Acute stridor in children

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Stridor is a harsh, vibratory sound produced when the airway becomes partially obstructed, resulting in turbulent airflow in the respiratory passages. It is symptomatic of underlying pathology and may herald life-threatening airway obstruction. This article reviews the assessment, common causes, and treatment of a child with a previously normal airway presenting with acute stridor.

Anatomy and physiology

Several anatomical and physiological features of the respiratory system in infants (age <1 yr) and young children render them susceptible to airway obstruction. The upper and lower airways are small, prone to occlusion by secretions, and susceptible to oedema and swelling. As resistance to laminar airflow increases in inverse proportion to the fourth power of the radius (Poiseuille's law), a small decrease in the radius of the airway results in a marked increase in resistance to airflow and the work of breathing.

The support components of the airway are less developed and more compliant than in the adult. The ribs are cartilaginous and perpendicular relative to the vertebral column, reducing the effect of the 'bucket handle' movement of the rib cage. In addition, the intercostal muscles and accessory muscles of ventilation are immature. As a result, children are more reliant on the diaphragm for inspiration. Increased respiratory effort causes subcostal and sternal recession, and the mechanical efficiency of the chest wall is reduced. Higher metabolic rate and increased oxygen demand mean children with airway compromise can deteriorate very quickly. Also, with a smaller functional residual capacity and fewer fatigue-resistant fibres in the diaphragm, there is little respiratory reserve at times of stress.[1]

Assessment

A stridulous, distressed child should be disturbed as little as possible as crying and agitation increase respiratory effort and might precipitate complete airway obstruction. Therefore, the child should be allowed to adopt the posture in which they are most comfortable and the parents should be present. Examination of the throat and i.v. cannulation should not be attempted.

The phase of stridor may indicate the level at which the airway is compromised (Table 1).[2] It is important to appreciate that the volume of stridor does not correlate with the degree of obstruction. For example, a progressive decrease in volume may signify a decreasing conscious level.

Examination may be limited to inspection for signs of increased work of breathing (Table 2), although a pulse oximeter probe is usually well tolerated and gentle examination of the chest may be possible. Laboratory tests are not helpful in the early assessment of a child with acute stridor. Radiology may be useful in less acute cases, but it should only be performed at the bedside with minimal disturbance to the child. Children with symptoms and signs of severe airway obstruction require urgent examination of the airway under anaesthesia to determine the cause and secure the airway. Collaboration with an experienced ENT surgeon is essential for optimal management of these cases.

Viral croup

Viral croup (laryngotracheobronchitis) is responsible for more than 80% of cases of acute stridor in children. It is usually due to infection with parainfluenza viruses, but it may also be caused by influenza viruses type A or B, respiratory syncytial virus or rhinovirus. It commonly affects children age 6 months to 3 yr with a peak incidence at 2 yr. Only 2% of cases are admitted to hospital each year, of which only 0.5–1.5% will require intubation.[2,3] Infection results in subglottic and tracheal swelling causing stridor and other signs of respiratory obstruction. In the more severe cases, it also affects the small airways, resulting in bronchial constriction, oedema, and atelectasis.

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Table 1 Correlation between phase of stridor and probable level of airway obstruction

Stridor	Level of obstruction
Inspiratory	Above the cords (extrathoracic); e.g. croup, epiglottitis
Expiratory	Below the cords (intrathoracic); e.g. foreign body
Biphasic	At or below the cords (intra or extrathoracic); e.g. foreign body, bacterial tracheitis

Croup develops 2–3 days into an upper respiratory tract infection and is characterized by a barking cough (which gives the condition its name), low-grade fever, inspiratory stridor, and increased respiratory effort progressing to fatigue, hypoxia, and hypercarbia. A scoring system for croup is useful in assessing severity. Ongoing, serial bedside assessment of a child with croup is key to determining the success of medical treatment or need for further interventions, such as admission to a high dependency unit or tracheal intubation (Table 3).[4]

Treatment includes humidification of respiratory gases, oxygen, steroids, and nebulized epinephrine.[5] Nebulized epinephrine is believed to reduce oedema by alpha-adrenergic-mediated vasoconstriction of the inflamed subglottic mucosa. In North America, racemic epinephrine containing equal amounts of the L- and D-isomers (L-isomer 30 times more active) is widely used. In the UK, only L-epinephrine is available. The usual dose of racemic epinephrine is 0.5 ml of a 2.25% solution diluted to 4 ml with saline or water, repeated hourly, as necessary. This dose contains approximately 5 mg of active L-epinephrine, equivalent to 5 ml of (non-racemic) epinephrine 1/1000.[6] Accordingly, in many UK hospitals, moderate to severe croup is treated with nebulized epinephrine 1/1000, 0.5 ml kg^{-1} to a maximum of 5 ml, repeated if necessary after 30–60 min. The improvement in the croup score after this can be dramatic, but the effects are short-lived; repeated administration is required. Although cardiovascular side-effects are rare, ECG monitoring is recommended.

Table 2 Signs of increased work of breathing

Ventilatory frequency	
Infant	>50 bpm
Child	>30 bpm
Effort	
Infant	Head bobbing; nasal flaring
Child	'See-saw' chest and abdomen; recession: subcostal, sternal, intercostal, tracheal tug; nasal flaring
Posture	
Infant	Arching backwards
Child	Tripod position
Noise	Infants grunt to generate auto-CPAP; wheezing can occur with an inhaled foreign body; stridor
Ineffective breathing	Hypoxaemia and hypercarbia produce tachycardia, sweating, restlessness and confusion, agitation and anxiety, pallor, or mottling
Impending respiratory arrest	Decreased conscious level, slowing ventilatory frequency, episodes of apnoea, silent chest despite vigorous respiratory effort, bradycardia

Table 3 Croup score.[4] Mild croup, 0–3; moderate croup, 4–6 (transfer to HDU); severe croup, 7–10 (patient requires tracheal intubation)

Score	0	1	2
Breath sounds	Normal	Harsh, wheeze	Delayed
Stridor	None	Inspiratory	Inspiratory and expiratory
Cough	None	Hoarse cry	Bark
Recession/flaring	None	Flaring, suprasternal recession	Flaring, suprasternal and intercostal recession
Cyanosis	None	In air	In oxygen 40%

Steroids reduce symptoms and improve croup scores 6–24 h after initial dosing, thereby reducing the need for intubation or shortening the time to extubation. Randomized controlled trials have shown that parenteral dexamethasone 0.6 mg kg^{-1}, oral dexamethasone 0.6 mg kg^{-1}, and nebulized budesonide 2 mg have similar efficacy in treating croup. As oral administration of dexamethasone is quicker and causes less distress than nebulization or parenteral administration, it may be preferable for children admitted to hospital with moderate–severe croup.[5] In the PICU setting, dexamethasone 0.1 mg kg^{-1} 6 h^{-1} is recommended.[3]

Heliox is a 70:30% helium–oxygen mixture that is less dense than air or 100% oxygen. The lower gas density facilitates laminar (rather than turbulent) flow in narrowed airways allowing more efficient ventilation in a child with a compromised airway. In a randomized controlled trial, heliox administration resulted in similar improvements in croup score as administration of nebulized epinephrine.[5] However, limiting the percentage of oxygen that can be administered to 30% could be a disadvantage in children with marked bronchial involvement.

Severe respiratory obstruction, as indicated by a persistently high croup score, despite ongoing medical treatment (e.g. score $\geq$ 7, Table 3), is an indication for tracheal intubation. Once preparations for intubation have been made, the patient should be escorted to theatre by an anaesthetists or other person capable of managing the airway. Oxygen should be given by mask if required. Historically, halothane was the agent of choice for induction and maintenance of anaesthesia in upper airway obstruction; however, sevoflurane is safe and effective in this situation and it is appropriate that anaesthetists use the agent with which they are most familiar.[7]

The child should be anaesthetized using only the volatile agent in oxygen. It is not necessary to apply full monitoring before induction, but an oxygen saturation probe and end-tidal carbon dioxide monitoring are invaluable. Inhalation induction will take longer than usual in children with severe upper airway obstruction, since alveolar ventilation is restricted. Spontaneous ventilation should be maintained and applying continuous positive airway pressure (CPAP) via a T piece will help to splint the airway open. I.V. access should be established as soon as possible after the patient loses consciousness. Once the pupils are small and central, the level of anaesthesia is usually sufficient to allow laryngoscopy and tracheal intubation.[8]

Visualization of the larynx should not be a problem; however, a smaller tracheal tube than normal for the age is usually required because of subglottic oedema. Oral intubation is preferred initially as it allows rapid control of the airway. When the child is fully oxygenated and clinically stable, the oral tube is replaced with a nasal one of the same size. I.V. sedation (e.g. midazolam $2–8 \, \mu g \, kg^{-1} \, min^{-1}$ and morphine $20–40 \, \mu g \, kg^{-1} \, h^{-1}$) should be commenced before discontinuing anaesthesia and continued in the PICU along with CPAP, humidified air–oxygen, and i.v. fluid therapy. Arm splinting should be considered in the awake, sedated child to prevent self-extubation. In cases with extensive small airway involvement, intermittent positive pressure ventilation (IPPV) and frequent suctioning of the tracheal tube will be required. Antibiotics are reserved for patients with evidence of bacterial infection. Extubation may be attempted when a leak develops around the tracheal tube. Owing to the variable nature of the disease, this can take 2–10 days.[5]

Postintubation croup

Stridor and other signs of respiratory obstruction due to subglottic oedema may occasionally occur after routine intubation, usually with an unduly large tracheal tube. Clinical signs generally appear within 30 min of extubation and respond well to treatment with nebulized epinephrine and i.v. dexamethasone. Day case patients treated for postintubation croup should be observed in hospital for at least 2 h before discharge, as signs of respiratory obstruction may recur. Persistent or recurrent signs of airway obstruction are an indication for admission to hospital.

Epiglottitis

Epiglottitis is a life-threatening emergency caused by bacterial infection of the epiglottis, aryepiglottis, and arytenoids. In children, it is almost always due to infection with *Haemophilus influenzae* type b (Hib), although it can be caused by beta-haemolytic strepto-cocci, staphylococci, and pneumococci. Since the introduction of Hib vaccine in 1992, childhood epiglottis has become rare. During a recent resurgence in Hib infections, a corresponding increase in the number of children with epiglottitis was observed, although numbers were still much smaller than those reported before Hib vaccination.[9] In a recent retrospective study, 10% of children presenting with epiglottis were found to have Hib infection, despite having being vaccinated.[10] These findings highlight the importance of considering acute epiglottitis in the differential diagnosis of a child presenting with upper airway obstruction. This is particularly relevant since, in future, fewer doctors will be familiar with the symptoms and signs of the disease.

Epiglottitis is distinguished from croup by its fulminant onset and the toxic appearance of its victims. It occurs usually in children aged 2–6 yr, with a peak incidence at 3 yr. Clinically, there is an abrupt onset of high fever, sore throat, dysphagia, stridor, and drooling. Speech may be muffled or lost and there is an absence of spontaneous cough. Typically, the child looks ill and may prefer to sit leaning forwards, mouth open, and with tongue and jaw pro-truding in order to open the airway. In contrast to viral croup, the signs of respiratory obstruction in epiglottitis are unlikely to be relieved by administration of nebulized epinephrine. The disease can be rapidly progressive with the use of the accessory muscles, cyanosis, and alteration in conscious level indicating severe respiratory compromise.[3]

Many hospitals have a protocol for managing epiglottitis; the key is to secure the airway under inhalation anaesthesia in a manner similar to that described for severe viral croup. An experienced ENT surgeon should be standing by in case an immediate bronchoscopy or tracheostomy becomes necessary. Anaesthesia will normally be induced in the sitting position and i.v. cannulation is only attempted once the child is sufficiently obtunded. At laryngoscopy, the epiglottis will be red and swollen and the arytenoids and other supraglottic tissues inflamed, so that the glottic opening may be extremely difficult to visualize. In these circumstances, an abrupt manual chest compression may open the expiratory passage momentarily or produce a few bubbles of expired gas. A tracheal tube 0.5–1.0 mm (ID) size smaller than normal for the child's age should be chosen to assist passage through the partially obstructed glottic opening. Stiffening the tube with a stylet or by refrigeration may also be helpful. Successful intubation can usually be achieved by directing the tip of the oral tracheal tube to the spot where the glottic opening was visualized or the bubbles emerged. When the child is fully oxygenated, the oral tube is replaced with a nasal one. Laryngeal and blood cultures should be obtained during anaesthesia, after which antibiotic treatment should be commenced with an extended spectrum cephalosporin (e.g. ceftriaxone $80 \, mg \, kg^{-1} \, day^{-1}$ max $4 \, g \, day^{-1}$). The return of normal body temperature and an increased leak around the tracheal tube are indicators of recovery, which usually occurs about 48 h after the institution of antibiotic therapy. Direct examination under anaesthesia with propofol will confirm the presence of a normal epiglottis and the patient can be extubated when fully awake.[3]

Bacterial tracheitis

Bacterial tracheitis is uncommon, but after the introduction of the Hib vaccine, it may have overtaken epiglottitis as a cause of life-threatening respiratory obstruction in some paediatric centres.[11] The condition may occur at any age. The usual pathogens are *Staphylococcus aureus*, *Haemophilus influenzae*, streptococci, and Neisseria species.[3]

The signs and symptoms of bacterial tracheitis are frequently intermediate between those of viral croup and epiglottitis. After an antecedent upper respiratory tract infection of 2–3 days, the child becomes rapidly (8–10 h) and seriously ill with a high fever and respiratory distress. Coughing produces copious tracheal secretions and retrosternal pain. The voice may be hoarse and stridor prominent, but there is no dysphagia or drooling and the patient can usually lie flat. The larynx, trachea, and bronchi can become

acutely obstructed with purulent debris and inflammation with adherent pseudomembranes overlying friable tracheal mucosa.

Most patients with bacterial tracheitis will require tracheal intubation and the same protocol used for epiglottitis should be followed. At laryngoscopy, the epiglottis and supraglottic structures will appear normal, although slough and pus may be visible beyond the vocal cords. Bronchoscopy should be performed to remove the purulent debris from the trachea before intubation. After intubation, any remaining purulent material may be aspirated via suction catheters. Tracheal intubation may be required before anaesthesia and bronchoscopy if a child is in extremis. In these circumstances, initial intubation may result in complete respiratory obstruction due to blockage of the tracheal tube with inspissated pus and an immediate tube change will be required to clear the airway. Once the airway is secured, samples of blood and tracheal aspirate should be sent for culture and sensitivity tests. Ceftriaxone is a reasonable first line antibiotic therapy. Methicillin-resistant *Staphylococcus aureus* (MRSA) is an important consideration since treatment with vancomycin (15 mg kg^{-1} 8 h^{-1}, max 2 g day^{-1}) may be necessary. Further management in the PICU will include sedation, IPPV/CPAP, frequent suctioning of the tracheal tube to prevent occlusion, and fluid resuscitation. The average duration of tracheal intubation is 6–7 days. Antibiotic therapy and hospitalization may be required for up to 14 days.[3] Tracheal stenosis is a late complication.

Retropharyngeal or tonsillar abscess

A retropharyngeal abscess forms in the space between the posterior pharyngeal wall and the prevertebral fascia by lymphatic spread of infection from the sinuses, teeth, or middle ear. A tonsillar abscess forms in the potential space between the superior constrictor muscles and the tonsil. Staphylococci and streptococci are the usual causal agents. Subsequent oedema of the pharynx may result in inspiratory stridor. Children under the age of 6 are affected most commonly, presenting with neck swelling and pain, fever, drooling, dysphagia, trismus, and limited neck movement. The diagnosis is made clinically, but a lateral soft tissue neck film can confirm retropharyngeal thickening. Surgery is required in most cases and care must be taken during intubation to avoid abscess rupture and aspiration of pus into the airways.[7] The patient will usually be extubated at the end of the procedure. Subsequent treatment consists of i.v. hydration and broad-spectrum antibiotics.[3]

Foreign body aspiration

Aspiration of a foreign body, commonly food, has a peak incidence at 1–2 yr of age. A sudden onset of coughing, gagging, and choking are suggestive of foreign body aspiration and may necessitate basic life support manoeuvres for the choking child.[12] A foreign body at or above the vocal cords can cause complete obstruction of the upper airway, stridor, or a change or loss of voice, and the cough and dyspnoea with which foreign bodies lower down the airway present. Partial obstruction of a lower airway may cause air trapping behind the foreign body (ball and valve effect) with pneumothorax, surgical emphysema, and pneumomediastinum a possibility. In this situation, the usual inspiratory chest X-ray can appear normal; however, an expiratory film may reveal air trapping. Depending on the cause, location, and duration of airway blockage, collapse and consolidation of a lobe or an entire lung with bronchial breathing, inspiratory crackles, and expiratory wheeze may be found on examination. Without a clear history of choking, the symptoms can be difficult to differentiate from acute asthma. Indeed, a presentation of refractory asthma may be explained by a previously missed foreign body.

Rigid bronchoscopy with the patient breathing spontaneously under deep inhalation anaesthesia supplemented with up to 3 mg kg^{-1} of topical lidocaine will confirm the diagnosis and allow for removal of the foreign body. The severity of symptoms and degree of airway obstruction will dictate the urgency with which this is performed. Dexamethasone and nebulized epinephrine will help ameliorate airway oedema after a lengthy bronchoscopy.[3,8]

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Please see multiple choice questions 1–4