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A "Killer" Sore Throat: Inflammatory Disorders Of The Pediatric Airway

"It's only a kid with a sore throat." The triage nurse said at 0100. You had a full ED and she assured you that the 13-year-old with a recent extraction of her wisdom teeth was fine. You put the sore throat to the back of the rack and took care of "more serious"cases. When you saw the patient four hours later, her respiratory rate was 36, her pulse was 160, and she had retractions at rest. You noted a substantial swelling of her anterior neck. You started her on high-flow oxygen, stat paged the ENT doctor, set up for a possible cricothyrotomy or tracheostomy, ordered blood cultures, chest x-ray, and neck x-ray, and told the nursing supervisor to get an OR crew in soon.

Sore throats represent one of the top ten presenting complaints to the ED in the US.[1] Many emergency physicians are jaded by the healthy appearance of the vast majority of patients with a triage note indicating a "sore throat." Since triage is an inexact science, these diseases don't come to you as carefully labeled packages… but simply as sore-throats or possibly pharyngitis, URI, or Flu.

A sore throat may be the hallmark of some of the most life-threatening diseases that we see as emergency physicians. Within this garbage can of disease labeled "sore throat," are life-threatening infections such as epiglottitis, tracheitis, croup, diphtheria and several of the deep neck abscesses. These diseases aren't common…

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CME Objectives

Upon completing this article you should be able to:

1. Describe the anatomy of the throat.
2. Discuss the potential causes of sore throats in pediatric patients.
3. Discuss the treatment options available for bacterial tracheitis, croup, diptheria, epiglottitis, peritonsillar abscess, Retropharyngeal abscess, and Ludwig's angina.
4. Evaluate, diagnosis, and treat the pediatric patient presenting with a sore throat.

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but then, some of them really aren't rare either. What is terribly frightening is that these patients can deteriorate rapidly and sometimes irretrievably; even when the initial symptoms of the disease are mild. This article will talk about airway obstructing infectious diseases: sore throats that truly kill.

Abbreviations Used In This Article

BET – Best Evidence Topics
CBC – Complete blood count
CT – Computerized Tomography imaging
ENT – Ear Nose, and Throat
GBS – Group B streptococcus species
HIB - *H. influenza* type B
MRI – Magnetic Resonance Imaging
RPA – Retropharyngeal abscess
RSI – Rapid Sequence Intubation
WBC – White blood cell count

Critical Appraisal Of The Literature

Medline, Ovid, Best BETs (Best Evidence Topics), Google Scholar, and Google were all searched using the terms epiglottitis, pediatrics, children, Ludwig's angina, deep neck space infections, retropharyngeal abscess, peritonsillar abscess, croup, diphtheria, infectious airway obstruction, and bacterial tracheitis. The terms were used in Boolean combination and separately in each database as appropriate. Over 1600 articles have been published in the last ten years on the combination of these subjects.

As might be expected in this disease process, there are few large, prospective, randomized, placebo-controlled studies of these diseases and their treatments. There were multiple retrospective studies, analyses of case reports, and many individual case reports and short series. A few small prospective studies of treatments were found that compared one treatment entity with another. These studies are all noted in the body of the text.

Epidemiology, Etiology, Pathophysiology

Anatomy

Anatomic differences between the pediatric and the adult airway make children more susceptible to acute airway compromise from infectious diseases. Because of the potentially high morbidity and mortality rates of these infections, the emergency physician must have a good understanding of the anatomy of the throat, and the causes and treatments of sore throats that can obstruct the airway.

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 them can spread easily and rapidly. Spreading of infection in the neck occurs by continuity along the path of least resistance – the fascial plane and the potential spaces. This spread can include swelling that surrounds the airway, the great vessels, and the lower cranial nerves.

Unfortunately, each anatomist describes the layers of the neck and cervical fascia using different terminology that muddles an already complicated subject. It seems that every time you learn the nomenclature of the neck, another paper gives another set of synonyms. In this paper, the terms accepted by the otolaryngologist will be used since, if an infection is found, definitive therapy will most likely be the province of the otolaryngologist. There are two main divisions of the cervical fascia: the superficial layer and the deep layer. (Please see the table below).

The potential spaces of the neck can be divided into groups that relate to the hyoid bone. There are six suprahyoid spaces, one infrahyoid space, and five spaces that span the length of the neck.

The spaces that span the entire neck allow communication into the mediastinum and deep structures of the back and chest.

Neck spaces are interconnected with each other and also communicate with the mediastinum so infections can spread easily to a variety of areas. Common clinical conditions which can occur in these

Fascial Layers Of The Neck

The superficial cervical fascia has no subdivisions

The superficial cervical fascia lies beneath the skin and is superficial to the platysma muscle of the neck.

The deep cervical fascia has three subdivisions:

Anterior layer (superficial) - surrounds the sternocleidomastoid, trapezius muscles and strap muscles.

Middle (visceral) - envelopes the trachea, larynx, and hypopharynx.

Deep (prevertebral) fascia - runs posterior to the esophagus and great vessels, and ensheathes the prevertebral musculature.

spaces are retropharyngeal abscesses, parapharyngeal abscesses, and infections of the sublingual and submental space such as Ludwig's angina.

The upper airway consists of the nasal cavity, the oral cavity, and the pharynx. With epiglottitis, the pharynx is the area of infection.

The pharynx is a muscular tube that extends from the soft palate to the esophagus and the trachea. It contains the nasopharynx, the oropharynx, and the juncture of the 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, the cricoid cartilage, the proximal trachea, the vocal cords, and the 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; this is the most narrow part of the adult patient's airway. In the child, the subglottic area is the most narrow area in the airway.

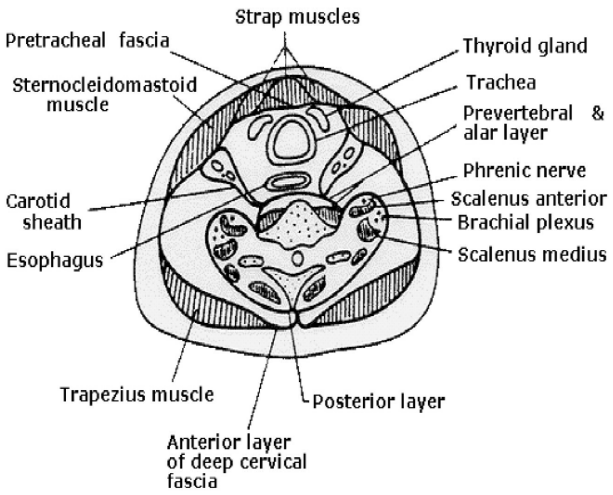
Any inflammation in the child's subglottic area greatly reduces the airway diameter. The magnitude of this airway compromise can be approximated if the provider remembers that the cross sectional area of a cylinder is proportional to the square of the radius (πr^2). The percentage of airway compromise can then be calculated by the following formula: πr^2 narrow / πr^2 normal x 100 = percentage of remaining airway compared with the normal airway. If a child's subglottic opening has a diameter of approximately 5m, only 0.5m of swelling will decrease the airway to 64% of the original cross section. (22/2.52 x 100).

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. This nerve 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-

Figure 1.



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Fascial Spaces That Span The Neck

The superficial space.
The prevertebral or retropharyngeal space is between the prevertebral musculature and prevertebral fascia. It is continuous with the mediastinum. It contains the retropharyngeal lymph nodes that typically atrophy after the age of five.
The danger space extends from the skull base to the diaphragm. The anterior border is the middle deep cervical fascia and the posterior border is the prevertebral layer of the prevertebral fascia.
The prevertebral space extends from the skull to the coccyx. The anterior border is the prevertebral layer of the prevertebral fascia. The posterior border is the anterior longitudinal ligament of the vertebral bodies.
The visceral vascular space is the potential space within the carotid sheath.

Suprahyoid Fascial Spaces

The temporal space contains the internal maxillary artery and the mandibular nerve.
The masticator space contains the masseter and the pterygoid muscles, the body of the mandible, and the inferior alveolar nerves and vessels.
The peritonsillar space is bounded by the capsule of the tonsil and the superior pharyngeal muscle medially.
The parotid space communicates with the parapharyngeal space above the parotid gland.
The pharyngomaxillary space is also known as the parapharyngeal space or the lateral pharyngeal space.
The submandibular space is bounded by the mandible anteriorly and laterally, and the lingular mucosa superiorly. This is the space affected by Ludwig's angina. Swelling is limited by the hyoid bone inferiorly.

hemolytic streptococci and *Staphylococcus aureus* microorganisms. Many abscesses are polymicrobial and include both gram-positive and gram-negative aerobic and anaerobic organisms. The most common of these are the Bacteroides I 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.

Differential Diagnosis Of The Life-threatening Sore Throat

Bacterial tracheitis

Description

This disease has multiple names: bacterial laryngotracheobronchitis, membranous laryngotracheobronchitis, and pseudomembranous croup.

Bacterial tracheitis is a disease of children that probably represents a superinfection of viral croup with *H. Influenza*, *M. catarrhalis*, *Corynebacterium diphtheriae*, *Streptococcal*, or *Staphylococcus aureus* species.[2,3] Some studies have shown occasional viral etiologies including parainfluenza viruses, RSV, measles, and enterovirus.[4,5] The bacterial mucosal infection of tracheitis is associated with the formation of exudate and copious purulent secretions. Untreated, up to 20% of these children's airways will become completely obstructed and the child will die.

Clinical Features

The clinical manifestations of bacterial tracheitis include features of both viral croup and epiglottitis. As with viral croup, a prodrome of URI may precede other symptoms. These symptoms are followed by the development of a croupy cough and upper airway obstruction with stridor. The patient often has a high fever and appears quite toxic. S/he has progressive respiratory distress which does not improve with the inhalation of racemic epinephrine and systemic steroids.[6] This may make the clinician lean towards 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 is frequently heard in these children.

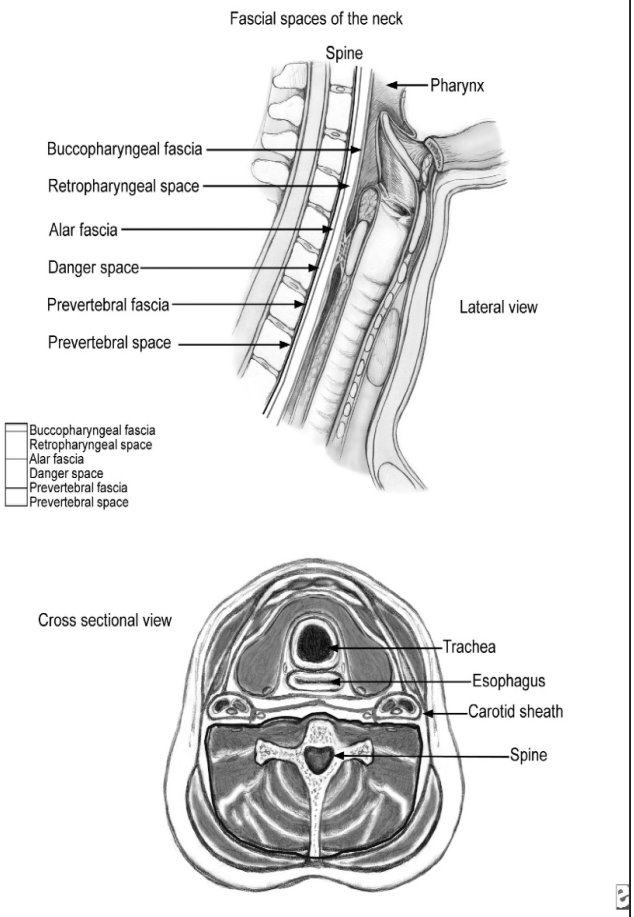
The disease is most prevalent in the winter months. Bacterial tracheitis has been reported from age 3 weeks to 16 years.[2,6] The mortality of tracheitis has been reported from 0 to 20% with the highest mortalities from the older literature.[3,6]

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.[7] The variety of terms has contributed only to a

Figure 2.



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sense of confusion about this infraglottic infection as multiple terms have been used by different authors to describe identical clinical conditions. The clinical term, "croup" is now used almost exclusively for non-bacterial croup.[8]

The original definition of croup was synonymous with diphtheria and had a mortality rate of about 25%. During the 1900's the term croup expanded to include other infections (including epiglottitis).[9] During the latter half of the 1900's, reports of viral etiologies became commonplace and bacterial causes of croup seemed to disappear.[10] The reasons for the shift from bacterial to viral infections is unknown, but may be due to a host of factors, including increased immunization, antibiotic usage, or even a change in bacterial virulence.

Spasmodic And Viral Croup

Spasmodic croup is a term used to describe an entity that shares many clinical features of viral croup. Spasmodic croup is generally considered less severe, more acute, and may be more responsive to simpler therapies.

Spasmodic croup has traditionally been differentiated from viral croup by absence of fever and a characteristic nocturnal onset. The viral prodrome may or may not be present. There is an ongoing debate about whether spasmodic croup and viral croup are separate conditions or simply different parts of a spectrum of disease.[11] The treatment for spasmodic croup is generally the same as for viral croup, and the etiologic organisms are also the same, so differentiating between the forms is not necessary.[11,12] [11]

Conditions Presenting With Sore Throat And Inability To Swallow Saliva

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

The exact frequency of viral croup in the general population is not known because many mild cases are often treated at home without seeing a healthcare provider or by following advice over the telephone. There is good epidemiologic data collected from pediatricians in outpatient practice.[13-15] There were no cases reported in the first month of life, but 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 to 2 year age group.[14] The male:female ratio of occurrence is 1.5:1 (about 40% more often in boys). After two years of age, the rate decreases and the disease becomes uncommon by school age. More than 80% of cases occur in the first five years of life. By age six, the incidence drops to < 0.5% per year. Although croup is much more common in younger children, it can be diagnosed in older children and adolescents.[16]

The microbiology of viral croup has been well established. Parainfluenza (types 1, 2, and 3) viruses represent about half of the documented cases of croup.[11,14,17] RSV is the cause of about 10% of cases of croup. Influenza causes about 6% of cases and is more common in older children. Measles was identified as a cause of croup in the 1980's but is rare now that children are routinely immunized.[18,19]

Mycoplasma I species may be identified in older children. *Mycoplasma pneumoniae* is a relatively rare cause of croup.

The disease clusters in spring and fall and mirrors the seasonal pattern of respiratory viruses in general. The fall peak coincides with parainfluenza virus infection rates and the winter peak mimics RSV infection rates.

Clinical Features

A case of viral croup usually starts with the signs and symptoms of a mild viral URI (low grade fever, sore throat, cough, and rhinorrhea).

Within a few hours, the distinctive barking cough, often described as being similar to a seal's bark, will develop. The child may also 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 identified clinically by cyanosis or simply restlessness.

Wheezes, expiratory rhonchi, and crackles may be heard in the lungs.

The disease is often worse at night. The child with croup should have no dysphagia and no drooling. Unfortunately mild (early) epiglottitis may mimic croup.

Clinical Croup Scores

The ideal croup score would assist in identifying and quantifying respiratory distress, help determine initial and subsequent therapy, and determine the need for admission. Multiple variants of croup "scoring" systems have been designed in an attempt to create an objective measure of severity and allow comparison between serial assessments of the patient. The Westley croup score is, perhaps, the most common scoring instrument that has been used in either the original or modified form in many randomized clinical trials.[20] In the determination of the severity of the child's airway obstruction, the score incorporates an assessment of the level of consciousness, color of the patient (presence or absence of cyanosis), stridor, presence of retractions, and air entry. Stridor at rest is an indicator of the highest severity of croup in all croup scoring systems. (See the table on page 7).

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.[21]

Most patients will do well in the outpatient setting. The annual incidence of croup in patients less than six years old ranges from 1.5% to 6% with admission rates from 1.5% to 31% depending on the admission practices and the severity of the illness in the population.[14,22] These figures may drop with the widespread adoption of steroids in treatment of croup. (See treatment section beginning on page 20).

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, then admission is warranted. Patients less than one year of age should be considered for admission because they have such small airways.[22] Children with stridor at rest should also 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.[23] A gentle and smooth intubation by the most skilled practitioner is appropriate. All trauma can worsen the airway obstruction and should be avoided.

Diphtheria (True Croup)

Description

Diphtheria is an infection of the throat, nose, ears, and occasionally skin. Pharyngeal infections may be toxic or asymptomatic depending on the presence of a toxin producing gene. Nasal, skin, and ear infections are often asymptomatic and may create a carrier state. *C. diphtheriae* has three biotypes: gravis, intermedius, and mitis. The most severe disease is associated with the gravis biotype, but any strain may produce toxin.

Friedrich Löffler first isolated the diphtheria bacillus in pure culture in Robert Koch's laboratory.[24] 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 that the organ damage was caused by the diphtheria toxin led to development of both an effective anti-toxin based therapy for actual infection and a toxoid vaccine to prevent infection.

C. diphtheria is a nonsporulating, unencapsulated, nonmotile, pleomorphic gram positive bacillus. *C. diphtheria* elaborates a toxin with 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 the most prominent effects are on the heart (myocarditis), nerves (demyelination), and kidneys (tubular necrosis).

Humans are the only known reservoir for *C. diphtheriae*. Asymptomatic nasopharyngeal carriers are common when immunization is slack. The primary modes of spread are via airborne respiratory droplets and direct contact with either respiratory

secretions or exudate from infected skin lesions. Fomites can play a role in transmission, and epidemics have been caused by contaminated milk. Most respiratory tract diseases occur in the colder months in temperate climates, 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.

Diphtheria is now rare in the US due to extensive immunization.[25] From 1980 through 2004, 57 cases of diphtheria were reported in the US, an average of 2 to 3 per year (range, 0 to 5 cases per year). Only five cases have been reported in the US since 2000.[26] The case fatality rate in the US from 1980 to 1995 was 10% with all deaths occurring in patients nine and under.[27] All four children who died had no vaccination and none of the patients who contracted the disease were adequately vaccinated.[27]

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. The widespread availability of diphtheria toxoid led to a marked decrease in the incidence of diphtheria and in circulating toxigenic *C. diphtheria* organisms resulting in less natural boosting of immunities.[28] Since 1990, epidemic diphtheria has reemerged in Russia and other areas where social disorganization has relaxed immunization practices. The 1993 to 1994 diphtheria epidemic in Russia had over 150,000 cases with over 5,000 deaths.[29] High case fatality rates, a large proportion of patients with complications, and occurrence in both young and older age groups characterized this outbreak.[30]

With immigration from these countries and declining rates of immunization both abroad and in the US, it is entirely possible that a US emergency physician may see this disease in his or her ED in the 21st century.[28]

Scoring System For Croup[20]

• 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. The highest number represents the most severe distress.

Clinical

The incubation period for patients with *C. diphtheria* is about one week. The disease may be quite mild or may be progressive and lethal. The onset of the disease is usually abrupt with low-grade fever, malaise, sore throat, pharyngeal injection, and the development of the membrane typically in one or both tonsils. 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. Chest films may show pneumonia.

Within the first few days of respiratory tract infection, local toxin causes tissue damage in the immediate area of the infection. 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 pseudomembrane involving the trachea and bronchi can cause respiratory distress. These patients may appear anxious and cyanotic, may use accessory muscles of respiration, and may have inspiratory intercostal, supraclavicular, and substernal retractions. Local swelling of nodes and neck tissues may give a "bull neck" appearance. In both adults and children, a common cause of death is suffocation after aspiration of the membrane.

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 intubation or tracheostomy, 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.

Systemic complications are due to 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.[31] Characteristically, the toxicity occurs after 1 to 2 weeks of illness, often when the local oropharyngeal disease is improving. Clinically, myocarditis can present acutely with congestive failure and circulatory collapse, or more slowly with progressive dyspnea, weakness, and cardiac dilatation. The EKG may show ST-T wave changes and first-degree heart block. These EKG changes can progress to more severe forms of block, atrioventricular dissociation, and other arrhythmias, which carry an ominous prognosis.[32,33]

Up to three-fourths of patients with severe diphtheria develop neuropathy.[31,34,35] Early in 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 causing oculomotor and ciliary paralysis are also common. Dysfunction of the 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 the disease in the throat. If the patient has recently been vaccinated, neuropathy may be milder.[36]

Mortality rates vary from 3.5% to 12% and have not changed in the last 50 years.[31] Rates are highest in very young and very old patients. Most deaths occur in the first 3 to 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.[37]

Epiglottitis (Supraglottitis)

Description

Epiglottitis, or more correctly supraglottitis, is cellulitis of the structures above the glottis: the epiglottis, the aryepiglottic folds, the arytenoid soft tissues, and the uvula. In children, the disease is usually confined to the epiglottis and closely surrounding structures; while in adults, the inflammation may also involve the prevertebral soft tissues, the valleculae, the base of the tongue, and the soft palate.[38]

A description of epiglottitis was recorded in the 13th century. The first accurate clinical description of epiglottitis was noted in 1900 by Theisen; he called it "angina epiglottidea anterior."[39] The association of acute laryngitis and *H. influenza* type b bacteremia was noted first in the English literature in 1941.[40]

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

Clinical

The classic pediatric clinical course is familiar to family physicians, emergency physicians, otolaryngologists, and pediatricians. The child presents with a rapid onset of severe sore throat, fever, toxic appearance, and difficulty managing secretions (drooling). The sore throat may be expressed as refusal to eat in the younger child. The combination of a sore throat and dysphagia is a significant clue to the presence of this disease. In fact, a severe sore throat is almost universal. Cough is not a prominent finding in either children or adults. Other findings include respiratory distress, anterior neck tenderness, and hoarseness.

The child goes from anxiety to tripod posturing (sitting and leaning forward with elbows on thighs) to respiratory compromise. This toxic clinical picture continues to rapidly progress to respiratory obstruction and then death within hours. The total duration of the disease prior to hospitalization is usually less than 24 hours and may be as short as two hours.[41]

The specific cause of the airway obstruction is not always clear. Several theories exist. 1) The swollen floppy epiglottis of the child obstructs the glottic opening. 2) Laryngospasm occurs from the inflammation surrounding the glottis. 3) 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.

Pathology

For many years, *H. influenza* type B (HIB) has been the most common etiology of epiglottitis in both children and adults. In 1994, 95% of epiglottitis cases in children and 53% of adult epiglottitis cases were caused by HIB.[42] This has changed with the increasing penetrance of HIB immunization.[43-45] The reported incidence has decreased from 3.47 cases per 100,000 children in 1980 to 0.63 cases per 100,000 children in 1990.[41]

It is important to remember that there are case reports of HIB epiglottitis occurring in children despite vaccination. Other serotypes of HIB may also cause serious invasive disease, including epiglottitis.[48] 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 group A, B, and C *streptococcus*.[49-51]

Group A streptococcus is probably the most common etiology in those areas where HIB immunization has been given. There has been an increasing emergence of severe infections in the US caused by group A streptococcus since the late 1980's. There is also an association of group A streptococcus with varicella as an etiology of epiglottitis.[52,53]

Of the pathogens, there is general agreement that HIB will cause an acute onset, correlates well with clinical severity, and is 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 epiglotittis, the course is longer with a mean intubation time of 6 to 7.5 days compared to the usual 1.5 to 3 days required for HIB.[50,51,54,55] There appears to be more involvement of the aryepiglottic folds, and the cherry red epiglottis found in HIB infection is replaced by a pale edematous epiglottis when streptococcal species cause the disease.[54] Other findings suggestive of streptococcus epiglottitis include a somewhat older age of onset, presence of fever, negative blood cultures, and slower resolution of the disease.[50,51,54,55]

Epiglotittis is also reported to occur in patients with acquired immunodeficiency syndrome.[56] A wider range of organisms can cause the disease, including *Aspergillus*, *Klebsiell*a, 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.[57-59] A history of ingestion of hot food or liquid may be elicited by the clinician. Smoking cocaine free-base (crack) may also precipitate thermal epiglottitis. Although these cases are described in the adult literature, much use of illicit drugs occurs in the adolescent population and this etiology of epiglottitis can occur in the older pediatric population.[59]

Clinical Findings Of Epiglottitis In The Child[168,169]

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

Note that these symptoms may be muted and the progression of the disease significantly slower when the etiology is other than HIB.

Peritonsillar Abscess

Desciption

Peritonsillar abscess or "quinsy" is the most common deep infection of the head and neck in the adult and older child. Peritonsillar abscess usually occurs in teenagers and young adults, but can occur at any age. It is more common in immunosuppressed, immunodeficient patients and diabetic patients. It can also occur after mononucleosis.[60-62] The incidence is about 30 per 100,000 people and it is uncommon in the very young child.

This disease develops by a spread of bacterial tonsillitis to the peritonsillar space between the tonsil and the surrounding superior constrictor muscles. It is often the endpoint of a spectrum of infection proceeding from tonsillitis to peritonsillar cellulitis and then on to peritonsillar abscess.[63] Rarely, a peritonsillar abscess may occur in a patient without any prior history of tonsillitis. The microbiology is predomi-

nantly a mixed infection with both aerobic and anaerobic bacteria. The streptococcal species are the most common (group A or group B). *Staphylococcus aureus*, fusobacterium, and anaerobic gram negative rods are also found.

Clinical Findings of Peritonsillar Abscess

- Deviation of tonsil toward midline with rotation of anterior or tonsillar pillar
- Dysphagia
- Enlargement of the tonsil
- Fever
- Fluctuance of the soft tissue between the upper pole of the tonsil and the soft palate
- Hoarse, "hot potato" voice
- Severe pain
- Trismus (in 60% of cases)[64]
- Refusal of food and, in severe cases, liquids

Complications

There is a real potential for serious complications in this relatively common illness. Local complications include extension of the abscess into the neck and possible catastrophic hemorrhage from erosion into the carotid artery. In some cases, the airway is compromised from laryngeal edema. Minor bleeding from the nose, ear, or mouth or ecchymosis of the neck usually heralds an arterial rupture with massive bleeding.[65] The abscess may cause septic thrombosis within the internal jugular vein. Mediastinitis, sepsis, and necrotizing fasciitis have all been described in this disease.[66] If rupture occurs, aspiration and subsequent pneumonia or lung abscess may occur.[67]

If the patient has a prior history of tonsillitis, about 20% of adults and 7% of children will have a recurrence of the abscess.[68] Because children have a lower recurrence rate than adults, tonsillectomy is recommended for adults, but may not always be needed in children.

Retropharyngeal Abscess

Description

A retropharyngeal abscess is an accumulation of pus in the retropharyngeal space. This area is bounded anteriorly by the posterior pharyngeal wall and posteriorly by the prevertebral fascia. The space is bounded superiorly by the base of the skull and inferiorly where the anterior and posterior layers of fascia fuse. (This fusion occurs at the level of C7-T1, within the superior mediastinum). This space 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 less than four years old and normally regress by the age of six.

In the pre-antibiotic era when retropharyngeal abscess was more common, several series reported that over 95% of cases occurred in children under the age of six.[69-72]

Abscess formation usually follows an upper respiratory infection with suppuration of the involved retropharyngeal lymph nodes. The disease can also occur as a result of trauma to the posterior pharynx.[73-75] A common mechanism of trauma occurs when a child with a stick or toy in their mouth falls and punctures or lacerates the posterior pharyngeal wall. The child is often seen in the ED for this initial insult. Use of crack cocaine has also caused retropharyngeal abscesses in young adults.[76] Other insults that can cause retropharyngeal abscess include endoscopy or endotracheal intubations.[77] In older children and adolescents, ingestion of a foreign body, a recent endoscopic procedure, and orotracheal intubation, or/and external neck trauma may cause retropharyngeal space infections.

Predisposing Factors For Retropharyngeal Abscess

- Adenoidectomy (recent)
- Croup
- Dental infections
- Intubation
- Nasal infections
- Otitis media
- Penetrating trauma (foreign body such as a pencil, popsicle stick, or fish bone)
- Peritonsillar abscess
- Pharyngitis
- Tonsillitis

Retropharyngeal Abscess Complications

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

The intense inflammation can also cause an acute inflammatory torticollis. The 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 can be dislocated by this intense muscle spasm.[82] A CT or MRI may be needed to determine the cause of the torticollis.

Submandibular Cellulitis And Abscess - Ludwig's Angina

Description

Ludwig's angina is a rapidly spreading cellulitis of the floor of the mouth involving the submandibular, sublingual, and submaxillary (submental) spaces. The submandibular space is divided into the sublingual space and the submaxillary space (also called the sub-mylohyoid space). The submaxillary space is further divided into two communicating compartments; the central submental space and the lateral submaxillary space. Since these spaces communicate with each other, they are effectively a single unit. Infection in these spaces is halted by the mandible anteriorly and the hyoid bone inferiorly. Swelling can only expand upwards and outwards causing distortion of the floor of the mouth (the "bull neck" appearance) and, finally, respiratory obstruction.

Wilhelm Frederick von Ludwig described this fast spreading, lethal, gangrenous induration of the connective tissues of the neck and floor of the mouth in 1836 in a case report of five patients.[83,84] Other causes of submandibular abscess include infection of the submandibular gland and suppuration of the submandibular lymph nodes.[85] In the pre-antibiotic era, the mortality exceeded 50% even with surgical drainage of the area. Aggressive surgical intervention and the introduction of antibiotics in the 1940's resulted in a reduction of the mortality of this disease to about 10%.

Ludwig's angina, or necrotizing fasciitis, usually begins in the submandibular space; dental disease is the most common cause.[86] An infected or recently extracted lower second or third molar are the most common causes.[87] Because the roots of these teeth protrude below the mylohyoid ridge, a periapical tooth abscess may easily spread into the submandibular space. Ludwig's angina may also occur after lacerations and infections of the floor of the mouth, salivary calculi, and mandibular fractures.

The most common pathogen is viridans group *Streptococcus*, followed by *Staphylococcus aureus*, and *Staphylococcus epidermis.*[88] Anaerobes, mostly bacteroidis, are implicated in about 40% of positive cultures. *E. Coli*, *Pseudomonas*, and *H. influenzae* have also been reportd.[88]

Clinical

Ludwig's angina is uncommon in children but certainly does occur.[84,88-90] In a study spanning a 13 year period, Ludwig's angina was diagnosed in 41 patients of whom 24% were children.[91] Ludwig's angina has been reported in infants as young as 12 days.[84] 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.[92] In children, Ludwig's angina can occur without any apparent precipitation cause.[91]

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

Figure 3. Retropharyngeal Space Enlargement

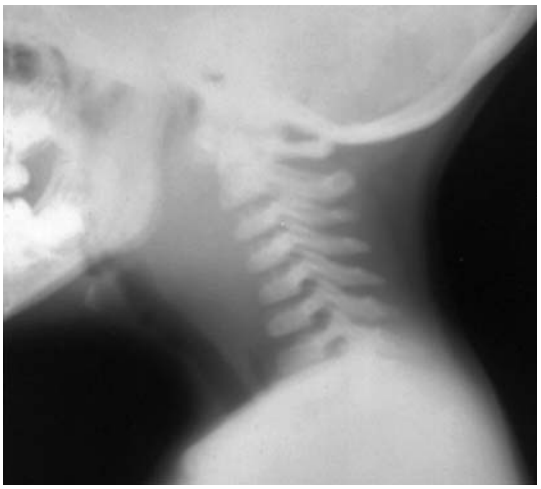

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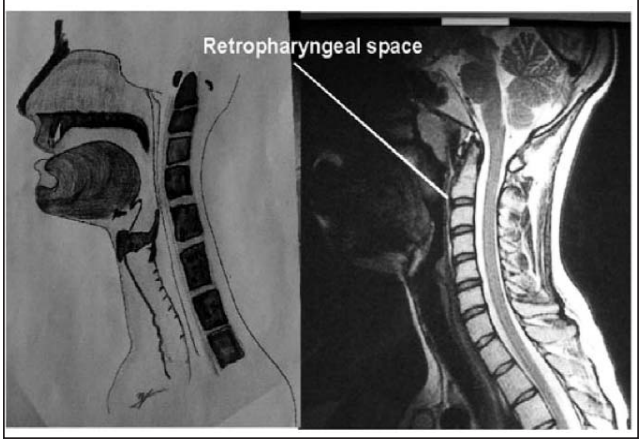
Figure 4.



secretions, and fever are frequently seen as well.

Ludwig's angina often progresses quite rapidly. The patient may suffer an acute respiratory arrest if forced to lie supine. The mortality in these cases may exceed 50%.[86,93] When Ludwig's angina is rapidly and appropriately treated, the mortality is between 0 and 8.5%. The causes of death include pneumonia, mediastinitis, sepsis, empyema, and respiratory obstruction.[88]

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.[94] If the abscess ruptures intra orally, then pus may be aspirated resulting in pneumonia and possible lung abscess. Tongue necrosis has also been reported.

Pre-Hospital Care

Initial medical care of the child with a sore throat and potential respiratory distress:

- Do not place anything in the mouth to visualize the pharynx.
- Do not start IV unless the child presents in impending arrest.
- Allow the child to be transported in a position of comfort such as the mother's arms, if this quiets child.
- Monitor EKG for changes in heart rate. Bradycardia signals deterioration.
- Transport rapidly, but without lights and sirens unless the child has impending arrest.

If the child appears to have croup, then there may be some benefit from saline mist from nebulizer mouthpiece. This benefit may be entirely psychological but the parents may expect it. If the child is wheezing, he or she may benefit from albuterol 2.5mg (3cc) or Xopenex™ 1.25mg (3cc) via nebulizer mouthpiece, mask, or aiming mist at child's face. Supplemental oxygen should be provided at 6L per minute. Administer oxygen by having a caregiver hold a mask or tube near the patient's mouth and nostrils. The EMS provider should not delay transport while waiting for a response from any of these therapies.

If the child has bradycardia, altered mental status, marked stridor/ventilatory distress, retractions, ineffective air exchange, and/or actual or impending respiratory arrest, then the child has an unstable airway. In the unstable child, using epinephrine (1:1,000) 3ml (3mg) via a nebulizer mask or aiming mist at the child's face with oxygen at 6L may be helpful.

If the child has a respiratory arrest, attempt to ventilate the child with bag-valve-mask using slow compressions of bag. In most patients, jaw-thrust combined with bag-valve-mask oxygenation usually provides adequate ventilation in patients with acute, infectious, upper-airway disease. The obstruction may possibly be overcome with gentle pressure during bagging, but in at least 3 cases, this has not worked.[95] Prepare for emergency intubation of the child, but do not attempt intubation unless unable to ventilate the child with the bag-valve-mask.

If unable to ventilate, the medic should stop the ambulance and attempt a single try at oral endotracheal intubation. Ensure that cricothyroidotomy and suction equipment are at hand. If this intubation is unsuccessful, the medic should perform needle cricothyroidotomy.

Emergency Department Evaluation

The ED evaluation is described in detail for each of the entities that compromise the mortally infectious sore throat. The history and physical examination are your opportunity to exclude a number of diseases in the child with a sore throat that can be serious or life-threatening.

Since the most important complication is airway obstruction, this is where the emergency physician should plan for problems. This must be the first priority for both adults and children. Put appropriately sized airway equipment at the bedside. Be sure to have both cricothyrotomy and needle airway equipment present. If the clinician is trained and feels comfortable with tracheostomy, this is an option.

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, ever. 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.

Do not transport the child without a secure airway; preferably already in the patient. A (distant) second choice is to have the airway equipment at the bedside in the ambulance, ready to go and with competent help to put it in. Securing a difficult airway in a critically ill child in a moving ambulance is not an easy task. Bright lights and cold steel may be need-

ed for this airway and these are not easy to acquire in most transport vehicles.

Diagnostic Studies

Specific Disease Entities

Diagnosis Of Bacterial Tracheitis

This disease should be suspected in a child with cough, fever, stridor, and a toxic appearance who does not respond to steroids and inhaled epinephrine. The child with bacterial tracheitis may have a requirement for supplemental oxygen that is not needed in most children with croup.[6]

The diagnosis of bacterial tracheitis is based on the presence of upper airway obstruction and one of the following: Visual inspection of the airway revealing purulent secretions exuding from below the vocal cords; radiographic evidence of intra-tracheal membranes; tracheal aspirate positive on gram stain or positive tracheal aspirate culture.[3]

Radiology

X-ray of the neck may show subglottic narrowing similar to croup's "steeple sign."[10] Other findings include opaque streaks or ragged, irregular margins of the airway on the lateral neck films. These tracheal irregularities may be seen in 20% to 80% of patients on lateral x-ray of the neck.[6] Chest x-ray may show interstitial infiltrates, pneumonia, bronchitis, or pneumonitis.

Culture

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 or by fiberoptic bronchoscopy.[96] This material should be sent for gram stain and cultures.

Other laboratory tests are of limited use in the diagnosis of bacterial tracheitis. The CBC may show a bandemia. There are reports of positive blood cultures when the causative organism is *H. influenza*, but this etiology is uncommon in the immunized child.

Diagnosis of Croup

Clinical

For the emergency physician, the typical case of croup is a diagnosis based on clinical findings rather than laboratory, radiographic, or microbiologic studies. Clinically, it is characterized by a distinctive cough (the so-called seal bark) that is often accompanied by a hoarse voice. In more severe cases, the child will have inspiratory stridor and respiratory distress. 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. The severity of croup ranges from a mild, self-limited disease (the cough) to severe airway obstruction requiring intubation and intensive care with the risk of death. If the child has signs of significant airway distress, then the setting should be completely controlled and additional diagnostic studies minimized.

Laboratory

Generally, the laboratory and radiology services do not contribute to the diagnosis of croup.

Nonspecific blood tests, such as WBC or erythrocyte sedimentation rates, may be elevated in quite a number of acute infectious diseases and 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 caused by bacterial infections.[97] In a relatively large study, about half of patients with bacterial or mixed viral and bacterial infections had a CRP of 20mg/L or more whereas only 35% of patients with viral infection alone had a CRP of more than 20mg/L.[98] This difference is clinically insignificant and does not appear to alter management. The non-utility of CRP has been validated by both data mining and retrospective studies.[99,100]

Likewise, 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 performed on nasal washings or sputum. In older children, nasopharyngeal swabs may show *Mycoplasma* species.

Radiology

X-ray studies are often ordered but are usually 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 may also help rule out epiglottitis and aspiration of a foreign body.

Diagnosis of Diphtheria

Culture

Culture is the gold standard for diagnosis of diphtheria. Unfortunately, diphtheria is not easily identified on blood agar used for streptococcal screening.

A culture medium containing tellurite is preferred because it provides a selective advantage for the growth of this organism. Swabs from specimens from asymptomatic carriers or contacts may have only small numbers of organisms that are easily obscured by the overgrowth of normal throat flora.

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 stain, distinctive black colonies with halos on Tindale's (telluride) medium, and the presence of metachromatic granules allows a presumptive identification of *C. diphtheriae*.

Polymerase Chain Reaction Detection

Not all colonies of diphtheria will produce toxin. Toxin production is normally demonstrated by Elek plate precipitin strips or by polymerase chain reaction testing for the toxin A subunit gene.[101,102] 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 start producing toxin.[103]

Diagnosis of Epiglottitis

Clinical *(See table on page 9).*

Pediatric patients may have a fulminant course with complete airway obstruction in as little as 30 minutes.[104] Patients who present with tachycardia, an increased white cell count, and a history of rapidly progressive sore throat are at a significantly increased risk of airway obstruction.[105] These patients should be treated aggressively.

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. This notion is supported only by anecdotal data. Note that the pediatric patient with advanced epiglottitis 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.[7,106] The emergency physician would still be wise to have airway management equipment and backup readily available when looking at a child's oropharynx or epiglottis in suspected cases of croup or mild epiglottitis.

Another clinical finding that may prompt the examiner to look at the epiglottis is swelling of the uvula. Uvular swelling may also be associated with contiguous swelling of the tonsils, hypopharynx, and the epiglottis.[107,108] Dysphagia should not be ascribed to swelling of the uvula without adequate evaluation of the rest of the airway.

Radiology

The lateral neck film has been thought to be useful in the diagnosis of the child with epiglottitis. These radiographs may be obtained with a portable machine in the ED or the operating room. Great care should be taken to not frighten the child during this procedure.

The emergency physician should stay with the patient at all times and ensure that intubation equipment is at the bedside. Do not send the patient to the x-ray suite if you can avoid it. Radiographs should be considered optional diagnostic studies, unless there is no possibility of direct visualization of the patient's epiglottis, such as in a small hospital without OR, ENT, or pediatric support.

Signs in children that have been described have included the "thumb sign" where the epiglottis is thickened and swollen, or swelling of the aryepiglottic folds. Unfortunately, these films are neither very sensitive (about 40%), nor specific (about 75%) for the diagnosis. The air pocket that runs parallel to the vallecula may be distorted (the vallecula sign).[109] 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, despite clinical findings on fiberoptic laryngoscopy.[110]

The radiologic diagnosis of epiglottitis is more subtle in the older patient. Findings may include minimal to massive enlargement of the epiglottis, enlarged aryepiglottic folds, and enlarged arytenoids. Indeed, radiographs may both delay and provide misleading information.[110]

If the clinical impression suggests epiglottitis, then the patient must be treated as if s/he has epiglottitis until the epiglottis and surrounding area has been directly visualized.

CT Scan

CT scanning may be used in the evaluation of the older patient with epiglottitis. It is probably most useful to exclude complications such as abscesses. Although CT is not recommended as the primary

means of establishing a diagnosis in the patient with epiglottitis, 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 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.

Fiberoptic Laryngoscopy

In the majority of cases, epiglottitis is diagnosed by findings on flexible laryngoscopy. In a child with a higher propensity for airway obstruction, this technique should be reserved for the operating room when everything is prepared for a rapid intubation. If an edematous epiglottis is seen, then the diagnosis is cinched. This does require a very cooperative patient without current airway compromise. It is certainly not recommended for a young child.

Indirect Laryngoscopy

Indirect laryngoscopy should not be attempted in a child with suspected epiglottitis.

Diagnosis Of Peritonsillar Abscess

Clinical

Patients with a peritonsillar abcess often have a toxic appearance. Physical examination is useful, but a clinical differentiation between peritonsillar abscess and peritonsillar cellulitis is very difficult.[111] The clinical picture of a peritonsillar abscess includes trismus, odynophagia, muffled voice, edema and erythema of the superior peritonsillar tissue, and deviation of structures away from the affected side.

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, there will be unilateral edema and swelling of the anterior tonsillar pillar and soft palate. Fluctuance and peritonsillar swelling are suggestive of a peritonsillar abscess. The soft palate is usually 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 muffled "hot potato" voice. This "hot potato" voice 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 is often 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 / Incision And Drainage

Needle aspiration or incision and drainage may be both diagnostic and therapeutic for a peritonsillar abscess. Unfortunately, failure to obtain purulent fluid on aspiration may mean that there really is no abscess 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 were used to maximize the chance of striking the abscess cavity.[112]

CT And Ultrasound

If the diagnosis is uncertain after the initial evaluation and after performing needle aspiration, imaging techniques may be useful. Both CT scan and intraoral ultrasound have been used to substantiate the diagnosis.[67,113-115] 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.[116 117] It is, however, considerably more expensive. Intraoral ultrasound is not yet widely available and the test's sensitivity is less than 90%.[118] Most radiologists do not have training in the interpretation of intraoral ultrasound.

Diagnosis Of Retropharyngeal Abscess

Clinical

In the child, the onset of symptoms is usually slowly progressive and often occurs after a mild upper respiratory infection. A history of trauma may be elicited. The child with a retropharyngeal abscess will appear toxic. Neck movement may be limited and suggest nuchal rigidity if the examiner does not adequately inspect the oral cavity.[119,120] Often the child will not move the head and neck to look up, preferring to do so only with the eyes.

The child will often have dysphagia and a muffled voice (the "hot potato" voice).[121] This muffled voice results from inflammation and swelling about the posterior pharyngeal wall. Many children will have trismus or be unwilling to open their mouth due to the pain.

On inspection, the examiner may see a bulging of the retropharyngeal wall.[122] This may appear one-sided. This bulging is often quite difficult to detect 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, fluctuance may be found. This should be performed very gently and only on a cooperative patient, since rupture of the abscess may occur.

In older children and adolescents, a retropharyngeal abscess usually occurs in the setting of an underlying illness, after an intraoral procedure, neck trauma, or a head and neck infection.[121,123] The most common presenting symptoms are still sore throat, dysphagia, and neck pain. Symptoms are often out of proportion to the examination of the neck.

The examiner should be wary. Most signs and symptoms of RPA are identical to those of acute epiglottitis. The most suggestive physical signs of both diseases are hyperextension of the neck, torticollis, muffled voice, and other signs of upper airway obstruction.

Radiology

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, although there is a great deal of controversy. In patients who had a clinical examination suggestive of a retropharyngeal abscess, there was scant correlation between the lateral neck films and findings on CT scan.[124] In another study, plain film exams were interpreted as normal in 7% and equivocal in 7%, despite surgically demonstrated pus.[119]

Lateral neck films may show an increase in the AP soft tissue space anterior to the vertebra, which is thought to represent widening of the retropharyngeal space. Unfortunately, such widening is nonspecific as failure to extend the neck may also cause widening of this space. Air density in the retropharyngeal space may indicate an abscess with a gas producing organism or communication with the esophagus or airway. Other helpful radiologic signs include loss of the normal lordotic curvature of the spine and evidence of a foreign body.

A retropharyngeal space measured from the most anterior aspect of CT to the soft tissues of the posterior pharyngeal wall that is greater than 7mm or a retrotracheal space larger than 14mm in a child suggests a mass.[125]

Ultrasound has been proposed for the diagnosis of a retropharyngeal abscess. In at least one study, this method had false negatives.[126] The proponents of ultrasound feel that the probe can differentiate between fluids and solids and provides a cheaper, non-irradiating diagnostic modality. The ultrasound can be used where appropriate, but should not be considered the final diagnostic test if the clinical suspicions warrant further study.

CT And MRI Scan

A CT scan of the neck, with emphasis on the retropharyngeal area, is the current method of choice for diagnosing a retropharyngeal abscess.[65,119,127] The CT scan delineates exactly which neck spaces are involved and localizes the lesion before surgical drainage is attempted.[128] CT is also useful in defining the vascular structures of the neck and their potential involvement. A MRI scan may be more useful for evaluation of the carotids.[129] Properly performed lateral neck films may show the thickened pre-vertebral soft tissue, but a CT scan of the neck and chest is necessary to determine the inferior limits of involvement.

Cultures

Abscesses have grown streptococcal species, *H. influenzae*, and *Neisseria* species.[130] Less common infections such as TB and syphilis have also been seen.[130]

Diagnosis Of Submandibular Cellulitis And Abscess – Ludwig's Angina

Clinical

In an early case, the external swelling may be limited. Physical examination in an advanced case reveals a tense, brawny swelling of the submental, sublingual, and bilateral submandibular spaces. The flexible tissues in the floor of the mouth can be distended superiorly and the patient may have displacement of the tongue superiorly and posteriorly. The tongue may literally bulge out of the mouth.

Fluctuance is rarely found. The patient does not often have trismus, and if it is present, then lateral pharyngeal space involvement should be suspected. These patients are at increased risk of mediastinal spread of the infection.

Radiology

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.[114] The patient may not be able to undergo CT or MRI until the airway is secured with tracheostomy or endotracheal intubation. It is inelegant to have the patient 'crash' in the radiology suite where intubation equipment for the difficult airway is not readily present.

The use of CT scan to determine the extent of the infection has also shown that the lateral pharyngeal spaces are often involved.[131] The infection is usually bilateral.

Treatment

General Airway

It has commonly been held and quoted in multiple textbooks that when a child has an obstructed airway due to an acute infection, the physician should attempt to assist respirations with a bag-valve-mask unit. The reasoning is that the obstruction is usually for inspiration and may be bypassed with forceful ventilation. Unfortunately, this has not been shown to be true in the only case report to date.[95] In this same paper, the authors posited that, perhaps, the child should be put in prone position so that gravity would allow the epiglottis to fall forward and vomitus would be expelled. This position goes against all training for both EMS and physician providers, but may prove more successful. This will require further study to validate. (In most patients, jaw-thrust combined with bag-valve-mask oxygenation usually provides adequate ventilation in the patients with acute infectious upper-airway disease).

If possible, perform the surgical airway in a controlled fashion. Get both anesthesia and ENT help. This airway WILL be difficult and help WILL be useful. The best place to get the airway is in the operating suite with neck prepped for a tracheostomy. Blind oral or nasal intubation or use of rapid sequence intubation should not be used as they may precipitate a failed airway crisis. This patient is not a candidate for RSI, unless a scrubbed ENT surgeon is standing by to do a tracheostomy.

Bacterial Tracheitis

Treatment

Close monitoring of the airway is the principle therapy for bacterial tracheitis and intubation is usually needed to prevent obstruction by the purulent exudate. Because of the overlap in symptoms, the emergency physician should consider the diagnosis of bacteria tracheitis in all children with viral croup.

Initial antibiotic therapy should target the common respiratory species such as *H. Influenza*, *M. catarrhalis*, *Corynebacterium diphtheriae*, *Streptococcal* or *Staphylococcus aureus*. [2,3] Cefuroxime or ceftriaxone are reasonable first line therapies.[6] Vancomycin may be added when gram stain results show gram positive cocci in areas where MRSA is common.[6] Alternatively, another antistaphylococcal agent such as nafcillin may be appropriate in areas where MRSA is not common.

In a review of the therapy for bacterial tracheitis, one major point does stand out.[10] Bacterial tracheitis is almost universally non responsive to the traditional medical therapies for viral croup. This includes humidification and racemic epinephrine. When the emergency practitioner has a child that appears toxic and does not respond to medical therapy for croup, s/he should start to think about bacterial tracheitis.

As noted previously, morbidity and mortality from bacterial tracheitis are primarily from respiratory obstruction. The copious secretions can cause respiratory distress and rapidly proceed to completly obstructing the airway causing respiratory arrest. Other reported complications include pneumonia and pneumothorax. Toxic shock syndrome has been noted when *Staphylococcus aureus* is the cause.

Disposition

All of these children must be admitted to the hospital with potential intubation in the operating room. Mortality rates are often cited in the 70% range, but in a meta-analysis of 11 studies comprising 177 patients, the mortality was only 3.4%.[10] Intubation and tracheostomy were necessary in the majority of patients. (The preference for intubation versus tracheostomy appears to be center-dependent.)

Intravenous antibiotics are appropriate in most cases. Antibiotic therapy should be directed towards the likely pathogens and may include penicillin or analogue such as ampicillin, methicillin, nafcillin, cloxacillin, dicloxacillin, gentamycin, chloramphenicol, or the third generation cephalosporins. Appropriate changes in antibiotic therapy can be made following the results of initial cultures.

Croup

Treatment

Many therapies have been advocated in the treatment of croup. Some of these therapies have persisted despite scant objective evidence of their value. Others are controversial despite substantial clinical research. The vast majority of patients with croup

will need only minimal treatment and will get better despite our intervention.

Humidification

Humidification and cool mist has been advocated as a mainstay of therapy for many years in the treatment of croup.[132] Steam generated by showers, baths, tea kettles, and croup tents have all 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 value about humidification exists; indeed, at least one controlled study contradicts the age-old advice.[133] This study of 140 children was performed with 40% humidity, 100% humidity with particles specifically sized to deposit in the larynx, and humidity by blow-by technique. 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

Steroids have also 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 is not known. Steroids may block allergic responses, reduce local inflammation, and reduce the subglottic edema.

In 1995, the first prospective study of steroids in outpatient management of croup was reported.[134] This randomized, blinded study showed that patients treated with dexamethasone required less medical care and parents reported earlier improvement in treated children.

In a 1960's randomized, placebo-controlled study steroids were shown to be effective, with significant improvement in hospitalized children.[135] A meta-analysis of nine randomized studies reported by Kairys et al showed that administration of corticosteroids was associated with significantly greater improvement at 12 and 24 hours after treatment.[136] There has been up to an 86% decrease in hospitalizations when croup is treated with steroids.[22] The incidence of endotracheal intubation was reduced by 80% in the treated group. Virtually no adverse effects of the use of corticosteroids were in these reviews.

Despite this evidence, other authors have concluded that routine use of steroids in children cannot be justified. Controversy exists concerning safety, the effects on hospitalization requirements, duration of hospitalization, requirement for subsequent medical visits, and requirements for intubation and intensive therapy.[10,11] Theoretical complications of steroid treatment include bacterial tracheitis and gastrointestinal bleeding.[11] Steroids should be used with caution if the child has a history of recent exposure to varicella (and hasn't been vaccinated).

A variety of dosages and regimens exist for steroids in croup.[137,138] Oral steroids have been evaluated for children with moderate and mild croup.[139,140] Although parental administration is the usual recommendation, both oral and nebulized agents have been evaluated.[11] Advocates of oral administration cite the discomfort of an IM injection, but neglect the significant possibility of early vomiting after administration. Dexamethasone from 0.6mg/kg as a single dose to a maximum of 8mg given orally, intramuscularly, or intravenously is the author's current recommendation. (Dexamethasone has an effective half-life of 48 hours as compared with 24 hours for prednisolone.) A recent randomized blinded study using nebulized dexamethasone suggests that this route is as effective as either oral or injected.[141] Nebulized budesonide has also been evaluated, but is not available in the US.[142]

Epinephrine

Like steroids, epinephrine is not a new therapy for croup.[20] 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 subsequent decrease in edema.

Use of racemic epinephrine has frequently been proposed and is widely thought to decrease the incidence of tachycardia and hypertension. Evidence to support these beliefs could not be found. In one study that compared racemic epinephrine and standard (non-racemic) epinephrine, there was not a significant difference.[143] Since standard epinephrine is readily available and much less expensive, there simply is no cogent reason to demand racemic epinephrine for the treatment of croup. The appropriate dose is racemic epinephrine 0.05-0.1ml per kg of normal saline delivered by nebulizer. An equivalent dose of epinephrine is 1:1000 epinephrine 5.0cc delivered by nebulizer. This may be repeated as

needed, but if more than one dose of epinephrine is used, consider admission.

Epinephrine is transient in effect 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 elegantly solved without admission by simply observing the child for about 3 to 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 follow-up of the patient.

Antibiotics

Antibiotics have not been shown to be of any benefit in the treatment of croup. There is usually no role for antibiotics in the management of this disease, but, in older children, the causative organism may be *Mycoplasma*. This will respond well to macrolides.

Heliox

The use of helium/oxygen (heliox) mixtures in the child with croup (and partial airway obstruction from any reason) may buy time for more help or better equipment.[144,145] A single randomized study of only 29 patients seems to support this, but larger studies would be appropriate before recommending this expensive therapy in every ED.[146] Heliox mixtures allow better oxygenation since the lighter gas flows more easily through the tighter airway passage. In at least one cited study, younger patients appeared to be better candidates for heliox mixtures, perhaps because of their smaller airways.[145]

Disposition

The vast majority of children with croup will be sent home. Of children who develop croup, only a few will require inpatient care and < 3% of those will require intubation.[106,147] In the ED, children who have no stridor at rest, normal air entry, good color, normal level of consciousness, and have received steroids can be safely discharged.

Before the use of epinephrine and steroids, the disease caused significant morbidity and occasional deaths. Inhaled epinephrine and steroids have drastically improved the outcome in the child with croup and have allowed most children to be sent home. The emergency physician should consider admission of the child who presents with stridor or cyanosis and/or fails to clear retractions after therapy.

Consider alternative diagnoses to croup when there is no cough, the child is drooling, has a toxic appearance, the oxygen saturation falls, and/or the child fails to respond to epinephrine nebulization and steroids. As noted earlier, a diagnosis of bacterial tracheitis or epiglottitis should be considered for this child.

Diphtheria

Treatment

Antibiotic therapy stops toxin production, improves the local infection, and prevents the spread of the organism to other people. Although several antibiotics, including penicillin, erythromycin, clindamycin, rifampin, and tetracycline, are effective, only penicillin and erythromycin are generally 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 to 40,000 units of antitoxin for pharyngeal or laryngeal disease of 48 hours' duration, and 40,000 to 60,000 units for nasopharyngeal lesions.[148] 80,000 to 120,000 units should be given for extensive disease of three or more days' duration and for anyone with brawny swelling of the neck. Diphtheria antitoxin is no longer licensed in the US, but a European-licensed product is available from the National Immunization Program of the Centers for Disease Control and Prevention by calling 404-639-8200.

Disposition

All of these patients will be admitted. Almost all of the younger patients will require intubation and treatment in the intensive care unit. The duration of treatment for diphtheria varies with the severity of the disease and the complications encountered.

Cardiac complications can be minimized by close observation in an ICU environment. The electrocar-

diograph monitoring will allow prompt initiation of electric pacing for conduction disturbances, drugs for arrhythmias, or digitalis for heart failure.

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

Prevention is the easiest treatment. The use of tetanus-diphtheria immunization every ten years will prevent, or 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.[149] Diphtheria is a reportable disease in all states in the US.

Epiglottitis

Treatment

Airway

Since the most important complication in epiglottitis is airway obstruction, this is where the emergency physician should plan for problems. The airway precautions noted in general airway management on page 17 should be strictly followed.

Helium-oxygen

The use of helium/oxygen (heliox) mixtures in a patient with epiglottitis may buy time for more experienced help or better equipment.[144] Details of helium-oxygen mixture are found in the section on croup treatment on page 19.

Antibiotics

Ampicillin 200mg/kg IV and chloramphenicol 100mg/kg IV has traditionally been recommended for treatment of epiglottitis. Second or third-generation cephalosporins are now recommended because of the increased prevalence of beta-lactamase producing *H. influenza*. Ceftriaxone has a long elimination half-life, excellent tissue penetration, and proven activity against *H. Influenza*.[150] Ampicillin combined with sulbactam would be a reasonable alternative. Vancomycin may be appropriate when staphylococcus is identified, given the high rates of MRSA.

Steroids

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

Epinephrine

Inhalation of epinephrine may decrease the size of the epiglottis and buy time.[151] This has been proven only anecdotally.

Disposition

All of these patients will be admitted. Almost all of the younger patients will require intubation and treatment in the intensive care unit. Some debate exists about the safety of monitoring the child in the intensive care unit without intubation when mild epiglottitis is present.

The duration of treatment for epiglottitis varies with the etiology. When given high doses of effective antibiotics, children with HIB epiglottitis can usually be extubated within 24 to 48 hours. In the reported cases of epiglottitis caused by the other three beta-hemolytic streptococci that are human pathogens, the mean duration of intubation was six days.[50,51,54,55]

Peritonsillar Abscess

Treatment

The initial assessment, stabilization, and management of a peritonsillar abscess do not initially depend on the diagnosis of peritonsillar abscess or peritonsillar cellulitis. If the patency of the upper airway is not immediately compromised, then the emergency practitioner has some time to decide whether the patient has cellulitis or an abscess.

Aspiration/Drainage

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).[111,152] 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 to 5 days.[153,154] There is no statistical difference between incision and drainage and needle aspiration in outcome.[155]

Needle aspiration has significant advantages over incision and drainage. These include: ability to proceed without general anesthesia, less trauma to surrounding structures, ready availability, and signifi-

cantly lower cost. In the patient with an uncomplicated peritonsillar abscess, needle aspiration will not only provide relief of symptoms, but 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.[65] Open drainage is also indicated for immunocompromised patients.

Abscess Tonsillectomy

The literature from the 1930's through the 1970's showed that abscess tonsillectomy was a safe procedure with three major advantages: it provides complete drainage of the abscess (including the lower pole and deep space abscesses), it prevents a recurrence of the abscess, and it (arguably) results in a shorter recovery period. Tonsillectomy fell out of favor in the late 60's and 70's and, as a consequence, abscess tonsillectomy fell out of favor.

About 30% of patients presenting with a periton-

Controversies/Cutting Edge

There are abundant controversies and cutting edges in the treatment of the various malignant causes of sore throats. Each of these controversies has been discussed in the appropriate section and is presented here to highlight the applicable point.

- **Should the clinician suspect diphtheria?** Although this disease has been rare in the United States, there has been a significant outbreak in the past decade in Russia. This outbreak spread to several European countries. With declining immunization rates in both elders and youngsters alike, the possibility of this very rapidly spreading communicable disease having a re-emergence in the US is real.
- **Should the clinician use oral steroids in the treatment of croup?** Certainly the research shows that there is significant shortening of the course and the severity of the disease is reduced with the use of both oral, intramuscular, and intravenous steroids. Likewise, the complications caused by a single dose of steroids are minimal, and the most significant warning is with recent exposure to varicella. Indeed, this author feels that the only real drawback to the use of oral steroids is the distinct possibility that the child will vomit the medication due to tussive emesis in the ED. In this event, intravenous and intramuscular routes are appropriate.
- **Should the clinician use cool mist in the treatment of croup?** While study of physiology shows that the gases inhaled are warmed to body temperature and humidified by the time they reach the supraglottic area, generations of physicians (and nurses and parents) have come to expect cool mist in the treatment of croup. Recent studies have shown that the expected physiology of respiration is, indeed, applicable to the child with croup and that there is no difference in outcome measured in either duration of stay or hospitalization when cool mist is used. The clinician must decide whether the vast inertia caused by decades of use must be overcome to save the few cents in equipment and sterile water required to administer the mist. Realization that the treatment is neither necessary nor particularly helpful will not assuage the parent who wants something done – NOW – for his/her child with croup and arrives with expectations of therapeutic cool mist.
- **The most critical cutting edge in the diagnosis of the malignant sore throat is the realization that the venerable cross-table radiographic examination of the soft tissues of the neck is inadequate for the diagnosis of epiglottitis and retropharyngeal abscess.** The false-negative rate of 10% in these studies means that the emergency physician may send 1 in 10 patients with this potentially fatal disease home. The best diagnostic technique for epiglottitis is visualization of the epiglottis and surrounding structure. The best diagnostic technique for visualization of any of the deep abscesses of the neck including both retropharyngeal and peritonsillar abscess is contrast-enhanced CT of the neck. This may require the physician's presence in the CT scanner with the child to ensure that the airway is not compromised during the time in the radiology suite.
- **Can epiglottitis occur that is not due to HIB in the child who has been adequately immunized?** Yes. In this case, the clinician who has been trained to expect a toxic child with a rapid course may be presented with a much less toxic, more indolent course. The findings of a cherry red epiglottis may also be absent in a non-HIB epiglottitis case.

Pitfalls to Avoid

1. **"The patient was a 17-year-old male. He began speaking in a muffled voice, and had an increasingly sore throat that started yesterday. He also had difficulty swallowing. His pulse was 120, respiratory rate 25, and blood pressure 130/75. His temperature was 101.5° F. The triage nurse thought he had strep, but the strep screen was negative in triage and there wasn't any exudate in the back of the throat. He admitted to a little crack three days ago. How was I to suspect a retropharyngeal abscess? He's way too old for that disease."**

   You are partly right. He is outside of the usual spectrum of retropharyngeal abscess. His muffled voice deserves closer inspection with either imaging or direct visualization. The association of crack smoking and retropharyngeal abscess in adults and adolescents is well reported.

2. **"The child is three, has a relatively toxic appearance and speaks in a muffled voice. He has an increasingly sore throat that started yesterday. His mother notes that he isn't eating and he is starting to drool. His temperature is 101.5°F, pulse is 160, and respiratory rate is 40. There is a lot of strep going around in the pre-school and the triage nurse did a strep screen. It was negative. The mother reported that she hasn't been real good on his shots - she's not a believer in immunizations. Why should I suspect epiglottitis? I've been reading that HIB epiglottitis is getting quite rare in children these days - I haven't seen one since residency and that was eight years ago."**

   When a classic disease has a classic presentation…and the child hasn't received immunizations… HIB is still a player. You will need to consider it in your differential, even though it's becoming much less common due to HIB immunization.

3. **The 18-month-old has a croupy cough and looks quite toxic. He's in your triage with a sore throat, is speaking in a muffled voice, has difficulty swallowing, has a pulse of 160, respiratory rate of 44, and a fever of 101.5°F. There are a few retractions but the child doesn't tripod, nasal flare, or belly breathe. You were thinking about epiglottitis with a child this sick, but the soft tissue of the neck was negative. The child has a bit of a barky cough and the chest x-ray showed a 'steeple sign' but was otherwise negative. He didn't respond to inhaled epi and cool mist. You consider croup, but the child appears more toxic than you usually see in croup. The charge nurse wants to give steroids and get the child out…it's a busy night.**

   Bad move. A little like epiglottitis… a little like croup…and not responsive to the usual treatments associated with croup. Maybe you need to consider bacterial tracheitis. You will need to admit this child, get IV antibiotics and carefully observe. You shouldn't depend on a soft tissue of the neck for epiglottitis. There is up to a 15% false negative rate with this study. You also need to look at the epiglottis to ensure that it isn't involved.

4. **The 18-year-old presented to triage yesterday with the complaint of a sore throat. He was speaking in a muffled voice, had a pulse of 140, a respiratory rate of 24, and a fever of 101.5°F. The triage nurse noted that the uvula was pushed to the right when she got a strep screen. The strep screen was negative, so she didn't prescribe any antibiotics. Your PA saw the patient and you countersigned the chart. He's back and feeling worse…now his temperature is 103.2°F and he can't swallow. He's had no fluids for about 16 hours. You examine the throat and see a massively deviated tonsil and uvula. How could both the triage nurse and the PA have missed the boat? The ENT surgeon is livid.**

   A peritonsillar abscess can progress rapidly and should be treated with antibiotics at a minimum. Aspiration, open drainage, and urgent tonsillectomies are considered by some ENT surgeons. You will need to consider this disease in your differential in a patient with a sore throat and muffled voice. A muffled voice deserves imaging or visualization of the airway. A deviated uvula, abnormal vital signs, and difficulty swallowing should clue you in to the possibility of a peritonsillar abscess.

5. **The six-year-old presented to your ED yesterday with a sore throat, fever of 101.5°F, pulse of 120, and respiratory rate of 25. He's having difficult swallowing, has a muffled voice, and the mother is worried about both strep and dehydration. She wants him to get a shot for his sore throat. She's not happy that the triage nurse told her that he wouldn't be getting antibiotics if the strep screen was negative and she already knows it's negative. The triage nurse noted that the child was in the ED four days ago when he was running with a pencil in his mouth and fell. The ED doctor then noted a bruise on the posterior pharynx. He was discharged with no further workup. You acceded to the mother's request and gave Bicillin LA… Today you've gotten a call from the other ED in town; the child's there, he's got a retropharyngeal abscess, and they want copies of the medical records for both visits.**

   Oops…. A recent retropharyngeal trauma, abnormal vital signs, and difficulty swallowing should have caused you to consider a diagnosis of a retropharyngeal abscess. A muffled voice deserves imaging or visualization of the airway.

6. **The child is six, has a relatively toxic appearance and speaks in a muffled voice. He has an increasingly sore throat that started four days ago. His mother notes that he isn't eating and he is starting to drool. His temperature is 101.5°F, pulse is 160, and respira-**

tory rate is 40. There is a lot of strep going around in the first grade. Your triage nurse did a strep screen. It's positive. You've read all of the horror stories about co-existent strep carriers and the absolute need to evaluate the child with a muffled voice…and are credentialed in fiberoptic laryngoscopy. You noted a pale, slightly swollen epiglottis when you took a look in this cooperative child. The epiglottis was surely swollen, but definitely wasn't 'cherry red' and the child's course is much more indolent than is usual for HIB epiglottitis. You've ruled out epiglottitis, right?"

Whew… you just diagnosed the disease: strep epiglottitis. This child may not need intubation but will need to be in the ICU and on IV antibiotics. With strep epiglottitis, the child will have a much more indolent course and may be intubated for a week's worth of IV antibiotics. The pale edematous epiglottis is characteristic of strep and other causes of epiglottitis. A cherry red epiglottis is associated with HIB only.

7. **You've got a 14-year-old recent Uzbekistan immigrant with a sore throat, muffled voice, abundantly exudative pharyngitis, and temperature of 101.5°F. You've not seen a case before, but you think you might have a case of diphtheria on your hands. A colleague suggests getting a Gram stain and then starting the child on intravenous penicillin or erythromycin, but you're not sure about this. You want to start off with a heavy duty cephalosporin and get antitoxin for the child.**

Good call…on the diagnosis. "Chinese characters" seen on gram stain may help in a presumptive identification of *C. diphtheriae*. Your colleague's therapy is right on the money...antibiotic therapy stops toxin production, improves the local infection, and prevents the spread of the organism to other people. Erythromycin and penicillin are the drugs of choice. Diphtheria antitoxin is not produced in the US, but the CDC has limited stocks of foreign antitoxin for specific cases. You need to get in touch and report the case anyhow.

8. **You've got a 2 AM crouper on your hands. The three-year-old has a fever of 101°F, sore throat, a typical 'seal-bark' cough, and some moderate retractions. Your chest x-ray shows a 'steeple sign' and you've given dexamethasone at 0.6 milligrams per kilogram PO. The child promptly vomited up the dexamethasone, so you repeated the dose IM. As you go to discharge the child, you start your litany about cool mist. The husband, a paramedic, interrupts you to point out that by the time the air gets to the supraglottic area it's already 100% saturated with water and raised to body temperature. You point out that this therapy has been used for croup for centuries. Who's right?**

Don't put big money on the concept that we've always done it that way, so it must be correct. Mr. Paramedic is correct. The utility of mist is like beauty… in the eye of the beholder. The science behind cool mist fails for the reasons that the paramedic enumerates. The best that can be said is that it helps everybody think they are doing something and everybody relaxes…so the child relaxes.

9. **The 16-year-old presented to triage yesterday with the complaint of a sore throat. He was speaking in a muffled voice, had a pulse of 136, a respiratory rate of 22, and a fever of 101.3°F. The triage nurse noted that the uvula was pushed to the left when she got a strep screen. The strep screen was positive, and the patient was allergic to penicillin, so the PA prescribed erythromycin. He's back and feeling worse…now his temperature is 104.1°F and he can't swallow. He's had no fluids for about 12 hours. You examine the throat and see rotation of the anterior tonsillar pillar, bulging tonsil and a slightly deviated uvula. Your ENT surgeon is not happy at this admission.**

Simply put - you were fooled by a positive strep screen. There is a large percentage of the population that are strep 'carriers.' Simply because the strep screen is positive doesn't stop another disease process from progressing. A peritonsillar abscess can progress rapidly and should be treated with antibiotics at a minimum. Aspiration, open drainage, and urgent tonsillectomies are considered by some ENT surgeons. You will need to consider this disease in your differential in a patient with a sore throat and muffled voice. A muffled voice deserves imaging or visualization of the airway. A deviated uvula, abnormal vital signs and difficulty swallowing indicates the possibility of a peritonsillar absess.

10. **"The three-year-old was a recent immigrant-adoptee from an orphanage in Russia. The adoptive mother was unsure about all immunizations, but was planning to get them updated within the month. The orphanage director provided her with a 'shot record' but it is in Russian and she didn't have a translation. The child had a sore throat that had been brewing since the flight from Russia three days ago. His temperature was 101.5°F, pulse 140, and respirations 25. He spoke in a muffled voice and had an abundant exudate on his tonsils…. How was I to suspect diphtheria?"**

Hmm… There has been an epidemic of diphtheria in Russia over the past few years…the child came from an area where the disease is much more prevalent than here...and has an exudative tonsillitis with a hoarse throat. You need to put this into your differential diagnosis when dealing with those who may have had no immunizations, come from a country with lax immunization policies, or come from a country where the disease is more prevalent.

sillar 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.[65]

Antibiotics

Regardless of the therapy chosen by the ENT surgeon, the patient will be started on intravenous antibiotics, rehydration, and discharged on oral antibiotics. Although penicillin will usually cover the organisms which cause peritonsillar abscess, cephalosporins are often used.[156,157] Close follow-up or hospitalization is indicated in all of these patients.

Disposition

There is some controversy about whether a patient can be treated in the ED and released or hospitalized. This depends partly on the preference of the ENT consultant and partly on the clinical picture of the patient. If the older and responsible patient has few symptoms of toxicity, a trial of 12 to 24 hours of antibiotics is appropriate. If the patient is toxic or has severe trismus, then more aggressive treatment is appropriate. If the patient has significant improvement of symptoms on a trial of antibiotics, then cellulitis is likely. In younger patients, admission and therapy with intravenous antibiotics is appropriate. This may be accomplished in an observation unit.

Retropharyngeal Abscess

Treatment

Oral intubation should be carried out with great caution to avoid rupturing the abscess. This could result in aspiration of the pus with attendant pneumonia, sepsis, or lung abscess. Blind oral or nasal intubation or use of rapid sequence intubation should not be used as they may precipitate a failed airway crisis.

Antibiotics

Children without airway compromise or other complications may initially be treated with intravenous antibiotics.[158] A retropharyngeal abscess will resolve in at least 20% of cases with antibiotic therapy alone.[159] Clindamycin with an aminoglycoside or a penicillinase-resistant penicillin combined with a third generation cephalosporin and metronidazole are appropriate first antibiotic choices.[159] Clindamycin and metronidazole should not be used alone.

Aspiration/Drainage

Therapy is needle aspiration of the abscess or surgical incision and drainage of the abscess. Needle aspiration is very painful, although it is performed by some ENT physicians under local anesthesia. Incision and drainage can be performed with an external approach or with a trans-oral approach.[159] The external approach has a longer recovery time, and there are possible complications to cranial nerves and great vessels. The intra-oral approach can be associated with aspiration of the abscess contents. Intraoral approach may not be possible with superior or lateral abscesses.

Disposition

Patients with a retropharyngeal abscess should be admitted to the hospital. These abscesses should not be drained in the ED. Surgical complications of 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 ready, suction at hand, and adequate sedation and analgesia is appropriate.

Submandibular Abscess/Cellulitis – Ludwig's Angina

Treatment

The three components of treatment of Ludwig's angina include protection of the airway, antibiotics, and surgical drainage of the abscess. Because airway compromise is the single most common cause of death in Ludwig's angina, protection of the airway is the first priority. Airway maintenance may be difficult in advanced cases. A number of methods have been used to manage the airway in these patients, including oral intubation, fiberoptic intubation, cricothyrotomy, and formal tracheostomy.

Airway Management

Tracheostomy has been considered the gold standard of airway management in Ludwig's angina.[160] Although this technique rapidly bypasses the supraglottic swelling, it is associated with a high complication rate and risks spreading the infection into the mediastinum.[161] Tracheostomy, intubation, or cricothyrotomy may be quite difficult in a patient with swelling, neck infection, and inability to lie flat.[88] Upright, awake nasal intubation with a flexible fiberoptic scope has recently been recommended as the most appropriate technique.[88] The best place to attempt this intubation may be in the operating room with a surgeon scrubbed for immediate tracheostomy.

Endotracheal intubation may be problematic due to swelling and distortion of the upper airway. A rapid sequence intubation is controversial and may be hazardous. These patients are not good candidates for rapid sequence intubation (RSI) techniques, since the swelling may preclude both ventilation and intubation in these patients. Routine orotracheal intubation is rarely applicable, since most patients who are candidates for this intubation approach are also candidates for simple observation and antibiotics.[162,163]

Cricothyrotomy or jet ventilation may also be quite difficult because of distortion of the anatomy by swelling. "Blind" nasotracheal intubation should not be performed because of the risk of precipitating an acute airway obstruction. Blind orotracheal intubation is not acceptable.

Antibiotics

High dose antibiotics are the mainstay of therapy. Once the airway has been protected, antibiotics alone will suffice for about 50% of patients.[164] 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.[164] Clindamycin may provide coverage of *Eikenella corrodens* and improve coverage of other antibiotics.[88]

Surgical Drainage

Surgical drainage was once recommended for all patients with Ludwig's angina, even though no purulent collections were found in many patients. Surgical drainage is now recommended for refractory cases or patients with rapid progression.[89] In one meta-analysis, 8 of 29 cases were successfully treated without surgical intervention using antibiotics and dexamethasone.[89] CT results can guide surgical drainage and may indicate surgical drainage if significant collections of pus are found.

Drainage can be intraoral or external. The intraoral approach should be reserved for the patient who only has an uncomplicated submandibular space infection that is limited to the sublingual compartment.

Steroids

Steroids have been suggested as an adjunct to decrease the spread of edema and decrease the need for artificial airways.[165] Dexamethasone is often recommended to decrease swelling and provide chemical decompression of the tissues surrounding the airway. It may also allow improved antibiotic penetration of the area. The usual dose is 10mg of dexamethasone intravenously followed by 4mg every six hours for 48 hours.

Disposition

All of these patients should be admitted to the hospital. Simple critical care unit observation and antibiotic therapy in a select group of patients is possible.[89,166] Observation is recommended only in a critical care unit where the airway can be controlled before compromise if the patient's disease progresses despite antibiotics.[167]

Summary

From the opening vignette...

Now you find yourself explaining to your hospital risk manager why this child is in the ICU and not doing well. You could blame it on the triage nurse, of course, but you saw the chart and missed the 120 pulse rate and the 36 respiratory rate in a child of 13. Fortunately the child recovers and the family is grateful that you saved her life. You are somewhat more sanguine about your part in this child's recovery.

Making the diagnoses of these "sore throats" can be extremely challenging. The differential diagnosis can be quite large and includes pharyngitis or "sore throat." The emergency physician should focus on why *this* sore throat is not like most sore throats, why *this* patient appears more toxic than expected, why the antibiotics given on a prior visit didn't work for *this* patient, and, of course, whether there is a potential for airway compromise in *this* patient. When the patient's voice is muffled, the emergency physician needs to consider the differential and consider imaging or direct visualization of the airway structures.

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

32. All of the following statements 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. Our second president may have died from epiglottitis.

33. The most common cause of death in children who have epiglottitis is:
a. Delay in care from misdiagnosis.
b. Laryngospasm.
c. Mediastinal infection and subsequent sepsis.
d. Concurrent illness.

34. The most common cause of early death from diphtheria is:
a. Myocardial disease.
b. Arrhythmia.
c. Paralysis of the diaphragm and respiratory muscles.
d. Airway obstruction.

35. Croup is characterized by:
a. A grey-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 due to a viral infection.
d. Ulcerated, partly necrotic tracheal mucosa due to a bacterial infection.

36. A retropharyngeal abscess in an adolescent 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.

37. A helium-oxygen mixture may be a useful temporizing measure in which of the following illnesses:
a. Croup.
b. Retropharyngeal abscess.
c. Ludwig's angina.
d. Peritonsillar abscess.

38. 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.

39. 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 "rebound" effect worsening of the clinical picture some 30 minutes to 2 hours after administration.
d. May be followed by discharge to home after 30 minutes of observation.

40. An 18-month-old child in the ED for moderate croup is receiving cool mist treatment. Which of the following statements is most accurate?
a. High-humidity cool mist treatment will lead to improvement in symptoms after 30 minutes.
b. High-humidity cool mist treatment will reduce the need for nebulized epinephrine therapy.
c. High-humidity cool mist treatment will increase the need for oral steroid treatment.
d. High-humidity cool mist treatment will not affect the likelihood of hospitalization.

41. Which statement does not usually indicate respiratory distress in a child?
a. Flaring nostrils and head bobbing.
b. Use of accessory muscles and leaning forward in a tripod position.
c. Muffled voice while speaking.
d. Speaking full sentences.

42. All of the following are consistent with the diagnosis of bacterial tracheitis EXCEPT:
a. Tachypnea.
b. High fever.
c. Stridor.
d. Drooling.
e. Minimal sputum production.

43. Which of the following infectious diseases is the LEAST LIKELY cause of stridor in an infant or child less than six years old?
a. Bacterial tracheitis.
b. Foreign body aspiration.
c. Croup.
d. Retropharyngeal abscess.
e. Peritonsillar abscess.

44. Oral dexamethasone is as effective as intramuscular dexamethasone in the diagnosis of croup.
a. True
b. False

45. Which of the following causes of upper airway obstruction is LEAST likely to result in an

acute precipitous deterioration of the patient in your ED?

a. Epiglottitis.
b. Diphtheria.
c. Croup.
d. Retropharyngeal abscess.
e. Bacterial tracheitis.

46. What is the most commonly found symptom or physical finding in the patient with a retropharyngeal abscess?

a. Muffled voice.
b. Neck pain.
c. Stridor.
d. Rapid respiratory rate.
e. None of the above.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Pediatric Emergency Medicine* Practice receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case-control studies
- Less robust RCTs
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. JAMA 1992;268(16):2289-2295.

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