Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

25 ACUTE INFECTIONS THAT PRODUCE UPPER AIRWAY OBSTRUCTION

IAN M. BALFOUR-LYNN, BSc, MD, MBBS, FRCP, FRCPCH, FRCS (ED), DHMSA, AND JANE C. DAVIES, MB, ChB, MRCP, MRCPCH, MD

Upper airway obstruction due to acute infection is not uncommon in children, and many parents have experienced an anxious night with a "croupy" child. Although infants and young children are most commonly affected because they have relatively narrow upper airways, older children and adults can also have significant symptoms. Fortunately, these are mostly due to self-limiting viral laryngotracheobronchitis (LTB), but there is also a group of bacterial infections (e.g., epiglottitis, bacterial tracheitis, diphtheria, retropharyngeal abscess, and peritonsillar abscess) that can occasionally cause significant obstruction. It is the job of the emergency physician, pediatrician, pediatric pulmonologist, or otorhinolaryngologist to diagnose more serious infections promptly so that treatment can be instituted early and disastrous obstruction avoided. It is also important to recognize when a simple viral LTB is causing significant problems so that appropriate treatment can be given immediately. This chapter is clinically orientated and outlines the principal infective causes of upper airway obstruction, with an emphasis on diagnosis and treatment. Confusion exists regarding the nomenclature for these disorders, with some using the term *croup* to refer to any inflammatory disorder of the upper airway, whereas others restrict its use to subglottic disease (i.e., LTB, which is usually of viral origin). Therefore, for the sake of clarity term *croup* will be largely avoided in this chapter.

The consequence of these upper airway infections is usually stridor, which is a clinical sign and should not be considered a definitive diagnosis. This chapter briefly outlines the principles behind what causes stridor, which should clarify why this condition mostly affects infants and young children. The appendix in Holinger and colleagues' *Pediatric Laryngology and Bronchoesophagology* discusses the physics of air flow and fluid dynamics.[1] The laws of fluid dynamics are based on flow through fixed tubes and may not always apply to dynamic airways *in vivo*. Normally, air flow through the upper airways is laminar, and the moving column of air produces slight negative pressure on the airway walls.[2] Inflammation resulting from infection causes a degree of airway narrowing, which increases the flow rate through the narrowed segment (the Venturi effect). This, in turn, causes a reduction in the pressure exerted on the airway wall. This is the Bernoulli principle. In other words, negative intraluminal pressure increases. This enhances the tendency of the airway to collapse inward, further narrowing the airway and causing turbulent air flow. The respiratory phase (inspiration or expiration) has a differential effect on air flow, depending on whether the obstruction is intrathoracic or extrathoracic (Fig. 25-1). Stridor is the sound made by rapid, turbulent flow of air through a narrowed segment of a large airway. It is most often loud, with medium or low pitch, and inspiratory. It usually originates from the larynx, upper trachea, or hypopharynx.[3] Progression of the disease process may make stridor softer, higher-pitched, and biphasic (inspiratory and expiratory). With the onset of complete obstruction, stridor may become barely audible as minimal air moves through the critically narrowed airway.[4]

The laryngeal anatomy of children makes them particularly susceptible to narrowing of the upper airways. The larynx of a neonate is situated high in the neck, and the epiglottis is narrow, omega-shaped (ω), and vertically positioned. The narrowest segment of the pediatric airway is the subglottic region (in adults, it is at the glottic level), which is encircled by the rigid cricoid cartilage ring. There is nonfibrous, loosely attached mucosa in this region that is easily obstructed in the presence of subglottic edema. Additionally, the cartilaginous support of the infant airway is soft and compliant, easily allowing dynamic collapse of the airways during inspiration. Young children have proportionally large heads and relatively lax neck support; this combination increases the likelihood of airway obstruction when supine.[5] Also, their tongues are relatively large for the size of the oropharynx. Simple mathematics shows why a small amount of edema has such a profound effect on the cross-sectional area and, hence, air flow. The diameter of the subglottis in a normal newborn is approximately 5 mm, and 0.5-mm edema in this region reduces the cross-sectional area to 64% of normal (area = $\pi \infty$ radius^2). Air flow is directly proportional to the airway radius to the fourth power (Poiseuille's law), so a small reduction in caliber has a major effect on flow rate. The same 5-mm airway with 0.5 mm edema will have a flow rate of only 41% of baseline, assuming that pressure remains unchanged—a situation that is not necessarily the case if the Bernoulli principle is in play. Because the caliber of the airway is almost inevitably reduced further in accord with the Bernoulli principle, and Poiseuille flow is not established, the flow rate is much further reduced and the work of breathing is greatly increased to maintain ventilation.

VIRAL LARYNGOTRACHEOBRONCHITIS

Etiology

The most common etiologic agents are the parainfluenza viruses (PIVs), of which PIV 1 is found most frequently and leads to epidemics. PIV 2 may account for

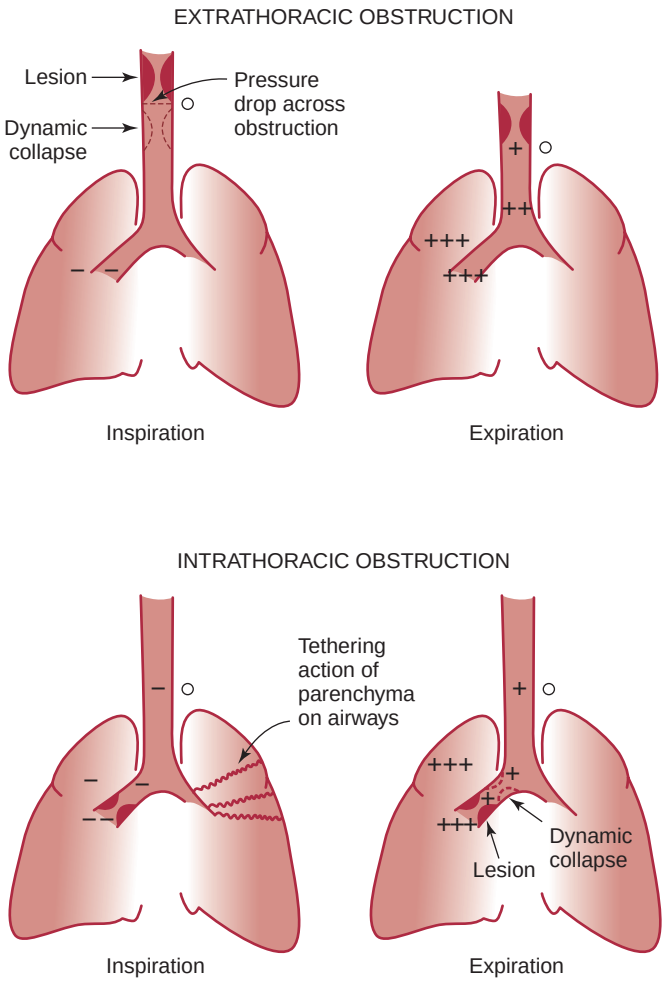

FIGURE 25-1. The effect of the respiratory phase on extrathoracic and intrathoracic obstruction is shown. During inspiration, negative intratracheal pressure (relative to atmospheric pressure) leads to dynamic collapse of the extrathoracic airway, thus worsening the effects of an extrathoracic obstructive lesion. In contrast, intrathoracic obstruction improves during inspiration because the elastic recoil of the lung parenchyma opens the intrathoracic airways. During expiration, intratracheal pressure is positive relative to atmospheric pressure, opening the extrathoracic trachea and lessening the obstructive effect of lesions. In contrast, intrathoracic obstruction worsens because of lower pressure in the airways relative to the surrounding parenchyma, collapsing the airways. (From Loughlin GM, Taussig LM. Upper airway obstruction. *Semin Respir Med.* 1979;1:131-146.)

many sporadic cases, and PIV 3 is a less common cause of viral LTB, usually targeting the epithelium of the smaller airways and leading to bronchiolitic illness. The PIVs belong to the *Paramyxoviridae* family, along with respiratory syncytial virus, measles, mumps, and the recently identified human metapneumovirus.[6] Together the PIVs account for more than 75% of viral LTB cases, although other respiratory viruses (e.g., respiratory syncytial virus, rhinovirus, influenza virus, adenovirus, coronavirus, and enteroviruses) can produce a similar clinical syndrome. Herpesviruses tend to cause a more severe and protracted form of the disease. LTB can also occur with some systemic infections, such as measles, and less commonly, Mycoplasma. In general, however, it is not usually possible to identify the cause of infection from the child's symptoms because severity does not correlate with any particular etiologic agent.

Epidemiology

Viral LTB is the most common cause of infective upper airway obstruction in the pediatric age group. Affected children are usually of preschool age, with a peak incidence between 18 and 24 months of age.[7] Reported incidence rates vary from 1.5% to 6%, but less than 5% of these require hospital admission, and only 1% to 2% of those admitted require endotracheal intubation and intensive care.[8] This proportion has fallen dramatically since the use of corticosteroids has become routine. There is a male preponderance in children younger than 6 years of age (1.4:1), although both sexes appear to be affected equally at an older age. Cases may occur in epidemics, with those caused by PIV 1 particularly presenting in fall and winter months and infection with other organisms (including PIV 2) occurring more commonly as isolated infections. Infection is via droplet spread or direct inoculation from the hands. Viruses can survive for long periods on dry surfaces, such as clothes and toys, emphasizing the importance of infection-control practices.

Pathophysiology

Infection affects the larynx, trachea, and bronchi, although swelling and inflammation in the subglottic area leads to the characteristic clinical features of viral LTB. In addition to relative differences in airway size (discussed earlier in the chapter), it is suggested that poor cell-mediated immunity in younger age groups also accounts for differences observed between adults and children.[9] The epithelium of the subglottis possesses abundant mucous glands, secretions from which can further narrow the airway lumen in response to infection. The PIVs are trophic for the respiratory epithelium, binding in particular to ciliated cells via an interaction between the viral hemagglutinin-neuraminidase protein and its receptor, sialic acid. Other viral proteins (the F protein in particular) are important in membrane fusion and the passage of viral particles between cells. Many strains of PIV are cytopathic, with infection leading to the formation of giant cells and cell death. As with many infective processes, the ensuing inflammatory response is involved in the evolution of symptoms. Both polymorphonuclear and monocytic leukocytes infiltrate the sub-epithelium, which leads to vascular congestion and airway wall edema. In addition, the symptoms of viral LTB are believed to be caused by the release of spasmogenic mediators, leading to decreased airway diameter. This may result from a type I hypersensitivity response to PIV, and some authors have postulated a role for anti-PIV-specific immunoglobulin E (IgE) in the development of airway narrowing.[10] These factors may play a relatively greater role in patients with recurrent (spasmodic) croup, and these patients may have hyperreactivity of the extrathoracic and intrathoracic airways.[11] The etiology of recurrent, spasmodic croup remains unclear, with authors expressing differing views on whether it is usually virus-related[12] or is a separate disease entity; suggested triggers include gastroesophageal reflux and anatomical abnormalities in addition to an allergic predisposition.[13]

Clinical Presentation and Diagnosis

Mild

Most children are affected mildly by viruses that cause LTB.[7] The exact incidence remains unknown because many of them do not receive medical attention, but are managed by parents at home. Children have a barking cough and a hoarse cry or voice; these symptoms are worse in the evening and at night. They may also have inspiratory stridor on exertion, but stridor at rest is usually absent, as are other signs of respiratory distress. There is most commonly a coryzal prodrome accompanied by a low-grade fever, but children are not particularly unwell or toxic. They remain interested in their surroundings, are playful, and still eat and drink.

Moderate

Features of moderate viral LTB include those discussed earlier, but with inspiratory stridor present at rest, as well as a degree of respiratory distress manifest by chest wall recession, tachypnea, and the use of accessory muscles of respiration. There is usually accompanying tachycardia, but children remain interactive and are able to take at least liquids orally.

Severe

Progression from moderate to severe infection can occur rapidly and may be precipitated by the distress caused by clinical examination. Worrisome signs include those of increasing respiratory distress, with the child appearing anxious or preoccupied and tired. Drooling may occur, and the child will often refuse liquids or be unable to coordinate swallowing and breathing. However, the child with viral LTB will not appear toxic, with high fever and flushed face, as do those with the classic signs of bacterial epiglottitis (Table 25-1). Another difference is in the nature of the cough; a harsh, barking cough is not commonly associated with epiglottitis (in which there is often a muffled cough and cry). Restlessness and agitation are

TABLE 25-1 DIFFERENTIATION OF PRINCIPAL INFECTIVE CAUSES OF UPPER AIRWAY OBSTRUCTION

	VIRAL LARYNGOTRACHEO-BRONCHITIS	EPIGLOTTITIS	BACTERIAL TRACHEITIS	DIPHTHERIA	RETROPHARYNGEAL ABSCESS
Principal Organisms	Parainfluenza 1–3 Adenovirus Respiratory syncytial virus	*Haemophilus influenzae* *Streptococcus*	*Staphylococcus aureus* *Moraxella catarrhalis* *H. influenzae*	Corynebacterium diphtheria	Mixed flora, including *S. aureus*, *Streptococcus*, *H. influenzae*, anaerobes
Age Range	6 mo–4 yr (peak, 1–2 yr)	2–7 yr	6 mo–8 yr	All ages	<6 yr
Incidence	Common	Rare	Rare	Rare if vaccinated	Uncommon
Onset	Insidious Usually follows upper respiratory tract infection	Rapid	Slow, with sudden deterioration	Insidious	Gradual
Site	Below the vocal cords	Supraglottis	Trachea	Tonsils, pharynx, larynx, nose, skin	Retropharyngeal space
Clinical Manifestations	Low-grade fever Nontoxic Barking (seal-like) cough Stridor Hoarseness Restlessness	High fever Severe sore throat Minimal nonbarking cough Toxic Stridor Drooling Dysphagia Muffled voice position	High fever Toxic Brassy cough Stridor Hoarse voice Neck pain Choking	Fever Toxic Stridor Sore throat Fetor oris Cervical lymphadenopathy Bull neck	Fever Sore throat Neck pain and stiffness (especially on extension) Dysphagia Stridor (less common) Drooling Retropharyngeal bulge
Endoscopic Findings	Deep red mucosa Subglottic edema	Cherry-red or pale and edematous epiglottis Edematous aryepiglottic folds	Deep red mucosa Ulcerations Copious, thick tracheal secretions Subglottic edema, with normal epiglottis and arytenoids	Gray, adherent membrane on the pharynx	N/A
Intubation	Occasional	Usual	Usual	Occasional	Unusual
Therapy	Corticosteroids Nebulized epinephrine	Intubation (1–3 days) IV antibiotics	Intubation (3–7 days) IV antibiotics Tracheal suction	Diphtheria antitoxin IV antibiotics Immunization during convalescence	IV antibiotics ± surgery

late signs of airway obstruction of any cause, as is cyanosis, pallor, or decreased level of consciousness. Pulse oximetry should be performed, but limitations must be recognized. Oxygen saturation may be well preserved until the late stages of severe viral LTB, and it can lead to significant underestimation of respiratory compromise in a patient who is receiving supplementary oxygen. Conversely, desaturation may be seen in children with relatively mild airway obstruction (presumably reflecting lower airway involvement and ventilation-perfusion mismatch).[14] Pulsus paradoxus is present in this group with severe disease, but in clinical practice, it is difficult to assess, and attempts to do so could worsen symptoms by causing distress.

Recurrent or Spasmodic Croup

Symptoms are similar to those of the more typical forms of viral LTB, but children are often older, do not have the same coryzal prodrome, and may be afebrile during the episode. There may be links with atopy, often with a positive family history. Attacks often occur suddenly, at night, and may resolve equally quickly. Treatment must be guided by the degree of severity, and is similar to that for viral LTB. Some practitioners prescribe oral or inhaled corticosteroids (via a spacer device) to be kept at home and administered by the parents in case of an episode, although there is a paucity of evidence for or against this practice.

Non-Infective Causes of Acute Airway Obstruction

There are a number of non-infective causes of upper airway obstruction, and these must be considered in the differential diagnosis of infective causes (Box 25-1). Foreign body inhalation is the most common non-infective cause in children. Symptoms may partly mimic those of viral LTB and will depend on the location of the foreign body, the degree of resultant airway obstruction, and (to a lesser extent) the nature of the foreign body. Onset of symptoms may be either acute or insidious; a large foreign body may cause severe obstruction, whereas a smaller one may simply lead to laryngeal and tracheal irritation and airway edema. In cases of severe airway obstruction, the voice may be lost and breath sounds quiet. This condition is an emergency and requires immediate visualization of the larynx and trachea and removal of the foreign body by a physician or surgeon experienced in this procedure. Occasionally, an unrecognized inhaled foreign body leads to chronic stridor. Acute upper airway obstruction may also result from the ingestion of caustic substances, with resulting pharyngeal burns, edema, and inflammation of the epiglottis, aryepiglottic folds, larynx, and trachea. This diagnosis is usually clear from the history. Rarely, angioneurotic edema may cause acute laryngeal swelling and airway obstruction. Patients appear nontoxic and may exhibit other signs of allergic disease, such as urticaria and abdominal pain. In hereditary angioneurotic edema due to C1 esterase deficiency, the family history may be positive, although the first presentation is more common in adults than in children.

There are numerous causes of chronic airway obstruction that are discussed elsewhere in this book. Confusion may arise when an upper respiratory tract infection unmasks a previously asymptomatic congenital abnormality. For example, mild subglottic stenosis may cause symptoms only with the additional burden of airway edema due to a simple viral upper respiratory infection. It is important to ensure that there is no history of intubation (which may have been brief, as in resuscitation of a newborn in the maternity unit) or of any coexisting signs (e.g., a cutaneous hemangioma) that may increase the index of suspicion for a congenital airway abnormality.

BOX 25-1 Infectious and Noninfectious Causes of Acute Upper Airway Obstruction

Infectious
Viral laryngotracheobronchitis
Epiglottitis
Bacterial tracheitis
Diphtheria
Retropharyngeal abscess
Peritonsillar abscess
Infectious mononucleosis

Noninfectious
Foreign body
Trauma
Caustic burns
Spasmodic croup
Angioneurotic edema

Who Should Be Evaluated?

Most children with viral LTB were previously well, have short, self-limiting symptoms, and make a full recovery. The lack of complete immunity and the variety of agents that can cause viral LTB mean that more than one episode is not uncommon, particularly in separate seasons. However, some children have symptoms that should lead to further clinical evaluation. These include multiple episodes, particularly if they are severe or frequent, symptoms that are particularly slow to resolve, and symptoms that occur between or in the absence of obvious infections. Evaluation of patients in this group is aimed at identifying an underlying airway abnormality that would predispose the child to more severe airway narrowing with viral infections, or that could cause problems independently of such infection. Investigation is usually centered on airway endoscopy. This must be performed in a unit and by an operator who is experienced in the technique because there is a risk in many of these conditions of exacerbating the airway obstruction. Spontaneous breathing is necessary to identify vocal cord problems or airway malacia, and anesthetic techniques must be carefully considered. If an inhaled foreign body is considered likely, rigid bronchoscopy is the study of choice. Additional studies that might be considered once the acute episode has resolved include plain lateral neck and chest radiographs, computed tomography or magnetic resonance imaging scan, contrast assessment of the upper airway (e.g., videofluoroscopy, barium swallow), and a

pH study. Polysomnography may help to determine the severity of chronic symptoms. Rarer causes of recurrent stridor (e.g., hypocalcaemia or angioneurotic edema) are diagnosed by blood testing.

Management of Viral Laryngotracheobronchitis

Management of viral LTB must be based on clinical assessment of severity. Such assessment should be based on the clinical features described earlier; there is no role for radiography in the assessment of acute airway obstruction. In skilled hands, plain lateral neck radiographs may demonstrate sites of obstruction, but this rarely influences management; it also wastes time and can be dangerous. The neck extension that is required could precipitate sudden worsening of airway obstruction, which can be fatal in severe cases. Several scoring systems have been devised,[15] and the most commonly applied system (the 17-point Westley scale, which assesses degree of stridor, chest retractions, air entry, cyanosis, and level of consciousness) has been well validated. However, these are mainly used in the context of clinical trials and are not a substitute for experienced clinical assessment.

Supportive Care

Children with mild croup can be managed at home. They should be treated with plenty of fluids and antipyretics as required. Because the vast majority of cases are of viral etiology, there is no role for the routine use of antibiotics in the absence of other features suggestive of bacterial infection. Parents should be warned that symptoms are usually worse at night.

Humidification

Both at home and in the hospital setting, humidified air (either steam or cool mist) has been used for more than a century to produce symptomatic relief from croup. Despite this, there is very little supportive published evidence; most early studies, some of which may have been underpowered, generally suggested no benefit.[16–18] A larger study of 140 moderately affected children showed no differences in signs or requirement for additional treatments with optimally delivered 100% humidity,[19] and the most recent Cochrane Systematic Review has also concluded there is no evidence of benefit.[20] Case reports have described severe burns caused by spilling of boiling water and facial scalds from the use of steam, so this type of treatment is not without the potential for harm.[21]

Corticosteroids

The use of corticosteroids has received much attention for more than a decade, and their therapeutic role is well established. Their mechanism of action, however, remains unclear, although is believed to relate to rapid-onset anti-inflammatory properties. The cumulative evidence strongly supports their use in children with moderate to severe symptoms, although there are still outstanding questions, including the optimal route of administration, the most appropriate dosing regimen, and the best oral agent.

The role of corticosteroids in the management of croup in children has been the subject of several Cochrane reviews, with the most recent update in November 2004.[22] In this review, the authors identified 31 studies that fulfilled their criteria for inclusion, namely, randomized controlled trials in children measuring the effectiveness of corticosteroids (any route of administration) against either a placebo or another treatment. A total of 3736 children were included, the majority from placebo-controlled trials. Outcome measures included the croup score (most commonly the Westley scale), the requirement for admission or return visit, the length of stay, and the requirement for additional therapeutic interventions. Overall, treatment led to an improvement in the croup score at 6 and 12 hours, but the improvement was no longer apparent at 24 hours. The length of time spent in either the emergency department or the hospital was also significantly decreased, as was the requirement for nebulized epinephrine. Importantly, and in contrast to the previous version of the Cochrane review, the authors concluded with funnel plots and other statistical methods that these results were not influenced by publication bias.[22] Since this publication, one further large randomized controlled trial (n = 720) studied mild croup (Westley score ≤ 2), and showed benefit of a single dose of dexamethasone, 0.6 mg/kg, in terms of return to medical care, resolution of symptoms, decreased loss of sleep by the child, and reduced parental stress.[23] In conclusion, the case for corticosteroids is now clear. In severe disease, rates of intubation are significantly decreased and the duration of intubation is reduced, and in moderate disease admission, the need for additional treatment and return visits are reduced.[8] More recent studies have focused attention on the optimal formulation, dose, and treatment regimen.

Optimal Route of Administration, Formulation, and Dosing Regimen

Studies included in the Cochrane review (discussed earlier in the chapter) and conducted since then have used the intramuscular, oral, or nebulized route to administer different corticosteroid preparations. This area has been recently well reviewed.[8] From the studies that have attempted to address the route of administration, nebulized, oral, and intramuscular routes appear, in general, to be roughly equivalent. Nebulization could potentially increase distress of the child and worsen upper airway obstruction, although it may be preferable in a child who is vomiting or having difficulty swallowing.

Similarly, studies using oral agents have used either dexamethasone or prednisolone, and both in varying doses. Many primary care physicians who visit homes do not routinely carry dexamethasone but do carry oral prednisolone. There is no strong evidence in support of one preparation over the other, although one recent study favored dexamethosone, which led to a reduced frequency of re-presentation.[24] In contrast, a recent Australian trial compared 1 mg/ kg prednisolone with two doses of dexamethasone (0.15 and 0.6 mg/kg) and found no difference in croup score, requirement for further treatment, or re-presentation.[25] With regard to dexamethasone, 0.6 mg/kg has been the dose most widely used, but

several recent studies have demonstrated that this dose may be higher than required and that 0.15 mg/kg is just as effective.[25–28] A practical approach might be to use dexamethasone, if available, at a dose of 0.15 mg/kg. If this preparation were not available at a home visit, prednisolone (at an equivalent dose of 1 mg/kg) could provide a useful substitute.

Nebulized epinephrine (adrenaline): Most clinical trials have used the racemic form of this drug,[29] although there is now evidence that the L-isomer used alone (which is the only available formulation in some units) may be equally effective.[30] The mechanisms of action are believed to be a combination of rapid reduction in airway wall edema and bronchodilation. It has a rapid onset of action (within 30 minutes), and the effect lasts for 2 to 3 hours. The recommended dose is 0.4 to 0.5 mL/kg (to a maximum of 5 mL) of the 1:1000 preparation that is put undiluted into the nebulizer pot. According to these studies, nebulized epinephrine has been shown to improve the croup score and reduce the likelihood of hospital admission, but it is less clear whether, when given with corticosteroids, it reduces the need for intubation. It should be used in any child who has severe signs and symptoms, and it should be considered for those with moderate signs and symptoms, depending on the signs of respiratory distress and possible response to corticosteroid administration. It can be administered in the home setting while awaiting an ambulance, but, clearly, any child requiring this treatment at home must be transferred promptly to the hospital for monitoring. Multiple doses may be administered, although the requirement for this must lead to consideration of the need for intensive care management. Although rebound worsening of symptoms after administration of nebulized epinephrine is often alluded to, in practice, this phenomenon does not appear to be a real risk. Traditionally, children treated with epinephrine have been admitted to the hospital, but recent studies have confirmed that discharge home is safe after 3 to 4 hours of observation if the child has made significant improvement.[31]

Other treatments for severe cases: Oxygen should be administered to any child with severe airway obstruction, even in the absence of severe hypoxia, because it will aid respiratory muscle function. As mentioned earlier, a child with severe respiratory distress and obstruction may have relatively normal pulse oximetry readings when breathing oxygen, which can be dangerous if misinterpreted by staff who are unaware of this limitation. Heliox (70% to 80% helium with 20% to 30% oxygen) has been used in both upper airway obstruction[32] and severe asthma, and it is the focus of a recent Cochrane review.[33] Only two small randomized controlled trials were identified, totaling 44 patients with severe croup. Heliox was compared with either 30% humidified oxygen or with 100% oxygen plus epinephrine. There was no additional benefit of Heliox, although further well-designed controlled trials were recommended. Some children with severe croup either do not respond to the usual therapies or are too severely compromised at presentation to permit their use. These children require urgent endotracheal intubation and mechanical ventilation to avoid potentially catastrophic complete airway obstruction and the serious sequelae of hypoxia and hypercapnia (e.g., hypoxic ischemic encephalopathy). Intubation should be performed by the most experienced person available, and it should be attempted with an uncuffed endotracheal tube one size smaller than the usual size for the child.[34] Facilities for immediate tracheostomy must be available at the time of intubation. Children may have coexisting lower airway and parenchymal involvement that impairs gas exchange and may lead to slower than expected clinical improvement after intubation. Rarely, pulmonary edema may develop after relief of airway obstruction, particularly if the disease course has been prolonged. Most children without severe parenchymal involvement require respiratory support for 3 to 5 days.[34] This is one context in which multiple rather than single doses of corticosteroids are often administered. The timing of extubation will depend on the development of an air leak, indicating resolution of airway narrowing.[34] Re-intubation rates of approximately 10% have been reported.[35]

EPIGLOTTITIS

Etiology

Historically, *H. influenzae* type B (HiB) was responsible for almost all (approximately 99%) cases of epiglottitis in otherwise healthy children. Since the introduction of HiB immunization, other organisms have been implicated, including groups A, B, C, and G β-hemolytic streptococcus. Other responsible organisms include *Haemophilus parainfluenzae, Staphylococcus aureus, Moraxella catarrhalis, Pneumococcus, Klebsiella, Pseudomonas,* Candida, and viruses (e.g., herpes simplex, varicella, PIV, influenza).[2,36]

Epidemiology

H. influenzae type B vaccines were first licensed in the United States in 1988, with widespread immunization programs in place by the early 1990s. Since then, reported cases of invasive HiB disease (including epiglottitis) in children younger than 5 years of age have declined by 99%.[37] The same pattern has been repeated in Europe, with significant reductions in the United Kingdom.[38,39] Immunization was introduced in the United Kingdom in 1992, and, with immunization coverage exceeding 90%, the decline in incidence was more than 95%.[39] In 1998, the incidence in those younger than 5 years of age was 0.6 per 100,000, compared with 31 to 36 per 100,000 in England and Wales before the introduction of the vaccine.[39] However in 2003 there was a resurgence of Hib infections in the United Kingdom, which led to the launch of a booster program. Ethnicity plays a part in vaccine efficacy. Data from 1996 to 1997 in the United States show that the average annual incidence of HiB invasive disease per 100,000 children younger than 5 years of age was 0.5 among non-Hispanic whites, 0.6 among Asians and Pacific Islanders, 0.7 among non-Hispanic blacks, 0.7 among Hispanic Americans, and 12.4 among Native Americans and Alaskan natives.[37] Nevertheless, cases of epiglottitis due to HiB continue to be reported,[40] as do cases due to other organisms.[36]

Cases of invasive HiB occur principally in non-immunized children, but also rarely in some true vaccine failures. Clinical risk factors for vaccine failure include prematurity, Down syndrome, malignancy, developmental delay, and congenital or acquired immunodeficiency, principally reduced immunoglobulin concentrations (IgG_2 subclass, IgA, IgM) and neutropenia.[41] However, these factors explain fewer than 50% of cases of vaccine failures.[41] An HiB IgG antibody titer of ≥0.15 µg/mL confers protection from disease, but, given the natural waning in antibody levels, it is estimated that a titer of ≥1.0 µg/mL should provide long-term protection.[40] However, there may sometimes be qualitative functional problems with antibody responses that are not yet fully elucidated.

Epiglottitis tends to occur in children 2 to 7 years of age, but cases have been reported in those younger than 1 year of age.[2] Since the introduction of HiB vaccine, the peak age distribution has increased slightly.[42] A recent review of a national U.S. dataset from 1998 to 2006 has shown that the mean age of a patient admitted with epiglottitis is 45 years and the national mortality rate is 0.89%; there is a decrease in admissions for those under 18 years of age (with greatest risk at <1 year) and an increase in the 46- to 64-year-old group.[43] Most cases occur in the fall and winter.

Pathophysiology

Although HiB has a low point-prevalence of nasopharyngeal carriage (1% to 5%), most young children become colonized with HiB in the first 2 to 5 years.[44] The relationship between asymptomatic carriage, immunity, and the development of invasive disease is not clearly understood. Viral co-infection may have a role in the transition from colonization to invasion.[45] Colonies of HiB organisms reside in the nasal mucosal epithelium and submucosa. Invasive disease occurs when organisms disseminate from the mucosa of the upper respiratory tract via the bloodstream; bacteremia increases over a period of hours, and metastatic seeding can occur.[44] Thus, although situated in close proximity to the nose, the supraglottic area is likely to be affected via the bloodstream; direct spread along mucosal surfaces may also play a part. This may account for the relatively high yield of positive blood cultures in epiglottitis and the relatively low incidence of epiglottitis among carriers of HiB.

Epiglottitis is more correctly called *supraglottitis*. It is a bacterial cellulitis of the supraglottic structures, particularly the lingual surface of the epiglottis and the aryepiglottic folds.[2] Destruction of the infected epithelial tissue results in mucosal ulcerations, which may appear on the epiglottis, larynx, and trachea. The submucosal glands are involved as well, with the formation of abscesses. Infection of the epiglottis itself causes a local inflammatory response that results in a cherry-red edematous epiglottis when caused by HiB (Fig. 25-2), although it tends to be pale and edematous and accompanied by edematous aryepiglottic folds when caused by *Streptococcus*.[36,37] As supraglottic edema worsens, the epiglottis is displaced posteriorly, and it may obstruct the airway.

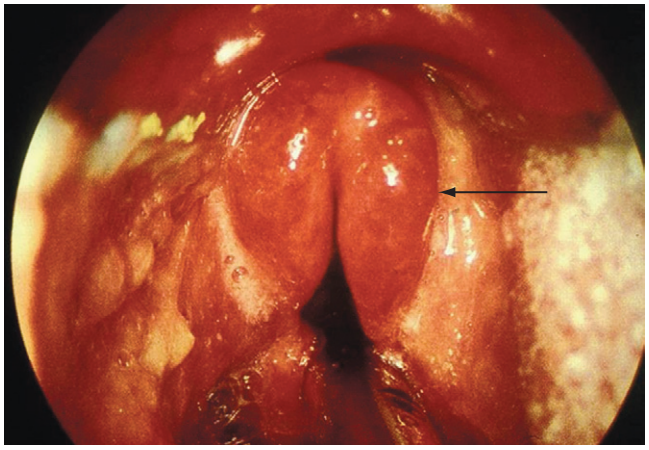

FIGURE 25-2. Swollen epiglottis *(arrow)* caused by acute epiglottitis in an intubated child. (From Benjamin B, Bingham B, Hawke M, et al. *A Colour Atlas of Otorhinolaryngology.* London: Taylor & Francis, 1995, p 292, with permission.)

Clinical Presentation and Diagnosis

Classic epiglottitis caused by HiB is a fulminant disease in an otherwise healthy child, who can be near death in a few hours. It is a medical emergency that can be alarming for the medical staff and devastating for the family. Epiglottitis clearly has not been eliminated, but due to its rarity there are concerns about a potential lack of familiarity with its management among emergency physicians, pediatricians, anesthesiologists, and otolaryngologists.[46] Up to 20% of infants with epiglottitis are misdiagnosed initially, usually with viral LTB.[4] Typically, there is a short history of fever, severe throat pain, stridor, and respiratory distress, but the symptoms progress rapidly. Children become toxic and tend to sit anxiously in the classic tripod position (sitting upright, with the chin up, mouth open, bracing themselves on their hands) as air hunger develops (Fig. 25-3). They often drool because they cannot swallow their secretions, and the voice is muffled due to pain and soft tissue swelling. Stridor may progress, and when marked, signals almost complete obstruction of the airways. Complete, fatal airway obstruction may occur suddenly and without warning. The most serious complication of this disease process (and any infective upper airway obstruction) is hypoxic ischemic encephalopathy resulting from respiratory arrest. This tragic complication is almost always preventable with clinical suspicion, prompt diagnosis, and correct management. However, a recent 13-year case series demonstrated that cardiac arrest occurred in 3 of 40 cases (7.5%), although there were no long-term sequelae.[40] Secondary sites of HiB infection may be present in approximately half of cases, and include meningitis, otitis media, pneumonia, and cellulitis; therefore, repeated physical examination during the admission is critical.[2] The pneumonia may contribute to poor gas exchange.

Generally, distinction from standard viral LTB is based on the older age of the child, the lack of history of upper respiratory tract infection, the speed of progression, the degree of toxicity, the extent of drooling, the use of the tripod position, and minimal cough (see Table 25-1).

FIGURE 25-3. Characteristic posture in a patient with epiglottitis. The child is leaning forward and drooling, with a hyperextended neck. (Courtesy of Dr. Robert Berg.)

However, it is important to remember that most of these symptoms can be present in acute severe upper airway obstruction from other causes.

The presentation and clinical course of epiglottitis caused by various types of β-hemolytic streptococcal pathogens are similar to each other, but they differ from those associated with HiB.[36] The onset of disease is more gradual, but the resolution of tissue damage and the time to recovery are longer, with a mean intubation time of 6 days.[36,47]

Management

The first priority and key response to the diagnosis must be to secure the airway in a controlled environment. Physical examination (especially of the throat) and cannulation or venipuncture should be deferred because emotional upset and crying may precipitate complete airway obstruction. When epiglottitis is suspected clinically, the child (and parents) should be approached in a calm and reassuring manner. Oxygen should be given, even if the mask is held at a distance from the child's face. The child should be taken to the operating room, anesthetic room, or pediatric intensive care unit, and held by a parent. The child should be accompanied by a senior medical team that is skilled in airway management and carrying a laryngoscope, an endotracheal tube, and a percutaneous tracheostomy tray. If complete airway obstruction develops suddenly, performance of a Heimlich maneuver may relieve the obstruction temporarily; alternatively, forward traction may be applied to the mandible.

Inhalational induction of anesthesia is preferred. Laryngoscopy should then be performed and the diagnosis confirmed, based on the appearance of the epiglottic region, as described earlier in the chapter (erythema and edema of the supraglottis). Endotracheal intubation is then achieved using an orotracheal tube, which is later changed to a nasotracheal tube because this is less likely to be displaced. Although tracheostomy is rarely necessary, a surgical team should be prepared to perform this immediately if intubation is unsuccessful. Once the airway is secured, the emergency is over, and the remaining studies can be performed. Intravenous cannulation and blood sampling can be done. The white cell count is increased, and blood culture findings are often positive (70% in one series).[40] Airway secretions and swabs from the epiglottic region should be sent for bacterial culture and viral detection. Urinary antigen testing may be useful for those already receiving antibiotics.[45]

Some authors have advocated the use of a lateral neck radiograph if the child is stable before intubation, claiming it to be the "single most useful study."[2,5] We strongly disagree, and this is not our recommendation because it can precipitate respiratory arrest as a result of complete obstruction. We take the same view as Goodman and McHugh, who state that "plain radiographs have no role to play in the assessment of the critically ill child with acute stridor."[48]

Intravenous antibiotics are started and must cover HiB and *Streptococcus;* the response is usually rapid.[2] A third-generation cephalosporin (e.g., ceftriaxone or cefotaxime) is usually given and may be changed once antibiotic sensitivities are available. Antibiotics have traditionally been given for 7 to 10 days; however, a randomized controlled trial showed that a two-dose course of ceftriaxone was as efficacious as 5 days of chloramphenicol.[45] Contacts of patients with HiB should be given appropriate prophylaxis, usually rifampicin. There is some empiric evidence that corticosteroids may improve the course of epiglottitis, but racemic epinephrine has not been shown to be of benefit. The duration of intubation for epiglottitis due to HiB averages 1 to 3 days,[5,40] but it is longer when caused by *Streptococcus;*[3] as always, there is great individual variation. A decision to extubate may be made when an air leak develops around the endotracheal tube, but repeat endoscopy may be useful to aid this decision. Again, facilities for emergency tracheostomy must be available. Some give dexamethasone before extubation to reduce postextubation stridor.[4]

BACTERIAL TRACHEITIS

Bacterial tracheitis has also been known as bacterial, or membranous LTB; nondiphtheritic laryngitis with marked exudate; and pseudomembranous croup.

Etiology

The most common pathogen is *S. aureus,* although other organisms implicated include HiB, α-hemolytic *Streptococcus, Pneumococcus,* and *M. catarrhalis*.[5] Occasionally, Gram-negative enteric organisms and *Pseudomonas aeruginosa* are isolated (the latter is associated with a more severe clinical course).[49] In a recent case series, *M. catarrhalis* (27%) was more common than S. aureus (22%), although this represents data from a single center over the course of 14 months.[50] One series of 94 cases over 10 years found that *M. catarrhalis* was associated with a greater rate of intubation: 83% versus 49% with other organisms, although they were a younger group.[51] In addition, PIV and influenza viruses are commonly isolated from tracheal secretions; measles and enteroviruses have also been detected. Although it may be a primary bacterial infection, bacterial tracheitis is considered secondary to primary viral LTB. Presumably, viral injury to the tracheal mucosa and impairment of local immunity predisposes to bacterial superinfection.

Epidemiology

Bacterial tracheitis is a rare disease, with the most recent large case series (from 1998) describing only 46 cases.[50] The peak incidence is during fall and winter, and it predominantly affects children 6 months to 8 years of age (mean 5 years of age). Most affected children were previously well, but it has been reported as a complication of elective tonsillectomy and adenoidectomy.[52] A large case series of life-threatening upper airway infections from Vermont in the United States between 1997 and 2006 showed that bacterial tracheitis has now superseded viral croup and epiglottitis, and was three times more likely as a cause of respiratory failure than the other two diagnoses combined.[53]

Pathophysiology

Bacterial tracheitis is characterized by marked subglottic edema, with ulceration; erythema; pseudomembranous formation on the tracheal surface; and thick, mucopurulent tracheal secretions. The thick exudate and sloughed mucosa frequently obstruct the lumen of the trachea and the main-stem bronchi.[2] The epiglottis and arytenoids are usually normal in appearance, although epiglottitis and bacterial tracheitis may coexist. Tracheal stenosis can be a complication, especially after prolonged intubation.[49]

Clinical Presentation and Diagnosis

The clinical picture is initially similar to that of viral LTB, with mild fever, cough, and stridor for several days. However, the patient's condition deteriorates rapidly, with a high fever and often a toxic appearance, with respiratory distress and airway obstruction. Other symptoms include choking episodes, orthopnea, dysphagia, and neck pain.[50] The clinical picture differs from that of epiglottitis in that its onset tends to be more insidious. Patients have a substantial brassy cough, are more able to lie flat, and tend not to drool (see Table 25-1).[5] Children are more ill than with simple viral LTB and do not respond to expected therapies (e.g., corticosteroids or nebulized epinephrine). There may be other co-infections, particularly pneumonia. Other reported complications include cardiopulmonary arrest, with subsequent hypoxic encephalopathy and seizures, pneumothorax, subglottic stenosis, septicemia, toxic shock syndrome, pulmonary edema, and adult respiratory distress syndrome.[54]

The white blood cell count shows polymorphonuclear leukocytosis, often with a left shift. A lateral neck radiograph may show a hazy tracheal air column, with multiple luminal soft tissue irregularities due to pseudomembrane detachment from the soft tissue, but radiographs should be taken only after the patient is stabilized and safe. There are, however, no clinical or radiographic features capable of confirming the diagnosis.[2] This must be confirmed by upper airway endoscopy and a positive bacterial culture.

Management

Diagnostic endoscopy, which should be done under general anesthesia, is also therapeutic because it enables removal of secretions and sloughed tissue from the airway lumen. Rigid endoscopy may be necessary, and sometimes the procedure must be repeated. Many patients (especially younger ones) require endotracheal intubation and mechanical ventilation to overcome airway obstruction (reports of 50% to 100% intubation rates),[55] usually for 3 to 7 days. Frequent tracheal suction is necessary. In a recent series, 57% of patients required intubation, which is lower than the rate previously reported.[50] The decision to extubate is based on clinical improvement, with reduction of fever, decreased airway secretions, and development of an air leak around the endotracheal tube. Corticosteroids may be given before extubation. Tracheostomy is required less often than in the past. Initially, intravenous broad-spectrum antibiotics are given, and these can be refined once cultures and antibiotic sensitivities are known, usually for 10 to 14 days. Mortality is now uncommon.

DIPHTHERIA

Etiology

Diphtheria is caused by toxigenic strains of the bacterium *Corynebacterium diphtheriae* and, less frequently, *C. ulcerans*. The organism may be isolated on bacterial culture of nasal and pharyngeal swabs, and serologic studies may detect antibodies to diphtheria toxin. Polymerase chain reaction can confirm *C. diphtheriae tox* genes.[56]

Epidemiology

Diphtheritic laryngitis was once the most common infectious cause of acute upper airway obstruction in children. Although it became uncommon due to widespread immunization programs started in the 1940s, it remains a serious disease in parts of the world. Large outbreaks occurred in the 1990s throughout Russia and

the independent countries of the former Soviet Union (nearly 50,000 cases were reported). In 2005, 36 countries reported almost 13,000 cases to the World Health Organization, and 80% were from India.[57] The last childhood deaths reported in the UK were 1 in 1994 and 1 in 2008.[58] Most life-threatening cases occurred in unvaccinated or inadequately immunized persons, and it is important for children traveling to these countries, particularly for extended periods, to be vaccinated. A list of endemic countries is available on the Centers for Disease Control website *(wwwnc.cdc.gov/travel/yellowbook/2012/chapter-3-infectious-diseases-related-to-travel/diphtheria.htm)*. Adults are particularly at risk because protective levels of diphtheria antibodies decrease progressively with time from immunization, so re-immunization is recommended before travel. The Third National Health and Nutrition Examination Survey of U.S. residents (1988 to 1994) indicated that fully protective levels (≥0.1 IU/mL) were found in 91% of children 6 to 11 years of age, but only in 30% of adults 60 to 69 years of age.[59]

Pathophysiology

Diphtheria is an acute disease, primarily involving the tonsils, pharynx, larynx, nose, skin, and, occasionally, other mucous membranes. In the milder, catarrhal form, there is no membrane formation, but in the more severe, membranous form, there is a characteristic lesion of one or more patches of an adherent grayish-white membrane, surrounded by inflammation. The toxin causes local tissue destruction at the site of membrane formation, which promotes multiplication and transmission of the bacteria.[60]

Clinical Presentation and Diagnosis

The incubation period is 1 to 6 days. Classic respiratory diphtheria is characterized by an insidious onset, and patients typically present with a 3- to 4-day history of upper respiratory infection. They have a fever, membranous pharyngitis with a sore throat, characteristic fetor oris, cervical lymphadenopathy, and sometimes edema of the surrounding soft tissues (bull-neck appearance). They may have a serosanguinous nasal discharge. Although not always present, the membrane is typically gray, thick, fibrinous, and firmly adherent, so it may bleed on attempted removal (Fig. 25-4). Laryngeal diphtheria most commonly occurs as an extension of pharyngeal involvement in children, leading to increasing hoarseness and stridor. The patient appears toxic, with symptoms of LTB, and signs of severe airway obstruction may develop quickly if the pharyngeal membrane dislodges and obstructs the airway. Complications include secondary pneumonia and toxin-mediated disease including myocarditis or cardiomyopathy, neuritis or paralysis, and adrenal failure with hypotension.[58] Cardiomyopathy can be predicted from a combination of a pseudomembrane score >2 (range is up to 4) and a bull-neck appearance.[61] Case fatality rates vary and can reach 20%, but the mortality rate was only 3% in a series of 676 patients (30% younger than 15 years of age) reported from the 1995 Kyrgyz Republic outbreak.[62] Deaths are usually a result of airway obstruction, myocarditis/cardiomyopathy, or sepsis (disseminated intravascular coagulation and renal failure).

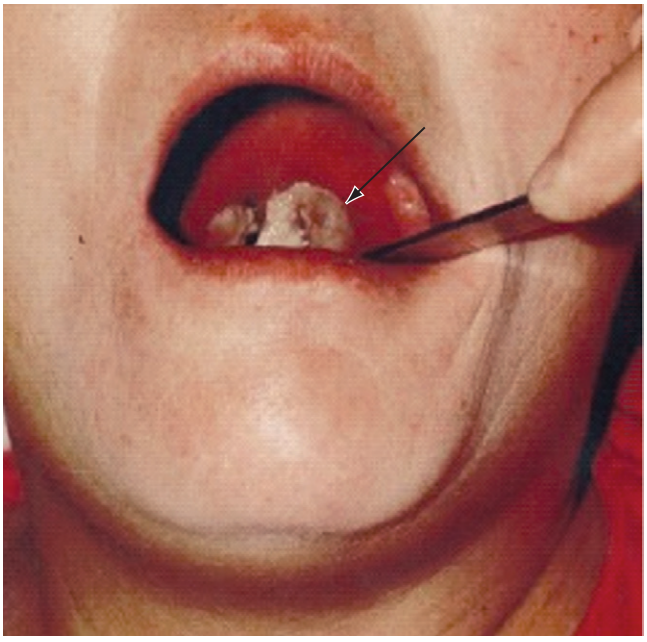

FIGURE 25-4. Diphtheritic membrane *(arrow)* extending from the uvula to the pharyngeal wall in an adult. (From Kadirova R, Kartoglu HÜ, Strebel PM. Clinical characteristics and management of 676 hospitalized diphtheria cases. Kyrgyz Republic 1995. *J Infect Dis.* 2000;181(Suppl 1):S110-S115. Photograph by P. Strebel.)

Management

Because diphtheria is now rare, a high index of suspicion is important to make the diagnosis and institute prompt treatment. Patients should be strictly isolated. Diphtheria antitoxin, which is a hyperimmune equine antiserum (available from the Centers for Disease Control and Prevention), should be administered without waiting for laboratory confirmation because it neutralizes circulating toxin, but not toxin bound to tissues. Antibiotics are not a substitute for antitoxin, but they are given to eradicate the organism, stop toxin production, and reduce the likelihood of transmission.[60] Intravenous penicillin or erythromycin is used, and once the child can swallow comfortably, treatment can be given orally, for a total of 14 days.[60] Mechanical ventilation and tracheostomy may be required. Intravenous dexamethasone has been given to children with laryngeal diphtheria and airway obstruction, and a small case series suggested that it was beneficial.[63] Because the disease may not confer immunity, patients should be given a diphtheria toxoid-containing vaccine during convalescence. Antibiotic prophylaxis (penicillin or erythromycin) is recommended for close contacts after nasal and pharyngeal specimens are taken, and immunization should be given to those who have not been vaccinated in the preceding 5 years.[60]

RETROPHARYNGEAL ABSCESS

Etiology

Retropharyngeal abscesses generally result from lymphatic spread of infection, although direct spread from adjacent areas, penetrating pharyngeal trauma (e.g., a fall with a pencil in the mouth), or foreign bodies can also play a role.[5] It has also been reported as a complication of adenoidectomy and adenotonsillectomy.[64,65] Infection is usually due to mixed flora, including *S. aureus* (methicillin-sensitive and resistant), various streptococcal species, HiB, and anaerobes.[66]

Epidemiology

The majority of cases occur in children younger than 6 years of age, probably due to the fact that retropharyngeal lymph nodes are so abundant at this age (they tend to atrophy later in life). Analysis of the U.S.-based Kid's Inpatient Database (KID) for 2003 covering 36 states revealed 1321 admissions with a mean age of 5.1 years, and 63% were boys; there were no deaths reported.[67] Another large series reported a median age of 3 years, with 75% of those affected younger than 5 years of age and 16% younger than 1 year of age.[66] In a large series from 1995 to 2006, there was a linear increase in incidence through the period.[68]

Pathophysiology

The retropharyngeal space (between the posterior pharyngeal wall and the prevertebral layer of deep cervical fascia) contains loose connective tissue and lymph nodes that drain the nasopharynx, paranasal sinuses, middle ear, teeth, and adjacent bones. The space extends from the base of the skull down to vertebra C7 or T1. Acute bacterial infection in this region may start as retropharyngeal cellulitis with localized thickening of the tissues, which can progress to purulent inflammation of the tissues and retropharyngeal adenitis. However, when this process is caused by lymphatic spread, it starts as adenitis. If liquefaction of one of the nodes occurs, an abscess can form and is usually contained within the inflammatory rind of the infected node.

Clinical Presentation and Diagnosis

Presentation is often nonspecific, and there may be overlap with the presentation of croup, epiglottitis, tracheitis, and peritonsillar abscess (see Table 25-1).[5] Children with acute epiglottitis tend to appear more toxic and progress to respiratory distress more rapidly.[69] Excluding causes secondary to foreign bodies or trauma, patients usually have a history of viral upper respiratory infection that lasts several days and then worsens. Children then have high fever, sore throat, dysphagia, poor feeding, neck pain, and stiffness. Limitation of neck extension and torticollis are more common than limited neck flexion.[66] Although the occurrence of neck signs with fever may suggest meningitis, children tend not to be as toxic as those with meningitis. Further deterioration may lead to extrathoracic airway compromise, with drooling, stridor, and respiratory distress. The classical picture of stridor and airway obstruction seems to be less common now, and in a series of cases reported by pediatricians from 1993 to 1998, only 3% of 64 children in Salt Lake City had stridor or wheezing.[66] However in a series of cases from 2002 to 2007 from the same center, but published by the otolaryngologists, 14/130 (11%) presented with airways obstruction, and 50% required intubation, with an average age of 1.4 years.[70] Older series have reported a higher incidence of stridor. Reported rates include 71% in patients younger than 1 year of age, 43% in patients older than 1 year of age, and no stridor in those older than 3 years of age in a series of 31 children in Sydney, Australia[71]; a rate of 56% in 17 patients in Denver[72]; and a rate of 23% in 65 children in Los Angeles.[73] It is possible that the spectrum of disease is changing, but it is more likely that the diagnosis is being made earlier, before the airways are compromised.[66] Sometimes a retropharyngeal mass is visible in the mouth, seen as an asymmetrical bulge of the posterior pharyngeal wall (Fig. 25-5), or a neck mass (marked lymphadenopathy or parapharyngeal abscess) is visible and palpable. Complications include rupture of the abscess with aspiration, asphyxiation or pneumonia, extension to a mediastinal abscess, Lemierre's syndrome, and vascular complications (e.g., thrombophlebitis of the internal jugular vein and erosion through the carotid artery sheath).[64,69]

Once the child is stable and safe, a lateral neck radiograph with the neck in full extension may confirm the diagnosis. Widened prevertebral soft-tissue and air-fluid levels in the retropharyngeal space are all indicators.[5] The radiograph must be taken in the correct position (true lateral orientation, with the neck in extension, and, if possible, during full inspiration) to ensure that the

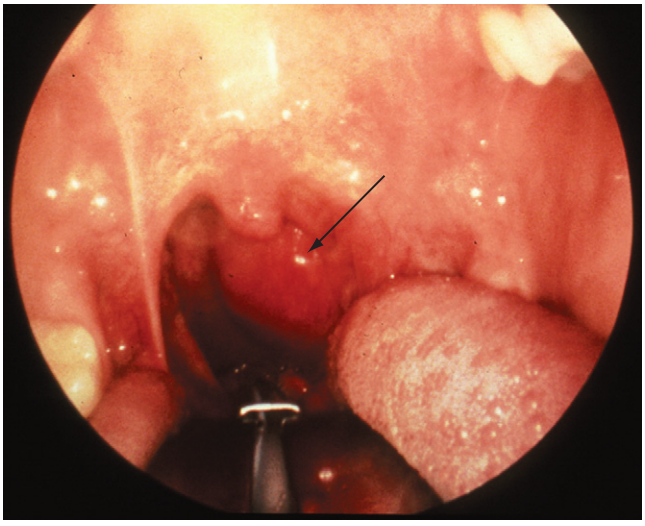

FIGURE 25-5. Retropharyngeal abscess behind and to the left of the uvula *(arrow)* is shown. The tongue is pressed down and to the left with a tongue depressor. (Photograph courtesy of the Otolaryngology Teaching Set, Department of Ear, Nose & Throat, Great Ormond Street Hospital for Children NHS Trust, London.)

retropharyngeal space is not falsely thickened.[66] A contrast-enhanced computed tomography scan is useful because it differentiates a fully developed abscess from cellulitis and delineates the full extent of the abscess. Blood cultures findings are usually negative, but the white blood cell count will be increased.

Management

Traditionally, management involves surgical drainage of the abscess (plus antibiotics). However, more cases are being managed by intravenous antibiotics alone, although surgery must be considered early if there is a compromised airway. In one older series, 25% of patients required no surgery,[72] but in the Salt Lake City series that covered 1993 to 1998, 58% of 64 patients had antibiotics alone, with no treatment failures.[66] Data from KID 2003 (discussed earlier in the chapter) showed that 57% of cases admitted did not require surgical drainage.[67] Many children start treatment with antibiotics before the diagnosis is made, and certainly retropharyngeal cellulitis can be treated with antibiotics alone. When necessary, surgical drainage is performed through the intraoral route. Care must be taken to avoid aspiration of the infected material. Occasionally, if there is extension lateral to the great vessels, external drainage through the neck is necessary. Mortality is rare now, with no deaths in recent reports.

PERITONSILLAR ABSCESS (QUINSY)

Etiology

A peritonsillar abscess is usually a complication of acute tonsillitis, but it may follow pharyngitis or a previous peritonsillar abscess. The infection usually involves mixed bacterial flora, with *Streptococcus pyogenes* the predominant organism. In a small series of children younger than 5 years of age, *Streptococcus viridans* was the most common organism detected.[74] In a large series of 457 children and adults in Israel, while *Streptococcus pyogenes* was the most common organism isolated, there was a sharp rise in anerobes cultured, particularly *Prevotella* and *Peptostreptococcus*.[75]

Epidemiology

Peritonsillar abscess is the most common deep-space head and neck infection in both adults and children. However, it is more common in young adults than in children. It tends to affect older children, and in one large, 10-year series, the mean age was 12 years of age, with two thirds of those affected older than 10 years of age.[76] In this series, 62% of cases occurred on the left side versus 38% on the right (with no obvious explanation).[76] There is no seasonal predilection. The reduction of antibiotic prescribing to children by general practitioners in the United Kingdom from 1993-2003 has not been accompanied by an increase in hospital admissions for peritonsillar abscess.[77]

Pathophysiology

Peritonsillar abscess is believed to arise from the spread of infection from the tonsil or the mucous glands of Weber, located in the superior tonsillar pole.[76] There is a spectrum of peritonsillar cellulitis that may then result in a collection of pus located between the tonsillar capsule (pharyngobasilar fascia), the superior constrictor, and the palatopharyngeus muscle.[76] There is a risk of spread through the muscle into the parapharyngeal space or other deep neck spaces. The abscess pushes the adjacent tonsil downward and medially, and the uvula may be so edematous as to resemble a white grape.

Clinical Presentation and Diagnosis

The child, who is already affected by acute tonsillitis, becomes more ill with a high fever and has a severe sore throat or neck pain, as well as marked dysphagia with referred earache. Absent or decreased oral intake can lead to dehydration, particularly in younger children.

Cervical lymphadenopathy is almost always present. The uvula is edematous and deviated to one side, and there is fetor oris. A striking feature may be trismus, with limited mouth opening. Examination may be difficult in a young, uncooperative child who refuses to open the mouth. The white blood cell count will be elevated.

The relevance of this condition to pediatric pulmonologists is that acute enlargement of the tonsils can cause airway compromise, and a ruptured abscess can lead to aspiration of infected material and subsequent pneumonia. In one large series of 169 children under 18 years of age, 8% presented with airway compromise.[78]

Management

Children may need intravenous fluids. Antibiotics are necessary, and intravenous penicillin is as effective as broad-spectrum antibiotics, although additional anaerobic coverage should be considered.[79] Analgesia is important. Corticosteroids are not uncommonly used but are of no obvious benefit (or harm).[78] Treatment often involves needle aspiration or incision and drainage; a meta-analysis of 10 studies with 496 patients revealed an average 94% success rate with simple needle aspiration.[80] Nevertheless, an initial period of medical treatment is appropriate in the absence of airway compromise and systemic toxicity, especially in younger children.[81] There is some debate among otolaryngologists about the role and timing of tonsillectomy, and peritonsillar abscess is no longer considered an absolute indication for tonsillectomy, although a history of recurrent tonsillitis prior to developing the peritonsillar abscess leads to a higher recurrence rate.[82]

INFECTIOUS MONONUCLEOSIS

Infectious mononucleosis is caused by Ebstein-Barr virus (EBV) and is common in adolescents and young adults. The clinical syndrome is characterized by fever, fatigue,

malaise, lymphadenopathy, and sore throat. Diagnosis is confirmed by positive EBV serology, and positive heterophile antibodies (Monospot), although the latter are only positive in 50% of children and 90% of adults. The illness is usually self-limiting, but malaise and exhaustion can persist.

Some degree of airway obstruction is not uncommon (reported in 25% to 60% cases[58]), but significant airway compromise is rare, occurring in an estimated 1% to 3.5% cases.[83] Nevertheless, given the high frequency of EBV infection, this small proportion still represents many patients. Acute upper airway obstruction may occur, but the cardinal signs of acute obstruction (stridor and respiratory distress with recession and tachypnea) can be absent until late in the process.[84] Obstruction arises from a combination of inflammation and hypertrophy of the palatal and nasopharyngeal tonsils, edema of the pharynx and epiglottis, and pseudomembrane formation in the large airways.[85] Peritonsillar abscess formation is a rare complication that can further compromise the airway, and it is now believed that this is not significantly associated with the use of corticosteroids, which contradicts earlier reports.[86]

Management is with systemic corticosteroids (in the presence of obstruction) and supportive care, which may include ventilation. A tracheostomy is sometimes necessary. If corticosteroids do not help, the role of acute tonsillectomy has been advocated,[87] but it is controversial due to the high risk of perioperative bleeding.[85]

Suggested Reading

Bjornson CL, Johnson DW. Croup. *Lancet*. 2008;371:329–339.
Jenkins IA, Saunders M. Infections of the airway. *Pediatr Anesthesia*. 2009;19(suppl 1):118–130.
Johnson D. Croup. *Clin Evid*. 2004;(12):401–426.
Loftis L. Acute infectious upper airway obstructions in children. *Semin Pediatr Infect Dis*. 2006;17:5–10.

References

The complete reference list is available online at www.expertconsult.com