viral infections without symptoms in the intervening period (known as viral-associated wheeze or wheezy bronchitis). Comprehensive guidelines exist in most countries for the management of acute wheeze. Intubation is avoided if at all possible because subsequent ventilation of the acute asthmatic is notoriously difficult.

### Tracheomalacia and bronchomalacia

Tracheomalacia or bronchomalacia implies weakness of the large cartilaginous airways with subsequent collapse under pressure. The collapse of the bronchi occurs in expiration when the thoracic pressure is slightly higher than the pressure in the fast-flowing air in the major bronchi. The process is exaggerated when the gradient across the airway is increased, for example in active expiration when the use of muscles in expiration generates a high positive intrathoracic pressure. Children with tracheomalacia or bronchomalacia are likely to have chronic symptoms but may present acutely during respiratory infections. Positive-pressure support, either non-invasively or invasively, is usually helpful. Airway emergencies are unusual but 'dying spells' may occur in severe tracheomalacia or in rarer situations such as

tracheal stenosis (which is a fixed narrowing as opposed to the dynamic collapse seen in tracheomalacia). The management of severe tracheobronchomalacia or stenosis is usually restricted to specialized centres. Outcomes are traditionally poor but have improved significantly with specialization of teams and the introduction of 'slide tracheoplasty'. The first case of tracheal transplant in a child has been reported with a partially denuded donor trachea seeded with stem cells from the child.

### Foreign bodies

Foreign bodies tend to lodge in the narrowest parts: nose, larynx or bronchi. A small object such as a bead in the nose can remain undetected, producing unilateral rhinorrhoea only. Impaction in the larynx produces dramatic symptoms with powerful reflexes to protect the airway. Failure to clear the object can result in complete and often fatal airway obstruction. Airway obstruction secondary to foreign body aspiration can occur at any age, but is most common and most likely to be fatal in the pre-school child. Treatment of the choking child forms part of the basic life support algorithms put forward by the Advanced Life Support Group.

## Airway obstruction occurring in the operating theatre

Airway obstruction in the operating theatre environment can often be anticipated. Problems can occur:

- at induction of anaesthesia and during intubation
- during anaesthesia
- after extubation.

## Recognizing airway obstruction in the ventilated or anaesthetized child

The ventilatory responses which generate the signs of airway obstruction in the spontaneously breathing child are blunted in the anaesthetized child and absent in the paralysed child. The anaesthetist needs to remain alert to changes in tidal volumes or airway pressures as well as hypercarbia and hypoxaemia. Monitoring of tidal volumes, end-tidal carbon dioxide and oxygen saturations are vital. The only clinical signs may be reduced chest expansion and diminished breath sounds. The 'feel' during hand ventilation will change as compliance changes.

Airway obstruction may be detected earlier when tidal volumes and airway pressures are closely monitored. These parameters can also be used to monitor the response to treatment of children ventilated for airway obstruction, for example the degree of leak around the endotracheal tube in a child ventilated for croup. Flow–volume curves in ventilated children with lower airways obstruction (such as asthma) will show incomplete expiration, with flow continuing at the end of expiration. This is indicative of potential dynamic hyperinflation or 'breath stacking'. Intrinsic positive end-expiratory pressure (PEEP) will be increased. The characteristic 'scooped' slope on the expiratory limb of the flow–volume loop may be apparent.

## Typical problems encountered in the operating theatre

### Induction and intubation

Many causes of chronic airway obstruction will be treated surgically (Table 2). Problems with securing an effective airway may be predicted because of reduced visualization (large tonsils or palatal shape), reduced movement (at jaw or cervical spine), narrowing of the airways (adenoidal hypertrophy, choanal stenosis, subglottic stenosis or congenital tracheal stenosis) or abnormal anatomy.

Subglottic stenosis is often an acquired condition secondary to prolonged or repeated intubation, and is most frequently seen in ex-preterm infants who have been intubated for infantile respiratory distress syndrome. However, it can also occur congenitally, or with a relatively short history of intubation. Clues include difficulty with extubation previously, or a history of stridor or recurrent croup.

Congenital stenosis is rare and may first be detected because of failure to pass an endotracheal tube. Children coming to theatre with other congenital anomalies, particularly of the heart or lungs, may have associated airway anomalies.

Adenoidal and tonsillar hypertrophy are the commonest cause of chronic upper airway obstruction in pre-school children, manifest usually by disordered breathing in sleep or breathing difficulties during upper respiratory tract infections. A significant history of sleep-disordered breathing in a child undergoing adenotonsillectomy should alert the anaesthetist to a potentially difficult airway. All operating theatres should have an algorithm for the management of the difficult airway, even if encountered unexpectedly. However, most anaesthetists will have anticipated problems.

### During anaesthesia

After intubation, the upper airway is bypassed and airway obstruction will only usually occur in the lower airways, provided the endotracheal tube is not dislodged. Upper airway

### Common management strategies in chronic airway obstruction

| | |
|---|---|
| Choanal atresia | Surgical resection and stenting |
| Adenotonsillar hypertrophy | Adenotonsillectomy |
| Pierre Robin sequence | Nasopharyngeal airway, tracheostomy, mandibular distraction |
| Laryngomalacia | Laryngoplasty, aryepiglottoplasty |
| Tracheomalacia | Aortopexy, tracheostomy and positive-pressure support, stenting *in extremis* |
| Tracheal stenosis | Resection of stenosed segment, slide tracheoplasty, patch tracheoplasty, tracheal transplant |
| Bronchomalacia | Tracheostomy and positive-pressure support, stenting *in extremis* |
| Asthma | Anti-inflammatory therapy (inhaled corticosteroids, leukotriene receptor antagonists) |
| CF and Non-CF bronchiectasis | Antibiotics and airway clearance (mucolytics and physiotherapy) |

**Table 2**

obstruction (particularly laryngospasm) can still occur with alternative airway adjuncts such as a laryngeal mask or nasopharyngeal airway. Minimizing the risk of anaesthesia in children with asthma requires a systematic approach.[1,5] Mild asthma is unlikely to be a major problem, although it is probably sensible to pretreat with inhaled bronchodilators if these are commonly used by the child. In moderate-to-severe asthma, it is useful to emphasize the importance of complying with anti-inflammatory treatment such as inhaled corticosteroids in the run-up to surgery. Adrenal suppression is increasingly recognized in children with asthma who are frequently on prolonged courses of oral steroids or high-dose inhaled corticosteroids. Other children may have an idiosyncratic reaction to anaesthetic gases, or an allergic reaction to a particular drug.

Laryngospasm and bronchospasm (either in isolation or as part of anaphylaxis) may present with airway obstruction. A similar structured approach is required in these situations with slightly different treatments (Table 3). Protocols, algorithms (such as those developed by the Australian Patient Safety Foundation — http://www.apsf.com.au/crisis_management/Crisis_Management_Start.htm) and simulation training are widely used to ensure optimal performance during an anaesthetic emergency. The Managing Emergencies in Paediatric Anaesthesia group in the UK (mepa.stipa.org.uk) has also developed an algorithm for the management of complete and partial laryngospasm based on recognition of the problem; use of manoeuvres to convert complete obstruction to partial obstruction (pressure in the laryngeal notch and mandibular pull); increasing the depth of anaesthesia; and finally paralysis with suxamethonium and atropine.[6]

Lower airways obstruction is characteristic of cystic fibrosis and other conditions causing chronic suppurative airways disease such as primary ciliary dyskinesia. Obstruction is caused by tenacious airways secretions, bronchiectatic airways and occasionally bronchospasm. Maximizing medical therapy and physiotherapy prior to anaesthesia minimizes risks. Depending on the severity of lung disease, the nature of the procedure and the duration of the anaesthetic, the child may be admitted for intravenous antibiotics for several days prior to the procedure. This can ameliorate the detrimental effects of atelectasis associated with anaesthesia, and also the effects of immobility and reduced compliance with regular physiotherapy which can be seen after some surgical procedures. During anaesthesia, assisted ventilation helps reduce atelectasis. Tracheal intubation permits

**Management of unexpected acute airway obstruction in the operating theatre**

| | Laryngospasm | Bronchospasm | Anaphylaxis |
|---|---|---|---|
| Oxygen | Increase to 100% | Increase to 100% | Increase to 100% |
| Anaesthesia | Deepen anaesthesia<br>May need paralysis and intubation | Deepen anaesthesia | Stop all drugs (potentially allergenic) |
| Surgery | Cease stimulation | Cease stimulation | Inform surgeon |
| β-agonists | Consider nebulized epinephrine if laryngeal oedema suspected | Salbutamol:<br>2.5—5.0 mg nebulized<br>OR<br>0.01 mg/kg in 1 ml via ETT<br>OR<br>i.v. bolus 15 μg/kg over 10 min and infusion (1—5 μg/kg/min).<br>Consider reducing bolus to 5 μg/kg in children less than 2 years. | Epinephrine 10 μg/kg (1:1000 solution i.m.) |
| Steroids | Consider dexamethasone (0.15 mg/kg) if laryngeal oedema suspected | Hydrocortisone 2—4 mg/kg i.v. | Hydrocortisone 2—4 mg/kg i.v. |
| Antihistamines | Not indicated | Not indicated | Chlorphenamine i.v.:<br>250 μg/kg (less than 6 months)<br>2.5 mg (6 months to 6 years)<br>5 mg (6 to 12 years)<br>10 mg (more than 12 years).<br>Consider ranitidine i.v.<br>(1 mg/kg, max. 50 mg) |
| Other | Atropine 0.01 mg/kg | Consider i.v. magnesium sulphate (40 mg/kg; max. 2 g) over 20 min in children over 2 years. | Volume expansion<br>Stop all other potentially allergenic medicines and infusions |

ETT, endotracheal tube; i.m., intramuscularly; i.v., intravenous.

**Table 3**

suctioning of secretions and better control of the airway. Atropine can dry secretions excessively and ketamine exacerbate secretions and coughing.[1] Antibiotics are often continued either intravenously or orally postoperatively.

Removal of an aspirated foreign body is usually undertaken by rigid open-tube bronchoscopy. A ventilating bronchoscope with grasping forceps is used. Gaseous or careful intravenous induction can be used to maintain spontaneous ventilation. Atropine can help diminish airway secretions and reflexes. Effective local anaesthesia of the trachea and larynx is needed. Coughing may dislodge the foreign body. Attempts to retrieve the foreign object may push it further distally producing a ball-valve effect (especially with positive-pressure ventilation), or move it proximally where, if dropped, it may precipitate complete airway obstruction or provoke strong vagal reflexes. Anaesthesia with maintenance of spontaneous ventilation is common practice to prevent hyperinflation and barotrauma from a potential ball-valve effect of the foreign body. Once the object is grasped, the bronchoscope, forceps and object are withdrawn and ventilation taken over with bag and mask. The operator and anaesthetist must be prepared to advance the bronchoscope again rapidly if the object is dropped. It may be necessary to push the object distally in this instance to clear the larynx or trachea and allow ventilation.[1]

Pulmonary hypertension can develop in any child with chronic airway obstruction and is an additional risk factor for complications including death during anaesthesia.[7]

### Following anaesthesia

Laryngeal oedema following anaesthesia is common and usually mild. The risk and severity of laryngeal oedema is increased by recent or current upper respiratory tract infections, use of an endotracheal tube rather than a laryngeal mask, and any procedure such as bronchoscopy which involves instrumentation of the larynx. Problems can be prevented by deferring elective surgery in any child with a current upper respiratory tract infection, particularly if the patient or procedure is considered to be high risk. If post-extubation stridor does not quickly settle, it can be helped by nebulized epinephrine, nebulized budesonide or systemic steroids (the same treatments used for infective laryngotracheobronchitis).

Tracheomalacia is often managed relatively easily with positive-pressure ventilation during anaesthesia. However, problems can quickly become manifest in recovery when normal negative pressure breathing resumes, particularly if prolonged anaesthesia has resulted in atelectasis with reduced lung compliance, or prolonged intubation has caused airway oedema.

Post-obstructive pulmonary oedema (POPE) can occur after an episode of acute airway obstruction. It is most commonly encountered following laryngospasm, either during intubation or other instrumentation of the airway or shortly after extubation. The cause of acute POPE is possibly related to changes in hydrostatic forces secondary to the high negative intrathoracic pressures generated in an attempt to breathe against an obstructed airway. POPE can also develop following the relief of chronic airway obstruction (e.g. following tonsillectomy). In either case, the treatment is supportive, usually requiring increased oxygen and occasionally pressure support, either invasively or non-invasively. The oedema usually resolves within hours, but it can progress to acute respiratory distress syndrome and deaths have been reported. In theory, specific treatments such as diuretics, β-agonists or steroids can be used but their value is unclear. ◆

## REFERENCES


1 Bingham R, Lloyd Thomas A, Sury M. Hatch & Sumner's textbook of paediatric anaesthesia. 3rd edn. London: Hodder Arnold, 2007.

2 Daniel SJ. The upper airway: congenital malformations. *Paediatr Respir Rev* 2006; **7S:** S260–3.

3 Hammer J. Acquired upper airway obstruction. *Paediatr Respir Rev* 2004; **5:** 25–33.

4 Bjornson CL, Johnson DW. Croup. *Lancet* 2008; **371:** 329–39.

5 Doherty GM, Chisakuta A, Crean P, Shields MD. Anesthesia and the child with asthma. *Paediatr Anaesth* 2005; **15:** 446–54.

6 Hampson-Evans D, Morgan P, Farrar M. Paediatric laryngospasm. *Paediatr Anaesth* 2008; **18:** 303–7.

7 Carmosino MJ, Friesen RH, Doran A, Ivy DD. Perioperative complications in children with pulmonary hypertension undergoing noncardiac surgery or cardiac catheterization. *Anesth Analg* 2007; **104:** 521–7.


## FURTHER READING


Pasterkamp H. The history and physical examination. In: Chernick V, Boat TF, Wilmott RW, Bush A, eds. Kendig's disorders of the respiratory tract in children. 7th edn. Philadelphia: Saunders Elsevier, 2006.

British Thoracic Society. BTS/SIGN asthma guideline 2011, http://www.brit-thoracic.org.uk/guidelines/asthma-guidelines.aspx (accessed 30 Nov 2011).