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The emergency physician commonly diagnoses and treats pediatric ear, nose, and throat (ENT) infections. The typical pediatric patient with an uncomplicated superficial infection has complaints that refer to the location of tissue invasion; however, younger children may be an exception and may present with fever and/or irritability. The physical examination typically reveals the site of infection. Rarely, a patient may display toxicity that is disproportionate to the localized infection, with evidence of a compromise of vital structures. These circumstances should lead emergency physicians (EPs) to pursue additional diagnostic investigations. This article will review common ENT infections, potential patient complications, and treatment strategies.

—The Editor

Common Pediatric ENT Infections

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Introduction

Traditional emergency department (ED) treatment of uncomplicated superficial ENT infections is medical rather than surgical. The EP judges whether viral, bacterial, or fungal agents are the likely offending microorganisms and if antibiotics are warranted. In the case of pharyngitis, initial antimicrobial therapy may be guided by rapidly returned microbiologic techniques. In other superficial infections, the initial antimicrobial therapy is expectant. The choice of antibiotic is based on clinical experience, recent use of antibiotics, patient allergies, and studies that have defined the predictable spectrum of organisms. The incidence of adverse outcomes with superficial ENT infections is low. The majority of infections remain in the original or proximal structures until the

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inflammatory process is resolved. However, infection may extend through contiguous structures or via hematogenous and lymphatic spread to involve distant areas. The EP must be cognizant of the potential for complications, and should provide appropriate instruction to discharged patients that would alert them to the possibility of treatment failure.

This article discusses three common pediatric ENT infections, pharyngitis, otitis media, and sinusitis, and discusses the typical and atypical clinical manifestations of these infections, as well as their diagnostic and therapeutic choices. It also highlights complications that may be life threatening — instances in which rapid recognition and therapy in the emergency setting are mandatory.

Pharyngitis

Definition and Anatomy. Pharyngitis is an inflammatory syndrome involving oropharyngeal structures. The oropharynx is defined posteriorly by the prevertebral fascia, laterally by the buccinator muscle groups, superiorly by the base of the skull, and inferiorly by the vocal cords. The potentially inflamed anatomical structures with pharyngitis include the lingual tonsils, palatine tonsils, adenoids, soft palate, and posterior pharyngeal wall.

Scope of the Problem. After viral upper respiratory infections (URIs), pharyngitis is one of the most common reasons for a child to see a physician.[1] Pharyngitis due to group A beta-hemolytic streptococcus (GABHS) is one of the most common bacterial infections seen in clinical practice.[2] After otitis media, pharyngitis is the most common reason for which a child is prescribed an antibiotic.[3]

Pharyngitis (sore throat) is among the most frequent complaints that cause a pediatric patient to visit the ED. ED encounters with pharyngitis involve all age groups; however, in preverbal children the parents may presume the child is having throat pain because of difficulty feeding. The majority of these visits will be non-urgent or urgent. Emergent encounters are rare. Emergency encounters may involve an overt or impending airway compromise, an extension of the infection into deeper planes within cervical structures, bacteremic extensions, or delayed development of acute rheumatic fever or glomerulonephritis.

Pathophysiology. The agents causing pharyngitis are typically spread by person-to-person contact. Pathogens are predominately transmitted by close-range airborne dissemination, such as a cough or sneeze, but also may be directly transferred by oral secretions (e.g., kiss, orogenital sex, pre-chewing of food with transfer to a child, adult "cleansing" by their own oral secretions of a pacifier, sharing of utensils, drinking from the same cup or bottle). Indirect transfer to hands of a future host may occasionally occur from fomites that are contaminated with oral secretions. In all circumstances of pharyngitis, the pathogen attaches to the epithelial cells of the oropharynx. The transferred agent competes with the resident flora and colonizes tissue. Invasion of tissue initiates a spectrum of symptoms and signs.

Bacteriology. One causative agent is identified in most pharyngitis cases, though multiple causative agents may be recovered.[4] As an example, with Epstein-Barr pharyngitis there may be recovery of GABHS as a copathogen in 10% of patients. More than 90% of recovered pathogens in the pediatric population include GABHS, adenoviruses, influenza A and B viruses, parainfluenza viruses, Epstein-Barr virus, enteroviruses, and *Mycoplasma pneumoniae*.[5] The recovered pathogen is closely related to age, season, and fluctuations in community outbreaks of contagion.

Incidence in Preschool-age Patients. Viral pharyngitis tends to occur more frequently in preschool-aged children than does bacterial disease. Parainfluenza viruses, adenoviruses, influenza viruses, rhinoviruses, respiratory syncytial virus, metapneumovirus, cytomegalovirus, and enteroviruses predominate in children < age 3-4 years. Bacterial agents cause pharyngitis in children ages 3 or younger in approximately 30% of cases. The most common bacterial pathogen in children < age 3 is GABHS. Unfortunately, misinformation continues to be passed to physicians who are told that GABHS pharyngitis is nonexistent in very young children.[6] Evidence-based literature reflects that GABHS pharyngitis has been reported to occur in infants as young as 8 weeks.[7] Glenzen et al reported a 3% isolation of GABHS in children < age 3.[5] Schwartz et al found a 15% recovery rate of GABHS in symptomatic children between ages 5 and 35 months.[8] Stillerman found that 24% of his symptomatic patients < age 3 had a positive throat culture for GABHS.[9] Other bacterial pathogens in preschool-aged children include

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Table 1. Parenteral Penicillins for GABHS Pharyngitis

		AGE-BASED		WEIGHT-BASED	
Drug	**Brand**	**Age (years)**	**Units**	**Weight (#=s)**	**Units**
Benzathine-pcn G	Bicillin L-A	< 6	600,000	< 60	600,000
		> 6	1.2 million	> 60	1.2 million
			OR 25,000 units/kg		
Benzathine + Procaine pcn G	Bicillin C-R 900/300	< 6	750,000	< 60	750,000
		6-12	1.2 million	60-140	1.2 million
			OR 25,000 units/kg of the benzathine component		

Key: pcn = penicillin
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Mycoplasma pneumoniae and *Chlamydia pneumoniae. Candida albicans* typically causes more buccal stomatitis, but pharyngeal and soft palatal lesions occur in the very young.

Incidence in School-age Patients. Viral pharyngitis occurs more frequently in school-aged children than does bacterial disease; viral estimates are in the 60% range. Parainfluenza viruses, influenza viruses, and enteroviruses are the predominate viral pathogens. Epstein-Barr virus has bimodal peaks between the fourth and seventh years, and again in late adolescence. Herpes viruses have their onset and peak in late childhood through early adolescence. *Mycoplasma pneumoniae, Chlamydia pneumoniae,* and non-group A streptococci are common throughout late childhood. *Neisseria gonorrhea, Treponema pallidum,* and *Chlamydia trachomatis* are found in children who engage in orogenital sex. *Arcanobacterium haemolyticum* typically affects the adolescent age group. All of these bacterial agents are far less common than GABHS, which is the primary bacterial cause of pharyngitis in children between the ages of 5 and 15. In this age group, GABHS, in non-epidemic circumstances, is responsible for at least 15% of pharyngitis.[10]

Season. Seasonal predilection occurs, with several pathogens associated with pharyngitis. Influenza virus, parainfluenza virus, adenovirus, rhinovirus, and respiratory syncytial virus cause pharyngitis with increased frequency in late autumn through mid-to-late winter. GABHS pharyngitis peaks between late winter and early spring. Coxsackie and echovirus account for the majority of pharyngitis cases from late spring through autumn.

Epidemics. Periodically, there are outbreaks of pharyngitis. The surges of oropharyngeal infection are the result of intrafamilial spread and close-quartered contacts. Secondary spread of GABHS in households, daycare centers, and classrooms leads to winter epidemics. Up to 50% of pediatric patients with pharyngitis during these epidemics are infected with GABHS.[11] Other pathogens encountered with sporadic clustering include *Neisseria meningitidis* and *Mycoplasma pneumoniae.*

Clinical Presentation. Patients with pharyngitis present with a limited number of chief complaints. The patient exposure history and past medical history; history of present illness, including review of systems; and physical examination may provide insight as to the pathogen causing pharyngitis. Scoring systems are of utility when estimating the likelihood of GABHS.

Chief Complaint. In preverbal children, or those with reduced verbal skills, the chief complaint will come from the caretaker who is interpreting behaviors. The parent witnesses either reduced feeding, refusal to feed, excess oral secretions, drooling, or painful facial expressions with swallowing or coughing. The parent presumes their child has a sore throat. Other parental observations may lead to alternate chief complaints, such as change in voice, bad breath, or fever. Verbal children with pharyngitis capably express pain that is located in the throat, posterior tongue, submental region inferiorly to the hyoid bone, posterior neck, or ears. Alternate chief complaints seen with pharyngitis include manifestations attributed to the central nervous system, such as lethargy or headache. Gastrointestinal manifestations can cause concern. Complaints include loss of appetite, nausea, vomiting, or abdominal pain, which may precede complaints of sore throat. Trouble breathing or cough may be expressed chief complaints. Enlarged cervical lymph nodes or rash may be the chief concern of the parent or patient.

Exposure, Past Medical History. Secondary spread of pharyngitis commonly has been reported for contacts exposed to various viral and bacterial agents. The absolute risk of infection in a patient who is exposed to a respiratory pathogen in closed environments is not known. However, it is appropriate to consider that a symptomatic patient with pharyngitis exposed to an infected individual has acquired the same pathogen.[12] Common pathogens also may be transmitted by sexual practices.

Patients with a distant history of pharyngitis may or may not have the same viral or bacterial pathogen causing a current case of pharyngitis. Patients with a recent history (< 60 days) of documented GABHS pharyngitis are at increased risk for clinical recurrence.[13] This holds true even if the patient was treated with antibiotics. Oral antibiotics prescribed for 10 days are effective in the management of GABHS for most patients. There may be persistence of GABHS pharyngitis or recurrence as a result of noncompliance, re-exposure to GABHS-infected peers or family members, re-exposure to asymptomatic carriers of GABHS, or bacterial resistance to eradication. The 30-day and 60-day treatment failure rates have steadily increased over the past decades and are now reported to be approximately 25% and 35%, respectively.[10]

Table 2. Suitable Oral Therapeutics for GABHS Pharyngitis

AGENT	DOSE (MG/KG/DAY)	DIVIDED DOSE
Amoxicillin	40	BID-TID
Amox./clavulanic acid	43.8/6.2	BID
Cephalexin	25-50	BID-QID
Cefadroxil	30	QD
Cefuroxime	20-30	BID
Cefpodoxime*	10	BID
Cefprozil	20	BID
Cefixime	8	QD
Ceftibuten	9	QD
Cefdinir*	14	QD
Clarithromycin	15	BID
Clindamycin	13-20	TID-QID
Azithromycin*	12	QD
E. estolate	20-40	BID-QID
E. ethylsuccinate (EES)	40	BID-QID
Penicillin V	25-50	BID-QID

Key: E = erythromycin
*FDA approval for 5-day course of therapy.

History, Including Review of Systems. The constellation of symptoms with pharyngitis may provide clues to the likely pathogen. The complaints can be divided into constant and inconstant features. The prominent and almost uniform complaint, irrespective of the offending pathogen, is sore throat. The onset is acute or subacute, and the intensity of discomfort is variable. Pain often is aggravated by swallowing (GABHS, EBV [Epstein-Barr virus], herpes), speaking (EBV, *C. diphtheriae*), or coughing (adenovirus, parainfluenza viruses, influenza viruses, RSV [respiratory syncytial virus], rhinovirus). Refusal to feed, excess saliva, or drooling (herpes) is common in infants. Fever is a common accompanying complaint in all age groups. The temperature range with pharyngitis is quite variable. Common, nonspecific, associated complaints of malaise, fatigue, decreased activity, and headache are shared features of many pathogens. Cough, myalgia, and arthralgia are seen with influenza A and B, parainfluenza, *Neisseria meningitidis,* and *Mycoplasma pneumoniae* infection.

Inconstant symptoms fall under umbrellas of respiratory, gastrointestinal, rheumatologic, and dermatologic features. Nasal congestion, watery or red eyes (adenovirus), cough (*Chlamydia, Mycoplasma*), and noisy breathing, including stridor and wheezing (RSV, metapneumovirus, parainfluenza viruses, adenovirus), are possible. Anorexia, nausea, vomiting, and abdominal pain are possible. The pain is generally nonlocalized except with GABHS, which may localize to the right upper quadrant in association with hydrops of the gallbladder or the right lower quadrant, simulating acute appendicitis. Joint aches and swelling may accompany pharyngitis *(N. gonorrhea, Mycoplasma* spp.*).* A rash with pharyngitis may precipitate a patient encounter.

Physical Examination. Vital signs confirm the febrile state. Patients typically look uncomfortable, but are not toxic. Patients may assume fixed posturing if there is a mass effect compromising the airway. An erythroderma, rash, etc., if present, may take several forms. The rash may be erythema multiforme-like *(Mycoplasma* spp.), scarlatiniform (GABHS, *Arcanobacterium),* or urticarial (GABHS, *Mycoplasma* spp.*).* The oral mucosa exhibits localized derangements. The gingiva may be hyperemic (mixed anaerobes) or evidence shallow ulcers (herpes, enteroviruses). Small vesicles may be present on the palate (enteroviruses). Petechiae may be observed on the palate (GABHS, EBV, rubeola, rubella) or uvula (GABHS). The uvula may be edematous and erythematous (GABHS).[14] The tonsillar tissue may be enlarged and erythematous. Follicular lesions (adenovirus, enteroviruses) or exudative lesions (EBV, GABHS) may be seen. A membrane may be present over the tonsillar tissue (*C. diphtheriae*). Painful, swollen submental or anterior lymph glands are not pathognomonic for a specific organism (e.g., GABHS), but in the context of an appropriate exposure and history of present illness will lend support to a presumptive diagnosis.[15]

Scoring Systems. Several scoring systems have been developed in adults and children to improve the presumptive diagnosis of GABHS using clinical symptoms and signs. In a pediatric suburban practice, Breese analyzed nine factors (including a peripheral white blood cell count) and their relative weights in calculating the probability of GABHS pharyngitis.[16] Breese determined that GABHS was the pathogen in 70% of children with sore throat who had fever, absence of rhinorrhea and cough, who had physical examination changes of the tonsils (erythema, petechiae, or exudates) and tender cervical lymphadenopathy.[16] Although Breese's sentinel findings have been challenged by others in recent decades, the reliability of pediatric clinical diagnosis has been validated using clinical attributes. The febrile child with a sore throat who has absence of coryza but the presence of positive physical findings, which include scarlatiniform rash, tonsillar swelling, and tender anterior cervical lymph nodes, has a probability for GABHS of 95%. Excluding the scarlatiniform rash, the remaining variables identify GABHS with a probability of 65%. In the presence of coryza, absence of rash, and absence of tender enlarged lymph nodes, the probability of GABHS falls to < 15%.[17]

Diagnostic Testing. There is considerable debate over the utility of laboratory studies in patients with pharyngitis. At one extreme, clinicians rely upon scoring systems and clinical acumen while they shun lab testing. With this approach, they are cost effective but at the expense of over diagnosing GABHS.[18,19] At the other extreme, clinicians discount the reliability of history and physical examination and argue for laboratory testing in all patients, independent of the clinical picture.[20] Other clinicians suggest an intermediate strategy. They estimate the clinical likelihood of GABHS before incorporating a throat swab for rapid streptococcal antigen testing.[21]

For reliable results from antigen detection and/or throat culture, both tonsillar pillars should be swabbed. There are several generations of rapid tests on the market for identification of GABHS. The older techniques employed latex agglutination methods and enzyme-linked immunosorbent assays. They yielded a median sensitivity of 78% and median specificity of 97%. Because of the low sensitivity, the American Heart Association,

Table 3. Suitable Oral Therapeutics for Otitis Media

AGENT	DOSE (MG/KG/DAY)	DIVIDED DOSE
Amoxicillin	80-90	BID
Amox./clavulanate	90/6.4	BID
Cephalexin	50	QID
Cefuroxime	30	BID
Cefpodoxime	10	BID
Cefprozil	30	BID
Cefixime	8	BID
Ceftibuten	9	QD
Cefdinir	14	QD-BID
Loracarbef	30	BID
Clarithromycin	15	BID
Azithromycin*	10 day 1; 5 thereafter	QD
EES/sulfisoxazole	50/150	QID
Tmp./sulfamethoxazole	4/20	BID

Key: EES = erythromycin ethylsuccinate; tmp. = trimethoprim
* 5-day course of therapy

the Infectious Disease Society of America, and the Canadian Pediatric Society have all recommended that a patient with a negative rapid test should have a throat culture submitted.[22] With more studies of the newer techniques, which employ optical immunoassay and nucleic acid probes, it may ultimately be recommended that backup throat cultures be abandoned.[21,23]

Beyond a throat swab for GABHS, other diagnostic tests should be driven by the patient's clinical condition. If gonococcal pharyngitis is suspected, submit a throat swab that can be plated on chocolate ager. A complete blood count and serologic test for Epstein-Barr virus may be useful for the patient with a compatible presentation. A blood culture is optional in a toxic-appearing patient and mandated in a septic-appearing patient. Imaging is warranted for the pediatric patient with pharyngitis who has increased work of breathing and signs of upper airway obstruction. A plain, portable, upright, lateral neck film is of utility to discriminate the causes of upper airway obstruction considered in the differential diagnosis.

Differential Diagnosis. The differential diagnosis of pharyngitis includes both noninfectious and infectious diseases. The noninfectious causes include the rare circumstance of tumor (lymphoma, nasopharyngeal angiofibroma), trauma (penetrating or blunt with retropharyngeal hematoma), and inflammatory conditions (Kawasaki disease, Stevens-Johnson syndrome). All of the oropharyngeal structures, including the lingual tonsils, palatine tonsils, soft palate, and posterior pharyngeal wall, can be superficially infected and present with similar symptoms. Other superficial oropharyngeal surfaces (adenoids, epiglottis, aryepiglottic folds, uvula) or deeper plane structures (peritonsillar, parapharyngeal, retropharyngeal space) can be infected, causing overlapping features with pharyngitis. However, the symptoms with more extensive infection are more disabling, such as inability to handle secretions, intense pain with swallowing, and reduced desire to speak. Physical examination of a child with a more extensive infection often are more striking and include toxicity, fixed upright posturing, drooling, stridor, and mass.

Treatment. Antibiotic therapy for non-GABHS pharyngitis has not been shown to reduce the duration of symptoms or its complications.[24] In contrast, antibiotic therapy will shorten the duration of symptoms and reduce some of the complications of streptococcal pharyngitis. Depending upon circumstance, empiric antibiotic therapy may be initiated based upon physician estimate for the likelihood of GABHS pharyngitis. Alternately, the physician may treat the patient with a positive rapid antigen detection test or a positive throat culture.

Antibiotic treatment for GABHS pharyngitis may be provided by a single injection of either Bicillin L-A or Bicillin C-R. There are both age and weight recommendations for dosing. *(See Table 1.)* Bicillin C-R, which adds a procaine component to the injection, does not hasten the resolution of signs or symptoms when compared to benzathine penicillin alone. However, Bicillin C-R is less painful. To render patients culture negative and clinically improved within a 1-2 day timeframe, there must be at least 25,000 units/kg of benzathine component. As 1.2 million units provides 900,000 units of benzathine in 2 mL, this amount is sufficient to treat the average 12-year-old who weighs in the neighborhood of 106 pounds. Children beyond age 12, or who weigh in excess of 140 pounds, should not be treated for GABHS pharyngitis with Bicillin C-R, but with 1.2 million units of Bicillin L-A.[25]

Several oral antibiotic agents are equally efficacious or superior to parenteral penicillins in eradicating the organism and creating clinical cure. They include aminopenicillins and various cephalosporins. See Table 2 for specific doses, frequency, and duration. Macrolides are recommended with caution. The rates of macrolide resistance nationwide are in the range of 3.8-8.3%, but various regions may have a resistant rate of up to 48%.[26]

Children who have a recurrence of GABHS pharyngitis shortly after completing a course of oral antimicrobial agents can be retreated with the parenteral route. If they have been treated with penicillin or an aminopenicillin, recurrence therapy should be with clindamycin or a cephalosporin.[27]

Corticosteroids have been used as adjuvant therapy for GABHS pharyngitis. Beyond late adolescence, intramuscular dexamethasone has been shown to improve the severity of symptoms when given in conjunction with antibiotics.[28] Oral dexamethasone, at 0.6 mg/kg as a single dose given to children between 5-16 years of age, decreased the time to clinically significant pain relief by 4 hours without altering the time to complete pain relief in those with GABHS pharyngitis.[29] The reduction from 13 hours to 9 hours for pain relief was achieved without increasing complications. The cost for the marginal clinical impact has been debated.

Supportive care holds benefit for patients with pharyngitis from all causes. The pain from pharyngitis can be ameliorated by oral analgesics. Pain may additionally be improved by topical anesthetics supplied in the form of sprays or lozenges.

Complications. The complications from pharyngitis from all pathogens are uncommon, but include mechanical and infectious disturbances. All pathogens have the capacity to derange oropharyngeal structures sufficiently to preclude normal oral intake or lead

Table 4. Suitable Oral Therapeutics for Sinusitis

AGENT	DOSE (MG/KG/DAY)	DIVIDED DOSE
Amoxicillin	80	BID-TID
Amox./clavulanate	43.8/6.2	BID
Cefuroxime	30	BID
Cefpodoxime	10	QD-BID
Cefprozil	20	BID
Cefixime	8	BID
Cefdinir	14	QD-BID
Clarithromycin	15	BID
Clindamycin	20-40	TID
Azithromycin*	10 day 1; 5 thereafter	QD

* 5-day course of therapy

to airway obstruction. This is especially true with mononucleosis. All pathogens may expand into potential cervical spaces and fascial planes. These infectious processes may envelope vital structures within the neck, including the great vessels and cranial nerves. Further, these deep neck infections may be associated with bacteremia and spread to distant sites. Mononucleosis may be associated with polymicrobial bacteremia, hepatitis, encephalitis, and neutropenia. GABHS pharyngitis is associated with several non-suppurative complications. These inflammatory or immunologic events can be dermatologic or systemic. The skin changes include erythema nodosum, erythema marginatum, erythema annulare, and angioedema.[30] The systemic complications include toxic shock syndrome, carditis, arthritis, chorea, rapid chaotic eye movements, and uncontrollable movements of muscles (opsoclonus-myoclonus syndrome), obsessive-compulsive disorder, tics, bradykinesia, and rigidity.[31-33] These neurologic and psychiatric complications appear long after the clinical features of pharyngitis have resolved.

Disposition. When comfort care, hydration, and parental observation are available, a home-going disposition is appropriate for the pediatric patient with pharyngitis. Hospitalization may be warranted in selected circumstances, which include impending upper airway obstruction, dehydration, immunocompromise, or concern for septicemia.

Otitis Media

Definition and Anatomy. Otitis media is defined as inflammation of the middle ear. In acute otitis media (AOM), symptoms, when present, are of recent origin and physical examination of the middle ear reveals effusion. The middle ear is directly connected to the mastoid air cells, which are posterior, and the eustachian tube, which is anterior. The eustachian tube connects the middle ear to the nasopharynx. The tympanic membrane lays laterally, which separates the middle ear from the outer external auditory canal.

Scope of the Problem. After respiratory tract infections, otitis media is the most common affliction of childhood. The disease process results in the largest number of ambulatory visits to offices and EDs.[34]

Otitis media occurs in all age groups, from newborns through adulthood. The highest incidence of AOM is between 6 and 24 months of age. Sixty to eighty percent of infants by age 1 and 80-90% of children between age 2 and 3 will have at least one occurrence of AOM.[35] Thereafter, the incidence declines.

Pathophysiology. The pathogenesis of AOM is the result of eustachian tube dysfunction. The eustachian tube, as well as the mastoid and middle ear, are air filled and lined with respiratory mucosa. Typically, an URI or allergic diathesis causes congestion of the respiratory mucosa. The eustachian tube obstructs, resulting in a build up of negative pressure in the middle ear. Mucosal secretions escalate. Viruses and bacteria that have colonized in the nasopharynx reach the middle ear via reflux, aspiration, or insufflation during nose blowing, swallowing, or crying. An inflammatory response ensues.[36]

Risk Factors. There are multifactorial predispositions for the development of AOM. The most important predisposition is young age, particularly age younger than 2 years. It is unexplained that the young at greatest risk are those of male gender. Other groups are at risk as the result of exposure to contagion. Those at lower socioeconomic status and those who attend daycare with four or more children have greater nasopharyngeal colonization with pathogens associated with AOM. Infants who use pacifiers, infants who are bottle fed, and infants who are exposed to tobacco smoke are at increased risk for AOM. Genetic factors are apparent. Those of Native American descent and Eskimos are at increased risk for AOM.[37]

Bacteriology. The microbiology of AOM has been established by multiple studies that have utilized cultures from tympanocentesis fluid. From these data, national trends and regional variations have emerged as a result of alterations in nasopharyngeal carriage states and the administration of vaccines.

Viral agents alone, bacterial agents alone, or co-pathogenic recovery of viral and bacterial pathogens have been found in various studies.[38] The most common viral agents include respiratory syncytial virus, adenovirus, influenza virus, and rhinovirus.[39] The bacterial organism responsible for causing most cases of AOM is *Streptococcus pneumoniae*. Since the introduction of the polyvalent pneumococcal conjugate vaccine (PCV-7/Prevnar), nasopharyngeal colonization and recovered tympanocentesis fluid have shifted to non-vaccine serotypes.[40,41] Of the pneumococci recovered, there is a higher incidence of penicillin resistance, particularly among children in group daycare.[42]

Since the introduction of *Haemophilus influenzae* type B vaccination, most *H. influenzae* isolates are non-typable. *Moraxella catarrhalis* is the third-most common pathogen. Ninety percent of the *M. catarrhalis* isolates and 50% of the *H. influenzae* isolates produce beta-lactamase.[43] Group A beta-hemolytic streptococcus (GABHS) and *Staphylococcus aureus* each account for fewer than 10% of cases. Gram-negative bacilli are rare isolates that are more commonly recovered in AOM in the first months of life.[44]

Clinical Presentation. Children found to have AOM may be asymptomatic or may present with a variety of complaints. The manifestations are to some extent dependent upon age.

History, Including Review of Systems. The constellation of symptoms with AOM can be divided into specific and nonspecific

features. The most common specific symptom is otalgia. The earache is throbbing, constant, and often interferes with or precludes normal activities, including sleep. The pain is intensified when the patient is supine, such that affected infants will prefer to be maintained upright. Parents suspect otalgia in infants who they observe to be pulling on the ears (or are just irritable). Verbal children may complain of pain that is referred to the throat or head. Verbal children may experience abnormal buzzing sensations in the ear or have frank loss of hearing with or without a sensation of dizziness.[45-47] The most common nonspecific symptom is fever, which occurs in approximately half the patients. Of those who are febrile, the range is quite variable.[48] Gastrointestinal complaints, such as refusal to feed, reduced feeding, nausea, vomiting, or diarrhea, may be seen. Cold symptoms, such as rhinorrhea or cough, may be antecedent or accompany AOM. Conjunctivitis may be an accompanying feature of AOM, especially with *H. influenzae* infections.[49]

Physical Examination. With the exception of fever and the attendant tachycardia of the febrile state, vital signs are normal. Patients look uncomfortable, but are not toxic in appearance. There is no swelling over the mastoid prominence. The ear is not displaced anteriorly or inferiorly by mastoid soft tissue swelling (as in mastoiditis). Extension of the pinna creates no pain (as in otitis externa). The external auditory canal is free of an inflammatory response. The tympanic membrane is altered from its pearly grey or pink appearance. Erythema may occur bilaterally with crying and as a nonspecific finding, but is of significance when unilateral in patients with compatible symptoms of AOM. The tympanic membrane should be distended and often bulges first in the posterior-superior quadrant.[50] A single bullous or multiple bullae may be present on the tympanic membrane. Bubbles and air-fluid levels are seen when an effusion is present. A layering of pus may be visible behind the tympanic membrane. Negative and positive pressure, applied via insufflation and release of a bulb, shows decreased or absent mobility of the drum. In the patient without PE tubes, perforation of the tympanic membrane with purulent drainage indicates AOM.[50-56]

Diagnostic Testing. In uncomplicated cases, laboratory tests are not warranted for the diagnosis or management of AOM.[57] In circumstances of extreme otalgia, pain relief may be provided via myringotomy. The fluid aspirated through the tympanic membrane should be submitted for culture and sensitivity information. Nasopharyngeal cultures do not adequately predict the bacteriology of middle ear effusion.[58] A peripheral blood count is of no benefit. The range of leukocytosis or distribution of neutrophils does not predict the presence or absence of otitis media.[59]

Differential Diagnosis. The differential diagnosis for otitis media includes conditions that cause earache and those diseases that distort normal anatomy of the middle ear. Earache can be seen in the early course of Bell's palsy and in the course of parotitis, otitis externa, gingivitis, pharyngitis, uvulitis, retropharyngeal abscess, lingual tonsillitis, palatine tonsillitis, mastoiditis, sinusitis, and dental pathology, including apical abscess. Erythema or vesiculation of the tympanic membrane may occur in the course of chickenpox and Ramsay-Hunt syndrome. Erythema of the drum may be present with crying, following cerumen removal, with otitis externa, and with URI.

Treatment. Medical management is customary for ED therapeutics of AOM. Analgesia is an important consideration. Patients may have unremitting pain that requires immediate attention. In circumstances in which acetaminophen or nonsteroidal anti-inflammatory drugs fail to alleviate pain, narcotic analgesia may be necessary. Immediate pain relief can be achieved with a topical analgesia, such as benzocaine or various homeopathic remedies, including combinations of belladonna, pulsatilla, aconite, chamomilla, calcarea carbonica, hepar sulphuris, and mercurous.[60]

Otitis media need not be treated with antibiotic medicines. The natural history is resolution of the infectious process with diminution of symptoms over a 3-5 day timeframe. The Europeans, for over a decade, have demonstrated that children with mild symptoms (absence of fever or vomiting) improved without antibiotic management. This conservative approach was taken in the United States in several studies and a meta-analysis demonstrated that a majority of patients reported improvement within three days without receiving antibiotics.[61] A multidisciplinary expert panel convened by the American Academy of Pediatrics (AAP) and the American Academy of Family Physicians jointly issued a clinical practice guideline in May 2004.[56] The guidelines offered an option for observation without immediate antibiotic therapy in selected children. Observation was an option in those children without severe illness (temperature $\leq$ 39C° and mild pain) and older than age 2 years. Observation was suitable for children > 6 months of age with an uncertain diagnosis and mild illness. In both circumstances, observation was appropriate only if follow-up examination within a 2-3 day period was assured. Despite the dissemination of these guidelines, practice-based physicians have used observation only in about 10% of their cases.[62] Because of the difficulties in assuring follow-up from emergency patients, emergency physicians are logically inclined to use antibiotic therapy as a matter of routine.

The AAP guidelines suggest amoxicillin as the first-line antimicrobial treatment *(see Table 3)* at a high-dose regimen (80 mg/kg/day). Greater than 80% of children will respond.[63] Should a patient remain symptomatic 48-72 hours after beginning therapy, switching to an antibiotic with beta-lactamase activity is recommended.[64] A patient who has completed a course of antibiotics, and returns symptomatic with a recurrent bout of otitis media, should be treated with a different class of antibiotics. For those treated by the oral route, a short course of 5-7 days is an option in children older than age 6 with mild or moderate symptoms.[65] Azithromycin therapy is suitable for a 5-day timeframe. Other oral antibiotic therapies are provided for 10 days. Parenteral therapy within intramuscular ceftriaxone is an alternate to oral therapeutics. Although a single injection may be suitable, outcomes are improved with three injections.[25]

Complications. The usual course of AOM, even without antibiotic therapy, is spontaneous resolution. Antibiotics increase the cure rate. Failures do occur and recurrence typically occurs

with relapsing of infection shortly after antimicrobial therapy has been completed. The complications of chronic, recurrent infections are hearing loss and language disorders.[45] The local suppurative complications of otitis media include labyrinthitis, mastoiditis, cranial osteomyelitis, brain abscess, and lateral sinus thrombosis. Local suppurative complications can lead to mass effect with the creation of otic hydrocephalus or impinge on the seventh cranial nerve, leading to facial palsy. Meningitis can result as an extension of pathogens toward the brain substance or with a bacteremic spread to the meninges.

Disposition. Inpatient management for otitis media is infrequent. Neonates with otitis media, particularly those who are febrile or who have had prolonged stays in the nursery, often are managed as inpatients. Individuals of all ages suspected to have a complication of otitis media may be hospitalized. Otherwise, the majority of patients are treated as outpatients. Follow-up instructions generally note that patients should be seen near the completion of therapy. Patients who do not improve within 48-72 hours after treatment has begun are encouraged to be reassessed earlier.

Sinusitis

Definition and Anatomy. Sinusitis is defined as inflammation of paranasal sinuses that is generally caused by bacterial infection.[66] The challenge in the ED is differentiating a true bacterial sinus infection from a simple viral URI or recurrent URIs. Sinusitis is considered acute if it lasts less than 30 days with complete resolution, subacute if it lasts from 30-90 days, and chronic if symptoms are present for more than 90 days.[67]

Maxillary and ethmoid sinuses are present at birth. The sphenoid and frontal sinuses develop around age 4 and age 7, respectively.[66,68] Each sinus drains into the nasal cavity through a narrow ostium. The sphenoid and posterior ethmoid sinuses drain into the superior meatus. The ostium from the maxillary sinus joins with the frontal and anterior ethmoid ostia to drain into the middle meatus, forming the osteomeatal complex. The maxillary sinus ostium sits superior to the sinus itself so the drainage does not have the benefit of gravity. Most cases of sinusitis in children affect the maxillary sinus.[66]

Scope of the Problem. Children can experience as many as 6-8 viral URIs yearly, often in succession during the winter months. Prolonged URIs may be a prelude to sinusitis. It may be difficult to differentiate recurrent or prolonged URIs from a true bacterial sinus infection. However, it is estimated that 5-13% of children who have prolonged URIs will have a secondarily-complicated bacterial infection of the paranasal sinuses.[69]

Pathophysiology. The sinuses are usually sterile reservoirs that rely on mucus drainage and ciliary action to clear any transient bacteria. In acute sinusitis the ability to maintain sterility is overcome. Although 80% of acute sinusitis is preceded by viral URIs, the majority of the remaining infections are preceded by allergic rhinitis.[67] Obstruction of the osteomeatal complex is the most important inciting factor. When the sinus is blocked by mucus or inflamed mucosa, oxygen in the space is resorbed, creating a negative pressure that favors the movement of nasal bacteria into the sinus.[68] Decreased ciliary function, increased mucus production, and viscosity caused by the URI or allergic reaction contribute to poor bacterial clearance. The bacteria are able to proliferate in the closed off space.[66]

Different causative factors have been identified for chronic and recurrent sinusitis that include adenoid vegetations, airway pollutants, GERD (gastroesophageal reflux disease), immunologic defects, primary ciliary dyskinesia, cystic fibrosis, and structural abnormalities of the sinuses.[70,71]

Bacteriology. The connection between the nasal cavity and the sinuses is similar to that between the middle ear and the nasal cavity. It is logical, then, that the same pathogens responsible for AOM also are recovered in a majority of cases of sinusitis. *Streptococcus pneumoniae* is the most common organism recovered via sinus aspirated cultures in patients with acute and subacute sinusitis.[68] Twenty-five to fifty percent of the isolates are penicillin resistant.[72] *Moraxella catarrhalis, Haemophilus influenzae,* and viridans streptococci *(S. viridans)* are additional common pathogens that cause acute sinusitis.[68] Fifty percent of *H. influenzae* and 100% of *M. catarrhalis* are beta-lactamase positive.[67] *Staphylococcus aureus,* gram-negative enteric organisms, anaerobes, and fungi have been implicated in cases of chronic disease.[72]

Clinical Presentation. Children with sinusitis present with respiratory manifestations, with or without constitutional complaints. The history of present illness is dependent upon the child's capacity to verbalize complaints.

History, Including Review of Systems. Uniformly, patients of all ages have inflammation of the nasal passages. This rhinitis causes a nasal discharge, which may be of variable consistency. Nasal drainage may be watery to thick and clear to colored. Fluid may emanate from the nose or drain posteriorly, causing a reflexive cough that often worsens with nighttime recumbency.[73] The nasal drainage or postnasal drainage should be present for ≥ 7-10 days. If purulent, the discharge should be present for at least 3-4 consecutive days.

In children, beyond rhinitis and cough, other symptoms are infrequent compared to the adult population. A child rarely may have an altered facial sensation or facial pain that is aggravated by stooping. Hemilateral or bilateral frontal headaches are rare.[73] Verbal patients may complain of fatigue, halitosis, sore throat, or toothache.[72] Younger children may seem to be irritable, anorectic, or less active compared to their baseline.[66] Throughout all age groups, fever is an inconstant feature in children.[72]

Physical Examination. Patients may not look well, but they are not toxic in appearance. Fever, when present, is generally low grade. The nasal mucosa is inflamed. A unilateral or bilateral nasal discharge is present and/or draining posteriorly into the oropharynx. The posterior pharyngeal structures may be erythematous. The breath may be malodorous. Nontender anterior cervical lymphadenopathy may be noted. With ethmoid sinusitis, patients may have unilateral or bilateral painless periorbital edema.[73] Sinus percussion tenderness is inconstant but may be

noted with maxillary or frontal sinusitis.[67] Other examination features will be present only in circumstances of complications. These include local extensions of infection beyond the sinus antrum or bacteremic extension to distant sites.

Diagnostic Testing. By consensus opinion, the diagnosis of sinusitis is established on the basis of history and physical examination.[74] Transillumination of the sinuses is not of value in diagnosing pediatric sinusitis.[75] Imaging techniques are available to support a clinical suspicion, but their value is debated.[76,77] Plain x-rays of the sinuses, either as a single Water's view or a three-view series, reveal mucosal thickening; this implies inflammation or edema of the mucosa. Unfortunately, in the absence of opacification or air-fluid levels, it does not necessarily imply infection.[78] Up to a 75% discrepancy has been reported when plain radiography is compared with computerized tomography (CT) in the pediatric population.[79] CT scan offers excellent visualization of the sinuses. The CT radiologic signs include fluid level, total opacification, and mucosal thickening of ≥ 5 mm. These changes are seen in approximately 75% of patients who are clinically diagnosed with acute sinusitis.[80] Unfortunately, these CT abnormalities are seen in pediatric patients who have no signs or symptoms of sinusitis. Further, a CT scan cannot distinguish mucosal thickening noted in asymptomatic children with URIs from those patients with bacterial sinusitis.[79,80] The Pediatric Subcommittee on Management of Sinusitis discourages the use of radiographs, and the American College of Radiology and the Sinus and Allergy Health Partnership concur.[67] Radiographs, specifically CT scanning, should be used in children who do not improve after appropriate medical management or in whom intracranial complications are suspected.

Transnasal antral puncture may reveal the nature of infecting organisms in 70-85% of sinusitis cases.[74] The procedure is invasive, painful, time consuming, and not practical for use in routine cases of suspected sinusitis. Surface cultures of the nose or pharynx have no predictive value in sinusitis.[81]

Differential Diagnosis. The differential diagnosis for a child with prolonged nasal discharge includes allergic diathesis, viral URIs, and when unilateral, a retained nasal foreign body. With the addition of cough, the differential includes reactive airway disease, gastrointestinal reflux, and bronchitis.[68]

Treatment. Antibiotics remain the mainstay of treatment for acute pediatric sinusitis despite a marginally-favorable influence over placebo. In one study, 80% of patients had resolution of symptoms within one week, regardless of whether they were treated with an antibiotic or placebo.[82] The Agency for Health Care Policy and Research report compiled data from trials comparing antibiotic treatment to placebo or no treatment and reported 90% improvement in patients with antibiotics and 66% improvement without antibiotics.[83]

The AAP Subcommittee on Sinusitis recommends starting antibiotics to foster rapid recovery, prevent suppurative complications, and minimize exacerbations of bronchospasm in those with underlying asthma.[67] The optimum duration of antibiotic treatment has not been established by any prospective study. A 10-day course of amoxicillin remains the drug of choice in children in whom the incidence of beta-lactamase producing pathogens is < 20%.[84] In areas of high rates of resistance, or in children who have clinically failed to respond to amoxicillin, alternate antimicrobials are suitable. *(See Table 4.)*

The role for adjunctive therapies in acute sinusitis is controversial. Proof of their efficacy is minimal. Oral alpha-adrenergic decongestants, such as pseudoephedrine, can improve nasal stuffiness. Topical alpha-adrenergic decongestants reduce the sensation of nasal obstruction. Such agents should not be used for more than three consecutive days; otherwise, there is a risk of tachyphylaxis. Nasal saline drops or irrigation has been shown to improve mucociliary clearance in the nose, but has not been demonstrated to hasten recovery.[74] Oral antihistamines are of no proven benefit in acute sinusitis. In those with an allergic diathesis as a precipitant, antihistamine may be of benefit.[70] Additionally, in patients with allergic-mediated sinusitis, topical corticosteroids in combination with antibiotics demonstrate an improvement of symptoms in infants and children with recurrent sinusitis.[85]

Complications. Acute sinusitis, if left untreated, may lead to subacute or recurrent sinusitis. Acute sinusitis may be associated with recurrent otitis media, and in those who are predisposed, exacerbation of asthma.[71,72] Other complications of acute sinusitis are rare. They are related to spread of infection beyond the sinus cavity. Extension of frontal sinus infection via diploic veins may extend to the inner and outer tables of the frontal bone and along the marrow. Anterior extension results in a subperiosteal abscess (Pott's puffy tumor). Frontal sinusitis also may lead to cranial osteomyelitis, epidural abscess, subdural abscess, and brain abscess.[70] Purulent material from an ethmoid sinusitis can lead to periorbital cellulitis with erythema and edema of the superior lid. With invasion of the orbit and the development of orbital cellulitis, there is eye pain, chemosis, proptosis, and ophthalmoplegia. The most potentially catastrophic complication of frontal ethmoid or sphenoid sinusitis is septic cavernous sinus thrombosis. Clinical features include those seen with orbital cellulitis, along with sensory changes in the distribution of the trigeminal nerve and visual disturbance.[86]

Disposition. Patients who are mild to moderately ill, but able to tolerate oral medications, can be discharged home with follow-up. Those who are not able to tolerate oral medications, appear severely ill, or who are suspected of a suppurative complication should be admitted to the hospital and provided intravenous antibiotics.

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The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

a.) Quickly recognize or increase index of suspicion for specific conditions;

b.) Describe the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed;

e.) Provide patients with any necessary discharge instructions.

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CME Questions

61. Between late winter and early spring, the most common cause of pharyngitis in a school-aged child is:
    A. *Mycoplasma pneumoniae.*
    B. group A beta-hemolytic streptococcus.
    C. influenza virus.
    D. coxsackie virus.

62. The most common bacterial pathogen recovered in children < age 3 with pharyngitis is:
    A. group A beta-hemolytic streptococcus.
    B. *Mycoplasma pneumoniae.*
    C. *Neisseria meningitidis.*
    D. *Moraxella catarrhalis.*

63. The pediatric patient with a sore throat, who has an absence of cold symptoms but the presence of findings that include tonsillitis and tender cervical lymphadenopathy, has a high probability for which pathogen?
    A. *Neisseria meningitidis*
    B. Epstein-Barr virus
    C. *Corynebacterium diphtheriae*
    D. Group A beta-hemolytic streptococcus

64. A definitive therapeutic dose of injectable penicillin for group A beta-hemolytic *Streptococcus* pharyngitis in a 10-year-old, who weighs 140 pounds, is 1.2 million units of which of the following preparation?
    A. 900,000:300,000 Bicillin C-R
    B. 600,000:600,000 Bicillin C-R
    C. Bicillin L-A
    D. Procaine penicillin

65. A 2-year-old treated with amoxicillin at 40 mg/kg/day for otitis media remains symptomatic 72 hours into therapy. Otitis persists on examination. The patient's therapy should be changed to which of the following regimens?
    A. Clarithromycin 30 mg/kg/day for 10 days
    B. Azithromycin 10 mg /kg/day for 5 days
    C. Ceftriaxone 100 mg/kg as a single injection
    D. Cefdinir 14 mg/kg/day for 10 days

66. Within the first 2 years of life, the location for an acute sinusitis is most commonly confined to which of the following sinuses?
    A. Ethmoid
    B. Sphenoid
    C. Frontal
    D. Maxillary

67. The germs responsible for the majority of acute sinusitis cases are:
    A. *Haemophilus influenzae* type b.
    B. *Staphylococcus epidermides.*
    C. *Streptococcus pneumoniae.*
    D. *Neisseria meningitidis.*

68. By consensus opinion of sinusitis experts, the diagnosis of sinusitis is established:
    A. on the basis of history and physical examination.
    B. with transillumination.
    C. by plain sinus radiographs.
    D. with CT scanning.

69. The physician should consider which underlying condition in the patient with recurrent, unilateral purulent nasal discharge?
    A. Allergic diathesis
    B. Retained nasal foreign body
    C. Immune deficiency state
    D. Cystic fibrosis

70. Antibiotics are recommended for acute sinusitis. Adjunctive therapies with suggested, but unproven efficacy, include all but which of the following?
    A. Topical corticosteroids
    B. Oral antihistamines
    C. Oral corticosteroids
    D. Topical alpha-adrenergic decongestants

Answers: 61. B; 62. A; 63. D; 64. C; 65. D; 66. D; 67. C; 68. A; 69. B; 70. C

In Future Issues: **Metabolic Emergencies**

PEDIATRIC Emergency Medicine Reports
The Practical Journal of Pediatric Emergency Medicine

Common Pediatric ENT Infections

Parenteral Penicillins for GABHS Pharyngitis

		AGE-BASED		WEIGHT-BASED	
Drug	**Brand**	**Age (years)**	**Units**	**Weight (#=s)**	**Units**
Benzathine-pcn G	Bicillin L-A	< 6	600,000	< 60	600,000
		> 6	1.2 million	> 60	1.2 million
			OR 25,000 units/kg		
Benzathine + Procaine pcn G	Bicillin C-R 900/300	< 6	750,000	< 60	750,000
		6-12	1.2 million	60-140	1.2 million
			OR 25,000 units/kg of the benzathine component		

Key: pcn = penicillin

Suitable Oral Therapeutics for GABHS Pharyngitis

AGENT	DOSE (MG/KG/DAY)	DIVIDED DOSE
Amoxicillin	40	BID-TID
Amox./clavulanic acid	43.8/6.2	BID
Cephalexin	25-50	BID-QID
Cefadroxil	30	QD
Cefuroxime	20-30	BID
Cefpodoxime*	10	BID
Cefprozil	20	BID
Cefixime	8	QD
Ceftibuten	9	QD
Cefdinir*	14	QD
Clarithromycin	15	BID
Clindamycin	13-20	TID-QID
Azithromycin*	12	QD
E. estolate	20-40	BID-QID
E. ethylsuccinate (EES)	40	BID-QID
Penicillin V	25-50	BID-QID

Key: E = erythromycin
*FDA approval for 5-day course of therapy.

Suitable Oral Therapeutics for Otitis Media

AGENT	DOSE (MG/KG/DAY)	DIVIDED DOSE
Amoxicillin	80-90	BID
Amox./clavulanate	90/6.4	BID
Cephalexin	50	QID
Cefuroxime	30	BID
Cefpodoxime	10	BID
Cefprozil	30	BID
Cefixime	8	BID
Ceftibuten	9	QD
Cefdinir	14	QD-BID
Loracarbef	30	BID
Clarithromycin	15	BID
Azithromycin*	10 day 1; 5 thereafter	QD
EES/sulfisoxazole	50/150	QID
Tmp./sulfamethoxazole	4/20	BID

Key: EES = erythromycin ethylsuccinate; tmp. = trimethoprim
* 5-day course of therapy

Suitable Oral Therapeutics for Sinusitis

AGENT	DOSE (MG/KG/DAY)	DIVIDED DOSE
Amoxicillin	80	BID-TID
Amox./clavulanate	43.8/6.2	BID
Cefuroxime	30	BID
Cefpodoxime	10	QD-BID
Cefprozil	20	BID
Cefixime	8	BID
Cefdinir	14	QD-BID
Clarithromycin	15	BID
Clindamycin	20-40	TID
Azithromycin*	10 day 1; 5 thereafter	QD

* 5-day course of therapy

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