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"It's only a sore throat," the triage nurse comments as she slides the patient's chart into the rack. You take a quick glance at the triage note and put the chart at the back of the stack, turning your attention to charts with "more serious" initial diagnoses: chest pain, abdominal pain, dislocated shoulder, and others. Unfortunately, something went wrong, because 24 hours later you find yourself explaining to the hospital risk manager why the patient with the sore throat is now in the intensive care unit (ICU), intubated, and is not doing well.

The fact is, many emergency physicians are somewhat jaded by the healthy appearance of the vast majority of patients who enter the department with a triage note indicating that "sore throat" is the patient's chief complaint. However, because triage is an inexact science, potentially life-threatening conditions will not always present to you in distinctly labeled packages such as retropharyngeal abscess, epiglottitis, or diphtheria, but simply as a "sore throat," or perhaps "pharyngitis," "upper respiratory infection," or "flu syndrome."

The point is, a sore throat may be the hallmark of some of the most serious or life-threatening conditions encountered by emergency physicians. (See Table 1.) The category loosely labeled as "sore throat" includes infections such as epiglottitis, tracheitis, croup, and diphtheria, as well as several entities best characterized as deep neck abscesses. Although these infectious conditions are not common, they are not rare either. It should be emphasized that even when the initial disease symptoms are mild, these patients can deteriorate rapidly—making these conditions deceivingly treacherous.

To prevent misdiagnosis and optimize outcomes in patients who have serious underlying causes of their sore throats, this issue reviews life-threatening causes of sore throat, emphasizing physical and radiological findings that confirm a specific diagnosis. Management strategies are presented in detail for heat-of-battle application.

— The Editor

"Killer" Sore Throat: Prompt Detection and Management of Serious and Potentially Life-Threatening Causes of Pharyngeal Pain

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General Principles

Anatomy. Because of the potentially high morbidity and mortality rates associated with pharyngeal and parapharyngeal infections, the emergency physician must cultivate an excellent understanding of the anatomy of respiratory tract, from larynx to bronchus. From a clinical, anatomical perspective, the respiratory tract from the larynx to the bronchus is composed of connective tissue, cartilage, muscle, and mucosa. The neck contains several

potential spaces and fascial planes. Infection in any of these compartments can spread rapidly. Extension and complications can include or be caused by swelling that surrounds and impinges on the airway, the great vessels, and/or the lower cranial nerves. Because spaces in the neck communicate with the mediastinum, contiguous spread also can produce massive infections in the chest.

The upper airway consists of the nasal cavity, the oral cavity, and the pharynx. In supraglottitis, the pharynx is the site of infection. The pharynx is a muscular tube that extends from the soft palate to the esophagus and the trachea. It contains the nasopharynx, oropharynx, and juncture of pharynx and larynx. Anteriorly, the pharynx contains the epiglottis, the arytenoid cartilages, and the cricoid cartilage. The hypopharynx extends from the hyoid bone to the esophagus and trachea.

The larynx consists of the thyroid cartilage, cricoid cartilage, proximal trachea, vocal cords, and arytenoid folds. The larynx maintains airway patency, protects the airway when swallowing, and provides the vocal mechanisms. The main cartilage of the larynx is the thyroid cartilage. Inferior to the thyroid cartilage is the cricoid cartilage. The glottic opening is the space between the vocal cords; it is the narrowest part of the adult patient's airway.

The supraglottic area is defined anteriorly by the epiglottis, laterally by the arytenoid folds, and posteriorly by the interarytenoid folds. The surface of the larynx contains the superior laryngeal branch of the vagus nerve; it is the major motor nerve of the larynx. Disturbance of this nerve causes laryngeal spasm and may contribute to autonomic activity during intubation.

Microbiology. The most common pathologic organisms found in the oropharynx and airway are group A, beta-hemolytic streptococci and *Staphylococcus aureus*. Many abscesses are polymicrobial and include both gram-positive and gram-negative aerobic and anaerobic organisms. The most common of these are the *Bacteroides* species. Group A streptococci and oral anaerobes are the most common organisms found in the peritonsillar and retropharyngeal abscesses. Retropharyngeal cellulitis may progress to a retropharyngeal abscess. Additional causes of morbid "sore throats" include infections such as tuberculosis, AIDS, mononucleosis, and cat scratch disease.

Pharyngitis

Many patients with the diagnosis of sore throat, who may have potentially lethal complications, present to the emergency department (ED) with the "triage diagnosis" of pharyngitis. It is appropriate to briefly discuss this baseline diagnosis. Pharyngitis is caused by an infection or irritation of the pharynx and tonsils. It is rarely found in infants younger than age 1 and also is uncommon in those younger than age 2. The illness incidence peaks in children between 4 and 7 years of age, but can recur throughout life. It is more common in settings where people congregate and during the winter months.

Pharyngitis may be caused by a wide variety of microbial agents. The relative frequency of each of these agents varies greatly and depends on a number of epidemiologic factors including the age of the patient, season of the year, patient's occupation, and part of the country where the patient lives. The most common bacterial cause is group A beta-hemolytic *Streptococcus*, which causes only 15% of all cases of pharyngitis. The most common cause of pharyngitis overall is viral infection. Most of these agents produce a self-limited infection with no significant sequelae. All causes of pharyngitis can progress to suppurative complications, including cervical adenitis, sinusitis, and otitis media. Retropharyngeal abscess and peritonsillar abscess may evolve after pharyngitis and are covered separately in subsequent sections.

Peritonsillar Abscess

Peritonsillar abscess is the most common deep infection of the head and neck. This disease develops by spread of bacterial tonsillitis to the peritonsillar space between the tonsil and the surrounding superior constrictor muscles. It often is the clinical end point associated with a spectrum of infection that begins with tonsillitis, proceeds to peritonsillar cellulitis, and culminates with

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Table 1. Conditions Presenting with Sore Throat and Inability to Swallow Saliva

- Epiglottitis
- Peritonsillar abscess
- Retropharyngeal abscess
- Ludwig's angina
- Abscesses in the deep neck space
- Allergic drug reactions
- Lingual tonsillitis
- Ingested foreign body with or without perforation
- Pharyngeal zoster
- Botulism
- Tetanus
- Stevens-Johnson syndrome
- Toxic epidermal necrolysis
- Inhalation or aspiration of toxic chemicals
- Tumors or trauma to the larynx
- Diptheria

Table 2. Clinical Findings of Peritonsillar Abscess

- Severe pain
- Fever
- Dysphagia
- Trismus
- Hoarse, "hot potato" voice
- Enlargement of the tonsil
- Uvula deviated to the unaffected side
- Deviation of tonsil toward midline with rotation of anterior tonsillar pillar
- Fluctuance of the soft tissue between the upper pole of the tonsil and the soft palate

peritonsillar abscess.[1] Rarely, a peritonsillar abscess may occur in a patient who has no history of tonsillitis. Peritonsillar abscess usually occurs in teenagers and young adults, but may occur at any age. It is more common in patients who are immunosuppressed, immunodeficient, or diabetic. It also can occur after mononucleosis.[2-4]

Clinical Findings. Clinical symptoms include progressive sore throat. *(See Table 2.)* The pain increases and becomes unilateral, with pain radiation to the ear on the affected side. The patient may complain of increasing dysphagia. In severe cases, the patient may not only refuse food, but also may be unable to swallow liquids. Eventually, the patient will have difficulty with secretions and opening the mouth (trismus). Trismus will be the presenting symptom in about 60% of all cases of peritonsillar abscess.[5]

Diagnosis. These patients often have a toxic appearance. Physical examination is useful, but the clinical differentiation between peritonsillar abscess and cellulitis is very difficult.[6] The patient's trismus may make intraoral examination more difficult or even preclude examination of the mouth. When the practitioner examines the patient's mouth, unilateral edema and swelling of the anterior tonsillar pillar and soft palate will be present. Fluctuation and peritonsillar swelling are suggestive of a peritonsillar abscess. The soft palate usually is erythematous and edematous on the affected side. There may be deviation of the uvula to the unaffected side. The patient may have mild drooling and a "hot potato" (muffled) voice, which results from the inflammation of the palate and the subsequent limitation of movement.

Bilateral cervical lymph nodes are common, often with more tenderness on the affected side. Although trismus often is present in patients with a peritonsillar abscess, absence of trismus is not diagnostic. Examination of the throat in children can be difficult in the presence of trismus and may require judicious use of an oral topical anesthetic solution.

Needle aspiration or incision and drainage both may be diagnostic and therapeutic for patients with a peritonsillar abscess. Unfortunately, failure to obtain purulent fluid on aspiration may mean that no abscess is present, or simply that the needle missed the pus. In one study of 43 children with a suspected diagnosis of a peritonsillar abscess, a three-point needle aspiration technique of the superior tonsillar pole, the medial portion of the tonsil, and the inferior pole of the tonsil was used to maximize the chance of striking the abscess cavity.[7]

If the diagnosis is uncertain after the initial evaluation and after performing needle aspiration, imaging techniques may be useful. Both computed tomography (CT) scan and intraoral ultrasound have been used to substantiate the diagnosis.[8,9] CT scanning is more commonly available and interpretation is more uniform. CT scans can identify the presence and extent of an abscess cavity and will differentiate peritonsillar abscess from cellulitis in most cases.[10,11] It is, however, considerably more expensive. Intraoral ultrasound is not yet widely available and the test's sensitivity is less than 90%.[12] Most radiologists do not have training for interpretation of intraoral ultrasound.

Treatment. The initial assessment, stabilization, and management of a peritonsillar abscess may not be linked to the diagnosis of peritonsillar abscess or peritonsillar cellulitis. That is, if the patency of the upper airway is not immediately compromised, the emergency physician has some time to determine whether the patient has cellulitis or an abscess.

There is some controversy as to whether the patient can be treated in the ED and released or requires hospitalization. This partially depends on the preference of the ear, nose, and throat (ENT) consultant and partly on the clinical picture of the patient. If an older and responsible patient has few symptoms of toxicity, a trial of 12-24 hours of antibiotics is appropriate. If the patient is toxic or has severe trismus, more aggressive treatment is appropriate. Cellulitis is the likely diagnosis if the patient has significant improvement of symptoms on a trial of antibiotics. In younger patients, admission and therapy with intravenous antibiotics is appropriate.

Other treatment options include aspirating the pus with a needle in the ED, incision and drainage of the abscess in either the operating room or the ED, and an emergent tonsillectomy ("quinsy" tonsillectomy).[6] In most cases of peritonsillar abscess, needle drainage is the procedure of choice. About 90% of uncomplicated patients will be cured with antibiotics and one or two needle aspirations over a course of 3-5 days.[13,14]

Needle aspiration has significant advantages over incision and drainage, including: ability to proceed without general anesthesia, less trauma to surrounding structures, ready availability, and significantly lower cost. In the patient with uncom-

plicated peritonsillar abscess, needle aspiration not only will provide relief of symptoms, but also will establish a diagnosis.

Needle aspiration may not be suitable for younger children. They may require aspiration under general anesthesia in a controlled environment. Open surgical drainage is more appropriate for children who have signs of sepsis or airway compromise.[15] Open drainage also is indicated for immunocompromised patients.

About 30% of patients presenting with peritonsillar abscess will have an indication for tonsillectomy, such as recurrent tonsillitis. These patients may be treated with either needle aspiration and delayed tonsillectomy or acute abscess tonsillectomy.[15] Regardless of the therapy chosen by the ENT surgeon, the patient will be started on intravenous antibiotics, rehydrated, and discharged on oral antibiotics. Although penicillin usually will cover the organisms that cause peritonsillar abscess, cephalosporins often are used.[16,17] Close follow-up or hospitalization is indicated for all of these patients.

Submandibular Cellulitis and Abscess—Ludwig's Angina

Clinical Pathology. An infection of the submandibular space may involve either the sublingual or submandibular compartment. The submandibular space is divided into the sublingual space and the submaxillary space. The submaxillary space (also called the submylohyoid space) is further divided into two communicating compartments: the central submental space and the lateral submaxillary space. Because these spaces communicate with each other, they are effectively a single unit. Wilhelm Frederick von Ludwig described a fast-spreading, lethal, gangrenous induration of the connective tissues of the neck and floor of the mouth in 1836.[18] Submandibular abscess may be caused by infection of the submandibular gland and/or suppuration of the submandibular lymph nodes.

Ludwig's angina, or necrotizing fasciitis, usually begins in the submandibular space; dental disease is the most common cause.[19] An infected or recently extracted lower second or third molar are the most often involved teeth.[20] Because the roots of these teeth protrude below the mylohyoid ridge, a periapical tooth abscess easily may spread into the submandibular space. Ludwig's angina also may occur after lacerations and infections of the floor of the mouth, salivary calculi, and mandibular fractures. The key to the diagnosis of Ludwig's angina is involvement of all of the submandibular spaces. The most common pathogen is viridans-group Streptococcus, followed by *Staphylococcus aureus* and *Staphylococcus epidermis.* Anaerobes, generally *Bacteroides*, are implicated in about 40% of positive cultures.

Clinical. The typical patient is between the ages of 20 and 40 and has dental caries but is otherwise healthy. There is a 3:2 male preponderance. Although most patients are healthy, the disease has been associated with diabetes, systemic lupus erythematosus, and neutropenia from multiple causes.

The patient will present as acutely ill-appearing and anxious. The most common complaints of Ludwig's angina are mouth and neck pain, sore throat, swelling, and dysphagia. In some patients, the complaint will be pain in the floor of the mouth. Voice changes, odynophagia, inability to handle oral secretions, and fever frequently are seen. Ludwig's angina often progresses quite rapidly and may result in life-threatening upper airway obstruction. The patient may suffer an acute respiratory arrest if forced to lie supine. The mortality in these cases may exceed 50%.[21] When Ludwig's angina is rapidly and appropriately treated, the mortality is between 0% and 8.5%.

Diagnostic Studies. Soft-tissue films of the neck may demonstrate marked edema of the submental and submandibular soft tissues. Air in the soft tissues may be noted if the infection is due to gas-forming bacteria. Both CT and MRI will better define the abscess and cellulitis.[9] The patient may not be able to undergo CT scan or magnetic resonance imaging (MRI) until the airway is secured with tracheostomy or endotracheal intubation. Use of CT scan to determine the extent of the infection also has shown that the lateral pharyngeal spaces often are involved.[22] The infection usually is bilateral.

Management. The three components of treatment of Ludwig's angina include airway protection, antibiotics, and surgical drainage of the lesion. Because airway compromise is the single most common cause of death in Ludwig's angina, protection of the airway is the first priority. Several methods have been used to manage the airway in these patients, including oral intubation, fiberoptic intubation, cricothyrotomy, and formal tracheostomy.

Awake fiberoptic intubation may be obscured by secretions or pus, but it may be a viable alternative in the seated patient. The best place to attempt this intubation may be in the operating room with a surgeon scrubbed for immediate tracheostomy.

Cricothyrotomy or jet ventilation also may be quite difficult because of distortion of the anatomy caused by swelling. "Blind" nasotracheal intubation should not be performed because of the risk of precipitating an acute airway obstruction. These patients are not candidates for rapid sequence intubation techniques.

Simple critical care unit observation and antibiotic therapy in a select group of patients is possible.[23-25] Observation is recommended only in a critical care unit, where the airway can be controlled before compromise occurs if the patient's disease progresses despite antibiotics.[26]

High-dose antibiotics are the mainstay of therapy. Once the airway has been protected, antibiotics alone will suffice for about 50% of patients.[27] Penicillin is considered by many authors to be the drug of choice. Increasing antibiotic resistance may prompt the use of second- or third-generation cephalosporins, and anaerobic coverage may be provided by the addition of metronidazole or clindamycin. Alternatively, ampicillin-sulbactam may be used.[27]

Surgical drainage once was recommended for all patients with Ludwig's angina, even though many would not have any purulent collection. Surgical drainage is now usually reserved for refractory cases or patients with rapid progression. CT results can guide surgical drainage and may indicate a need for surgical drainage if significant collections of pus are found.

Drainage may be conducted intraorally or externally. The intraoral approach should be reserved for the patient who only

Table 3. Predisposing Factors for Retropharyngeal Abscess

- Penetrating trauma: Foreign body such as pencil, Popsicle stick, or fishbone
- Tonsillitis
- Pharyngitis
- Peritonsillar abscess
- Croup
- Otitis media
- Nasal infections
- Dental infections
- Intubation
- Adenoidectomy (recent)

Table 4. Complications of Retropharyngeal Abscess

- Rupture of abscess
- Airway rupture
- Asphyxiation
- Aspiration pneumonia
- Spinal rupture
- Lateral pharyngeal space rupture
- Inferior rupture into mediastinum
- Airway compromise

has an uncomplicated submandibular space infection that is limited to the sublingual compartment. Steroids have been suggested as an adjunct to decrease the spread of edema, as well as the need for artificial airways. Although there may be some merit in the use of steroids for Ludwig's angina, there are insufficient data in the literature to recommend this adjunct.

Complications. Mortality from Ludwig's angina may be related to rapid upper airway obstruction or to extension of the cellulitis into the mediastinum, resulting in mediastinitis and pericarditis.[28] If the abscess ruptures intraorally, pus may be aspirated and result in pneumonia and possible lung abscess. Tongue necrosis also has been reported.

Retropharyngeal Abscess

Anatomy. A retropharyngeal abscess is an accumulation of pus in the retropharyngeal space. This area is bound anteriorly by the posterior pharyngeal wall and posteriorly by the prevertebral fascia. The space is bound superiorly by the base of the skull and inferiorly at the fusion of the anterior and posterior layers of fascia. (This fusion occurs at the level of C_7-T_1, within the superior mediastinum.) This space was described by Grodinsky and Holyoke in 1938 and contains no important anatomic structures except lymph nodes.

Retropharyngeal lymph nodes lie in two vertical chains on either side of the midline in the retropharyngeal space. They receive lymph drainage from the oropharynx, nasopharynx, teeth, maxillary sinuses, and the eustachian tube. The retropharyngeal lymph nodes offer a path for the spread of infection from this space into the mediastinum. These lymph nodes are most prominent in children younger than age 4 and normally regress by age 6.

Infection of the retropharyngeal space is most likely to occur in the pediatric population; adult disease is distinctly uncommon. In the pre-antibiotic era, when retropharyngeal abscess was more common, several series reported that more than 95% of cases occurred in children younger than age 6.[29-32] The disease also can occur as a result of trauma to the posterior pharynx.[33-35] A common mechanism of trauma occurs when a child with a stick or toy in his or her mouth falls and punctures or lacerates the posterior pharyngeal wall. The child often is seen in an ED for this initial insult. The use of crack cocaine has caused retropharyngeal abscess in adults.[36] Other insults that can cause retropharyngeal abscess include endoscopy or endotracheal intubation.[37] *(See Table 3.)*

Clinical Presentation. In the child, the onset of symptoms is usually slowly progressive and often occurs after a mild upper respiratory infection. A history of trauma as previously described also may be elicited. The child with a retropharyngeal abscess will appear toxic. Neck movement in these patients may be limited and may suggest nuchal rigidity if the examiner does not adequately inspect the oral cavity. The child often will have dysphagia and a muffled voice—the "hot potato" voice. This muffled voice results from inflammation and swelling about the posterior pharyngeal wall. Many children will have trismus or be unwilling to open their mouths because of pain.

On inspection, the examining physician may see bulging of the retropharyngeal wall, which may appear one-sided. This bulging often is difficult to detect both because of the small size of the child and the pooling of secretions in the hypopharynx. If the child allows the examiner to palpate the posterior pharynx, fluctuation may be detected. Palpation should be performed very gently, and only on a cooperative patient, because rupture of the abscess may occur.

In the adult, retropharyngeal abscess usually occurs in the setting of underlying illness (i.e., after an intraoral procedure, neck trauma, or a head and neck infection). The most common presenting symptoms are sore throat, dysphagia, and neck pain. Symptoms often are out of proportion to the neck examination findings.

Diagnosis on clinical grounds may be difficult, particularly in the adult. Only a minority of adults will have visible swelling of the posterior pharynx on physical examination. A fluctuant area in oropharynx or hypopharynx may be palpated. Again, this should be performed in a gentle manner.

Diagnostic Studies. Lateral neck x-rays have been used for many years to evaluate the neck mass, but are now considered less effective than CT scan of the neck. In patients who had a clinical examination that was suggestive of retropharyngeal abscess, there was scant correlation between the lateral neck films and findings on CT scan.[38]

Lateral neck films may show an increase in the anteroposterior (AP) soft-tissue space anterior to the vertebra, which is thought to represent a widening of the retropharyngeal space. Unfortunately, such widening is nonspecific, as failure to extend the neck also may cause widening of this space. Air density in the retropharyngeal space may indicate an abscess caused by a gas-producing organism or communication with the esophagus or airway.

CT scan of the neck, with focus on the retropharyngeal area is the current method of choice for diagnosis of a retropharyngeal

abscess.[15] The CT scan delineates exactly which neck spaces are involved and localizes the lesion before surgical drainage is attempted.[39] CT also is useful for defining the vascular structures of the neck and evaluating their potential involvement. MRI scan may be more useful for evaluation of the carotids.[40]

Management. Patients with a retropharyngeal abscess should be admitted to the hospital. Therapy should include aspiration of the abscess or surgical drainage of the abscess. Children without airway compromise or other complications initially may be treated with intravenous antibiotics.[41] Clindamycin and an aminoglycoside or a penicillinase-resistant penicillin combined with a third-generation cephalosporin and metronidazole are appropriate first-line antibiotic choices.[42]

These abscesses should not be drained in the ED. Surgical complications associated with drainage of a retropharyngeal abscess include both difficult intubation and rupture of the abscess during the procedure. Control of the airway is paramount in this disease, and the controlled environment of an operating room with all tools for airway management ready—suction, adequate sedation, and analgesia—is appropriate. Procedures to relieve an acute airway obstruction include endotracheal intubation, tracheostomy, cricothyrotomy, and fiberoptic intubation.

Complications. Complications of a retropharyngeal abscess include extension into the mediastinum, involvement of the great vessels of the neck, rupture of the abscess (often with aspiration of the contents), and asphyxia from mass effect.[43,44] *(See Table 4.)* The most common of these complications is airway obstruction.[45,46] Extensive inflammation may can cause an acute inflammatory torticollis. Spasm of the sternocleidomastoid muscle will cause posturing of the head, with the occiput rotated to the affected side. In marked cases, the atlantoaxial joint may be dislocated by this intense muscle spasm.[47] CT scan or MRI may be needed to determine the cause of the torticollis.

Diphtheria (True Croup)

Description. Diphtheria is an infection that affects the throat, nose, ears, and occasionally, the skin. Nasal and ear infections often are asymptomatic. Pharyngeal infections may produce significant signs or symptoms or they may be asymptomatic. Diphtheria does not invade tissues apart from those of the surface epithelial cells at the site of the local lesion.

Diphtheria was one of several upper respiratory illnesses viewed collectively as "croup." The clinician-pathologist Pierre Bretonneau first described its unique clinical characteristics in an epidemic in southern France in 1821.

During the first half of the 20th century, diphtheria was a major worldwide health problem, with multiple epidemics that yielded to vigorous public health control measures. Since 1990, epidemic diphtheria has reemerged in the former Soviet Union and other areas where social disorganization have relaxed immunization practices. The 1993-1994 diphtheria epidemic in Russia included more than 150,000 cases.[48] With increasing immigration from these countries, and declining rates of immunization both abroad and in the United States, it is possible that physicians in the United States may see more cases in emergency medicine practice.

Friedrich Loffler first isolated the diphtheria bacillus in pure culture in Robert Koch's laboratory.[49] He also noted that the organisms remained in the membrane without invading the tissues of the throat, and theorized that neurologic and cardiologic manifestations of the disease were caused by a toxin elaborated by the organism. Recognition of organ damage caused by the diphtheria toxin led to development of both an effective antitoxin-based therapy for actual infection and a toxoid vaccine to prevent infection.

Corynebacterium diphtheriae is a nonsporulating, unencapsulated, nonmotile, pleomorphic gram-positive bacillus. *C. diphtheriae* elaborates a toxin with a molecular weight of 61,000 Daltons. The major virulence of *C. diphtheriae* results from the action of its potent exotoxin, which inhibits protein synthesis in mammalian cells but not in bacteria. This toxin cleaves into two subunits: the A subunit attaches to cells, while the B subunit is the lethal factor. The toxin affects all cells in the body, but it most prominently affects the heart (myocarditis), nerves (demyelination), and kidneys (tubular necrosis).

Diphtheria is now rare in the United States due to extensive immunization.[50] About 5-10 cases per year occur, and these are most frequently seen in immigrants (legal or illegal) who have not been adequately immunized.[51] Some rare cases occur when immunity declines in otherwise immunized individuals.

Humans are the only known reservoir for *C. diphtheriae.* Asymptomatic nasopharyngeal carriers are common when immunizations lapse. The primary modes of infection occur via airborne respiratory droplets and direct contact with either respiratory secretions or exudate from infected skin lesions. Fomites may play a role in transmission, and epidemics have been caused by contaminated milk. Most respiratory tract disease occurs in the colder months in temperate climates, and is associated with crowded indoor living conditions and hot, dry air. Multiple factors promote the spread of diphtheria, including poor hygiene, overcrowding, and inadequate medical care.

Clinical Presentation. The incubation period is about one week. The disease may be quite mild or progressive and lethal. The onset of frank disease usually is abrupt, with low-grade fever, malaise, sore throat, pharyngeal injection, and the development of a membrane (typically on one or both tonsils). If left untreated, the disease spreads with subsequent extension to the tonsillar pillars, uvula, soft palate, oropharynx, and nasopharynx. Diphtherial pharyngitis may spread downward into the larynx. Symptoms then include hoarseness, dyspnea, respiratory stridor, and a brassy cough.

Within the first few days of respiratory tract infection, local toxin causes tissue damage in the immediate area of the infection. As noted earlier, direct invasion of the tissues by diphtheria is unusual. Most commonly, the local toxin production induces a dense necrotic coagulum composed of fibrin, leukocytes, erythrocytes, dead respiratory epithelial cells, and diphtheria organisms. Removal of this adherent gray-brown pseudomembrane reveals a bleeding, edematous mucosal surface.

Edema and a membrane involving the trachea and bronchi can cause respiratory distress. The patient will appear anxious and cyanotic; will use accessory muscles of respiration; and will have inspiratory intercostal, supraclavicular, and substernal retractions. Local swelling of nodes and neck tissues gives a "bull neck" appearance. The edema and adenopathy can cause respiratory embarrassment, and if the airway is not promptly protected by intubation and mechanical membrane removal, patients become exhausted and die. In both adults and children, a common cause of death is suffocation after aspiration of the membrane.

Systemic complications are caused by the diphtheria toxin, which has its most striking effects on the heart and nervous system.

In 50% of cases, the patient will develop a toxic myocarditis from the effects of the toxin. Characteristically, the toxicity occurs after 1-2 weeks of illness, often when the local oropharyngeal disease is improving. Clinically, myocarditis can present acutely with congestive heart failure and circulatory collapse, or more slowly with progressive dyspnea, weakness, and cardiac dilatation. The electrocardiogram (ECG) may show ST segment-T wave changes and first-degree heart block. These ECG changes can progress to more severe forms of block, atrioventricular dissociation, and other arrhythmias, which carry an ominous prognosis.[52]

Up to three-fourths of patients with severe disease develop neuropathy.[53] Early in the course of the disease, local paralysis of the soft palate and posterior pharyngeal wall are common and are caused by local toxin release. This local paralysis may be manifested by regurgitation of swallowed fluids through the nose. Later cranial neuropathies that cause oculomotor and ciliary paralysis also are common. Dysfunction of facial, pharyngeal, or laryngeal nerves can contribute to the risk of aspiration. Peripheral neuritis develops later, from 10 days to 3 months after the onset of disease in the throat.

Mortality rates vary from 3.5% to 12% and have not changed in the last 50 years. Rates are highest in the very young and very old. Most deaths occur in the first 3-4 days, from asphyxia or myocarditis; a fatal outcome is rare in a fully immunized individual. The experience in Russia in the 1990s was similar.[54]

Diagnosis. Culture is the gold standard for diagnosis of diphtheria. Because routine methods of throat culture do not promote the isolation and identification of *C. diphtheriae,* the laboratory must be alerted to use selective media when the disease is suspected. The combination of "Chinese characters" as seen on Gram's stain, distinctive colonies with halos on Tindale's medium, and the presence of metachromatic granules allows a presumptive identification of *C. diphtheriae.*

Not all colonies of diphtheria will produce toxin. Toxin production normally is demonstrated by Elek plate precipitin strips or by polymerase chain reaction testing for the toxin A subunit gene.[55] Unfortunately, when immunization policies are not enforced, there is an increase in the number of toxin-bearing cultures found, and non-toxin bearing *C. diphtheriae* can begin producing toxin.

Management. Antibiotic therapy inhibits toxin production, improves the local infection, and prevents spread of the organism to other people. Although several antibiotics, including penicillin, erythromycin, clindamycin, rifampin, and tetracycline, are effective, only penicillin and erythromycin generally are recommended. Because erythromycin is marginally superior to penicillin in eradicating the carrier state, some authorities prefer it for initial treatment. There is no cogent reason why newer macrolides, available in intravenous forms, cannot be used.

Diphtheria antitoxin is a hyperimmune antiserum produced in horses. It has been the standard of therapy since 1989. Diphtheria antitoxin antibodies will only neutralize toxin before the toxin enters the cells. It is critical to administer diphtheria antitoxin as soon as a presumptive diagnosis has been made. The Committee on Infectious Diseases of the American Academy of Pediatrics recommends 20,000-40,000 units of antitoxin for pharyngeal or laryngeal disease of 48 hours duration; 40,000-60,000 units for nasopharyngeal lesions.[56] For extensive disease of three or more days duration and for anyone with brawny swelling of the neck, 80,000-120,000 units should be given. Diphtheria antitoxin is no longer licensed in the United States, but a European-licensed product is available from the National Immunization Program of the Centers for Disease Control and Prevention, (404) 639-8200.

Patients should receive toxoid immunization in the convalescent stage of their disease because clinical infection does not always induce adequate levels of antitoxin. Close contacs whose immunization status is incomplete or unclear should promptly receive doses of toxoid that are appropriate for their ages and complete the proper series of immunizations.

Prevention is of paramount importance. The use of tetanus-diphtheria immunization every 10 years will prevent or will markedly decrease the seriousness of the infection. Recommendations from the Immunization Practices Advisory Committee, published by the Centers for Disease Control and Prevention in 1991, remain current in the prevention of diphtheria.[57] Diphtheria is a reportable disease in all U.S. states.

Complications. The diphtheria toxin can damage the airway, heart, nervous system, and kidneys. Early in the disease, respiratory and cardiac complications are the biggest threats. Airway obstruction can result from aspiration of the pharyngeal membrane, its direct extension into the larynx, or external compression by mass effect from enlarged nodes and edema. For this reason, many experts recommend early tracheostomy or intubation, particularly when the larynx is involved. This provides lower access for mechanical removal of tracheobronchial membranes and avoids the risk of aspiration of the membrane. Cardiac complications can be minimized by close observation in an ICU environment. The electrocardiograph monitoring will allow prompt initiation of electric pacing for conduction disturbances, drugs for arrhythmias, or digitalis for heart failure.

Croup (Viral or "False" Croup)

Description. Croup is a viral infection of the upper airway, also called laryngo-tracheo-bronchitis (LTB). Classifications of

croup based on anatomy, pathology, and microbiology have resulted in such terms as croup syndrome, true croup, false croup, viral croup, spasmodic croup, recurrent croup, pseudomembranous croup, acute subglottic laryngitis, spasmodic laryngitis, laryngotracheitis, LTB, and acute infective LTB.[58] The variety of terms has only contributed to confusion about this infraglottic infection, as multiple terms have been used by different authors to describe identical clinical conditions. The clinical term now is used almost exclusively for non-bacterial croup.[59]

The original definition of croup (so-called true croup) was synonymous with diphtheria and had a mortality rate of about 25%. During the 1900s the term "croup" expanded to include other infections (including epiglottitis).[60] During the latter half of the 1900s, reports of viral etiologies became commonplace and bacterial causes of croup seemed to disappear.[61] The reasons for this shift from bacterial to viral infections is unknown, but may be due to host factors, increased immunization, antibiotic usage, or even a change in bacterial virulence.

Mortality rates attributed to children with a harsh barking cough may be reflected in the anxious attitudes of the parents, grandparents, or great-grandparents who accompany the child with croup to the ED. Fortunately, the viral variant rarely is as serious as it sounds.

Spasmodic and Recurrent Croup. Spasmodic croup is a term used to describe an entity that shares many clinical features of viral croup. Spasmodic croup generally is considered less severe, more acute, and may be more responsive to simpler therapies. There is an ongoing debate about whether spasmodic croup and viral croup are separate conditions or simply different parts of a spectrum of disease. Although there is a perceived difference between spasmodic and viral croup in the minds of some authors, there appears to be no significant clinical difference between the two entities.

Spasmodic croup traditionally has been differentiated from viral croup by the absence of fever and a characteristic nocturnal onset. The viral prodrome may or may not be present. The treatment for spasmodic croup is generally the same as that used for viral croup, so differentiating between the forms is not necessary.[62] The attack tends to subside quickly but may recur; it may be more common in children with allergies.

The exact frequency of viral croup in the general population is not known because many mild cases may be treated at home, without the patient seeing a health care provider, or by following advice dispensed over the telephone.

With regard to viral croup, epidemiologic data have been collected from pediatricians in outpatient practice.[63,64] There were no cases reported in the first month of life and an increasing incidence was reported during the first two years of life. The peak incidence was 5.6 per 100 male children and 3.66 per 100 female children in the 1-2 year age group. The male:female ratio of occurrence was 1.5:1. After age 2, the rate decreased and the disease became uncommon when the children reached school age. More than 80% of cases occurred in the first five years of life.

The microbiology of viral croup has been well established. Parainfluenza (types 1, 2, and 3) viruses represent about one-half of the documented causes.[64] Respiratory syncytial virus (RSV) is the cause of about 10% of cases. Influenza causes about 6% of cases and is more common in older children. *Mycoplasma* species may be identified in older children. Measles was identified as a cause of croup in the 1980s but is rare now that children are routinely immunized.[65,66] The disease clusters in spring and fall and mirrors the seasonal pattern of respiratory viruses in general. The fall peak coincides with that of parainfluenza virus infection, and the winter peak mimics RSV infection rates.

Clinical Features. A case of viral croup usually begins with the signs and symptoms of a mild viral upper respiratory infection (i.e., low-grade fever, sore throat, cough, and rhinorrhea).

Within a few hours, the distinctive barking cough—often described as a seal's bark—will develop. The child also may develop inspiratory stridor, hoarseness, and retractions. The degree of respiratory distress ranges from mild to life-threatening. The examiner may note tachypnea; suprasternal, subcostal, and infracostal retractions; and decreased air entry. Hypoxemia may be marked by cyanosis or simply restlessness. Wheezes, expiratory rhonchi, and crackles may be heard in the lungs. The disease often is worse at night. The child with croup should have no dysphagia and no drooling.

Unfortunately, mild (early) epiglottitis may mimic croup. Multiple variants of croup "scoring" systems have been designed in an attempt to create an objective measure of severity and to allow comparison between serial assessments of the patient. The ideal croup score would assist in identifying and quantifying respiratory distress, help determine initial and subsequent therapy, and determine need for admission. The most commonly used systems include color of the patient (presence or absence of cyanosis), air entry, retractions, level of consciousness, and stridor. Stridor at rest is an indicator of the highest severity of croup in all croup scoring systems. *(See Table 5.)*

Diagnosis. For the emergency physician, the typical case of croup is a diagnosis based on clinical findings rather than laboratory, radiographic, or microbiologic studies. The severity of the presentation and the apparent toxicity of the child will be paramount in determining the necessity of further studies or airway management. If the child has signs of significant airway distress, the treatment setting should be completely controlled and additional diagnostic studies minimized soas to not exacerbate the child and cause airway obstruction.

Nonspecific blood tests, such as white blood cell (WBC) count or erythrocyte sedimentation rates, may be elevated in quite a number of acute infectious diseases and may add little to the evaluation of the patient. C-reactive protein (CRP) has been reported to be elevated in those cases of middle or lower respiratory tract infections which are caused by bacterial infections.[67] In a relatively large study, about one-half of patients with bacterial or mixed viral and bacterial infections had a CRP of 20 mg/L or more, and only 35% of patients with viral infection alone had a CRP of more than 20 mg/L.[68] This differ-

Table 5. Scoring System* for Croup[75]

LEVEL OF CONSCIOUSNESS	
Normal or sleeping	0
Disoriented	5
CYANOSIS	
None	0
With agitation	4
At rest	5
STRIDOR	
None	0
With agitation	1
At rest	2
AIR ENTRY	
Normal	0
Decreased	1
Markedly decreased	2
RETRACTIONS	
None	0
Mild	1
Moderate	2
Severe	3

* Zero represents the normal state or absence of the sign and the highest number represents the most severe distress.

ence is clinically insignificant and does not appear to alter management.

Moreover, microbiologic studies may point to a specific etiology for croup, but the results are unlikely to alter management. Respiratory specimens for RSV or parainfluenza can be harvested from nasal washings or sputum. X-rays often are ordered but usually are not needed to make the diagnosis of viral croup. Plain films of the neck can help confirm the diagnosis of laryngotracheitis. These films may show air trapping and dilated hypopharynx and the classic "steeple" sign secondary to subglottic narrowing. The films also may help rule out epiglottitis.

Management. Many therapies have been advocated for the treatment of croup. Some of these therapies have persisted despite scant objective evidence of their value. Others are controversial despite substantial clinical research.

Humidification and cool mist have been advocated as a mainstay of therapy for many years in the treatment of croup.[69] Steam generated by showers, baths, tea kettles, and croup tents all have been suggested. The additional moisture is thought to prevent drying out of mucous membranes, loosen thickened secretions, and decrease inflammation in the larynx and pharynx. Despite the widespread use of humidification, little objective evidence of its value exists.[70] Since inspired air becomes completely saturated with water before reaching the larynx, there is little additional water vapor added by humidification of the air.

In most cases, the warm moisture is not unreasonable as long as it is well tolerated by the child. If the child becomes uncomfortable when kept in the moisture, the putative benefits are far outweighed by increased oxygen consumption caused by agitation.

Steroids also have been advocated for the treatment of viral croup for many years. Nearly 50 years after the first proposal to use steroids in croup, the exact mechanism of benefit still is not known. However, steroids may block allergic responses, reduce local inflammation, and reduce the subglottic edema.

Steroids were shown to be effective, with significant improvement in hospitalized children, in a 1960s randomized, placebo-controlled study.[71] A meta-analysis of nine randomized studies showed that administration of corticosteroids was associated with significantly greater improvement at 12 and 24 hours after treatment.[72] The incidence of endotracheal intubation was reduced by 80% in the treated group. Virtually no adverse effects of the use of corticosteroids have been reported in these reviews.

In 1995, the first prospective study of steroids in outpatient management of croup was reported.[73] This randomized, blinded study showed that fewer patients who were treated with dexamethasone required additional medical care and that parents reported earlier improvement in treated children.

Despite this evidence, other authors have concluded that routine use of steroids in children cannot be justified. Controversy exists concerning the effects on hospitalization requirements, the duration of hospitalization, the requirement for subsequent medical visits, and requirements for intubation and intensive therapy.[61]

A variety of dosages and regimens exists for steroids in croup. Although parenteral administration is the usual recommendation, both oral and nebulized agents have been evaluated. Advocates of oral administration cite the discomfort of an intramuscular injection, but neglect the significant possibility of early vomiting after oral administration. Dexamethasone 0.6 mg/kg as a single dose given intramuscularly or intravenously is this author's current recommendation. A recent, randomized, blinded study using nebulized dexamethasone suggests that this route is as effective as either oral or injection. Nebulized budesonide also has been evaluated, but is not available in the United States.[74]

Like steroids, epinephrine is not a new therapy for croup.[75] Although the early studies used intermittent positive pressure breathing, nebulized epinephrine shows similar benefits. Epinephrine is thought to cause vasoconstriction of the inflamed mucous membranes and a subsequent decrease in edema. Use of racemic epinephrine frequently has been proposed and is widely thought to decrease the incidence of tachycardia and hypertension. Evidence to support these beliefs could not be found. Indeed, in one study that compared racemic epinephrine and L-epinephrine, there was not a significant difference.[76] Since L-epinephrine is readily available and much less expensive, there simply is no cogent reason to demand racemic epinephrine for the treatment of croup.

Table 6. Clinical Findings of Epiglottitis in the Adult[99,100,107,111]

- Gradual onset of symptoms
- Rapid progression
- Severe sore throat (95-100%)
- Dysphagia (76%)
- Pain on swallowing (95%)
- Pain on palpation of the larynx
- High fever (88%)
- Muffled voice (50%)
- Shortness of breath or respiratory difficulty
- Drooling (relatively uncommon)
- "Upright" positioning
- Ear pain
- Toxic appearance

Table 7. Clinical Findings of Epiglottitis in the Child[111,112]

- Sudden onset of symptoms
- Rapid progression
- High fever
- Muffled, "hot potato" voice
- Inspiratory stridor
- Sore throat
- Dysphagia
- Drooling
- "Tripod" positioning
- Toxic appearance

The effect of epinephrine is transient, and croup scores may return to pretreatment levels in some patients in less than two hours ("the rebound effect"). Ten years ago, admission was thought to be mandatory if epinephrine was used because of this possibility. This quandary can be solved without admission by simply observing the child for about 3-4 hours. It is unclear from the literature how widespread the use of epinephrine is in patients who are discharged home from the ED. Use of epinephrine followed by discharge would require a period of observation in the ED followed by close patient follow-up.

Antibiotics have not been shown to be of any benefit in the treatment of croup.

The use of helium/oxygen (heliox) mixtures in the child with croup may buy time for more help or better equipment.[77] Heliox mixtures allow better oxygenation since the lighter gas flows more easily through the tighter airway passage.

Complications and Disposition. Airway obstruction occurs because of subglottic edema, tracheal and bronchial inflammation, and increased mucosal secretions. This combination narrows the airway and restricts airflow with both hypoventilation and depletion of oxygen reserves, hypercarbia, hypoxemia, and respiratory failure.

Most patients will do well in the outpatient setting. Fewer than 5% of children will require hospitalization, and only 10% of those hospitalized will require ICU services. Only 1% of children with croup will require intubation. The mortality rate of patients hospitalized with croup is about 1%.

Children with croup should be admitted if they have severe or unusual symptoms, poor response to inhaled epinephrine, or a toxic appearance. If the child worsens during observation or if multiple epinephrine treatments are required, admission is warranted. Patients younger than age 1 should be considered for admission because they have such small airways. Children with stridor at rest also should be considered for admission. In all cases, the child's parents or caretakers must be reliable and have adequate access to a telephone or transportation.

When endotracheal intubation is required for the child with croup, the physician should choose a tube about two sizes smaller than usual.[78] A gentle and smooth intubation by the most skilled practitioner is appropriate. All trauma can worsen the airway obstruction and should be avoided.

Epiglottitis (Supraglottitis)

Epiglottitis, or more correctly supraglottitis, is characterized by cellulitis of the structures above the glottis: the epiglottis, the aryepiglottic folds, the arytenoid soft tissues, and the uvula. In children, the disease usually is confined to the epiglottis and closely surrounding structures, whereas in adults, the inflammation also may involve the prevertebral soft tissues, the valleculae, the base of the tongue, and the soft palate.[79] *(See Table 6.)* Supraglottitis should be considered in the differential diagnosis of any adult patient who presents with a sore throat and dysphagia.

Interestingly, a description of supraglottitis was recorded in the 13th century. The first accurate clinical description of epiglottitis was noted in 1900 by Theisen.[80] The association of acute laryngitis and *H. influenzae* type b (HIB) bacteremia was noted first in the English literature in 1941.[81]

Although epiglottitis generally is considered by recent generations of physicians to be a pediatric disease, historically it has been deemed a disease of adults. Indeed, President George Washington probably died from epiglottitis in 1796. It was only between 1950 and 1980 that it more commonly was diagnosed in children and described as a childhood disease. In the early 1980s, the ratio of adults to children with epiglottitis was about 1 to 2.6. By the mid 1990s, that ratio had changed to 1 adult case for every 0.4 pediatric cases.

The classic pediatric clinical course is familiar to family physicians, emergency physicians, otolaryngologists, and pediatricians. The child presents with an abrupt onset of severe sore throat, fever, and drooling. *(See Table 7.)* The sore throat may be expressed as refusal to eat in the younger child. The disease progresses rapidly. The child goes from anxiety to tripod posturing (sitting and leaning forward with elbows on thighs) to respiratory compromise. This clinical picture continues to rapidly progress through respiratory obstruction to death within hours. The total duration of the disease prior to hospitalization usually is fewer than 24 hours and may be as short as two hours.

The specific cause of the airway obstruction is not always clear, and several theories exist: The swollen floppy epiglottis of the child obstructs the glottic opening; laryngospasm occurs from the inflammation surrounding the glottis; the smaller airway diameter causes respiratory fatigue and the patient develops respiratory distress. It has been noted that it is rather unlikely for a swollen, edematous, rigid epiglottis to fall onto or be aspirated into the glottis as a means of respiratory obstruction.

Epiglottitis in the adult progresses more slowly. The combination of sore throat and dysphagia is a significant clue to the presence of the disease. Indeed, a severe sore throat is almost universal. Dysphagia occurs in about 75%, fever in about 90%, and a muffled voice in about 50% of adults with epiglottitis.[82] Cough is not a prominent finding in either children or adults. Other findings include difficulty managing secretions, respiratory distress, anterior neck tenderness, and hoarseness.

Unlike the pediatric disease, the adult will require active airway management in only about 15% of cases.[83] Unfortunately, it is not possible to accurately predict which 15% of the cases will require acute airway management. The disease often will continue to progress while oral antibiotics are taken.

Microbiology. For many years, HIB has been the most common etiology of epiglottitis in children and adults. Indeed, 95% of cases of epiglottitis in children and 53% of cases in adults in 1994 were caused by HIB.[84] This has changed with the increasing penetrance of *H. influenzae* immunization.[85,86] There has been a recent increase in adult supraglottitis in comparison to pediatric epiglottitis. The incidence of acute supraglottitis in adults has remained stable at about 1.8 cases per 100,000 adults, while the incidence in children has plummeted. This is most likely due to the routine *H. influenzae* immunization of children in Western countries since December 1987.[87] Unlike epiglottis in children, which nearly always is caused by *H. influenzae,* acute epiglottitis in the adult can be caused by a broad microbiologic spectrum.[88]

It is important to remember that there are case reports of HIB epiglottitis occurring in children despite vaccination. Other serotypes of *H. influenzae* also may cause serious invasive disease, including epiglottitis.[89] Rarer causes of epiglottitis include *Streptococcus pneumoniae; Staphylococcus aureus; Haemophilus parainfluenzae; Klebsiella pneumoniae; Neisseria meningitidis;* varicella zoster; herpes simplex; parainfluenza virus; influenza type b; and groups A, B, and C *Streptococcus*.[90-92]

Group A *Streptococcus* probably is the most common etiology in those areas where *H. influenzae* immunization has been given. There has been an increasing emergence of severe infections in the United States caused by Group A *Streptococcus* since the late 1980s. There also is an association of Group A *Streptococcus* with varicella as an etiology of epiglottitis.[93,94]

In the adult patient, blood cultures are positive in only a minority of cases. A throat culture may be positive in about 25% of cases. The high rate of negative blood cultures may reflect prior oral antibiotic therapy (frequently found), self-limited bacteremia, or a viral cause.

Among the pathogens, there is general agreement that HIB will cause an acute onset and will correlate well with clinical severity, and that these patients are likely to require aggressive airway management. This is particularly true in the pediatric patient. If organisms other than HIB become a more common cause for epiglottitis, the characteristics of the disease and the type of antimicrobial treatment required may change.

When Group A streptococcal disease causes supraglottitis, the course is longer, with a mean intubation time of 7.5 days compared to the usual 1.5-3 days required for *H. influenzae*. There appears to be more involvement of the aryepiglottic folds.

Other findings suggestive of streptococcus epiglottitis include an older age of onset, presence of fever, negative blood cultures, and slower resolution of the disease. Supraglottitis also is reported to occur in patients with acquired immunodeficiency syndrome (AIDS). A wider range of organisms can cause the disease, including aspergillus, *Klebsiella*, and candida when the patient is immunocompromised. Although the pathogens are similar, the disease is much more aggressive in the patient with AIDS. Acute thermal epiglottitis may result from a direct thermal insult to the epiglottis.[95-97] A history of ingestion of hot food or liquid may be elicited by the clinician. Smoking cocaine free-base (crack) also may precipitate thermal epiglottitis.

Clinical Presentation. Clinical diagnosis of epiglottitis must be swift in both adults and children. The toxic and anxious appearance of the child with epiglottitis, rapid onset and equally rapid course, and tripod posture should prompt the clinician to think of this disease. In adults and children, the sore throat and odynophagia are the most prominent symptoms of epiglottitis. Both adult and pediatric patients may have varying degrees of respiratory distress, inspiratory stridor, hoarseness, muffled voice, and/or drooling.

Patients may have a fulminant course with complete airway obstruction in as little as 30 minutes.[98] Patients who present with tachycardia, an increased WBS count, and a history of rapidly progressive sore throat are at significantly increased risk of airway obstruction.[99] These patients should be treated aggressively.

The absence of perceived acuity in the presentation may significantly delay the diagnosis of the adult with epiglottitis. In one study, only sitting erect and stridor were useful early predictors of airway compromise in the adult.[98] Other studies include dyspnea, tachypnea, stridor, retractions, and cyanosis as manifestations of advanced disease. The clinician should not wait for these symptoms to appear before attempting airway intervention.[100]

It is commonly accepted that in cases of epiglottitis, examination of the child's oropharynx or epiglottis may cause laryngeal spasm and convert respiratory distress to respiratory obstruction. Note that the pediatric patient with advanced supraglottitis and respiratory distress may have sudden and relatively unpredictable airway obstruction, which may explain tales of abrupt decompensation during examination. Catastrophes from this examination in mild cases have not been reported in any of the reviewed literature, so this disaster may be more folklore than a documented complication of examination.[58,101] The emergency physician would be wise to have airway management equipment and backup readily available for his or her look at the child's oropharynx or the epiglottis in suspected cases of croup or mild epiglottitis.

In the adult, this risk is much smaller. Indirect laryngoscopy repeatedly has been demonstrated to be safe in the adult patient. Indeed, indirect laryngoscopy is appropriate in any patient with both sore throat and odynophagia. If a swollen, cherry red

epiglottis is seen, the diagnosis should be clear. This does require a very cooperative patient without airway compromise. It is most certainly not recommended for the child.

Another clinical finding that may prompt the examiner to look at the epiglottis is swelling of the uvula. Uvular swelling also may be associated with contiguous swelling of the tonsils, hypopharynx, and the epiglottis.[102,103] Dysphagia should not be ascribed to swelling of the uvula without adequate evaluation of the rest of the airway.

Lateral Radiograph of the Neck. The lateral neck film has been thought to be useful in diagnosis of the epiglottitis in the child. These radiographs may be obtained with a portable machine in the ED or operating room. Great care should be taken to not frighten the child during this procedure. The prudent emergency physician will stay with the patient and ensure that intubation equipment is at the bedside. If possible, the patient should not be sent to the x-ray suite. Radiographs should be considered optional diagnostic studies, unless there is no possibility of direct visualization of the patient's epiglottis.

Signs in children that have been described have included the "thumb sign," which occurs when the epiglottis is thickened and swollen or there is swelling of the aryepiglottic folds. Unfortunately, these films are neither very sensitive (about 40%) nor specific (about 75%) for the diagnosis. Indeed, radiographs may both delay diagnosis and provide misleading information.[104]

The radiologic diagnosis of supraglottitis is more subtle in the adult patient. Findings may include minimal to massive enlargement of the epiglottis, enlarged aryepiglottic folds, and enlarged arytenoids. The air pocket that runs parallel to the vallecula may be distorted (the vallecula sign).[105] Prevertebral soft-tissue swelling and ballooning of the hypopharynx may be present. At times the epiglottis may appear quite normal on radiographs of the neck.

Multiple studies have shown that there also is a significant risk (about 12-15%) of false-negative radiographs in the adult. Some reports suggest that films should not be emphasized in the diagnosis of this disease.[82,106,107] If the clinical impression suggests supraglottitis, the patient must be treated as if he or she has supraglottitis until the epiglottis and surrounding area has been visualized directly.

Fiberoptic Laryngoscopy. In the majority of cases, supraglottitis is diagnosed by findings on flexible laryngoscopy. In the adult, this can be performed in the ED. In the child, with the higher propensity for airway obstruction, this technique should be reserved for the operating room when everything is prepared for a rapid intubation.

Because nearly one-half of adults with epiglottitis have a coexisting pharyngitis on oral examination, any adult with severe sore throat and odynophagia deserves at least indirect visualization of the epiglottis with a mirror.[98] Adults generally tolerate indirect laryngoscopy well and this procedure can be safely performed in the adult without risk of precipitating acute airway obstruction. Indirect laryngoscopy should not be attempted in the child with suspected epiglottitis.

CT Scan. CT scanning may be used for the evaluation of the adult with epiglottitis. It probably is most useful to exclude complications such as abscesses. Although CT scan is not recommended as the primary means of establishing a diagnosis in the patient with supraglottitis, it is indicated in the patient when a direct inspection of the airway is not feasible. It should be limited to the patient who has a stable airway.

The CT scan displays the thickening of the epiglottis, the aryepiglottic folds, and the tissues around the glottis. The radiologist may note obliteration of the pre-epiglottic fat, thickening of the platysma, and abscess formation.

Management. Management of the adult patient with acute epiglottitis (supraglottitis) is controversial. There are many who believe that the adult form of epiglottitis is less aggressive than the pediatric variety. These physicians advocate a conservative approach with close monitoring and intravenous antibiotics. The opposing faction believe that adult epiglottitis has a high mortality rate when the airway is not aggressively protected. These physicians advocate tracheostomy or intubation in any patient with the disease. A third group feels that there is a subset of patients with rapid development of symptoms and a more severe course. The history of rapid onset of severe symptoms and tachycardia are the usual historical clues cited for identification of this group of patients. These patients almost surely will need aggressive airway management.

Airway. Since the most dreaded complication is airway obstruction, it should be the first priority for both adults and children. Have appropriately sized airway equipment ready at the bedside. Also be sure to have both cricothyrotomy and tracheostomy equipment present. Leave the patient in the most comfortable position possible. For a child, this may be in a parent's arms. Do not leave the patient alone. An appropriate medical attendant who can manage the airway must be at the patient's bedside at all times. This is usually the physician or someone who is trained in aggressive and invasive airway techniques.

As noted above, adults may require only observation and antibiotics.[82] In one larger series of adult epiglottitis, active airway intervention was necessary in only nine of 57 patients.[107] Eight of the patients were managed with intubation and one with awake tracheostomy. Not one patient who was initially observed required later intubation.

With an obstructed airway, the physician should attempt to assist respirations with a bag-valve-mask unit. The obstruction usually hinders *inspiration* and may be bypassed with forceful ventilation. If possible, perform the surgical airway in a controlled fashion. Get help from both anesthesia and ENT consultants. This airway *will* be difficult and help *will* be useful. The best place to get the airway is in the operating suite with the patient's neck prepped for a tracheostomy. This patient is not a candidate for rapid sequence intubation (RSI), unless a scrubbed ENT surgeon is standing by to do a tracheostomy. The patient should not be transported without a secure airway. The use of helium/oxygen (heliox) mixtures in a patient with epiglottitis may allow time for the arrival of more experienced help or bet-

ter equipment.[77] Heliox mixtures allow better oxygenation because the lighter gas flows more easily through the tighter airway passage.

Antibiotics. Ampicillin 200 mg/kg IV and chloramphenicol 100 mg/kg IV traditionally have been recommended for epiglottitis treatment. Second- or third-generation cephalosporins are now recommended because of the increased prevalence of beta-lactamase producing *H. influenzae.* Ceftriaxone has a long elimination half-life, excellent tissue penetration, and proven activity against *H. Influenzae*.[108] Ampicillin combined with sulbactam would be a reasonable alternative.

Steroids. The use of steroids to decrease inflammation has been recommended, but their use is controversial. There is little anecdotal evidence and there are no prospective studies that show that corticosteroids are useful for treatment of this disease.

Epinephrine. Inhalation of epinephrine may decrease the size of the epiglottis and allow more time for stabilization.

Bacterial Tracheitis

Description. Bacterial tracheitis probably represents a superinfection of viral croup with *H. influenzae, Streptococcus* or *Staphylococcus aureus* species. Some studies have shown occasional viral etiologies, including parainfluenza viruses, RSV, measles, and enterovirus.[109,110] The bacterial mucosal infection of tracheitis is associated with formation of an exudate and copious purulent secretions. If left untreated, up to 25% of these children would develop a completely obstructed airway and die.

Clinical Features. The clinical manifestations of bacterial tracheitis include features of both viral croup and epiglottitis. As with viral croup, a prodrome of upper respiratory infection may precede other symptoms. These symptoms are followed by the development of a croupy cough and upper airway obstruction. The patient often has a high fever and appears quite toxic. This may make the clinician lean toward a clinical diagnosis of epiglottitis. A harsh barking cough, quite like that seen in croup, will be noted as a distinguishing factor from epiglottitis. Patients with bacterial tracheitis have a slower course and less drooling than the patient with epiglottitis.

Auscultation of the lungs may reveal rhonchi or wheezes caused by the excessive tracheal exudates and by localized infiltrates in the lungs. Retractions are common. Stridor frequently is heard in these children.

Diagnosis. X-ray of the neck may show subglottic narrowing similar to croup's "steeple sign."[61] Other findings include opaque streaks or irregular margins on the lateral neck films. Chest x-ray may show interstitial infiltrates, pneumonia, bronchitis, or pneumonitis. Definitive diagnosis is made by direct visualization of the exudate in the trachea. This can be done during intubation of the child as purulent exudate is suctioned from the glottic opening. This material should be sent for Gram's stain and cultures. Other laboratory tests are of little use.

Management. Morbidity and mortality from bacterial tracheitis are primarily due to respiratory obstruction. The copious secretions may cause respiratory distress and rapid progression to complete airway obstruction and respiratory arrest. Other reported complications include pneumonia and pneumothorax. Toxic shock syndrome has been noted when *Staphylococcus aureus* is the cause.

Mortality rates are often cited in the 70% range, but in a meta-analysis of 11 studies comprising a total of 177 patients, the mortality rate was only 3.4%.[61] Intubation and tracheostomy were necessary in the majority of patients. (The preference for intubation vs tracheostomy appears to be center-dependent.)

In review of therapy for bacterial tracheitis, one major point does stand out.[61] Bacterial tracheitis almost universally is not responsive to the traditional medical therapies for viral croup. This includes humidification and racemic epinephrine. When the emergency physician has a child who appears toxic and does not respond to medical therapy for croup, he or she should start to think about bacterial tracheitis.

Close monitoring of the airway is the principle therapy and intubation usually is needed to prevent obstruction by the purulent exudate. Antibiotic therapy should be directed toward the likely pathogens and may include a penicillin, or analogue such as ampicillin, methicillin, nafcillin, cloxacillin, or dicloxacillin; gentamycin; chloramphenicol; or the third-generation cephalosporins. Appropriate changes in antibiotic therapy can be made following the results of initial cultures.

Because of the overlap in symptoms, the emergency physician should consider the diagnosis of bacteria tracheitis in all children with viral croup. Suspicion should be highest in those children who appear unusually toxic or fail to respond to standard therapies for croup.

Disposition. All of these children must be admitted to the hospital and have intubation performed in the operating room, as was described for epiglottitis.

Summary

Making the diagnosis of life-threatening "sore throats" can be extremely challenging. The differential diagnosis can be complex and includes pharyngitis or "sore throat." The emergency physician should focus on why this sore throat is different from other presentations (i.e., why the patient appears more toxic than expected, why the antibiotics given on a prior visit were not successful, and of course, whether there is a potential for airway compromise). With careful assessment and appropriate management of the patient's airway problems and infection, it is possible to reduce the mortality and morbidity associated with these diseases.

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Physician CME Questions

73. All of the following statements regarding epiglottitis are true *except*:
    A. Epiglottitis is more common in adults than in children.
    B. Children are more likely to require emergency airway management than adults.
    C. Early epiglottitis may mimic croup.
    D. Supraglottitis should be excluded from the differential diagnosis of any adult patient who presents with a sore throat and dysphagia.

74. In the adult with epiglottitis, which of the following would provide clues to the presence of the disease?
    A. Sore throat and dysphagia
    B. Fever
    C. Muffled voice
    D. All of the above

75. The *single most common* cause of death in Ludwig's angina is:
    A. myocardial disease.
    B. arrhythmia.
    C. paralysis of the diaphragm and respiratory muscles.
    D. airway compromise.

76. Croup is characterized by:
    A. a gray-green pseudomembrane covering the tissues of the throat.
    B. spasm of the glottis with closure of the crowded aryepiglottic folds.
    C. inflammation of the subglottic tissues.
    D. ulcerated, partly necrotic tracheal mucosa due to a bacterial infection.

77. A retropharyngeal abscess in an adult may be caused by all of the following *except*:
    A. trauma to the back of the oropharynx.
    B. inhalation of crack cocaine.
    C. intubation attempts.
    D. inflammation of the anterior paracervical nodes.

78. A helium-oxygen mixture may be a useful temporizing measure in which of the following illnesses?
    A. Croup and epiglottitis
    B. Retropharyngeal abscess
    C. Ludwig's angina
    D. Peritonsillar abscess

79. Corticosteroids for viral croup:
    A. are not effective unless they are given parenterally.
    B. should be given for at least five days in combination with an antibiotic.
    C. may alleviate symptoms and shorten the course of the disease.
    D. appear to have no literature support.

80. Nebulized epinephrine:
    A. has no place in the treatment of epiglottitis.
    B. requires the racemic form in order to be effective.
    C. may be followed by the "rebound effect," which worsens the clinical picture some 30 minutes to two hours after administration.
    D. may be followed by discharge to home after 30 minutes of observation.

In Future Issues: Drugs of Abuse

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

"Killer" Sore Throat

Conditions Presenting with Sore Throat and Inability to Swallow Saliva

- Epiglottitis
- Peritonsillar abscess
- Retropharyngeal abscess
- Ludwig's angina
- Abscesses in the deep neck space
- Allergic drug reactions
- Lingual tonsillitis
- Ingested foreign body with or without perforation
- Pharyngeal zoster
- Botulism
- Tetanus
- Stevens-Johnson syndrome
- Toxic epidermal necrolysis
- Inhalation or aspiration of toxic chemicals
- Tumors or trauma to the larynx
- Diptheria

Clinical Findings of Peritonsillar Abscess

- Severe pain
- Fever
- Dysphagia
- Trismus
- Hoarse, "hot potato" voice
- Enlargement of the tonsil
- Uvula deviated to the unaffected side
- Deviation of tonsil toward midline with rotation of anterior tonsillar pillar
- Fluctuance of the soft tissue between the upper pole of the tonsil and the soft palate

Predisposing Factors for Retropharyngeal Abscess

- Penetrating trauma: Foreign body such as pencil, Popsicle stick, or fishbone
- Tonsillitis
- Pharyngitis
- Peritonsillar abscess
- Croup
- Otitis media
- Nasal infections
- Dental infections
- Intubation
- Adenoidectomy (recent)

Complications of Retropharyngeal Abscess

- Rupture of abscess
- Airway rupture
- Asphyxiation
- Aspiration pneumonia
- Spinal rupture
- Lateral pharyngeal space rupture
- Inferior rupture into mediastinum
- Airway compromise

Scoring System* for Croup[75]

LEVEL OF CONSCIOUSNESS	
Normal or sleeping	0
Disoriented	5
CYANOSIS	
None	0
With agitation	4
At rest	5
STRIDOR	
None	0
With agitation	1
At rest	2
AIR ENTRY	
Normal	0
Decreased	1
Markedly decreased	2
RETRACTIONS	
None	0
Mild	1
Moderate	2
Severe	3

* Zero represents the normal state or absence of the sign and the highest number represents the most severe distress.

Clinical Findings of Epiglottitis in the Adult[99,100,107,111]

- Gradual onset of symptoms
- Rapid progression
- Severe sore throat (95-100%)
- Dysphagia (76%)
- Pain on swallowing (95%)
- Pain on palpation of the larynx
- High fever (88%)
- Muffled voice (50%)
- Shortness of breath or respiratory difficulty
- Drooling (relatively uncommon)
- "Upright" positioning
- Ear pain
- Toxic appearance

Clinical Findings of Epiglottitis in the Child[111,112]

- Sudden onset of symptoms
- Rapid progression
- High fever
- Muffled, "hot potato" voice
- Inspiratory stridor
- Sore throat
- Dysphagia
- Drooling
- "Tripod" positioning
- Toxic appearance

Supplement to *Emergency Medicine Reports*, May 7, 2001: "'Killer' Sore Throat: Prompt Detection and Management of Serious and Potentially Life-Threatening Causes of Pharyngeal Pain." *Author:* **Charles Stewart, MD, FACEP,** Emergency Physician, Colorado Springs, CO. *Emergency Medicine Reports' "Rapid Access Guidelines."* **Editor-in-Chief:** Gideon Bosker, MD, FACEP. **Vice President and Group Publisher:** Brenda Mooney. **Editorial Group Head:** Valerie Loner. **Managing Editor:** Suzanne Zunic. For customer service, call: **1-800-688-2421.**