

Respiratory Emergencies in Children

Alexandre T Rotta MD and Budi Wiryawan MD

Introduction

General Assessment Skills and Management

Croup (Laryngotracheobronchitis)

Spasmodic Croup

Epiglottitis

Bacterial Tracheitis

Retropharyngeal Abscess

Foreign Bodies

Inhalational Injuries

Summary

Acute obstructive respiratory emergencies in children are a common cause of emergency department visits. The severity of these conditions ranges from mild, self-limited disease to life-threatening forms of rapidly progressive airway obstruction. A high index of suspicion is necessary for prompt diagnosis and treatment. This review discusses general principles of assessing and managing respiratory emergencies in children, as well as clinical characteristics and treatment of specific conditions such as croup, epiglottitis, bacterial tracheitis, retropharyngeal abscess, foreign bodies, and inhalational injuries. Key words: pediatric, respiratory, emergency, croup, epiglottitis, bacterial tracheitis, retropharyngeal abscess, foreign bodies, burns, inhalation, helium. [Respir Care 2003;48(3):248–258. © 2003 Daedalus Enterprises]

Introduction

Respiratory failure is the most common cause of cardiopulmonary arrest in pediatric patients. Therefore, prompt recognition, assessment, and expert management of respiratory emergencies are critical to obtaining the best possible outcome. Anatomical differences between pediatric and adult patients render children more susceptible to acute airway com-

promise. Young children have proportionally larger heads, prominent occiputs, and relatively lax cervical support, which increases the likelihood of airway obstruction in the supine position. A relatively large tongue in comparison to a small oropharynx further contributes to this problem.

The subglottis is the narrowest segment of the pediatric airway, in contrast to the glottis in adults. The subglottic region is completely encircled by the cricoid cartilage, thus restricting its ability to freely expand in diameter. Furthermore, the subglottic airway contains loosely attached connective tissue that can rapidly cause substantial reduction in airway caliber should inflammation and soft tissue edema develop. The change in air flow resulting from reduction in airway diameter can be predicted by the application of Poiseuille's law:

$$\dot{V} = (\Delta P \pi r^4) / (8 n L)$$

in which \dot{V} is flow, ΔP is the pressure gradient between the 2 ends of a tube (airway), r is the radius, n is the viscosity

Alexandre T Rotta MD and Budi Wiryawan MD are affiliated with the Division of Pediatric Critical Care Medicine, The Children's Hospital of Buffalo, and State University of New York at Buffalo, Buffalo, New York.

Alexandre T Rotta MD presented a version of this report at the 31st RESPIRATORY CARE Journal Conference, Current Trends in Neonatal and Pediatric Respiratory Care, August 16–18, 2002, in Keystone, Colorado.

Correspondence: Alexandre T Rotta MD, Division of Pediatric Critical Care, The Children's Hospital of Buffalo, 219 Bryant Street, Buffalo NY 14222. E-mail: arotta@buffalo.edu.

of the medium, and L is the length. Considering that the change in airway flow is directly proportional to the airway radius elevated to the fourth power, an airway with a diameter of 7 mm that develops a 0.5 mm edema will have a flow of 54% of baseline, assuming pressure remains unchanged ($3^4/3.5^4 \times 100$).

There are many potential causes of respiratory emergencies in children, including infections, inflammatory and allergic processes, foreign body aspirations or ingestions, trauma, chemical or thermal injuries, neoplasms, neurological syndromes, and congenital anomalies. This review discusses the emergency recognition and management of common conditions presenting with acute onset and that can rapidly progress to airway compromise, obstruction, and respiratory failure.

General Assessment Skills and Management

Although many specific causes of airway emergencies will be discussed in this review, certain basic principles of assessment and management can be universally applied. Prompt recognition of the obstructive respiratory emergency and then correct management and precise treatment of the inciting condition are critical to achieving optimal clinical outcome.

Several objective and subjective observations can be used in evaluating a respiratory emergency and gauging the severity of airway compromise. Subjective impressions of a child's general well being are very important and can be derived from the overall level of alertness, response to the surrounding environment, and interaction with the parents or with the examiner. Young or nonverbal children pose a particular challenge, as decreased interaction with the environment and an altered level of consciousness may be the only noticeable early presenting signs of a rapidly progressive respiratory process. Older children may be mature enough to voice specific areas of discomfort, fatigue, respiratory difficulty, or sense of impending doom. Tachypnea is one of the earliest objective signs of respiratory compromise in children. Unfortunately, this important clinical clue may be missed by a clinician who is not familiar with pediatric respiratory patterns or with the age-dependent range of respiratory rates in children, spanning rates as low as 12 breaths/min in adolescents to 50 breaths/min in newborns.¹

Under normal conditions, breathing should appear effortless, even at the higher respiratory frequencies of newborns and infants. The use of accessory respiratory muscle groups and nasal flaring correlates well with the development of obstruction and respiratory difficulty in children. Stridor is also a common presenting sign of extrathoracic airway obstruction and warrants prompt investigation. Stridor occurs when the laminar flow through the extrathoracic airway is disrupted by a narrowing or partial obstruction,

creating a Venturi effect, which is the acceleration of flow observed through a narrowed segment of a tube. This flow acceleration results in the development of a more negative intraluminal airway pressure (Bernoulli principle), which further contributes to the collapse of the deformable extrathoracic airway. The resulting turbulence and vibration of the airway, which characteristically occurs during inspiration, are perceived as stridor on physical examination.² Further progression of the disease process, fixed obstruction at the cricoid level, or a foreign body in the extrathoracic airway can lead to biphasic stridor. Obstructions of the intrathoracic airways generally present as expiratory wheezes, although narrowing of the intrathoracic extrapulmonary (central) airways can present as a low-pitched sound that resembles an expiratory stridor.³

Cyanosis is a late sign and is consistent with impending respiratory failure. Therefore, although the presence of cyanosis mandates immediate attention, its absence should not be construed as a sign of stability in the child who presents with other symptoms of respiratory embarrassment.

A more objective sign in the assessment of patients with obstructive airway emergencies, and who are instrumented with invasive arterial pressure monitoring in the intensive care setting, is pulsus paradoxus, which is a dynamic physiologic decrease in systolic blood pressure observed during inspiration, compared to the higher systolic blood pressure during expiration. Breathing in the presence of an extrathoracic airway obstruction results in the generation of excessive negative intrathoracic forces and exacerbates the pulsus paradoxus. Longitudinal observation of the magnitude of the pulsus paradoxus provides an interesting objective measurement of disease progression and response to treatment (Fig. 1).

The stable child with signs and symptoms of airway compromise warrants a careful and detailed history in order to establish the presence or absence of pre-existing conditions, fever, trauma, choking, ingestions, exposure to allergens, concomitant illnesses, as well as onset and duration of the presenting signs and symptoms. The unstable child must be evaluated and treated rapidly, with emergency stabilization of the airway receiving the highest priority. Patients with airway obstruction leading to respiratory arrest should promptly be ventilated with a combination of bag-valve-mask technique and proper head positioning (slight neck extension, chin lift and jaw thrust maneuvers). A patent airway should then be established under optimal conditions by the staff member most experienced in handling difficult airways. Spontaneously breathing children should be offered supplemental 100% oxygen in a non-threatening manner, keeping the patient as calm as possible while preparing to deliver definitive treatment.

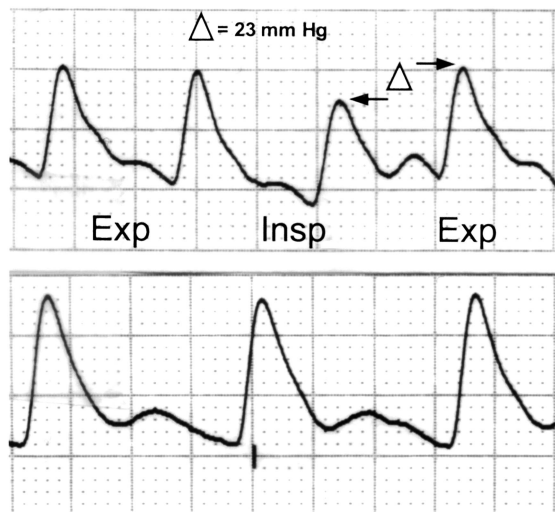


Fig. 1. Pressure recordings from a radial artery catheter of a spontaneously breathing child with croup and post-extubation stridor. Upper panel: An abnormally high pulsus paradoxus of 23 mm Hg is measured as the difference (Δ) in systolic blood pressure between expiration (Exp) and inspiration (Insp). Lower panel: Normal physiologic variation of the systolic blood pressure as a function of the respiratory cycle after racemic epinephrine, steroids, heliox, and 12 hours post-extubation.

Croup (Laryngotracheobronchitis)

Croup or laryngotracheobronchitis (LTB) is the most common cause of infectious airway obstruction in children,^{4,5} with an annual incidence of 18 per 1,000 children in the United States.^{6,7} It primarily affects children between the ages of 6 months and 4 years, with a peak incidence of 60 per 1,000 among children 1–2 years of age.^{6,7} LTB is epidemic in nature, with peak incidence during early fall and winter,^{8–10} although sporadic cases may be seen throughout the year. The most common etiologic agent is the parainfluenza virus type 1, although parainfluenza types 2 and 3, influenza A and B, respiratory syncytial virus, adenovirus, *Mycoplasma pneumoniae*, herpes simplex type 1, and numerous other organisms have also been implicated.^{8,10–13}

LTB has a broad disease severity spectrum. Most children who are seen in clinics and emergency departments return home for supportive care. Hospitalization rates ranging from 1 to 30% have been reported for patients in the more severe end of the spectrum.¹⁴ Endotracheal intubation and mechanical ventilation are needed in approximately 2% of hospitalized children,⁷ although the need for intubation appears to be diminishing because of the increasing use of glucocorticoids.¹⁵ The typical LTB patient presents with a several-day history of upper respiratory-type symptoms, progressing to hoarseness, the characteristic barking (seal-like) cough, and stridor. The stridor is most commonly an inspiratory sound, with biphasic stridor



Fig. 2. Typical radiographic appearance of laryngotracheobronchitis (croup) showing symmetric narrowing of the subglottic area (steeple sign).

indicating more severe narrowing of the airway. Breath sounds are generally clear except for transmitted upper airway sounds. The presence of biphasic stridor, nasal flaring, intercostal and suprasternal retractions, tachypnea, and low pulse oximetry values should be seen as signs of impending respiratory collapse. Low-grade fever is a common finding with LTB patients. The hemogram may show leukocytosis with a viral differential, although a normal white cell count is commonly found. The diagnosis of classic viral LTB should be made clinically, posing little challenge to the experienced practitioner. In fact, only approximately 2% of patients initially diagnosed with croup are given an incorrect diagnosis.¹⁶ Radiographic examination of the soft tissues of the neck may help establish the diagnosis of croup, while ruling out other important conditions such as epiglottitis, hemangioma, congenital abnormality, foreign body, or retropharyngeal abscess. The classic radiographic finding of LTB on the frontal view is a narrowing of the subglottic area, commonly known as the steeple sign (Fig. 2). The absence of this finding, however, does not rule out the diagnosis of LTB, since as many as half of patients may have normal neck radiographs.^{5,8} When visible, the subglottic narrowing is dynamic and is more accentuated during inspiration, because of the more negative intraluminal airway pressure during inspiration.¹⁷

LTB is usually self-limited and frequently requires only supportive care. Less than 10% of LTB patients require hospitalization^{8,10} and management is largely dependent on the severity of respiratory symptoms. Traditionally, patients with LTB have been treated with humidified air, as either heated or cool mist. The rationale for this practice includes soothing the inflamed mucosa, decreasing the amount of coughing due to mucosal irritation, and liquefying secretions for easier expectoration.⁵ Unfortunately, no scientific evidence exists to support the idea that humidified air has any effect on the subglottic mucosa or that it positively influences patient outcome.¹⁸ Furthermore, mist tents can increase respiratory distress by provoking anxiety and upsetting the patient because of separation from the parents. Another potential disadvantage of the mist tent is the reduced ability to closely observe the child because he or she is behind a plastic wall. Despite the lack of objective benefit, humidified air continues to be used by many in the treatment of LTB, largely based on anecdotal evidence.

Nebulized racemic epinephrine is an important treatment for LTB patients, as the vasoconstrictive α adrenergic effect on the mucosal vasculature is highly effective in rapidly reducing airway edema.^{11,19,20} Racemic epinephrine contains both levo (L) and dextro (D) epinephrine isomers, of which the L form is the active component. The racemic form is the most common form of treatment administered via nebulization, although L epinephrine is as effective as racemic epinephrine without greater adverse effects, when appropriate concentrations are used.²¹ Treatment with racemic epinephrine causes rapid improvement in clinical status (in 10–30 min) and appears to decrease the need for intubation.²² The effect, however, is transient and disappears within 2 hours of administration.^{11,19,20} A single dose may relieve the symptoms of some children in considerable distress, but others may need repeated doses. In the past, all children with LTB treated with racemic epinephrine were admitted to the hospital for observation, because of the fear of rebound airway edema. Recent studies, however, suggest that these patients may be safely discharged home from the emergency department after a 2–3 hour observation period, provided they are stridor free, show no signs of respiratory distress, and that the parents can provide reliable monitoring and return to the hospital if necessary.^{23–25}

The use of corticosteroids for LTB has been the topic of substantial debate.^{26–31} Recent studies have shown substantial improvement in children with severe LTB treated with corticosteroids.^{30,31} In these studies corticosteroids were beneficial regardless of the route of administration.^{30,31} The study by Johnson et al³⁰ compared patients treated with placebo, nebulized budesonide, or a dose of 0.6 mg/kg dexamethasone either orally, intramuscularly, or intravenously, and found a lower hospitalization rate

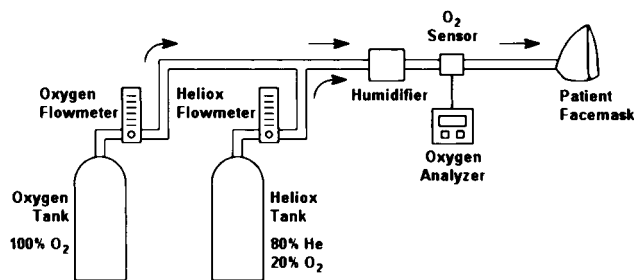


Fig. 3. Schematic diagram of a heliox delivery system.

among patients treated with any type of steroid, compared to placebo. The precise mechanism of action of steroids in LTB is not known. Corticosteroids may work because of their anti-inflammatory activity, through inhibition of the synthesis or release of inflammatory mediators such as interleukin 1, interleukin 2, tumor necrosis factor α , platelet-activating factor, and metabolites of arachidonic acid. However, the rapid response observed after corticosteroid treatment suggests that another mechanism may play a role. Corticosteroids decrease permeability of the capillary endothelium and stabilize lysosomal membranes.³² These actions have been known to decrease the inflammatory reaction and reduce submucosal edema. Although adverse effects from short-term corticosteroids for LTB are rare, physicians must be aware of potential complications, such as bacterial tracheitis, gastrointestinal bleeding, and oral candidiasis.³³

The administration of a mixture of helium and oxygen (heliox) can be of benefit in the treatment of selected patients with severe forms of LTB.³⁴ Helium is a very light, odorless, tasteless, noncombustible, and physiologically inert gas. It has a very low gas density (0.1785 g/L) in comparison to room air (1.20 g/L). Therefore, a mixture of helium and oxygen can create a respirable gas with a density lower than that of an oxygen/nitrogen mixture or oxygen alone (1.43 g/L). The low density of heliox reduces the gas turbulence in the airways (and around the obstruction) and the pressure gradient needed to generate respiratory flow, thus decreasing the work of breathing and benefiting the patient who is suffering an airway obstruction.³⁴ Effective mixtures of helium and oxygen contain between 80:20 and 60:40 parts of each gas, respectively. Therefore, patients with a high oxygen requirement (greater than 40%) are unlikely to benefit from this therapy. Heliox should only be administered from a pre-mixed heliox cylinder with 80% helium and 20% oxygen, so as to prevent the possibility of asphyxia by accidentally delivering 100% helium, as would be the case if the oxygen cylinder were empty. Higher oxygen concentration can be obtained by blending in oxygen from an oxygen cylinder with the heliox (Fig. 3).

In patients with the most severe forms of LTB that do not respond to supportive treatment, nebulized epinephrine, corticosteroids, heliox, endotracheal intubation, and ventilation may be necessary. In these cases, an endotracheal tube with a diameter smaller than recommended for the patient's age and size should be used. Extubation can usually be accomplished within 2 or 3 days, when an air leak around the endotracheal tube is detected.

Endoscopy is indicated for patients who fail to develop an air leak within 7 days, those who follow an uncharacteristic clinical course, and those who have frequent and recurrent episodes of presumed LTB. Endoscopy should also be considered in patients who have signs and symptoms of LTB but who are younger than 6 months of age, because of the higher likelihood of anatomical abnormality, gastroesophageal reflux, or laryngeal hemangioma in these patients.

Spasmodic Croup

The term "spasmodic croup" describes a condition clinically similar to LTB. Patients with spasmodic croup typically have a barking cough and stridor, but lack fever and a viral prodrome. Its onset tends to be abrupt, usually at night, and it often improves within hours either with cool humidified air or without intervention. The precise pathogenesis of spasmodic croup is unknown, although it is thought to be allergic or angioneurotic in origin, as opposed to infectious.^{5,8,32} Treatment for severe forms of spasmodic croup is the same as for viral LTB.

Epiglottitis

Epiglottitis is a serious, life-threatening infection of the extrathoracic airway and is an airway emergency. The term epiglottitis is somewhat misleading, since the process is actually a cellulitis of supraglottic structures, including the posterior lingual surface and surrounding soft tissues, the epiglottis and the aryepiglottic folds, thus making supraglottitis a more appropriate designation. Supraglottitis is classically described in children between 2 and 8 years of age, although it can occur at any age.^{16,35-38}

Haemophilus influenzae type B is the most common causative organism,³⁹ although many other agents have been reported, including viruses,⁴⁰ group A β -hemolytic *Streptococcus*,⁴¹ pneumococci,⁴² staphylococci,⁴³ *Klebsiella*,⁴³ *Pseudomonas*,⁴⁴ and *Candida*.⁴⁵ The introduction of the conjugated *Haemophilus influenzae* type B vaccine dramatically decreased the incidence of supraglottitis in children < 5 years old, from 41 cases per 100,000 children in 1987 to 1.3 per 100,000 in 1997 (Fig. 4).⁴⁶ Despite that significant decrease in the number of cases, supraglottitis has not been completely eliminated and requires a high index of suspicion for the diagnosis, particularly since

organisms other than *Haemophilus influenzae* type B currently account for a greater percentage of cases in the immunized population, and the disease may present with atypical features.³⁹

Children with supraglottitis classically present with high fever, irritability, throat pain, and extrathoracic airway obstruction, with signs of respiratory distress. These symptoms show rapid progression, generally evolving in a matter of hours. The affected child is often described as toxic-appearing and anxious, preferring to rest in the tripod position (upright sitting position leaning forward and supported by both hands, with the chin up and an open mouth) in order to maximize airway diameter. Blackstock et al⁴⁷ described the "4 Ds" of supraglottitis: drooling, dyspnea, dysphagia, and dysphonia. Unfortunately, not all signs and symptoms are necessarily present in all cases.³⁷ Most patients, however, will have some degree of difficulty handling their secretions because of severe odynophagia. Speech may also be altered by pain or soft tissue swelling, and the voice may sound muffled. Stridor is a late finding and indicates impending complete airway obstruction. The white blood cell count is usually elevated, with neutrophilic predominance and a left shift. Concomitant infections, such as otitis media, cellulitis, pneumonia, or meningitis, are present approximately 50% of the time.³

The diagnosis of supraglottitis is made by directly inspecting the supraglottic region, a procedure that should be performed under optimal and controlled conditions in the operating room. Attempts to directly visualize the posterior pharynx and epiglottis during the initial examination in the emergency department should be strongly discouraged. The concern is that this type of manipulation can increase the risk of complete airway obstruction by subjecting the involved area to local trauma and by creating further emotional distress in the patient. When the diagnosis of supraglottitis is suggested by history and physical examination, a physician capable of controlling the difficult airway should monitor the patient at all times, including during stages of the evaluation that take place outside of the emergency department, such as in the radiology suite. All anxiety-provoking procedures should be postponed or abandoned altogether, including intraoral examination and phlebotomy. In fact, the child should be allowed to stay in the most comfortable position, usually held by a parent, until enough information is obtained to rule out or establish the diagnosis. Radiographs are useful because they can confirm the diagnosis as well as rule out the presence of other conditions such as croup, retropharyngeal abscess, or foreign body. The single best exposure is a lateral neck radiograph obtained with hyperextension during inspiration (Fig. 5). The classic findings include a round and thick epiglottis (thumb sign), loss of the val-

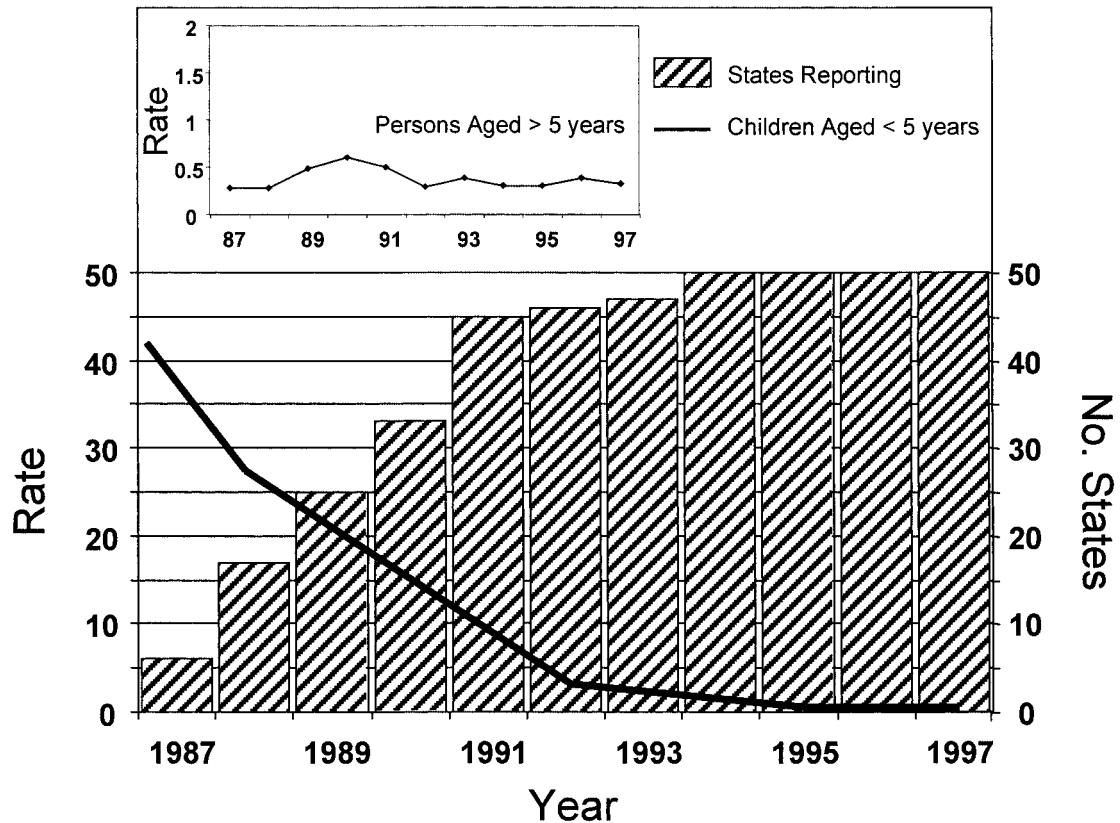


Fig. 4. Incidence of *Haemophilus influenzae* invasive disease among children < 5 years old and persons > 5 years old. Vertical bars represent the number of states reporting *Haemophilus influenzae* surveillance data in the United States, 1987–1997. Rates are per 100,000 persons. (Adapted from Reference 46.)

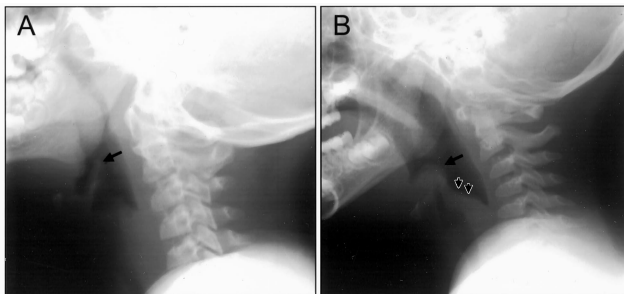


Fig. 5. Panel A: Normal lateral neck radiograph showing a thin epiglottis (arrow). Panel B: Lateral neck radiograph of a patient with supraglottitis, showing loss of cervical lordosis, enlarged epiglottis (thumb sign, arrow), and thickened aryepiglottic folds (arrowheads).

lecular air space, and thickening of the aryepiglottic folds.

The cornerstone in the treatment of a child with supraglottitis is obtaining and maintaining an adequate airway. Whenever supraglottitis is suspected, arrangements should be made for emergency endoscopy in the operating suite. Proper equipment, such as adequately sized endotracheal tubes, rigid bronchoscopes, and tracheotomy supplies, must be available. Spontaneous breathing is preferred, and once

anesthesia is achieved, the supraglottic structures should be directly visualized. Typical findings include edema and erythema of the supraglottic structures, including the epiglottis, arytenoids, and aryepiglottic folds.⁴⁸ An orotracheal tube should be placed and specimens obtained from the supraglottic region for culture and sensitivity. Blood samples should also be obtained at this time for hemogram and cultures. Whenever possible, the orotracheal tube should be changed to a nasotracheal tube because of the greater stability of a nasotracheal tube. Occasionally, the older patient with epiglottitis may not require intubation, and may be managed successfully with supplemental oxygen, antibiotics, and very close monitoring in the intensive care setting.⁴⁹

Once the airway is secured and cultures are obtained, the patient should be started on antibiotics and transferred to the intensive care unit. Until culture and sensitivity results from blood and supraglottic specimens are available, the child should be treated with broad-spectrum antibiotics effective against β -lactamase-producing *Haemophilus influenzae*. Second- or third-generation cephalosporins, such as cefuroxime, ceftriaxome, and ampicillin/sulbactam, are reasonable alternatives. Continued

antibiotic treatment should be adjusted based on cultures and sensitivities.

The patient should be kept intubated until there is evidence of clinical improvement and the development of an audible air leak around the endotracheal tube. Alternatively, examination of the supraglottic area via flexible laryngoscope is often adequate to assess clinical progress. The mean duration of intubation ranges between 30 and 72 hours,^{36,49} with some clinicians advocating the use of dexamethasone to reduce the incidence of post-extubation stridor.⁵⁰

Bacterial Tracheitis

Bacterial tracheitis, also known as bacterial laryngotracheobronchitis or pseudomembranous croup, was first described in detail by Jones et al in 1979.⁵¹ This is a rare disease, with a peak incidence during fall and winter months,^{51,52} predominantly in children between the ages of 6 months and 8 years (mean age of 5 years).^{51,52} This condition is characterized by marked subglottic edema and thick mucopurulent (membranous) secretions. The most common pathogen is *Staphylococcus aureus*, although several others have been implicated, including *Haemophilus influenzae*, α -hemolytic streptococcus, pneumococcus, and *Moraxella catarrhalis*.⁵¹⁻⁵³

The clinical presentation of bacterial tracheitis is generally more insidious than that of supraglottitis, with the typical patient presenting with a several-day history of viral upper-respiratory symptoms such as low grade fever, cough, and stridor (similar to LTB). This is followed by a period of rapid deterioration, when the patient develops a high fever, toxic appearance, and evidence of airway obstruction. These patients are generally more toxic than those with LTB. They often differ from those with supraglottitis, because patients with bacterial tracheitis have a substantial cough, appear comfortable when flat, and tend not to drool.⁵³ The hemogram is marked by polymorphonuclear leukocytosis, often with a left shift. Radiographically, bacterial tracheitis may be indistinguishable from LTB, with the neck radiograph showing marked subglottic narrowing (steeple sign). In some cases, the tracheal air column may appear hazy, with the presence of multiple intraluminal irregularities representing pseudomembrane detachment from soft tissue.⁴⁸ Another clue to the diagnosis is a patient who appears more ill than the usual case of LTB, while failing to respond to conventional LTB treatment such as racemic epinephrine and corticosteroids.^{52,53} Patients with evidence of severe respiratory distress should be diagnosed in the operating room, in the same fashion as those with supraglottitis. The accepted standard for the diagnosis of bacterial tracheitis is the endoscopic finding of subglottic edema with ulcerations, er-

ythema, and pseudomembrane formation in the trachea, in conjunction with a positive bacterial culture.

Treatment consists of endotracheal intubation to maintain a patent airway, antibiotics, and endoscopic removal of secretions and dead tissue from the airway lumen. Broad-spectrum antibiotics should be used, considering the need to cover against *Staphylococcus aureus*. A reasonable initial combination is oxacillin and ceftriaxone, but definitive antibiotic coverage should be guided by the Gram-stain results, cultures, and sensitivities. Antibiotic treatment is generally continued for 10–14 days, and intubation is often required for 3–7 days. The decision to extubate should be based on clinical improvement, as assessed by defervescence, decreased airway secretions, and the development of an air leak around the endotracheal tube.

Retropharyngeal Abscess

Retropharyngeal abscesses are serious infections of the normally sterile retropharyngeal space. Under normal conditions, this space contains loose connective tissue and lymph nodes that drain the nasopharynx, paranasal sinuses, middle ear, teeth, and adjacent bones. Retropharyngeal abscesses generally result from lymphatic spread of infection, although direct spread from contiguous areas, trauma, or foreign bodies can also play a role. Retropharyngeal abscesses are more common in young children,⁵⁴ with the vast majority of cases occurring in patients younger than 6 years of age.^{55,56} This may be due to the fact that retropharyngeal lymph nodes are abundant in young children but suffer progressive involution and atrophy in older patients.

The typical presentation of a retropharyngeal abscess is often nonspecific. Considerable clinical overlap with several other conditions, such as croup, epiglottitis, tracheitis, and peritonsillar abscess, is frequently observed. The diagnosis of meningitis can be incorrectly entertained with a patient who has a retropharyngeal abscess, because of the presence of a stiff and tender neck caused by irritation of the pre-vertebral soft tissue, thus simulating meningeal irritation signs. Presenting symptoms are usually vague, with most patients showing evidence of a viral upper respiratory infection for several days prior to worsening of the clinical picture. The nonspecific symptoms give rise to high fever, sore throat, poor feeding, and neck stiffness.^{54,57} Further progression of the disease process is marked by evidence of extrathoracic airway compromise, including drooling, stridor, and respiratory distress. In some patients a retropharyngeal mass may be seen during oral examination.^{54,57} Forceful introduction of tongue depressors in repeated attempts to visualize this finding should be discouraged because of the risk of abscess rupture resulting in aspiration of purulent material.



Fig. 6. Lateral neck radiograph of a patient with a retropharyngeal abscess showing a widened retropharyngeal space and air pockets (arrowheads) within the soft tissue.

The diagnosis can be confirmed by the typical appearance of the inspiratory lateral neck radiograph in full extension (Fig. 6). Abnormally increased thickness of the pre-vertebral soft tissue in the proper clinical context indicates an infection of the retropharyngeal space. The presence of gas or air fluid levels within the retropharyngeal space, as well as foreign bodies and the loss of normal cervical lordosis, are other important clues potentially obtained from the lateral neck radiograph. Computed axial tomography plays a major role in diagnosing retropharyngeal abscesses, as it is useful in defining the precise anatomical extension and differentiating a true abscess from cellulitis. Blood cultures are generally negative in patients with retropharyngeal abscesses, although the white blood cell count and erythrocyte sedimentation rate are often elevated. Culture of the abscess material usually yields a polymicrobial flora including *Staphylococcus aureus*, various streptococcal species, and anaerobes.^{56,58}

The first line of treatment is antibiotic therapy, which alone is effective in approximately 25% of cases.⁵⁹ In cases refractory to initial therapy, drainage of the abscess is indicated. Careful endotracheal intubation followed by intraoral surgical drainage is considered the standard of care in most institutions. The use of computed-tomography-guided needle aspiration has been reported as an alternative to surgical drainage, with the advantage of causing less trauma to surrounding tissues and the possibility of avoiding general anesthesia.⁵⁴ Regardless of the mode

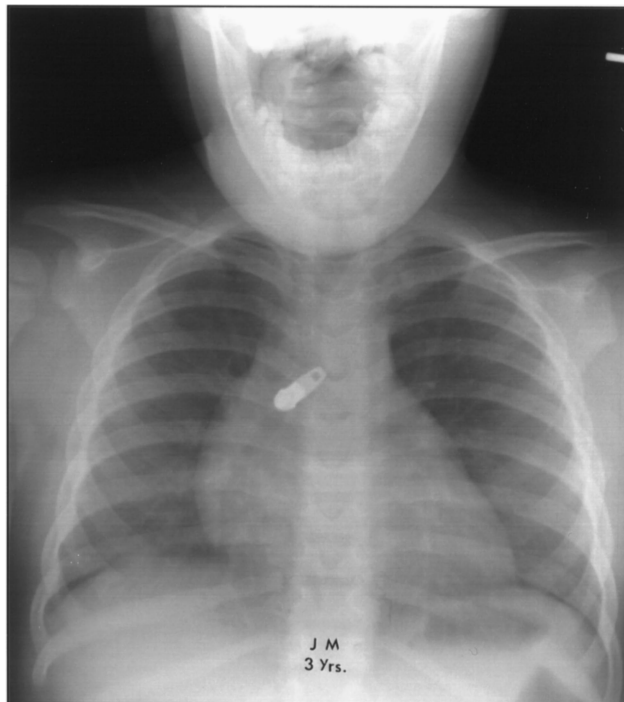


Fig. 7. Frontal radiograph of a patient with a metallic foreign body lodged in the right main bronchus.

of drainage chosen, great care should be exercised during the instrumentation to avoid aspiration of purulent or infectious material into the airway.

Foreign Bodies

Foreign body aspiration or ingestion can lead to partial obstruction of the airway or a catastrophic complete obstruction that can lead to death in a matter of minutes. The victim of an airway obstruction caused by a foreign body is generally asymptomatic prior to the event. Airway compression or obstruction by the foreign body lead to the acute onset of symptoms. Clinical symptoms and signs include coughing fits and increased respiratory difficulty, such as the use of accessory muscles, nasal flaring, stridor, or wheezing, depending on the anatomical location and severity of the obstruction. A foreign body lodged in the extrathoracic airway typically causes inspiratory or biphasic stridor. An intrathoracic foreign body is associated with expiratory or biphasic wheezing. The clinical presentation of these types of airway obstructions may be complicated by the fact that signs and symptoms will change, depending on the location of the foreign body and whether it migrates up or down the airway over time. For instance, a toddler may be very symptomatic after a choking spell with severe stridor and retractions due to a subglottic foreign body, only to have no evidence of respiratory distress and mild expiratory wheezing when the object moves into

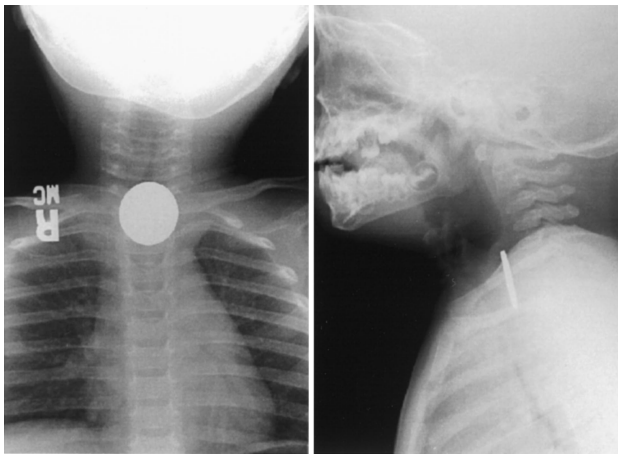


Fig. 8. Frontal and lateral neck radiographs of a child who presented with inspiratory stridor and drooling, showing a metallic disk (coin) lodged in the thoracic inlet. The coronal orientation of the foreign body is typical of an esophageal position. This impression is confirmed by the obvious esophageal position of the foreign body on the lateral neck radiograph.

the right main bronchus (Fig. 7). Signs and symptoms of airway obstruction are not exclusive of airway foreign bodies. An ingested object that is unable to progress down the esophagus can cause inflammation and compression of the posterior wall of the trachea and lead to a clinical picture almost indistinguishable from that of a foreign body lodged in the airway (Fig. 8).

Radiopaque foreign bodies can be easily diagnosed by radiograph. Frontal and lateral views of the neck and chest should be used to further localize the object to the esophagus or airway. Flat foreign bodies, such as coins, that enter the esophagus tend to assume a coronal orientation, owing to the fact that the narrowest diameter of the esophageal lumen at rest is antero-posterior. Esophageal foreign bodies will generally obstruct in one of 3 sites: immediately distal to the pyriform sinus, at the level of the aortic arch, or at the cardia. The diagnosis of a nonradiopaque foreign body poses a much greater challenge. Intrathoracic foreign bodies located beyond the carina may be diagnosed by dynamic radiographic (fluoroscopic) examination or by the use of inspiratory and expiratory radiographs showing asymmetric pulmonary aeration. Young children unable to cooperate with instructions to inspire and forcefully exhale can be studied in the right and left lateral decubiti, since the dependent side simulates the lung aspect during exhalation. A patient who has a highly suggestive clinical picture should undergo a diagnostic and therapeutic endoscopy, despite a negative radiograph.

A patient who has partial airway obstruction and who is spontaneously breathing should be given 100% supplemental oxygen and be kept calm. A physician skilled in airway intervention should accompany the patient at all times, including time spent in the radiology department or

in transport to the operating suite, since a partially obstructive foreign body can move and provoke an acute obstruction and respiratory arrest. Complete airway obstruction must be treated as an emergency, with the goal of reestablishing a patent airway and respiratory flow. Unconscious infants with witnessed or highly suspected foreign body airway obstruction should be straddled over the arm of the rescuer with the head lower than the trunk. Five back blows should be delivered with the heel of the hand between the shoulder blades, followed by turning the patient and delivering 5 chest thrusts in the same manner as external chest compressions are performed.⁶⁰ Blind finger sweeps should generally be avoided in infants and children, as they may force the foreign body further down the airway. Older children and adults with foreign body airway obstructions should be given the Heimlich maneuver: a series of subdiaphragmatic thrusts with the purpose of forcing air out of the lungs in what can be described as an “artificial cough.”^{61,62} With an unconscious adult patient, a finger-sweep maneuver can be performed by introducing the index finger deep into the throat in a “hooking” motion in attempt to retrieve the foreign body. Regardless of the size of the patient, direct visualization of an airway foreign body during emergency intubation may allow for its removal with the use of a Magill forceps or a Kelly clamp.

Inhalational Injuries

Inhalational injuries are frequently associated with major burns in victims of fires in enclosed spaces. In fact, inhalational injuries are responsible for approximately 50–80% of the mortality attributed to burns.^{63–65} Inhalational injuries lead to morbidity and mortality by 3 distinct but frequently overlapping processes: airway thermal burn, asphyxia (carbon monoxide or cyanide poisoning), and pulmonary injury from smoke inhalation.

Thermal injury to the airway by inhalation of hot gas is generally limited to the supraglottic airway. This sparing of the subglottic airway and trachea is related to a protective reflex of vocal cord closure upon exposure to heat.⁶⁶ In addition, air is a poor conductor of heat, which, coupled with the efficient heat exchange characteristics of the upper airway, contributes to the cooling of hot inspired gas before it reaches the lungs. Substantial direct pulmonary injury, however, is known to occur when steam is inhaled. This is because steam has approximately 4,000 times the heat-carrying capacity of dry air⁶⁷ and easily overwhelms the upper airway’s ability to equilibrate the temperature of the inspired gas.

The highest risk for airway thermal injuries occurs in victims of fires in enclosed spaces, patients who lose consciousness at the scene, those with burns to lips or nose, soot in the mouth or nostrils, and the early development of hoarseness, stridor, or respiratory difficulty. Patients with

airway thermal injuries generally develop rapid-onset airway edema and should undergo tracheal intubation early, before the inflammatory reaction progresses to airway obstruction or makes intubation more difficult or impossible. As in other severe obstructive airway diseases, such as supraglottitis, the endotracheal tube should be maintained in place until resolution of the edema and demonstration of an air leak. Serial flexible laryngoscopies can be of use in following the disease process.

Summary

Processes that lead to obstructive respiratory emergencies are common in the pediatric population. Many of these entities can progress rapidly, creating a life-threatening situation. The clinician must maintain a high index of suspicion and make a rapid and precise diagnosis, oftentimes based solely on the patient's brief history or a limited examination. In the setting of obstructive airway emergencies, appropriate treatment needs to be implemented without delay. This generally involves assuring the presence of a patent airway while time, supportive therapies, and (when appropriate) antibiotics contribute to the resolution of the baseline process.

REFERENCES

- Weiner DL. Respiratory distress. In: Fleisher GR, Ludwig S, editors. Textbook of pediatric emergency medicine, 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2000:553–564.
- Lusk RP, Khosla S. Principles of fluid dynamics. In: Holinger LD, Lusk RP, Green CG, editors. Pediatric laryngology and bronchoesophagology. Philadelphia: Lippincott-Raven; 1997:381–391.
- Dunham ME, Holinger LD. Stridor, aspiration and cough. In: Bailey BJ, editor. Head & neck surgery-otolaryngology, vol 1, 2nd ed. Philadelphia: Lippincott-Raven; 1998:1131–1148.
- Zalzal GH. Stridor and airway compromise. *Pediatr Clin North Am* 1989;36(6):1389–1402.
- Skolnik NS. Treatment of croup: a critical review. *Am J Dis Child* 1989;143(9):1045–1049.
- Geelhoed GC, Macdonald WB. Oral and inhaled steroids in croup: a randomized, placebo-controlled trial. *Pediatr Pulmonol* 1995;20(6):355–361.
- Yates RW, Doull IJ. A risk-benefit assessment of corticosteroids in the management of croup. *Drug Saf* 1997;16(1):48–55.
- Cunningham MJ. The old and new of acute laryngotracheal infections. *Clin Pediatr* 1992;31(1):56–64.
- Tan AK, Manoukian JJ. Hospitalized croup (bacterial and viral): the role of rigid endoscopy. *J Otolaryngol* 1992;21(1):48–53.
- Denny FW, Murphy TF, Clyde WA Jr, Collier AM, Henderson FW. Croup: an 11-year study in a pediatric practice. *Pediatrics* 1983;71(6):871–876.
- Westley CR, Cotton EK, Brooks JG. Nebulized racemic epinephrine by IPPB for the treatment of croup: a double-blind study. *Am J Dis Child* 1978;132(5):484–487.
- Myer CM. Inflammatory diseases of the pediatric airway. In: Cotton RT, Myer CM, editors. Practical pediatric otolaryngology. Philadelphia: Lippincott-Raven; 1999:547–559.
- Baugh R, Gilmore BB Jr. Infectious croup: a critical review. *Otolaryngol Head Neck Surg* 1986;95(1):40–46.
- Henrickson KJ, Kuhn SM, Savatski LL. Epidemiology and cost of infection with human parainfluenza virus types 1 and 2 in young children. *Clin Infect Dis* 1997;18(5):770–779.
- Geelhoed GC. Sixteen years of croup in a Western Australian teaching hospital: effects of routine steroid treatment. *Ann Emerg Med* 1996;28(6):621–626.
- Mauro RD, Poole SR, Lockhart CH. Differentiation of epiglottitis from laryngotracheitis in the child with stridor. *Am J Dis Child* 1988;142(6):679–682.
- Swishchuk LE. Upper airway, nasal passages, sinuses, and mastoids. In: Swishchuk LE, editor. Emergency radiology of the acutely ill and injured child, 2nd ed. Baltimore: Williams & Wilkins; 1986:127–140.
- Bourchier D, Dawson KP, Fergusson DM. Humidification in viral croup: a controlled trial. *Aust Paediatr J* 1984;20(4):289–291.
- Gardner HG, Powell KR, Roden VJ, Cherry JD. The evaluation of racemic epinephrine in the treatment of infectious croup. *Pediatrics* 1973;52(1):52–55.
- Kristjansson S, Berg-Kelly K, Winso E. Inhalation of racemic adrenaline in the treatment of mild and moderately severe croup: clinical symptom score and oxygen saturation measurements for evaluation of treatment effects. *Acta Paediatr* 1994;83(11):1156–1160.
- Waisman Y, Klein BL, Boenning DA, Young GM, Chamberlain JM, O'Donnell R, Ochsenschlager DW. Prospective randomized double-blind study comparing L-epinephrine and racemic epinephrine aerosols in the treatment of laryngotracheitis (croup). *Pediatrics* 1992;89(2):302–306.
- Landau LI, Geelhoed GC. Aerosolized steroids for croup. *N Engl J Med* 1994;331(5):322–323.
- Kelley PB, Simon JE. Racemic epinephrine use in croup and disposition. *Am J Emerg Med* 1992;10(3):181–183.
- Prendergast M, Jones JS, Hartman D. Racemic epinephrine in the treatment of laryngotracheitis: can we identify children for outpatient therapy? *Am J Emerg Med* 1994;12(6):613–616.
- Ledwith CA, Shea LM, Mauro RD. Safety and efficacy of nebulized racemic epinephrine in conjunction with oral dexamethasone and mist in the outpatient treatment of croup. *Ann Emerg Med* 1995;25(3):331–337.
- Martensson B, Nilsson G, Torbjar J-E. The effect of corticosteroids in the treatment of pseudo-croup. *Acta Otolaryngol Suppl* 1968;158:62–71.
- Kuusela AL, Vesikari T. A randomized double-blind, placebo-controlled trial of dexamethasone and racemic epinephrine in the treatment of croup. *Acta Paediatr Scand* 1988;77(1):99–104.
- Super DM, Cartelli NA, Brooks LJ, Lembo RM, Kumar ML. A prospective randomized double-blind study to evaluate the effect of dexamethasone in acute laryngotracheitis. *J Pediatr* 1989;115(2):323–339.
- Klassen TP, Feldman ME, Watters LK, Sutcliffe T, Rowe PC. Nebulized budesonide for children with mild-to-moderate croup. *N Engl J Med* 1994;331(5):285–289.
- Johnson DW, Jacobson S, Edney PC, Hadfield P, Mundy ME, Schuh S. A comparison of nebulized budesonide, intramuscular dexamethasone, and placebo for moderately severe croup. *N Engl J Med* 1998;339(8):498–503.
- Klassen TP, Craig WR, Moher D, Osmond MH, Pasterkamp H, Sutcliffe T, et al. Nebulized budesonide and oral dexamethasone for treatment of croup: a randomized controlled trial. *JAMA* 1998;279(20):1629–1632.
- Cressman WR, Myer CM 3rd. Diagnosis and management of croup and epiglottitis. *Pediatr Clin North Am* 1994;41(2):265–276.
- Stroud RH, Friedman NR. An update on inflammatory disorders of the pediatric airway: epiglottitis, croup, and tracheitis. *Am J Otolaryngol* 2001;22(4):268–275.
- McGee DL, Wald DA, Hinchliffe S. Helium-oxygen therapy in the emergency department. *J Emerg Med* 1997;15(3):291–296.

35. Rosenfeld RM, Fletcher MA, Marban SL. Acute epiglottitis in a newborn infant. *Pediatr Infect Dis J* 1992;11(7):594–595.
36. Baines DB, Wark H, Overton JH. Acute epiglottitis in children. *Anaesth Intensive Care* 1985;13(1):25–28.
37. Losek JD, Dewitz-Zink BA, Melzer-Lange M, Havens PL. Epiglottitis: comparison of signs and symptoms in children less than 2 years old and older. *Ann Emerg Med* 1990;19(1):55–58.
38. Brilli RJ, Benzing G 3rd, Cotcamp DH. Epiglottitis in infants less than two years of age. *Pediatr Emerg Care* 1989;5(1):16–21.
39. Senior BA, Radkowski D, MacArthur C, Sprecher RC, Jones D. Changing patterns in pediatric supraglottitis: a multi-institutional review, 1980 to 1992. *Laryngoscope* 1994;104(11 Pt 1):1314–1322.
40. Grattan-Smith T, Forer M, Killham H, Gillis J. Viral supraglottitis. *J Pediatr* 1987;110(3):434–435.
41. Glenn GM, Schofield T, Krober M. Group A streptococcal supraglottitis. *Clin Pediatr* 1990;29(11):674–676.
42. Crysedale WS, Sendi K. Evolution in the management of acute epiglottitis: a 10-year experience with 242 children. *Int Anesthesiol Clin* 1988;26(1):32–38.
43. Murrage KJ, Janzen VD, Ruby RR. Epiglottitis: adult and pediatric comparisons. *J Otolaryngol* 1988;17(4):194–198.
44. Lacroix J, Gauthier M, Lapointe N, Ahronheim G, Arcand P, Girouard G. *Pseudomonas aeruginosa* supraglottitis in a six-month-old child with severe combined immunodeficiency syndrome. *Pediatr Infect Dis J* 1988;7(10):739–741.
45. Haberman RS, Becker ME, Ford CN. *Candida* epiglottitis. *Arch Otolaryngol* 1983;109(11):770–771.
46. Progress toward eliminating *Haemophilus influenzae* type b disease among infants and children—United States, 1987–1997. *MMWR Morb Mortal Wkly Rep* 1998;47(46):993–998.
47. Blackstock D, Adderley RJ, Steward DJ. Epiglottitis in young infants. *Anesthesiology* 1987;67(1):97–100.
48. Duncan NO, Sprecher RC. Infections of the airway. In: Cummings CW, editor. *Otolaryngology-head & neck surgery*, 3rd ed. St Louis: Mosby; 1998:388–400.
49. Loos GD. Pharyngitis, croup, and epiglottitis. *Prim Care* 1990 17(2):335–345.
50. Schroeder LL, Knapp JF. Recognition and emergency management of infectious causes of upper airway obstruction in children. *Semin Respir Infect* 1995;10(1):21–30.
51. Jones R, Santos JI, Overall JC Jr. Bacterial tracheitis. *JAMA* 1979;242(8):721–726.
52. Liston SL, Gehrz RC, Siegel LG, Tilelli J. Bacterial tracheitis. *Am J Dis Child* 1983;137(8):764–767.
53. Donaldson JD, Maltby CC. Bacterial tracheitis in children. *J Otolaryngol* 1989;18(3):101–104.
54. Broughton RA. Nonsurgical management of deep neck infections in children. *Pediatr Infect Dis J* 1992;11(1):14–18.
55. Coulthard M, Isaacs D. Retropharyngeal abscess. *Arch Dis Child* 1991;66(10):1227–1230.
56. Yeoh LH, Singh SD, Rogers JH. Retropharyngeal abscesses in a children's hospital. *J Laryngol Otol* 1985;99(6):555–566.
57. Thompson JW, Cohen SR, Reddix P. Retropharyngeal abscess in children: a retrospective and historical analysis. *Laryngoscope* 1988;98(6 Pt 1):589–592.
58. Asmar BI. Bacteriology of retropharyngeal abscess in children. *Pediatr Infect Dis J* 1990;9(8):595–597.
59. Morrison JE Jr, Pashley NR. Retropharyngeal abscesses in children: a 10-year review. *Pediatr Emerg Care* 1988;4(1):9–11.
60. Day RL, Crelin ES, DuBois AB. Choking: the Heimlich abdominal thrust vs back blows: an approach to measurement of inertial and aerodynamic forces. *Pediatrics* 1982;70(1):113–119.
61. Heimlich HJ. A life-saving maneuver to prevent food-choking. *JAMA* 1975;234(4):398–401.
62. Heimlich HJ, Hoffmann KA, Canestri FR. Food-choking and drowning deaths prevented by external subdiaphragmatic compression: physiological basis. *Ann Thorac Surg* 1975;20(2):188–195.
63. Heimbach DM, Waeckerle JF. Inhalation injuries. *Ann Emerg Med* 1988;17(12):1316–1320.
64. Young CJ, Moss J. Smoke inhalation: diagnosis and treatment. *J Clin Anesth* 1989;1(5):377–386.
65. Smith DL, Cairns BA, Ramadan F, Dalston JS, Fakhry SM, Rutledge R, et al. Effect of inhalation injury, burn size, and age on mortality: a study of 1447 consecutive burn patients. *J Trauma* 1994;37(4):655–659.
66. Moritz AR, Henriques FC, McLean R. The effects of inhaled heat on the air passages and lungs: an experimental investigation. *Am J Pathol* 1945;21:311.
67. Weiss SM, Lakshminarayan S. Acute inhalation injury. *Clin Chest Med* 1994;15(1):103–116.

Discussion

Anderson: Alex, part of your and my job is to take the transport calls from referring facilities, and the topic of epiglottitis comes up about twice a month. A well-meaning emergency physician refers a kid who does not have all the classic symptoms, but maybe a fever, a little drooling, and the most overcalled thumb sign that we see month after month—“Oh my God; he’s got epiglottitis.” If it is epiglottitis, they go to the operating room, get anesthesia, and get intubated, but now we have “gray area” situations where we’re not exactly sure what ad-

vice to give to the referring facility or to the transport team member. What advice do you give to our colleagues?

Rotta: That’s certainly a critical point, because as the disease becomes more rare and more atypical, we will tend to become more complacent and think of epiglottitis a disease of the past. The reality is that we still see cases of epiglottitis and they may be very atypical. I think that there are several ways to approach this, and there is not one right or wrong way. The approaches range from high-tech to simple. We are connected to some institutions that share the same radio-

graphic Web-based service that we use in our hospital, through which it is simple to look at a CT scan or, in the case of epiglottitis, a lateral neck radiograph, and give them our impression of whether that’s a compatible diagnosis as far as the radiograph can reveal.

Now, having said that, in the era of atypical epiglottitis, a large number of those patients will have atypical radiographs, so the radiograph is not a definitive discriminator. So the advice we give depends on factors such as how much do you trust the other person’s assessment and observations of the patient, because that person is your

eyes and ears. When we don't have a good feeling about what's happening on the other end of the line, our approach has been to respect the diagnosis from the referring institution and recommend that they go through the steps of diagnosing and treating epiglottitis, as we would in our institution, such as taking the patient to the operating room and having an ear, nose, and throat specialist look at the epiglottis and supraglottic area. If that is not available at that referring institution, we fly our team out there. We've flown a critical care attending and ear, nose, and throat attending to a couple of hospitals to gain control of a critical situation when epiglottitis was seriously in question. Now, this is certainly not a case where you would send a first-year fellow on transport who has never dealt with a case of epiglottitis.

When the referring physician says, "We think that this patient has epiglottitis but he is very stable and looks very good; we just need somebody else to take a look down the airway," we will send a transport team to bring this patient back, not necessarily securing the airway before leaving, which may sound preposterous, but the reality is that with the changing characteristics of epiglottitis, there are a number of patients in the older age group who will be managed throughout the entire disease process without intubation.

This is a disease that is changing, and we're still trying to figure out where and how to deal with some of the particular points. But, certainly, I think they will vary widely on the technology (such as Web-based radiography and telemedicine) and on your trust of the referring institution, and that institution's ability to perform full-service airway establishment.

Myers: My question moves a little bit out of the emergency room setting. *RESPIRATORY CARE* recently published a survey study of critical care fellowship directors' use of the endotracheal

tube air leak test (that is, air leaking around the tube's cuff) and those doctors' assessment of the value of that test. What's your opinion on what the leak test actually tells us or indicates what to do in an airway-obstructed patient?

REFERENCE

1. Foland JA, Super DM, Dahdah NS, Mhanna MJ. The use of the air leak test and corticosteroids in intubated children: a survey of pediatric critical care fellowship directors. *Respir Care* 2002;47(6):662-666.

Rotta: The leak test has a somewhat limited use with a patient who gets intubated for pulmonary causes. However, I would place more value on the development of an air leak in a patient who was intubated because of an acute, severe narrowing of an exothoracic airway, particularly if the air leak was not present when the endotracheal tube was originally placed. I would view the development of an air leak as improvement of the reversible airway obstruction.

I saw a patient with croup who was intubated, developed an air leak, and was extubated, but then developed very severe post-extubation stridor. In that case an air leak wasn't a good predictor of the patient being ready for extubation, and that person limped along for about 12 hours after extubation. So I think it's a valid sign of progress in the resolution of reversible edema. How much weight to put on that and the actual numbers are something I do not have right now.

Hansell: How do you handle a situation where a face mask is going to further upset a child suffering croup? Do you use the aerosol blow-by technique? From the practical perspective of the therapist who's trying to administer a treatment, it gets confounding. Also, do you routinely give steroids to kids who've had acute exacerbations and inflamed airways prior to extubation? How do you evaluate whether to give steroids prior to extubation?

Rotta: The first question relates to some thing that is part of the job of the emergency medicine physician: to serve as a mediator between therapies or strategies that do not always have the same common interest, although they may have the same common goal. In this case, that goal would be providing the best delivery of drug to a child who could potentially benefit from it, such as nebulized epinephrine for croup, while considering that the down side of doing so may be increasing anxiety and crying in a patient who already has an obstructive airway.

Our routine is to attempt to deliver the nebulized treatment via face mask, because it provides greater drug deposition. Occasionally, we still see aerosol delivery with the blow-by method. Now, one could argue that the deposition of medication to the area of interest is negligible with blow-by if the patient is screaming or crying.¹ Some would even say that the blow-by technique is completely ineffective,² underscoring what I just mentioned about the superiority of nebulization via face mask. However, in clinical medicine we sometimes have to make compromises. In cases where the pediatric patient is so upset or threatened by the application of a tight face mask, to the point of potentially suffering acute deterioration, of course we will try to promote a less threatening environment while attempting to reach the therapeutic goal, even if this means using the less desirable blow-by technique.

Regarding steroids before extubation, we routinely use steroids before extubation of patients who have been intubated for longer than 48 hours. That's largely based on the study by Anene et al of patients who are at relatively high risk for post-extubation stridor and failure.³ That study considered a broad subgroup of patients, not trying to exclude those who were at high risk for severe obstruction or extubation failure, such as those with pre-existing malformations (as was the case with many studies of post-extu-

bation stridor), and the evidence indicated that the use of steroids was associated with less stridor, lower obstruction scores, and less need for reintubation.

REFERENCES

1. Everard ML, Clark AR, Milner AD. Drug delivery from jet nebulisers. *Arch Dis Child* 1992;67(5):586-591.
2. Hess DR. Liquid Nebulization: emerging Technologies. Conference summary. *Respir Care* 2002;47(12):1471-1476.
3. Anene O, Meert KL, Uy H, Simpson P, Sarnaik AP. Dexamethasone for the prevention of post-extubation airway obstruction: a prospective, randomized, double-blind, placebo-controlled trial. *Crit Care Med* 1996; 24(10):1666-1669.

Wagener: If you have to use blow-by nebulized medication, you can compensate for the 80% decreased delivery by 5-fold increasing the dose. Thus you may have to treat more frequently or with higher doses, but it doesn't mean that blow-by doesn't work; it just means that it's less effective.

Concerning croup versus epiglottitis, I'm of the old school and rely primarily on history and physical examination for diagnosis. I have always thought that, generally, if the patient is coughing, the diagnosis is croup. In the patient who has snoring and a low-pitch stridor, I think supraglottitis. I also don't believe there is a place for radiographic diagnosis in a child with substantial respiratory distress: those patients first need an airway. A question about the croup patient who gets

intubated is, should he be kept on steroids while he is intubated or do you take him off steroids and start the steroids again before extubation?

Rotta: Our approach has been to keep those patients on steroids, and that is extrapolating from the literature that says that steroids benefit the unintubated patient.¹ We also factored in the idea that intubation for croup tends to be short intubation, so giving a 48-hour course of steroids generally coincides with priming those patients for extubation within a day or two. We've been burned on that approach with the occasional patient who gets intubated and is still intubated after 5 days, and is still on steroids, and now you have a patient who has received a much more prolonged course than you originally planned on. But those are the exceptions, and though I don't have any numbers to quote to you, those atypical patients generally had pre-existing airway risk such as pre-existing intubations and are patients who are later found to have airway hemangioma, subglottic stricture, or some airway comorbidity.

REFERENCE

1. Super DM, Cartelli NA, Brooks LJ, Lembo RM, Kumar ML. A prospective randomized double-blind study to evaluate the effect of dexamethasone in acute laryngotracheitis. *J Pediatr* 1989;115(2):323-329.

Kercsmar: A recent report in the *Journal of Pediatrics* made the com-

ment that parapharyngeal abscesses and retropharyngeal abscesses have become the new epiglottitis and that, in an era of a vaccinated population and in the absence of high-risk populations who do not respond to vaccination, you should consider those other conditions before epiglottitis.¹ I don't think we see much real epiglottitis anymore, but that retropharyngeal and parapharyngeal abscesses are out there, and we see them. Maybe that's where the emphasis should be with referrals and with teaching—that these are the things we need to look for, that they have not gone away, and, if anything, have increased in prevalence. I wonder if that's your experience.

REFERENCE

1. Lee SS, Schwartz RH, Bahadori RS. Retropharyngeal abscess: epiglottitis of the new millennium. *J Pediatr* 2001;138(3):435-437.

Rotta: I agree with that. I don't think we're seeing more retropharyngeal abscess, I just think they stand out more now that we don't see many cases of epiglottitis. In fact, retropharyngeal abscess was not originally supposed to be part of this presentation, but I added it because I think that, in the whole scheme of importance, it is right now the most important infectious obstructive airway disease, and that one needs to have a high index of suspicion in the emergency department and then deal with it in the intensive care unit.