Paediatric airway infections

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Initial management of respiratory distress

Difficulty in breathing is the commonest medical reason for paediatric attendance at emergency departments.1 Infections of the paediatric airway may combine the urgent challenge of managing airway obstruction, hypoxaemia, and systemic sepsis. While basic life support principles provide a sound framework for immediate management of respiratory distress, therapies will vary with the specific pathology encountered. It should be remembered that non-infective causes of respiratory distress may present in a similar fashion. Table 1 lists some of the infective and non-infective causes of respiratory distress in children.

Key points

• Difficulty in breathing is the commonest medical reason for paediatric attendance at emergency departments.
• Such presentations may present the combined challenges of airway obstruction, hypoxaemia, and systemic sepsis.
• Tracheal intubation, supportive therapies, and, where indicated, broad-spectrum antibiotics are appropriate management strategies in all severe cases of airway infection and obstruction.
• Corticosteroids improve symptoms in croup and reduce hospital stay, whereas nebulized adrenaline causes only a transient improvement.
• The life-threatening condition epiglottitis is increasingly rare in parts of the world with established vaccination programmes.

History

A sudden onset of symptoms suggests the presence of a foreign body, anaphylaxis, or trauma as the cause of respiratory distress. Infection has a longer time course and is commonly associated with a fever. The history may also help identify the location of the pathology, e.g. a change in voice is suggestive of inflammation or obstruction at a laryngeal/supraglottic level.

Examination

Having excluded complete or life-threatening airway obstruction, examination of the child should involve assessment of:
• Respiratory rate, pattern, and use of accessory muscles/recessions
• Breath sounds
• The presence and character of cough
• Signs of infection: fever, lymphadenopathy, sputum production
• The child’s position, e.g. head and neck held in a ‘sniffing’ position
• The ability to swallow/any drooling

**Investigations**

Assuming that airway obstruction is not present and the diagnosis proves elusive, imaging anteroposterior and lateral plain film X-rays, computed tomography (CT), and microbiology (blood or throat culture/viral swabs) may be of use.

**Special considerations**

**Induction of anaesthesia**

Should tracheal intubation be indicated there is no consensus on whether inhalational or i.v. induction is preferable. Certainly, a traditional view that there is no place for i.v. induction in the context of airway obstruction is debatable. Clinical expertise, the child’s condition, the likelihood of a difficult airway, and the risk of further agitating the child should all be accounted for when choosing the course of action. Table 2 lists some of the advantages of each respective method for induction of anaesthesia.

**Table 1 Causes of respiratory distress**

| Site             | Infective cause | Non-infective cause |
|------------------|-----------------|---------------------|
| Upper airway     | Uvulitis        | Foreign body        |
|                  | Epiglottitis    | Vocal cord dysfunction |
|                  | Croup           | Anaphylaxis         |
|                  | Retropharyngeal| Tumours             |
|                  | peritonsillar abscess |                  |
| Lower airway     | Tracheitis      | Asthma              |
|                  | Bronchioloitis  | Anaphylaxis         |
| Pulmonary        | Pneumonia       | Hilar tumours       |
|                  | Empyema         | Vascular abnormalities |
|                  |                 | Pulmonary oedema    |
|                  |                 | Pulmonary infiltrations |
|                  |                 | (e.g. fibrosis, oncological, |
|                  |                 | and autoimmune conditions) |

**Table 2 Potential advantages of both inhalational and intravenous induction**

| Advantages of inhalational induction | Advantages of intravenous induction |
|-------------------------------------|------------------------------------|
| Slow onset of anaesthesia in a sick patient with less chance of causing apnoea | Rapid induction of anaesthesia allows airway reflexes to be overcome very quickly |
| Complete airway obstruction will prevent deepening of anaesthesia, allowing patient to wake up | Familiar technique even for non-paediatric specialists |
| Does not require intravenous access before starting which may reduce agitation of the child | Depth of anaesthesia (hence lack of awareness) not dependent on patency of airway |
| Volatile anaesthetic agents cause bronchodilatation and reduce resistance in the respiratory tree | 100% oxygen can be administered |

**Heliox**

Turbulent flow occurs when the Reynolds number rises above a specific value for that airway. The Reynolds number is proportional to the radius of the airway, the average velocity of gas flow in the airway, and the gas density, and inversely proportional to gas viscosity. Narrowed airways will predispose to turbulent rather than laminar flow, as the average velocity in these segments is greatly increased. Failure to maintain laminar flow in airways will result in an increase in the work of breathing. Helium has a similar viscosity to oxygen and nitrogen, but a much lower density. Heliox is a mixture of 79% helium and 21% oxygen and its low density provides favourable flow dynamics in narrowed airways by keeping the Reynolds number low and hence supporting laminar flow.

**Croup**

Croup is viral laryngotracheitis although the term is often used to describe a range of pathologies including laryngotracheobronchitis and ‘spasmodic croup’ characterized by nocturnal paroxysms of stridor without fever or inflammation. Croup occurs due to swelling and oedema of the epithelial layer of the upper airway (Fig. 1).

**Aetiology**

Parainfluenza virus (types 1–3) is most commonly responsible for croup epidemics. Respiratory syncytial virus, adenovirus, and human coronavirus may also be responsible for croup.

**Presentation**

Children with croup aged 6 months to 3 years classically present with fever, stridor, ‘barking’ cough, and hoarseness.
Symptoms often start as nasal congestion and coryza, potentially worsening over the course of 48 h until respiratory distress and airway obstruction supervene.\textsuperscript{5} The presence of stridor is key in determining severity and its presence, particularly at rest, indicating severe airway obstruction. At this stage of airway obstruction, suprasternal, substernal, and intercostal recessions may all be seen. Hypoxia may develop in this context, and intervention is mandated.

Management

The mainstay of management may be supportive therapies, with tracheal intubation where indicated. Dexamethasone (oral or intramuscular injection, 0.15 mg kg\textsuperscript{-1}) and nebulized budesonide (2 mg) are effective in the treatment of all severities of croup, with improvement in Westley Croup Scores (a scoring system commonly used for research purposes, which classifies the severity of croup based on the presence of recession, stridor, cyanosis, level of consciousness, and air entry) seen as soon as 6 h after administration. Steroids also result in reduced hospital stay and readmission rates. It is important to note that the optimum dose and route of administration has yet to be determined.\textsuperscript{6}

Nebulized adrenaline causes a significant clinical improvement 30 min after administration, although this improvement is transient, conferring no benefit compared with no treatment 2 h after administration.\textsuperscript{7}

Epiglottitis

The thin cartilage of the epiglottis is covered on its anterior (superior) surface by stratified squamous epithelium, which extends onto the upper third of the posterior (inferior) surface, where it is replaced by the respiratory epithelium of the larynx. Unlike the posterior surface, the epithelium of the anterior surface of the epiglottis is only loosely adherent; in epiglottitis, oedema will collect beneath the epithelium on the anterior surface, narrowing the airway and causing stridor.

Aetiology

Infectious epiglottitis is most commonly caused by Haemophilus influenzae type b (Hib) and accordingly, case numbers have fallen in countries such as the UK, which have established childhood vaccination programmes.\textsuperscript{8} Cases still occur either because of different pathogens including other Haemophilus subgroups or Streptococcus species\textsuperscript{9} or by Hib itself because of the phenomenon of vaccination failure.\textsuperscript{10}

Presentation

The child with infectious epiglottitis classically has a short history of respiratory distress, drooling, stridor, and may adopt a ‘sniffing’ posture in an attempt to maximize their airway patency. Cough is commonly absent. This is a clinical emergency, and the child is typically extremely unwell with a high fever and odynophagia.\textsuperscript{11}

Management

The management principles will be those basic principles of initial management of respiratory distress combined with prompt administration of antimicrobial drugs. The additional respiratory distress that may be provoked by attempts at venous cannulation may necessitate delaying antimicrobial therapy and microbiological culture until after definitive control of the airway has been undertaken. When antibiotics are commenced, intravenous co-amoxiclav 30 mg kg\textsuperscript{-1} would be an appropriate initial agent.

The narrowing of the airway because of the oedematous epiglottis may make conventional tracheal intubation difficult and an ear, nose, and throat (ENT) surgeon should ideally be present during definitive airway management should tracheostomy be required.\textsuperscript{12} Routine intubation of children with epiglottitis has been shown to result in significantly lower mortality rates compared with those managed medically without an artificial airway.\textsuperscript{13} Intubation is usually required for 2–3 days before extubation is permissible. Mandatory criteria for extubation include demonstrable cuff leak around the tracheal tube, apyrexia, and the ability to swallow.\textsuperscript{11}

Uvulitis

Aetiology

Acute infection of the uvula may occur at any age and is most commonly caused by Group A Streptococcus, although other infective organisms that have been implicated include Hib.\textsuperscript{14} A range of non-infective causes of uvula inflammation have also been described including trauma, thermal injury, vasculitis, and allergic reactions that may produce similar clinical features.

Presentation

Uvulitis will cause fever, pain, dysphagia, drooling, but respiratory distress is uncommon unless epiglottitis is also present.\textsuperscript{15} A swollen, erythematous uvula with or without exudate would be expected with an infective aetiology.

Management

Treatment depends on the cause. As with all airway infections threatening airway patency, the basic principles referred to throughout this article apply. In the case of infective uvulitis, antimicrobial therapy is the mainstay of treatment and should be directed by culture and sensitivities.

Tonsillitis, peritonsillar, and retropharyngeal abscess

Acute airway compromise purely as a result of tonsillitis may occur, but this is rare without associated extra-tonsillar spread of infection.\textsuperscript{12} Figure 2 shows a significant degree of airway narrowing as a result of tonsillitis.
Aetiology

Infections of the tonsil and surrounding structures are commonly caused by multiple organisms. Those predominantly responsible include Group A Streptococcus, Staphylococcus aureus, and respiratory anaerobes such as Fusobacteria and Prevotella species.16

The palatine tonsils lie between the palatoglossal and palatopharyngeal arches and are covered by a fibrous capsule that separates the tonsil from the superior and middle constrictor muscles. Owing to loose connective tissue in this area, a potential space exists bordered by the arches and the superior constrictor.

The retropharyngeal space extends from the skull base to the posterior mediastinum, and its boundaries are formed by layers of the deep cervical fascia. Lymph nodes draining the nasopharynx, adenoids, sinuses, and middle ear run in this space.

Ordinarily, peritonsillar infection follows tonsillitis and advances through local cellulitis to organized abscess formation.17 A much rarer disease process, retropharyngeal abscess is associated with upper respiratory tract infection, which may be the precursor for abscess formation in up to 50% of cases. Other causative mechanisms are described, however, including trauma (notably laryngoscopy or dental procedures).18

Presentation

Owing to the proximity of the structures involved and the similar disease processes, it may not be surprising that manner of presentation of these infections is also very similar. CT with contrast should illuminate the site of infection but may not be necessary and indeed may not be sensible in the child with respiratory distress.

Peritonsillar abscess

Severe unilateral sore throat, fever, and change in voice are often present in peritonsillar abscess, but trismus (caused by spasm of the medial pterygoid muscle) may be the distinguishing feature from simple tonsillitis.19 Examination will likely reveal a swollen tonsil with potentially gross deviation of the surrounding uvula or palatal structures.17

Retropharyngeal abscess

Shown in Fig. 3, retropharyngeal abscess may produce fever, dysphagia, and change in voice accompanied by neck swelling. Severe infection can result in drooling, while movement of the neck may be so painful as to cause torticollis. Trismus has been described and chest pain would be expecting whether the infection has tracked down into the mediastinum.17 Airway obstruction is a rare consequence of severe infection.

Management

Surgical drainage of the abscess, antibiotics, and supportive care should be the mainstays of management. Antimicrobial therapy will be required and guided on local policy, but attention should be paid to the high likelihood of the presence of both Gram-positive bacteria and anaerobes.

In peritonsillar abscess, surgical removal of the tonsils in the acute stage is controversial owing to the high associated bleeding risk,20 but if pus has formed, the consensus seems to be in favour of tonsillectomy to avoid the risks of local vascular erosion or aspiration following spontaneous rupture.16

Airway obstruction is an emergency and may be challenging owing to anatomical distortion. Retropharyngeal abscess may make the glottis hard to visualize, and any attempt at securing an airway should occur with an ENT surgeon present. Inhalation induction has been suggested and a cuffed tracheal tube will help to prevent soiling of the lower airways.12

Bacterial tracheitis

Invasive, exudative bacterial infection of the trachea, bacterial tracheitis was initially described as possessing features of both croup and epiglottitis.21 The initial case series described a normal epiglottis but with significant subglottic oedema which is shown in Fig. 4.

Aetiology

Staphylococcus aureus seems to be the most common causative organism, but others implicated in the disease include Streptococcus species and H. influenzae.22

Presentation

Cough, stridor, and pyrexia are commonly rapidly progressive and the child with tracheitis often appears toxic.23 The rapidly advancing nature of the infection means that the potential for airway obstruction should always be considered.

Management

While airway maintenance in cases of airway obstruction is an obvious indication for intubation, the possibility for rapid deterioration means that the majority of children with tracheitis...
will require intubation and management on intensive care, regardless of their clinical condition on presentation. Intensive care management will include regular suctioning of tracheal secretions.\(^2\)

Endoscopy is important in both diagnosis and management by removing pseudomembranous exudates, which in turn may significantly improve symptoms.\(^3\) This also provides an opportunity to send specimens for culture. Depending on the clinical condition of the child, rigid bronchoscopy under general anaesthesia may be possible in the spontaneously ventilating child before tracheal intubation and transfer to intensive care. This can be very challenging in the sick or hypoxic child however, and if the patient is compromised by attempts at bronchoscopy and airway toilet, they should be intubated and airway debris removed via the use of suction catheters.

Antibiotic agent selection should again follow local guidelines, but the frequency of Staphylococcus and other Gram-positive organisms as the cause should guide initial broad-spectrum antimicrobial therapy. Intravenous co-amoxiclav 30 mg kg\(^{-1}\) is a suitable first-line agent, as is ceftriaxone. There is no evidence to support the use of glucocorticoids as they do not influence outcome.\(^4\)

### Summary

Paediatric airway infections remain a common cause of presentation to hospital. The above pathologies (in addition to many more not mentioned in this article) exist on a spectrum of severity, the most dramatic end of which is complete airway obstruction in addition to systemic toxicity. In addition to the principles that underlie detection and management of sepsis, basic strategies for initial airway management in threatened or current airway obstruction are vital to improving outcome. Initial management with a simple ABCDE stepwise approach will permit simultaneous assessment and therapy, regardless of whether infective or non-infective conditions are present.

### Declaration of interest

None declared.

### MCQs

The associated MCQs (to support CME/CPD activity) can be accessed at http://www.oxforde-learning.com/journals/ by subscribers to BJA Education.

### References

1. Armon K, Stephenson T, Gabriel V et al. Determining the common medical presenting problems to an accident and emergency department. Arch Dis Child 2001; 84: 390–2
2. The child with breathing difficulties. In: Samuels M, Wieteska S, eds. Advanced Paediatric Life Support: The Practical Approach, 5th Edn. Oxford: Blackwell, 2011; 70–90
3. Cherry JD. Clinical practice. Croup. N Engl J Med 2008; 358: 384–91
4. Riikkanen H, Rönkkö E, Nieminen T, et al. Respiratory viruses in laryngeal croup of young children. J Pediatr 2008; 152: 661–5
5. Cherry JD, Croup (laryngitis, laryngotracheitis, spasmodic croup, laryngotracheobronchitis, bacterial tracheitis, and laryngotracheobronchopneumonitis) and epiglottitis (supraglottitis). In: Cherry JD, Harrison GJ, Kaplan SL et al., eds. Feigin and Cherry’s Textbook of Paediatric Infectious Diseases, 7th Edn. Philadelphia, PA: Elsevier Saunders, 2014; 241–60
6. Russell KA, Liang Y, O’Gorman K et al. Glucocorticoids for croup. Cochrane Database Syst Rev 2011; 1: CD001955
7. Björnsson C, Russell K, Vandermeer B et al. Nebulised epinephrine for croup in children. Cochrane Database Syst Rev 2013; 10: CD006619
8. Midwinter KI, Hodgson D, Yardley M. Paediatric epiglottitis: the influence of the Haemophilus influenzae b vaccine, a ten year review in the Sheffield region. Clin Otolaryngol 1999; 24: 447–8
9. Shah RK, Roberson DW, Jones DT. Epiglottitis in the Hemophilus influenzae type b vaccine era: changing trends. Laryngoscope 2004; 114: 557–60
10. Heath FT, Booy R, Azzopardi HJ et al. Antibiotic concentration and clinical protection after conjugate vaccination in the United Kingdom. JAMA 2000; 284: 2334–40
11. Stroud RH, Friedman NR. An update on inflammatory disorders of the pediatric airway: epiglottitis, croup and tracheitis. Am J Otolaryngol 2001; 22: 268–75
12. Jenkins IA, Saunders M. Infections of the airway. Pediatr Anesth 2009; 19: 118–30
13. Cantrell RW, Bell RA, Morika WT. Acute epiglottitis: intubation versus tracheostomy. Laryngoscope 1978; 88: 994–1005
14. Kotloff KL, Wald ER. Uvulitis in children. Pediatr Infec Dis 1983; 2: 392–3
15. Wald ER. Uvulitis. In: Cherry JD, Harrison GJ, Kaplan SL et al., eds. Feigin and Cherry’s Textbook of Paediatric Infectious Diseases, 7th Edn. Philadelphia, PA: Elsevier Saunders, 2014; 165–6
16. Brook I. Microbiology and management of peritonsillar, retropharyngeal, and parapharyngeal abscesses. J Oral Maxillofac Surg 2004; 62: 1545–50
17. Goldstein NA, Hammerschlag MR. Peritonsillar, retropharyngeal, and parapharyngeal abscesses. In: Cherry JD, Harrison GJ, Kaplan SL et al., eds, Feigin and Cherry’s Textbook of Paediatric Infectious Diseases, 7th Edn. Philadelphia, PA: Elsevier Saunders, 2014; 167–74
18. Philpott CM, Selvadurai D, Banerjeeer AA. Paediatric retropharyngeal abscess. J Laryngol Otol 2004; 118: 919–26
19. Ungkanont K, Yellon RF, Weissman JL et al. Head and neck space infections in infants and children. Otolaryngol Head Neck Surg 1995; 112: 375–82
20. Sdralis T, Berkowitz RG. Early adenotonsillectomy for relief of acute upper airway obstruction due to acute tonsillitis in children. Int J Pediatr Otorhinolaryngol 1996; 35: 25–9
21. Jones R, Santos JJ, Overall JC Jr. Bacterial tracheitis. JAMA 1979; 242: 721–6
22. Tebruegge M, Pantazidou A, Thorburn K et al. Bacterial tracheitis: a multi-centre perspective. Scan J Infect Dis 2009; 41: 548–57
23. Graf J, Stein F. Tracheitis in pediatric patients. Semin Pediatr Infect Dis 2006; 17: 11–3
24. Eckel HE, Widemann B, Damm M et al. Airway endoscopy in the diagnosis and treatment of bacterial tracheitis in children. Int J Pediatr Otorhinolaryngol 1993; 27: 147–57