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Common Ear, Nose, and Throat Disorders Encountered in Emergency Practice: Expeditious Evaluation and Definitive Management

Part II: Facial Nerve Palsies and Oropharyngeal Infections

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Part I of this series focused on ear and nose disorders. Part II of this two-part series focuses on facial nerve palsies and oropharyngeal infections. The authors present a systematic approach to differential diagnosis and identification of etiologic agents responsible for such conditions as peritonsillar abscess, epiglottitis, and pharyngeal infections. Radiographic and bacteriologic findings are emphasized, and appropriate antibiotic therapy is presented. The authors have provided treatment tables that direct emergency practitioners toward outcome-effective therapy.

—The Editor

Facial Nerve Palsies

Bell's Palsy. Bell's palsy is an acute, monosymptomatic, unilateral, peripheral facial paresis of unknown etiology. The incidence is estimated at 32 per 100,000 population per year.[1] Bell's palsy is responsible for 60-75% of cases of acute facial paresis.[2]

Clinical Presentation. The onset of symptoms progresses over 48 hours and may or may not lead to complete paralysis. In addition to facial droop, patients complain of pain and numbness of the ear, midface, tongue, decreased hearing, phonophobia, hyperacusis, and taste disturbance.[2]

Most patients (70%) have complete paralysis of the facial nerve. On physical examination, paralysis of the facial nerve can be differentiated from deficit from supranuclear origin such as stroke or tumor. A central seventh cranial nerve palsy will spare the forehead musculature because of dual innervation of forehead musculature from both sides of the brain, so a patient will be able to wrinkle the forehead/raise the eyebrows symmetrically. A patient with Bell's palsy with complete paresis will have unilateral facial droop, inability to wrinkle the forehead, and have Bell's phenomenon. Bell's phenomenon (defective lid closure

= lagophthalmos) manifests when the patient attempts to close the eye; the lid doesn't close completely, so the eye can be seen rolling upward. Other associated symptoms are present variably. The remainder of the physical examination is normal.

Etiology. By definition, the cause of Bell's palsy is unknown; however, the evidence of a viral etiology, especially herpes simplex, is persuasive.[1-3] Ramsay Hunt syndrome, also known as herpes zoster oticus, is caused by reactivation of the dormant virus. It is characterized by unilateral facial paralysis, a herpetiform vesicular eruption, and vestibular cochlear dysfunction. The vesicular lesions may occur on the pinna, external auditory canal (EAC), tympanic membrane, oral cavity, soft palate, face, or neck.[4] In most cases, the vesicles occur prior to or concurrent with the palsy; however, in 15% of patients, the vesicles appear after the onset of facial paresis, making the diagnosis difficult.[1] Disease caused by herpes zoster tends to be more severe and is associated with increased rate of hearing loss (73% of patients).[1]

In a study of more than 2500 patients with peripheral nerve palsies, 38 different causes of facial nerve palsies were seen. The most common etiology was idiopathic (66%), neonatal/birth trauma (6%), and herpes zoster (5%). Other etiologies included trauma, diabetes, pregnancy, polyneuritis, parotid tumor, and multiple sclerosis.[1]

Although Bell's palsy is the most common form of peripheral nerve palsy, care should be taken not to misdiagnose any palsy as idiopathic. The symptomatology of Bell's is not specific, and if the acute phase of the palsy does not affect all branches, it is important to consider another etiology because a partial palsy of one or two branches strongly suggests disease localized distally from the stylomastoid foramen and may indicate a cancer in the parotid gland.[1]

Evaluation. The extent of diagnostic testing performed is based in the clinician's suspicion of disease other than Bell's palsy. If the history and examination are classic for Bell's, then no emergent diagnostic testing is needed. If there is upper facial sparing consistent with a central seventh nerve palsy, then the evaluation should include computed tomography (CT) or magnetic resonance imaging (MRI) of the brain to evaluate for stroke or tumor. Possible infectious causes, especially Lyme disease, need a more extensive evaluation, such as lumbar puncture and serologies. Other indications for a more extensive evaluation include a slow progression of symptoms, recurrent ipsilateral paralysis, other associated cranial nerve abnormalities, bilateral paresis, or associated systemic symptoms.[4]

Treatment. Most patients recover without treatment. Eighty-five percent of patients recover function within three weeks, and the remaining 15% after 3-5 months without treatment. Return of complete normal function was seen in 71% of patients.[1] Corticosteroid treatment is used widely for Bell's palsy, although the efficacy is not well established. The use of steroids is based on the belief that edema of the nerve confined within the facial canal is causing or contributing to the nerve injury, and steroids should minimize this.[4]

A recent Cochrane review concluded that the available evidence from randomized controlled trials does not show significant benefit from treating Bell's palsy with corticosteroids.[5] The American Academy of Neurology notes that available evidence suggests that steroids probably are effective.[6] If prednisone (Deltasone) is given, a typical regimen is 1 mg/kg/day for 7-10 days with or without a taper.[4]

The use of antiviral medication such as acyclovir (Zovirax) is becoming more widespread. A Cochrane review of the available data in 2001 is inconclusive regarding the benefit of antiviral therapy for Bell's palsy, noting that more data are needed from a large, multicenter, randomized, controlled, and blinded study with at least 12 months follow-up before a definitive recommendation can be made regarding the routine use of acyclovir.[7] The American Academy of Neurology notes that acyclovir combined with prednisone is possibly effective in improving functional outcomes.[6]

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Prognosis. Factors that are associated with higher rates of complete recovery of facial nerve function include incomplete paresis (vs total paralysis), early recovery, younger age of patient, normal taste, normal tearing, and lack of post-auricular pain.[1] More important than administering steroids or antivirals is prevention of ocular sequelae via eye protection. Corneal exposure and ulceration can occur if eye protection is not sufficient. Patients should be advised to use ophthalmic ointment with an eye patch at night. Corneal protection should be continued until full recovery of tearing and lid closure has occurred.[2] Patients should be referred to an otolaryngologist or neurologist for follow-up.

Throat Disorders

Pharyngitis. Pharyngitis is an inflammation of the pharynx and tonsils.[8] Although it can be transmitted through fomites or food, it most commonly is acquired through contact with respiratory secretions.[9] Most cases of pharyngitis result from a viral infection.[10,11] Of these, rhinovirus and adenovirus are most common. Less frequently encountered are Epstein-Barr virus, herpes simplex, influenza A and B, parainfluenza, and coronavirus. (*See Table 1.*) The most common bacterial causes are group-A beta-hemolytic Streptococcus (GABHS) and group C Streptococcus. Mycoplasma and *Arcanobacterium haemolyticum* are less common, while group G Streptococcus, *Neisseria gonorrhoeae* and *meningitidis*, Chlamydia, *Corynebacterium diphtheriae* and *ulcerans*, and anaerobes (Peptostreptococcus, Fusobacterium, and Bacteroides) are uncommonly found. (*See Table 2.*) Other rare causes are fungi (e.g., Candida, *Cryptococcus neoformans*, *Histoplasma capsulatum*, *Rhinosporidium seeberi*, *Blastomyces dermatitidis*, and *Paracoccidioides brasiliensis*) and parasites (e.g., *Toxoplasma gondii*).[12]

As a general rule, patients complain of throat pain and odynophagia. Because of shared innervation, tonsillar pain may radiate to the ears. On exam, the pharynx is erythematous and may have exudates. Tonsillar hypertrophy and cervical lymphadenopathy also may be present.[9] Classically, viral infections are more likely to be associated with myalgias, conjunctivitis, rhinorrhea, stomatitis, cough, and exanthems.[13] Although the typical presentation varies depending on the etiology, it is nearly impossible to determine the cause based solely on exam. The differential diagnosis for pharyngitis includes deep space infection, tumor, foreign body, chemical and thermal burns, drug reaction, pemphigus, allergic reaction, Stevens-Johnson syndrome, uvulitis, angioneurotic edema, esophagitis, gastroesophageal reflux, thyroiditis, epiglottitis, and cricoarytenoid arthritis.[9]

Because pharyngitis usually is an uncomplicated, self-limited infection, much of its treatment is supportive. Management consists of warm fluids, acetaminophen or ibuprofen to ease the fever and pain, and topical anesthetics like Cepacol (menthol and benzocaine). Antibiotics usually are indicated if a bacterial etiology is suspected. In addition, steroids can help decrease the duration of symptoms and alleviate pain.[9]

Complications include airway obstruction, sleep apnea, deep neck infections, necrotizing fasciitis, bacteremia, and sepsis.[9]

Specific Bacterial Etiologies. Group A beta-hemolytic Streptococcus. As mentioned earlier, GABHS is the leading bacterial cause of pharyngitis.[14] Its incidence peaks during the winter and early spring, and it most commonly afflicts children 5-15 years old.[10,14] Recent exposure to someone with GABHS pharyngitis increases the risk of contracting it.[9] Patients classically present after an incubation period of 2-5 days with fever and sore throat,[8] although they also may complain of headache, nausea, vomiting, and abdominal pain; cough, rhinorrhea, and coryza usually are absent.[9,12] Examination is remarkable for exudative tonsillitis, uvular erythema and edema, palatal petechiae, and tender anterior cervical adenopathy.[12] Another characteristic finding is a scarlatiniform rash. This erythematous, sandpaper-like rash starts in the axilla and inguinal folds and spreads over the whole body, including the palms and soles.[8] The Centor criteria, first described in 1981, uses four variables, tonsillar exudates, swollen tender anterior cervical nodes, lack of a cough, and history of fever, to help the clinician make a clinical diagnosis of pharyngitis due to GABHS. Unfortunately, patients with all four criteria only had a 56% probability have having GABHS.[15]

Since clinicians have a fifty-fifty chance of correctly diagnosing GABHS pharyngitis based on clinical findings alone,[8] some method of bacteriologic confirmation frequently is necessary. The most commonly used tests in the ED are throat cultures and rapid antigen detection tests (RADTs). Throat cultures often are considered the diagnostic standard because they have a 90-95% sensitivity for GABHS.[10] This sensitivity is affected by how the specimen is collected and cultured as well as by whether the patient recently has taken any antibiotics.[9] The two main drawbacks to throat cultures are that they cannot distinguish between acute infections and carrier states and that they require 24-48 hours for results.[8,16] RADTs avoid a long waiting period for results but are less sensitive than cultures (70-90% sensitive).[16] Thus, a negative RADT often is followed by a confirmatory throat culture.[10] However, recent studies have suggested that confirmation of negative RADTs may not be necessary in adults.[11]

Because of the limitations of these studies, physicians have tried to create clinical guidelines to determine who to test and/or treat for GABHS. Many of these algorithms and scoring systems are complicated and inaccurate.[9] Attia et al have proposed the following for children and adults. If a patient has tonsillitis and tender adenopathy without coryza (with or without a scarlatiniform rash), treat without testing. If he or she does not have a rash and lacks one of the aforementioned criteria, test and treat if positive. If he or she has coryza without a rash and without adenopathy, do not test or treat.[8] In contrast, Rosen's recommends treating children if they have positive RADTs or cultures. In adults, empiric treatment is recommended if the incidence of GABHS is high or if the clinical examination is highly suspicious for GABHS pharyngitis. If the probability of GABHS is low, treatment is based on positive test results.

Although GABHS infection often is self-limited, it is the only common cause of pharyngitis for which antibiotics are indicated.[10,11,14] Antibiotic therapy can hasten the resolution of symptoms, limit the spread of infection, and prevent suppurative complications.[14,16] Perhaps most importantly, antibiotics begun within nine days after the onset of symptoms can prevent the

Table 1. Various Viral Agents

ORGANISM	CLINICAL PRESENTATION	DIAGNOSIS TESTS	TREATMENT
Adenovirus	Fever, malaise, pharyngo-tonsillitis, and conjunctivitis.	None specific	Symptomatic because infection resolves in 6-7 days
Epstein-Barr virus (infectious mononucleosis)	Viral prodrome. Exudative pharyngitis, palatal petechiae, splenomegaly.[8,12,17] Acute infection may be followed by months of easy fatigability and malaise.[8]	CBC with atypical lymphocytosis [Marx], elevated liver transaminases common,[13] monospot test for heterophil antibodies (less sensitive in children)[9]	Steroids, hydration, pain control (e.g., ibuprofen [Advil, Motrin]). Avoid contact sports because of risk of splenic rupture.[9] Avoid ampicillin (Omnipen)/ amoxicillin (Amoxil) because of risk of rash.[12]
Herpes simplex virus	Painful, shallow ulcers on erythematous base on lips, gums, soft palate, buccal mucosa.[9,11] Fever and adenopathy common.	Serology, viral culture, cytopathology of fluid from vesicles	Symptomatic. Immunocompromised patients may require acyclovir (Zovirax), famciclovir (Famvir) or valacyclovir (Valtrex).[9]
Influenza A and B	Common in winter and early spring. High fever, myalgias, headache, cough. Tonsillar exudates and lymphadenopathy rare.[9,12]	Rapid antigen tests more timely than traditional viral culture or serology	Generally symptomatic because pharyngitis resolves in 3-4 days.
Coxsackie virus	Herpangina: Small vesicles and/or ulcers on erythematous base on tonsils, uvula, and soft palate; fever, coryza. Hand, foot, and mouth disease: Similar oropharyngeal findings with vesiculopustular lesions or shallow ulcers on their palms and soles.[8,12]	None specific	Symptomatic
Cytomegalovirus	Typical signs and symptoms of IM but a negative monospot test or Epstein-Barr virus titer.	CMV-specific IgG and IgM antibody titers	Symptomatic. Immunocompromised individuals may require ganciclovir (Cytovene) or foscarnet (Foscavir).[12]
HIV	Acute infection with a flu-like illness, including fever and sore throat.[11]	Usually enzyme-linked immunoassay followed by confirmatory Western blot	Anti-retroviral therapy

Key: CBC = complete blood count; CMV = cytomegalovirus

development of acute rheumatic fever.[16] Penicillin still is first-line therapy; however, failure rates have been as high as 35% due to poor compliance, reinfection, and co-pathogenicity with beta-lactamase-producing organisms.[14] Alternative regimens include clindamycin, first-generation cephalosporins, and macrolides.[8,14] (*See Table 3.*)

Rheumatic fever and glomerulonephritis (GN) deserve special mention. (*See Table 4.*) As mentioned above, acute rheumatic fever (ARF) can be prevented if antibiotic therapy is instituted early. ARF is characterized by a combination of carditis (peri-, myo-, or endo-), arthritis, skin nodules, and chorea about 2-6 weeks after pharyngitis.[12,17] In contrast, GN cannot be prevented by the administration of antibiotics. Patients with GN present with edema, hypertension, hematuria, and proteinuria about 10 days after the initial GABHS infection. Permanent renal failure can occur as a result.[12]

Peritonsillar Abscess. Peritonsillar abscess (PTA) is the most common deep space infection of the head and neck.[14] Modern-day PTAs first were described in the early 1700s.[18]

PTAs are most prevalent in previously healthy adults between 15 and 40 years of age.[14,19] Cases occur more frequently from November-December and April-May.[13] In previous studies, PTAs recur in 0-23% of cases.[19]

Table 2. Miscellaneous Agents

ORGANISM	PRESENTATION	TREATMENT
Groups C and G Streptococcus	Resembles GABHS pharyngitis. GN can follow (not ARF).	Penicillin, a cephalosporin, or erythromycin.[8]
Mycoplasma pneumoniae	Often in epidemics or in crowded conditions. Mild exudative pharyngitis with signs of a lower respiratory tract infection.	Erythromycin, doxycycline (Vibramycin), and tetracycline (Sumycin)[9]
Arcanobacterium haemolyticum	Common among adolescents and young adults.[9,11] GABHS-like infection with pharyngeal erythema and exudates, adenopathy, and a scarlatiniform rash.	Erythromycin[8]
Neisseria gonorrhoeae	Sexually active patients (especially those who practice fellatio).[8,35] In young children, rule out sexual abuse.[8] Often asymptomatic or sore throat and exudative or ulcerative tonsillopharyngitis. May not have concurrent genital infection. Intracellular gram-negative diplococci on tonsillar swab. Confirm by PCR or culture of the tonsils, blood, or genital area.[13,35]	Ceftriaxone (Rocephin) 125 mg IM x1, ciprofloxacin (Cipro) 500 mg PO x 1, or ofloxacin (Floxin) 400 mg PO x 1. Azithromycin (Zithromax) (1 gm PO x 1) or doxycycline (100 mg PO bid x 7 days) to treat any concurrent Chlamydial infection.[9,12]
Chlamydia	*C. pneumoniae*: Epidemics or crowded places. Recurrent and persistent pharyngitis, laryngitis, and lower respiratory tract infections. *C. trachomatis*: Sexually-transmitted disease. Asymptomatic or mild sore throat.	*C. pneumoniae*: Doxycycline, trimethoprim-sulfamethoxazole (Bactrim) and macrolides.[9] *C. trachomatis*: Treated like gonococcal pharyngitis.
Corynebacterium diphtheriae	Fever, mild sore throat, and dysphagia. Thick, gray pseudomembrane can cause stridor and respiratory distress. "Bull-neck" from severe inflammation and edema.[9,12] Toxin causes myocarditis, arrhythmias, polyneuritis, nephritis, hepatitis, and vascular collapse.[9,13]	Antitoxin and erythromycin or penicillin.
Anaerobes (Vincent's angina)	Associated with poor oral hygiene, malnourishment, leukopenia, or immunocompromise.[9,12] Severe halitosis, purulent exudates, and submandibular adenopathy.[12]	Clindamycin (Cleocin), penicillin, cephalosporins, and beta-lactamase-resistant penicillins.[12]
Candida	Associated with immunocompromise. Adherent white plaques in oropharynx, dysphagia, odynophagia. Hyphae on Potassium hydroxide (KOH) prep or culture on Sabouraud's agar.	Fluconazole (Diflucan), itraconazole (Sporanox), nystatin (**M**ycostatin), and clotrimazole (Lotrimin).[9]

Pathophysiology. The palatine tonsils are surrounded by the palatoglossal muscle anteriorly, the palatopharyngeal muscle posteriorly, and fibrous connective tissue laterally.[20] PTAs occur when pus collects between the tonsillar capsule, lateral pharyngeal wall, and the supratonsillar space. If the infection spreads into the surrounding muscles (such as the internal pterygoids), muscle spasm and trismus can result. If the abscess drains into the superior tonsillar crypt or along the soft palate, pus may flow into the mouth or throat.[18]

As a general rule, PTAs are considered a complication of follicular tonsillitis. In these cases, direct extension leads to peritonsillar cellulitis, tissue necrosis, and abscess formation. Other possible mechanisms include obstruction and infection of Weber's glands (salivary glands in the superior pole of the tonsillar fossae) or hematologic or lymphatic spread of bacteria.[18,20]

Most PTAs are found to contain mixed aerobic and anaerobic flora. The most commonly isolated organisms include *Streptococcus pyogenes*, Bacteroides, Peptostreptococcus, and *Staphylococcus aureus*. One study revealed beta-lactamase producing organisms in more than half of the PTAs that they cultured.[21]

Table 3. Antibiotic Regimens for GABHS Pharyngitis

PENICILLIN

- Penicillin V 250 mg PO bid-tid x 10 days (peds) or 500 mg PO bid-tid x 10 days (adults)
- Benzathine penicillin G 50,000 units/kg IM x 1 (maximum 1.2 million units)

CLINDAMYCIN

- Clindamycin (Cleocin) 8-25 mg/kg/day PO divided tid-qid (peds) or 150-450 mg PO qid x 10 days (adults)

CEPHALOSPORINS

- Cephalexin (Keflex) 12.5 mg/kg or 250 mg PO tid-qid x 10 days

MACROLIDES

- Azithromycin (Zithromax) 12 mg/kg PO qd x 5 days (maximum 500 mg)
- Clarithromycin (Biaxin) 7.5 mg/kg PO bid x 10 days (maximum 500 mg per dose)
- Erythromycin 40 mg/kg/day divided bid-qid x 10 days (maximum 1 gm/day)

Complications of GABHS pharyngitis can be divided into suppurative and nonsuppurative types. (*See Table 4.*)

Table 4. Complications of GABHS Pharyngitis

SUPPURATIVE

- Sinusitis
- Otitis media
- Peritonsillar abscess
- Deep neck infection
- Cervical adenitis

NONSUPPURATIVE

- Acute rheumatic fever
- Post-streptococcal glomerulonephritis
- Erythema nodosum
- Toxic shock syndrome

Clinical Presentation. Patients with PTAs often complain of fever, sore throat, malaise, muffled voice, odynophagia, and dysphagia. On exam, they have unilateral tonsillar hypertrophy and palatal edema. The affected tonsil is displaced inferiorly and medially, and the uvula is deviated away from it. In addition, there may be evidence of dehydration, drooling, halitosis, or trismus.[13,18,19,22] Differential diagnoses include peritonsillar cellulitis, infectious mononucleosis, herpes simplex tonsillitis, parapharyngeal or retropharyngeal abscess, foreign body, internal carotid artery aneurysm, and various neoplasms (e.g., lymphoma, leukemia).[13,18]

PTAs often are evident clinically. However, some studies have suggested the use of ultrasound to differentiate cellulitis from abscess, since the former can be treated medically while PTAs require drainage. In addition, ultrasound can localize any pus collection that needs to be drained. Studies have demonstrated sensitivities of 82-91% for ultrasound detection of PTAs.[23] Although CT has been shown to have 100% sensitivity in differentiating between cellulitis and abscess, routine use is not recommended due to expense and radiation exposure.[22-24] However, CT may be indicated in a stable patient in whom a thorough clinical examination is not possible, whose peritonsillar aspiration was not possible or was negative, or in whom a deep neck infection is suspected.[13,25]

The gold standard for diagnosis of PTA also is its first-line therapy: needle aspiration of pus. After the tonsillar mucosa is anesthetized with topical lidocaine gel or spray followed by an injection of 1-2 cc of lidocaine with epinephrine, an 18-gauge needle is inserted into the abscess. Because the carotid artery lies inferiorly and laterally, the needle should be directed medially and superiorly and no deeper than 1 cm. If no pus is obtained, aspiration can be attempted more inferiorly.[13] Any fluid obtained should be sent for Gram stain and cultures (aerobic and anaerobic).

Treatment. Before the introduction of antibiotics, most cases of PTA required surgical intervention.[18] At that time, many clinicians believed that tonsillectomy (either immediate or delayed) was the only method to completely drain the abscess and to decrease the rate of recurrence. In 1961, King advocated the use of needle aspiration as a viable alterative to surgery.[25] Subsequent studies have reported a 90-94% rate of resolution of PTAs with needle aspiration alone.[25] Currently, most adults can be treated as outpatients with needle aspiration, antibiotics, and pain medications. If needle aspiration is unsuccessful, alternative therapies include intraoral incision and drainage and tonsillectomy. Children, however, require inpatient therapy with intravenous fluids, antibiotics, and drainage under general anesthesia.[19,26]

Antibiotics are essential in controlling the local infection as well as preventing its spread into the deep neck spaces.[27] In general, penicillin still is the initial antibiotic of choice. Because of the prevalence of penicillin-resistant organisms, other possible drug regimens include clindamycin (Cleocin), cefotaxime (Claforan), cefoxitin (Mefoxin), metronidazole (Flagyl), or beta-lactamase-resistant penicillins.[21,26] Unfortunately, there is no clear association between the patient's clinical presentation and the type of infecting organism(s) to guide the initial choice of antibiotics.[27]

Uvulitis. This condition is characterized by swelling of the uvula. Patients often present with a sore throat, odynophagia, or dysphagia. Some even may complain of "something hanging down in the back of my throat."[28] Although sometimes associated with upper airway obstruction,[29] many cases present without evidence of respiratory distress.

Uvulitis can result from angioedema (hereditary, allergic, idiopathic), local trauma (e.g., endotracheal intubation, laryngeal mask airway, orogastric tube insertion), and infection.[30] In infectious cases, uvular erythema and swelling have been associated with both exudative pharyngitis and epiglottitis.[31-33] Furthermore, there seems to be an association between pediatric uvulitis and *Haemophilus influenzae* infection.[29,31] Although adults with concomitant epiglottitis have only mild uvular inflammation, children tend to have a more severe presentation. The association

between uvulitis and epiglottitis may be explained by the spread of inflammation from the epiglottis into the soft tissues of the neck and pharynx or by uvular cellulitis.[32]

The diagnosis of uvulitis may be based on clinical exam alone.[28] However, some authors advocate obtaining lateral neck x-rays to rule out epiglottitis.[31,34] Treatment depends on the suspected underlying etiology. For example, the case of uvulitis secondary to attempted orogastric tube placement resolved spontaneously without any intervention.[30] Allergic causes may benefit from steroids and antihistamines, while antibiotics are appropriate for suspected bacterial infections. Obviously, if there is any evidence of upper airway obstruction, aggressive airway management is key.

Laryngitis. Laryngitis is caused by inflammation of the larynx and vocal folds. Patients commonly present with hoarseness or aphonia following an upper respiratory tract infection.[36] Although this illness resolves spontaneously in a few days, patients who are in voice-demanding professions often will seek medical care acutely.[37]

Etiology. Laryngitis most commonly is associated with viral infections such as influenza, rhinovirus, and adenovirus; however, group A Streptococcus, pneumococcus, and *Staphylococcus aureus* can cause bacterial superinfection.[36] In addition, studies have shown patients with laryngitis to have a high rate of nasopharyngeal colonization with *Moraxella catarrhalis* and *Haemophilus influenzae*.[37] Uncommon causes include Klebsiella, Pseudomonas, tuberculosis, syphilis, and *Corynebacterium diphtheriae*. Patients who are immunocompromised also are at risk for fungal infections (e.g., Candida).[38]

Diagnosis. As a general rule, the diagnosis of laryngitis is based on history and physical exam. No other studies are needed unless there is a concern for epiglottitis or there is evidence of respiratory distress. Treatment is conservative and consists of voice rest, humidified air, and avoidance of irritants such as smoke. Antibiotics are indicated only if a bacterial infection is present and a specific pathogen is isolated.[39] Steroids may help decrease the duration of symptoms.[9] If hoarseness persists for more than 2-3 weeks, referral to an otolaryngologist is recommended.

Epiglottitis. George Washington is speculated to have died from epiglottitis in 1799.[40] However, "angina epiglottidea anterior" was not formally described until 1878 by Michel. In 1900, Thiesen wrote about similar cases that he renamed "acute infectious epiglottitis."[41] At first, this infection was found primarily in adults. By the 1950s-1960s, it had become a disease of childhood.[41] Since the introduction of the *Haemophilus influenzae* type B (HiB) vaccine in 1985, the incidence in children has been decreasing, and epiglottitis has again become more common in adults.[13,42] Despite modern advances in therapy, epiglottitis still is associated with a significant mortality (less than 1% in children and 6-7% in adults).[40] Unfortunately, several studies have shown that 35-70% of cases initially may be misdiagnosed.[13,40,43] Thus, the physician must suspect epiglottitis in any patient that has an acutely sore throat and dysphagia.[43]

Pathophysiology. Epiglottitis is characterized by inflammation of the epiglottis, aryepiglottic folds, and the loose connective tissue in the pre-epiglottic and paraglottic spaces.[13,44] Stridor and the concomitant risk of airway obstruction are thought to result from edematous supraglottic mucosa prolapsing into the glottis. Another proposed mechanism is that swelling of the supraglottic tissues impairs swallowing, leading to pooling of secretions.[43-45]

Epiglottitis traditionally occurs most commonly in children 2-8 years old. In adults, the peak incidence is 35-39 years of age.[46] It occurs more often during the spring and late fall.[41] There appears to be a male predominance in both children and adults.[41,46] In addition, epiglottitis is more prevalent in African-Americans.[13]

Although no organisms are isolated in most adult cases, the most common cause of epiglottitis overall is HiB.[13] Up to 36% of these cases may be ampicillin-resistant.[40] Epiglottitis also can be caused by bacteria such as Streptococcus, Staphylococcus, *Moraxella catarrhalis*, Klebsiella, *Mycobacterium tuberculosis*, *Haemophilus parainfluenzae,* and *Escherichia coli*. Viral etiologies include respiratory syncitial virus, varicella, adenovirus, and herpesvirus. Fungi such as Candida and Aspergillus also may be inciting agents.[13,46] Immunocompromised patients are at risk for atypical agents.[40,46]

As mentioned earlier, the HiB vaccine has changed the epidemiology of epiglottitis. In 1985 the vaccine was introduced in the United States for children older than 24 months. Since 1990, conjugate forms have been available for those 2 months of age and older. Even unvaccinated children seem to be provided some protection from HiB by herd immunity.[42] Gorelick and Baker reported an 84% decrease in the annual incidence of pediatric admits for epiglottitis since 1990. They also noted the emergence of infection in older children and caused by other bacteria, especially GABHS.[47]

Clinical Presentation. As a general rule, children with epiglottitis appear ill. They present with sore throat and fever (temperature greater than 101° F or 38.4° C) and progress in 4-8 hours to dysphagia, dyspnea, dysphonia, and drooling (the four Ds).[44,48] In young children, irritability or lethargy may prevail. If in extremis, the patient may have severe inspiratory stridor, suprasternal and infrasternal retractions, and cyanosis.[45,49]

In contrast, adults tend to have a more insidious onset and a more benign illness.[50] They may have a 1-2 day prodrome consistent with an upper respiratory tract infection. This is followed by sore throat, odynophagia, drooling, muffled voice, and anxiety. Fever may be absent in up to half of these patients on initial examination.[13,46] Stridor occurs more infrequently (possibly because the adult airway is larger and more rigid).[51]

Both children and adults may not be able to lie supine because of problems handling secretions. They classically prefer to tripod (leaning forward on outstretched arms) or assume the sniffing position (mouth open, neck extended) to keep their airways open and improve air entry.[40,46] Pain with palpation of the upper trachea or thyroid cartilage should raise the suspicion of epiglottitis.[46]

The differential diagnosis of epiglottitis includes PTA, tonsillitis, infectious mononucleosis, retropharyngeal abscess, angioneurotic edema, diphtheria, pertussis, croup, foreign body, and laryngeal tumor or trauma. Epiglottic edema also has been reported with toxin inhalation, hydrocarbon aspiration, and crack cocaine smoking.[40,43,51]

Diagnostic Evaluation. In patients who have no signs of respiratory distress and do not have a definitive diagnosis, radi-

ographs and/or laryngoscopy may be useful.[43,51] Lateral soft tissue neck x-rays are reported to have a sensitivity anywhere from 38% to 90% for epiglottitis.[40,51] Radiographic findings suggestive of epiglottitis include swelling of the aryepiglottic folds, edema of the prevertebral and retropharyngeal soft tissues, ballooning of the hypopharynx, and an enlarged epiglottis (thumb sign).[13,44,45] Ducic et al have suggested that a decrease in vallecular air space can improve the sensitivity of plain films to 98.2%. Furthermore, they reported an equal sensitivity among x-rays read by emergency physicians, radiology residents, and medical students after just 5 minutes of training about the vallecula sign.[52] Because x-ray interpretation ultimately is subjective, negative radiographs do not rule out epiglottitis.[46,51]

The gold standard of diagnosis is visualization of a red, swollen epiglottis.[46] Because of anecdotal reports of complete airway obstruction after tongue blade insertion, visual inspection is avoided unless the physician is prepared to establish a definitive airway.[44] Unlike in children, fiberoptic laryngoscopy generally is safe in adults and can be performed in the emergency department (ED) to view the epiglottis.[40]

Although some experts believe that blood and surface cultures of the epiglottis are not helpful in the acute management of illness, they can help confirm HiB infection;[46] however, they should not be collected until a definitive airway is established.[13] Before the introduction of the HiB vaccine, blood cultures were positive for HiB in more than 80% of children.[44] In general, bacteremia is less common in adults, but cultures positive for HiB may herald a more fulminant disease course.[51]

Treatment. Treatment depends on the degree of respiratory distress, progression of disease, and experience of the caregiver. The main goal of ED care is to avoid airway obstruction.[45] This involves constantly monitoring the patient, keeping him/her in a quiet environment, and allowing him/her to sit in the most comfortable position. Painful, invasive procedures and interventions (including IV hydration and antibiotics) should be avoided until a definitive airway is in place.[44,50] Supplemental humidified oxygen should be given, and immediate otolaryngologic consultation is necessary.

Controversy still exists as to whether all patients with epiglottitis need to have a definitive airway established and how best to accomplish this. Because only one-third of adults with epiglottitis require intubation, careful observation is an option in some patients.[46] Friedman et al have proposed a standardized protocol for adults. Patients without respiratory distress can be observed closely in the intensive care unit (ICU). Those with slight respiratory difficulty need intubation in the operating room (OR), while those with moderate to severe respiratory distress require immediate intervention.[53] Because children have a higher rate of requiring intubation, physicians tend to be more conservative about airway management in this patient population. Bank and Krug have created an algorithm for pediatric patients; unstable patients need immediate airway control. Those who are stable and have epiglottitis should have an examination of the airway in the OR with intubation as needed. Those who are stable with an uncertain diagnosis can be observed closely.[44]

Before the 1960s-1970s, tracheostomy was the airway of choice.[127] Other options have developed since, including nasotracheal or endotracheal intubation and cricothyrotomy. Ideally, intubation is performed in the OR in a controlled environment with provisions made for a surgical airway should attempts at intubation fail. Since emergency physicians often have to deal with less-than-ideal circumstances, they must be prepared to intubate unstable patients in the ED. Distortion of the anatomy is to be expected as a result of supraglottic edema.[13] Placing direct pressure on the chest may help guide the intubation by producing an air bubble at the opening of the airway.[44] If intubation is not possible, cricothyrotomy is the simplest and most efficacious rescue airway.[43]

Although ampicillin (Omnipen) used to be the antibiotic of choice, increasing incidence of beta-lactamase-producing HiB has led to a change in regimen.[46] Currently second- or third-generation cephalosporins such as cefuroxime, cefotaxime, and ceftriaxone are preferred.[13] Adjunctive steroids are controversial because there are no controlled studies about their use in epiglottitis.[51] However, many otolaryngologists still use them because they believe steroids help reduce airway edema.[13,51]

Patients with epiglottitis usually recover in 2-4 days without problems. Household contacts should receive rifampin prophylaxis.

Deep Neck Infections. Deep neck infections occur in potential spaces formed by the fascial planes of the neck. Although these infections have decreased in frequency since the advent of antibiotics, they still are associated with significant morbidity and mortality.[55] Parapharyngeal and retropharyngeal abscesses will be discussed separately from Ludwig's angina due to differences in their pathophysiologies and presentations.

Parapharyngeal Abscess. The parapharyngeal space is limited superiorly by the base of the skull, inferiorly by the hyoid, medially by the buccopharyngeal fascia, and laterally by the pterygoid muscles and mandible. It abuts the submandibular space anteriorly and the vertebral column and paravertebral musculature posteriorly.[56,57] It contains several important structures, including the internal and external carotids, internal jugular vein, deep lobe of parotid gland, sympathetic truck, and cranial nerves IX-XII.[57,58]

Twenty to 30 percent of parapharyngeal abscesses (PPAs) are the result of odontogenic infections. Other sources are gingivitis, pharyngitis, tonsillitis, and mastoiditis. These infections can spread via lymphatics or via the styloglossus muscle plane from adjacent areas (e.g., floor of mouth, parotid space, peritonsillar space, masseteric space, submandibular space).[58]

Retropharyngeal Abscess. The retropharyngeal space lies between the middle and deep layers of the deep cervical fascia and ends inferiorly when these layers fuse at the level of tracheal bifurcation (T1).[54,59] It is bounded superiorly by the skull base, anteriorly by the superior constrictor muscles, posteriorly by the alar fascia, and laterally by the carotid sheaths.[57,60] Although the retropharyngeal space is separated from the parapharyngeal space by the alar fascia, infection still can spread between them. In addition, the retropharyngeal space contains two paramedial chains of lymph nodes which drain the nasopharynx, adenoids, and posterior nasal sinuses.[13]

Retropharyngeal abscesses (RPAs) often result from infection and suppuration of these nodes.[55] This can be precipitated by

antecedent upper respiratory tract infection, sinusitis, otitis media, pharyngitis, or tonsillitis. Because these nodes usually atrophy by 3-4 years of age, this is thought to be the major mechanism in children. Most RPAs occur in young children, and a majority of these children are younger than 12 months old.[13] In contrast, most adult cases of RPA are related to local trauma, immunocompromise, foreign body ingestion, or procedural complications (e.g., endoscopy, intubation).[61]

Cultures from both PPAs and RPAs usually reveal a combination of aerobes and anaerobes. The most common aerobes are *Streptococcus viridans* and *pyogenes*. Other Streptococcus species, *Staphylococcus aureus*, Neisseria, Haemophilus, *Moraxella catarrhalis*, and Enterobacteriaceae also have been isolated. The most frequently found anaerobes are Bacteroides, Peptostreptococcus, and Fusobacterium.[42,48,63] Of note, 41-71% of cultures grew beta-lactamase-producing organisms.[64,65]

Clinical Presentation. Deep neck infections can manifest a variety of symptoms. Children may present with more nonspecific complaints such as fever, poor oral intake, breathing problems, and irritability.[59] In those younger than 1 year of age, stridor and neck swelling may be more common.[63] Older patients are more likely to complain of sore throat, neck pain, odynophagia, and dysphagia. Although adults present with evidence of airway obstruction less often than children do, the clinician must search for signs of respiratory distress in all patients (e.g., increased work of breathing, anxiety, cyanosis, stridor).

On exam, patients may have a muffled voice, trismus, drooling, torticollis, or tender lymphadenopathy. They may limit their neck motion due to pain.[55,61] More specifically, Craig et al described their patients with RPAs as moving their eyes only (instead of their heads) to look up and preferentially keeping their necks in a neutral position.[61] Although there may be medial displacement of the tonsil, bulging of the posterior or lateral pharynx and/or swelling at the angle of the mandible can help distinguish deep neck infections from PTAs.[58] Unfortunately, posterior pharyngeal swelling is present only in a minority of patients. The examination of the posterior pharynx often is difficult in children due to the size of their oropharynx and pooling of secretions. As a result, up to one-third of cases may be misdiagnosed initially.[60]

The rule to remember is that a negative exam in a patient complaining of severe throat or neck pain does not rule out a deep neck infection. Pain out of proportion to exam should, instead, increase the suspicion for a PPA or RPA.[59] In addition, clinical exam cannot distinguish a PPA from an RPA nor abscess from cellulitis.[57]

Diagnostic Evaluation. Deep neck infections usually can be diagnosed with a thorough clinical evaluation and selected imaging.[61] Lateral neck x-rays traditionally have been a mainstay in the workup. Findings suspicious for an RPA include thickened prevertebral soft tissue, foreign body, loss of normal cervical lordosis, and air or air-fluid level in the soft tissue. The prevertebral soft tissue is considered thickened if it is wider than the adjacent vertebral body, greater than 7 mm at C2, or greater than 22 mm at C6. (Note: In children, the cutoff is less than 14 mm at C6.)[55,66] Radiographs should be taken at end expiration with the neck in extension since soft-tissue thickness varies with respiration, crying, swallowing, and position.[55] Because plain films are reportedly only 88% sensitive for RPAs, they are being supplanted by other imaging modalities.[63]

CT now is the radiologic study of choice for deep neck infections.[57,65] With intravenous contrast, an abscess appears as an area of low attenuation with a ring-enhancing wall.[67,68] CT offers advantages over plain films, including the ability to differentiate abscess from cellulitis, ascertain abscess size and location, determine the relative position of the great vessels and other anatomic structures, identify other causes of retropharyngeal thickening, and detect internal jugular thrombosis.[13,57] Studies have shown CT to have a 90-95% sensitivity for deep neck infections.[56,57]

Studies are examining ultrasound and MRI as alternatives for the detection of deep neck infections. Although ultrasound is reliable in differentiating abscess from cellulitis and does not require any radiation exposure, a highly experienced interpretation is necessary.[56,65] MRI offers better soft-tissue definition than CT, can detect impending carotid artery rupture, and does not expose the patient to any radiation. However, it takes more time to perform, is more expensive, and usually is less available than CT.[56]

Treatment. Patients with deep neck infections require immediate otolaryngologic consultation. In addition, airway maintenance is a high priority.[58] Patients without any evidence of airway obstruction can be observed and monitored closely. However, if they have any respiratory distress, a definitive airway is needed. Although tracheostomies traditionally were considered the airway of choice, later studies have shown them to be necessary in only a minority of cases.[69] In most cases, intubation (endotracheal or fiberoptic nasotracheal) has become the preferred method. If the anatomy is severely distorted, this may be difficult, if not impossible. Thus, the physician always must be prepared to perform a surgical airway.

Controversy still exists as to the most effective means of treatment. Conventional wisdom holds that all deep neck infections require early surgical drainage.[56,58] However, studies have shown that antibiotics alone can cure 10-57% of patients.[55,70] In general, cellulitis responds well to antibiotics, whereas abscesses more commonly require drainage. This has led to a change in paradigm: Patients with CT findings of cellulitis alone can be managed with empiric IV antibiotics. Those with an abscess and respiratory distress need to go to the operating room. Those with an abscess without evidence of airway compromise can be managed either with a trial of antibiotics or immediate surgical drainage.[55,61]

Empiric antibiotic therapy for deep neck infections should cover normal oropharyngeal flora and beta-lactamase-producing organisms. Penicillin used to be the antibiotic of choice because of its low cost and its good penetration of infected tissues.[56] However, as penicillin resistance is becoming more prevalent, alternative regimens have developed: penicillin plus metronidazole, clindamycin, cefoxitin, aminoglycosides, imipenem, penicillin plus beta-lactamase inhibitor.[56,65]

Complications. Rupture of a deep neck abscess into the airway can lead to asphyxiation, pneumonia, empyema, or lung abscess. Other complications include airway obstruction, epidural abscess, atlanto-axial dislocation, cavernous sinus thrombosis,

mediastinitis, internal jugular thrombosis, pneumomediastinum, and carotid artery rupture.[55,59,71,72]

Internal jugular (IJ) thrombosis is the most common vascular complication of deep neck infections.[56] Patients present with fever, chills, and tenderness and swelling along the course of the IJ. These patients are at risk for bacteremia, septic thrombi, and pulmonary emboli. Medical management consists of anticoagulation and antibiotics. If this is not successful, the vein may need to be ligated and removed.

Carotid artery rupture is much less common but carries a mortality rate of 20-40%.[73] This occurs most commonly with the internal carotid and should be suspected when blood clots are found during abscess drainage.[56]

Ludwig's Angina. Ludwig's angina is a rapidly progressing cellulitis of the submandibular space that was first described by Wilhelm Frederick von Ludwig in 1836.[74]

Pathophysiology. The submandibular space is limited by the floor of the mouth (superiorly) and the superficial layer of the deep cervical fascia (inferiorly). This area is further subdivided by the mylohyoid muscle into the submaxillary and sublingual spaces, which communicate posteriorly. Because Ludwig's angina spreads along fascial planes, extension is limited inferiorly by the deep cervical fascia. Instead, infection spreads from the submaxillary area to the connecting sublingual space, causing induration of the floor of the mouth and elevation and protrusion of the tongue. In some cases, extension occurs along intrinsic tongue musculature into the parapharyngeal or retropharyngeal spaces or even into the mediastinum.[75,76]

Up to 90% of cases of Ludwig's angina arise from an odontogenic source. Of these, the second or third mandibular molars most commonly are involved because their roots extend below the mylohyoid line of the mandible, allowing infection to spread into the submaxillary space.[77] Other etiologies include oral lacerations, mandibular fractures, peritonsillar or parapharyngeal abscesses, submandibular sialoadenitis, and infected oral malignancies.[75,78] In a minority of cases, no source is identified.[79]

Although bacterial isolates in wound cultures are mixed in about 50% of cases, the most commonly isolated organism is Streptococcus, followed by Staphylococcus. Anaerobes are found in almost one-third of cases, with Bacteroides being the most prevalent.[80] Other reported organisms include gram-negative rods (such as Pseudomonas, *Escherichia coli*, Klebsiella, Haemophilus), Candida, Clostridium, Corynebacteria, *Enterococcus fecalis*, *Aerobacter aeruginosa*, and Peptostreptococci.[81]

Clinical Presentation. The most common chief complaints of patients with Ludwig's angina include neck swelling and tooth pain, followed by dysphagia and dyspnea. On exam, patients have a brawny or "woody" induration of the submental area and floor of the mouth as well as an elevated or protruding tongue.[79] Other signs and symptoms may include fever, trismus, and odynophagia.[75,78] Because of the risk of airway compromise in these patients, the clinician must search for signs of impending obstruction or respiratory distress, including tachypnea, increasing anxiety, stridor, cyanosis, difficulty managing secretions, and retractions.

Although Ludwig's angina is a clinical diagnosis, blood cultures may help in isolating a causative organism. CT scan may help determine the size, location, and extent of infection. In one study, physical exam plus CT scan had a 95% sensitivity and 80% specificity for detecting drainable collections of fluid.[82]

Treatment. The mainstays of treatment include airway management, IV antibiotics, and early surgical consultation. Because the most serious early complication of Ludwig's angina is airway obstruction, maintaining a secure airway is of the utmost importance. How this is accomplished still is somewhat controversial. First is the question of close observation vs. immediate intervention. Patients who present in the early stages of disease without any signs of respiratory distress or airway compromise may be observed.[83] Obviously those with impending obstruction or who require incision and drainage under anesthesia require airway control.[84] For those who require intervention, what is the best method of securing the airway? Classic training teaches that tracheostomy is the method of choice. Other options include orotracheal intubation with direct laryngoscopy, awake nasal intubation with flexible fiberoptic scope, or cricothyroidotomy.[78] In general, this decision should be made based on the condition of the patient, physician experience, and available resources.[83] Blind nasotracheal intubation should be avoided because of the risk of bleeding or abscess rupture.[81]

With the advent of antimicrobial therapy, the mortality of Ludwig's angina has decreased from greater than 50% to fewer than 10%.[79] High-dose penicillin usually is the initial drug of choice. Alternative regimens include combinations of penicillins, aminoglycosides, clindamycin, chloramphenicol, and metronidazole (for penicillin-resistant anaerobes). In addition, IV steroids may be useful to stop the progression of edema and prevent need for invasive airway procedures.[85,86]

Early surgical consultation is important to assess the need for surgical airway procedures and/or surgical decompression. Although incision and drainage used to be routine in all cases, it now commonly is reserved for patients whose infection persists or progresses despite optimal medical therapy or who have evidence of a localized abscess.

Complications. Complications of Ludwig's angina include airway obstruction, mediastinitis, empyema, pericarditis, pneumothorax, subphrenic abscess, pneumonia, sepsis, necrotizing fasciitis, and jugular vein thrombosis.[75,84]

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Physician CME Questions

71. Most cases of pharyngitis result from a viral infection.
    A. True
    B. False

72. Which of the following is *not* a suppurative complication of pharyngitis?
    A. Deep neck infection
    B. Acute rheumatic fever
    C. Peritonsillar abscess
    D. Otitis media
    E. Sinusitis

73. Treatment options for GABHS include all the following *except*:
    A. penicillin.
    B. cephalexin.
    C. tetracycline.
    D. clindamycin.
    E. macrolides.

74. All of the following are true regarding facial nerve palsy *except:*
    A. Most patients recover without treatment.
    B. Anti-viral medication is of proven benefit.
    C. The American Academy of Neurology notes that available evidence suggests that steroids are probably effective.
    D. A recent Cochrane review concluded no significant benefit with corticosteroids.

75. Which of the following is the most common deep space infection of the head and neck?
    A. Rheumatic fever
    B. Glomerulonephritis
    C. Peritonsillar abscess
    D. Uvulitis

76. Which of the following statements is true of PTAs?
    A. They often are evident clinically.
    B. They require drainage.
    C. On exam, patients with PTAs have unilateral tonsillar hypertrophy and palatal edema.
    D. All of the above

77. Overall, which of the following is the most common cause of epiglottitis?
    A. *Moraxella catarrhalis*
    B. *Haemophilus influenzae* type B (HiB)
    C. *Mycobacterium tuberculosis*
    D. *Escherichia coli*

78. CT is the radiologic study of choice for deep neck infections.
    A. True
    B. False

79. Which of the following statements is true regarding deep neck infections?
    A. Patients with deep neck infections require immediate otolaryngologic consultation.
    B. Empiric antibiotic therapy for deep neck infections should cover normal orpharyngeal flora and beta-lactamase-producing organisms.
    C. Internal jugular thrombosis is the most common vascular complication of deep neck infections.
    D. All of the above.

80. Which of the following usually is the initial drug of choice for treatment of Ludwig's angina?
    A. Aminoglycosides
    B. High-dose penicillin
    C. Clindamycin
    D. Chloramphenicol

CME Answer Key: 71. A; 72. B; 73. C; 74. B; 75. C; 76. D; 77. B; 78. A; 79. D; 80. B

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Ear, Nose, and Throat Disorders Part II

Various Viral Agents

ORGANISM	CLINICAL PRESENTATION	DIAGNOSIS TESTS	TREATMENT
Adenovirus	Fever, malaise, pharyngo-tonsillitis, and conjunctivitis.	None specific	Symptomatic because infection resolves in 6-7 days
Epstein-Barr virus (infectious mononucleosis)	Viral prodrome. Exudative pharyngitis, palatal petechiae, splenomegaly.[8,12,17] Acute infection may be followed by months of easy fatigability and malaise.[8]	CBC with atypical lympho-cytosis [Marx], elevated liver transaminases common,[13] monospot test for heterophil antibodies (less sensitive in children)[9]	Steroids, hydration, pain control (e.g., ibuprofen [Advil, Motrin]). Avoid contact sports because of risk of splenic rupture.[9] Avoid ampicillin (Omnipen)/ amoxicillin (Amoxil) because of risk of rash.[12]
Herpes simplex virus	Painful, shallow ulcers on erythematous base on lips, gums, soft palate, buccal mucosa.[9,11] Fever and adeno-pathy common.	Serology, viral culture, cyto-pathology of fluid from vesicles	Symptomatic. Immunocom-promised patients may require acyclovir (Zovirax), famciclovir (Famvir) or valacyclovir (Valtrex).[9]
Influenza A and B	Common in winter and early spring. High fever, myalgias, headache, cough. Tonsillar exudates and lymphadenopathy rare.[9,12]	Rapid antigen tests more timely than traditional viral culture or serology	Generally symptomatic because pharyngitis resolves in 3-4 days.
Coxsackie virus	Herpangina: Small vesicles and/or ulcers on erythematous base on tonsils, uvula, and soft palate; fever, coryza. Hand, foot, and mouth disease: Similar oropharyn-geal findings with vesiculopustular lesions or shallow ulcers on their palms and soles.[8,12]	None specific	Symptomatic
Cytomegalovirus	Typical signs and symptoms of IM but a negative monospot test or Epstein-Barr virus titer.	CMV-specific IgG and IgM antibody titers	Symptomatic. Immunocom-promised individuals may require ganciclovir (Cytovene) or foscarnet (Foscavir).[12]
HIV	Acute infection with a flu-like illness, including fever and sore throat.[11]	Usually enzyme-linked immunoassay followed by confirmatory Western blot	Anti-retroviral therapy

Key: CBC = complete blood count; CMV = cytomegalovirus

Miscellaneous Agents

ORGANISM	PRESENTATION	TREATMENT
Groups C and G Streptococcus	Resembles GABHS pharyngitis. GN can follow (not ARF).	Penicillin, a cephalosporin, or erythromycin.[8]
Mycoplasma pneumoniae	Often in epidemics or in crowded conditions. Mild exudative pharyngitis with signs of a lower respiratory tract infection.	Erythromycin, doxycycline (Vibramycin), and tetracycline (Sumycin)[9]
Arcanobacterium haemolyticum	Common among adolescents and young adults.[9,11] GABHS-like infection with pharyn-geal erythema and exudates, adenopathy, and a scarlatiniform rash.	Erythromycin[8]
Neisseria gonorrhoeae	Sexually active patients (especially those who practice fellatio).[8,35] In young children, rule out sexual abuse.[8] Often asymptomatic or sore throat and exudative or ulcerative tonsil-lopharyngitis. May not have concurrent genital infection. Intracellular gram-negative diplococci on tonsillar swab. Confirm by PCR or culture of the tonsils, blood, or genital area.[13,35]	Ceftriaxone (Rocephin) 125 mg IM x1, ciprofloxacin (Cipro) 500 mg PO x 1, or ofloxacin (Floxin) 400 mg PO x 1. Azithromycin (Zithromax) (1 gm PO x 1) or doxycycline (100 mg PO bid x 7 days) to treat any concurrent Chlamydial infection.[9,12]
Chlamydia	*C. pneumoniae*: Epidemics or crowded places. Recurrent and persistent pharyngitis, laryngitis, and lower respiratory tract infections. *C. trachomatis*: Sexually-transmitted disease. Asymptomatic or mild sore throat.	*C. pneumoniae*: Doxycycline, trimethoprim-sulfamethoxazole (Bactrim) and macrolides.[9] *C. trachomatis*: Treated like gonococcal pharyngitis.
Corynebacterium diphtheriae	Fever, mild sore throat, and dysphagia. Thick, gray pseudomembrane can cause stridor and respiratory distress. "Bull-neck" from severe in-flammation and edema.[9,12] Toxin causes myocarditis, arrhythmias, polyneuritis, nephritis, hepatitis, and vascular collapse.[9,13]	Antitoxin and erythromycin or penicillin.
Anaerobes (Vincent's angina)	Associated with poor oral hygiene, malnourishment, leukopenia, or immunocompromise.[9,12] Severe halitosis, purulent exudates, and submandibular adenopathy.[12]	Clindamycin (Cleocin), penicillin, cephalosporins, and beta-lactamase-resistant penicillins.[12]
Candida	Associated with immunocompromise. Adherent white plaques in oropharynx, dysphagia, odyno-phagia. Hyphae on Potassium hydroxide (KOH) prep or culture on Sabouraud's agar.	Fluconazole (Diflucan), itraconazole (Sporanox), nystatin (**M**ycostatin), and clotrimazole (Lotrimin).[9]

Differential Diagnosis of Facial Palsy

- Sarcoidosis
- Lyme disease
- Diabetes
- Fungal/tuberculus meningitis
- Carcinomatous meningitis
- Tumor/neuroma
- Bacterial infection of middle ear, mastoid, or external auditory canal (EAC)

Complications of GABHS Pharyngitis

SUPPURATIVE

- Sinusitis
- Otitis media
- Peritonsillar abscess
- Deep neck infection
- Cervical adenitis

NONSUPPURATIVE

- Acute rheumatic fever
- Post-streptococcal glomerulonephritis
- Erythema nodosum
- Toxic shock syndrome

Antibiotic Regimens for GABHS Pharyngitis

PENICILLIN

- Penicillin V 250 mg PO bid-tid x 10 days (peds) or 500 mg PO bid-tid x 10 days (adults)
- Benzathine penicillin G 50,000 units/kg IM x 1 (maximum 1.2 million units)

CLINDAMYCIN

- Clindamycin (Cleocin) 8-25 mg/kg/day PO divided tid-qid (peds) or 150-450 mg PO qid x 10 days (adults)

CEPHALOSPORINS

- Cephalexin (Keflex) 12.5 mg/kg or 250 mg PO tid-qid x 10 days

MACROLIDES

- Azithromycin (Zithromax) 12 mg/kg PO qd x 5 days (maximum 500 mg)
- Clarithromycin (Biaxin) 7.5 mg/kg PO bid x 10 days (maximum 500 mg per dose)
- Erythromycin 40 mg/kg/day divided bid-qid x 10 days (maximum 1 gm/day)

Complications of GABHS pharyngitis can be divided into suppurative and nonsuppurative types.

Complications of PTAs

- Airway obstruction
- Deep neck abscess
- Thrombophlebitis
- Abscess rupture and aspiration
- Epiglottitis
- Endocarditis
- Mediastinitis
- Submaxillary artery ulceration
- Sepsis

Radiographic Findings of Epiglottitis

- Swelling of aryepiglottic folds
- Edema of prevertebral and retropharyngeal soft tissues
- Ballooning of the hypopharynx
- Thumb sign
- Vallecula sign

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