

- media. A double-blind crossover study in pediatric practice. *N Engl J Med*. 1974;291:664–667
57. Schwartz RH, Puglise J, Rodriguez WJ. Sulphamethoxazole prophylaxis in the otitis-prone child. *Arch Dis Child*. 1982;57:590–593
 58. Schuller DE. Prophylaxis of otitis media in asthmatic children. *Pediatr Infect Dis*. 1983;2:280–283
 59. Liston TE, Foshee WS, Pierson WD. Sulfoxazole chemoprophylaxis for frequent otitis media. *Pediatrics*. 1983;71:524–530
 60. Varsano I, Volovitz B, Mimouni F. Sulfoxazole prophylaxis of middle ear effusion and recurrent acute otitis media. *Am J Dis Child*. 1985;139:632–635
 61. Gonzalez C, Arnold JE, Woody EA, et al. Prevention of recurrent acute otitis media: chemoprophylaxis versus tympanostomy tubes. *Laryngoscope*. 1986;96:1330–1334
 62. Paradise JL. Antimicrobial prophylaxis for recurrent acute otitis media. *Ann Otol Rhinol Laryngol*. 1981;90(suppl):53–57
 63. Goldstein NA, Sculerati N. Compliance with prophylactic antibiotics for otitis media in a New York City clinic. *Int J Pediatr Otorhinolaryngol*. 1994;28:129–140
 64. Prellner K, Fogle-Hansson M, Jorgensen F, Kalm O, Kamme C. Prevention of recurrent acute otitis media in otitis-prone children by intermittent prophylaxis with penicillin. *Acta Oto-Laryngologica*. 1994;114:182–187
 65. Heikkinen T, Ruuskanen O, Ziegler T, Waris M, Puhakka H. Short-term use of amoxicillin-clavulanate during upper respiratory tract infection for prevention of acute otitis media. *J Pediatr*. 1995;126:313–316
 66. Berman S, Nuss R, Roark R, Huber-Navin C, Grose K, Herrera M. Effectiveness of continuous vs. intermittent amoxicillin to prevent episodes of otitis media. *Pediatr Infect Dis J*. 1992;11:63–67
 67. Daly KA, Giebink GS, Lindgren B, et al. Randomized trial of the efficacy of trimethoprim-sulfamethoxazole and prednisone in preventing post-tympanostomy tube morbidity. *Pediatr Infect Dis J*. 1995;14:1068–1074
 68. Bernard PA, Stenstrom RJ, Feldman W, Durieux-Smith A. Randomized, controlled trial comparing long-term sulfonamide therapy to ventilation tubes for otitis media with effusion. *Pediatrics*. 1991;88:215–222
 69. Brook I, Gober AE. Prophylaxis with amoxicillin or sulfoxazole for otitis media: effect on the recovery of penicillin-resistant bacteria from children. *Clin Infect Dis*. 1996;22:143–145
 70. Klein JO. Lessons from recent studies on the epidemiology of otitis media. *Pediatr Infect Dis J*. 1994;13:1031–1034
 71. Niemela M, Uhari M, Mottonen M. A pacifier increases the risk of recurrent acute otitis media in children in day care centers. *Pediatrics*. 1995;96:884–888
 72. Heikkinen T, Ruuskanen O, Waris M, Ziegler T, Arola M, Halonen P. Influenza vaccination in the prevention of acute otitis media in children [see Comments]. *Am J Dis Child*. 1991;145:445–448
 73. Bluestone CD. Surgical management of otitis media: current indications and role related to increasing bacterial resistance. *Pediatr Infect Dis J*. 1994;13:1058–1063
 74. Giebink GS. Immunology: promise of new vaccines. *Pediatr Infect Dis J*. 1994;13:1064–1068
 75. Alho OP, Laara E, Oja H. What is the natural history of recurrent acute otitis media in infancy? *J Fam Pract*. 1996;43:258–264
 76. Bitar CN, Steele RW. Use of prophylactic antibiotics in children. *Adv Pediatr Infect Dis*. 1995;10:227–262

Pharyngitis—Principles of Judicious Use of Antimicrobial Agents

Benjamin Schwartz, MD*; S. Michael Marcy, MD‡; William R. Phillips, MD, MPH§; Michael A. Gerber, MD||; and Scott F. Dowell, MD, MPH*

ABSTRACT. Accurate diagnosis of group A streptococcal pharyngitis and appropriate antimicrobial therapy are important, particularly to prevent nonsuppurative sequelae such as rheumatic fever. Most episodes of sore throat, however, are caused by viral agents. Clinical findings cannot reliably differentiate streptococcal from viral pharyngitis and most physicians tend to overestimate the probability of a streptococcal infection based on history and physical examination alone. Therefore, diagnosis should be based on results of a throat culture or an antigen-detection test with throat culture backup. Presumptively starting therapy pending results of a culture is discouraged because treatment often continues despite a negative test result. Other bacterial causes of pharyngitis are uncommon and often can be diagnosed based on nonpharyngeal findings. Penicillin remains the drug of choice for streptococcal pharyngitis because of its effectiveness, relatively narrow spectrum, and low cost. No group A streptococci are resistant to β -lactam antibiotics. High rates of resistance to macrolides has been documented in several areas; in Finland, decreased national rates of macrolide use led to a decline in the proportion of

macrolide-resistant group A streptococci. *Pediatrics* 1998;101:171–174; group A *Streptococcus*, pharyngitis, diagnosis, antimicrobial therapy.

PRINCIPLES

1. Diagnosis of group A streptococcal pharyngitis should be made based on results of appropriate laboratory tests in conjunction with clinical and epidemiologic findings.
2. Antimicrobial therapy should not be given to a child with pharyngitis in the absence of diagnosed group A streptococcal or other bacterial infection.
3. A penicillin remains the drug of choice for treating group A streptococcal pharyngitis.

BACKGROUND AND JUSTIFICATION

Sore throat is one of the most common complaints in pediatrics, resulting in millions of physician office visits each year. Group A *Streptococcus* (*S pyogenes*), the leading bacterial cause of pharyngitis, accounts for ~15% of all cases.¹ Diagnosis and treatment of streptococcal pharyngitis are important because antimicrobial therapy initiated within 9 days of onset is effective in preventing acute rheumatic fever.² In addition, treatment of group A streptococcal infection may prevent suppurative complications, lead to more rapid resolution of illness, and prevent the spread of infection. Nevertheless, because most episodes of sore throat are caused

From the *Childhood and Respiratory Diseases Branch, National Centers for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia; †Kaiser Permanente, Panorama City, California; §Northwest Family Medicine, Seattle, Washington; and ||Connecticut Children's Medical Center, Hartford, Connecticut.

Reprint requests to (S.F.D.) Centers for Disease Control and Prevention, Mailstop C-23, 1600 Clifton Rd, NE, Atlanta, GA 30333.

PEDIATRICS (ISSN 0031 4005). Copyright © 1998 by the American Academy of Pediatrics.

by viral agents, treatment of all children with this illness would result in substantial unnecessary antimicrobial use. The recommendations that follow provide an approach to the diagnosis and treatment of children with pharyngitis that are consistent with judicious antimicrobial use.

EVIDENCE IN SUPPORT OF PRINCIPLES

Diagnosis of Group A Streptococcal Pharyngitis Should Be Made Using a Laboratory Test in Conjunction With Clinical and Epidemiologic Findings

Symptoms of classic streptococcal pharyngitis include acute onset of pharyngeal pain, dysphagia, and fever. Malaise, headache, abdominal pain, and vomiting occur commonly. Rhinorrhea, cough, hoarseness, conjunctivitis, and diarrhea are uncommon and strongly suggest a viral etiology. On examination, the pharynx is erythematous, a patchy exudate often is present on the posterior pharynx and tonsils, and palatal petechiae may be observed. The anterior cervical lymph nodes often are enlarged and tender.³

Unfortunately, these clinical findings are neither sensitive nor specific for group A streptococcal infection. When a diagnosis is based on clinical impression alone, physicians generally overestimate the probability that patients have streptococcal infection.⁴ Several schema have been developed to improve the ability to predict which patients will have group A streptococcal pharyngitis by scoring clinical and epidemiologic findings.^{2,5,6} None of these systems, however, identifies accurately children who need treatment and those who do not. Although the negative predictive value of a low score is good and may help guide a physician in deciding when a diagnostic test is needed, the positive predictive value of even the highest score is limited. In the evaluation of one system among adults, only 54% of patients in the most predictive group—those with a history of fever, tonsillar exudate, anterior cervical lymphadenopathy, and an absence of cough—had group A streptococci identified by culture.⁶

Because the clinical presentation of pharyngitis does not predict reliably the etiologic agent, when group A streptococcal infection is suspected, diagnosis should be based on results of a throat swab culture or antigen-detection test with culture back-up. Culture of a throat swab specimen is recommended as the standard for diagnosis.⁷ Some studies report the sensitivity of antigen-detection tests to be $\geq 90\%$ in carefully controlled clinical settings,⁸⁻¹¹ but such tests often have proved less sensitive in routine clinical practice.¹²⁻¹⁷ Consequently, the American Academy of Pediatrics recommends that if an antigen-detection test is negative in a child with suspected group A streptococcal pharyngitis, a culture also be performed.⁷ Because the specificity of antigen-detection tests is high, confirmation of a positive test is not required.

Throat cultures may be false-negative if specimens are obtained or cultured improperly. Samples should be obtained by vigorous swabbing of both tonsillar surfaces or fossae and the posterior pharynx; swabbing the soft palate and uvula should be avoided,

because it dilutes the inoculum.¹⁸ Culture methods are important as well. In one study, results of throat cultures performed in five physicians' offices were compared with a duplicate swab cultured at a reference laboratory. The sensitivities of cultures performed in the offices ranged from 73% to 100%; errors occurred both in isolating group A streptococci and in correctly identifying the organism.¹⁹ The sensitivity of culture also has been reported to vary depending on the laboratory methods used.^{17,20} For both culture and antigen detection, the sensitivity of the test is dependent on the quality of the specimen, how well the assay is performed, and the experience of the person reading the results.

Survey results indicate that many physicians initiate antimicrobial therapy for pharyngitis pending results of throat culture and that antimicrobial therapy often is continued despite cultures being reported as negative.²¹ This approach results in substantial antimicrobial overuse and obviates the benefits of performing a culture. If antibiotics are provided pending results of culture, physicians should be diligent in contacting parents if cultures are negative and should inform them to stop therapy and discard any remaining antibiotics.

Because early antimicrobial therapy may limit transmission of illness if the infection is caused by group A streptococci and may facilitate a child's return to school or day care, appropriate therapy should be initiated as soon as the diagnosis is supported by a laboratory test. It is unclear, however, whether immediate therapy offers a clinical benefit compared with symptomatic treatment,^{22,23} and no evidence suggests that early antimicrobial therapy decreases recurrent infection²⁴ or is necessary to prevent acute rheumatic fever.² Negative consequences of empirically starting therapy include selection of resistant bacterial pathogens, the risk of hypersensitivity or other adverse reactions, and cost. Use of a rapid antigen-detection test can help clinicians resist pressure for immediate therapy, because a negative result may facilitate immediate return to school or day care.

Antimicrobial Therapy Should Not Be Given to a Child With Pharyngitis in the Absence of Diagnosed Group A Streptococcal or Other Bacterial Infection

Viral agents cause most pharyngitis episodes. Even in patients with pharyngeal exudate and fever, group A streptococci account for a minority of infections. In one study, diagnostic tests for bacterial and viral pathogens were performed on 110 children who had exudative pharyngitis and fever and had not been treated previously with antibiotics. Group A streptococci were isolated from only 12% of children, whereas viral infection was documented from 31%. In addition, viral agents for which diagnostic testing was not available, including rhinovirus and coronavirus, may have accounted for infection in some of the children in whom no etiologic agent was identified.²⁵ The predominance of viral infection was especially noted among children who were <3 years of age—a

group in whom classical group A streptococcal pharyngitis occurs less often.

Pharyngeal irritation occurs frequently in persons with rhinovirus, corona virus, parainfluenza, influenza, adenovirus, and Epstein–Barr virus infection.²⁶ The signs and symptoms of pharyngitis associated with these viral infections overlap substantially with those of group A streptococcal pharyngitis; however, differences in clinical presentation also may exist. Children with viral pharyngitis often have prominent extrapharyngeal signs and symptoms such as nasal discharge, cough, and hoarseness. Adenoviral infection, a common cause of prolonged exudative pharyngitis, may be accompanied by conjunctivitis (pharyngoconjunctival fever), whereas an Epstein–Barr virus infection may have other signs of infectious mononucleosis (eg, generalized lymphadenopathy, splenomegaly). Coxsackie viruses and herpes simplex viruses often cause stomatitis as well as pharyngitis; vesicular or ulcerative lesions may be noted on examination.²⁶

Bacteria other than group A *Streptococcus* are rare causes of pharyngitis, and many such infections can be recognized by extrapharyngeal signs.¹ Other β -hemolytic streptococci (groups C and G) may be carried in the pharynx asymptotically or may cause infection resembling that caused by group A streptococci; the course of these infections is self-limited, and rheumatic fever does not occur. These β -hemolytic streptococci could be identified by culture but not by an antigen-detection test. *Neisseria gonorrhoea* pharyngitis is rare and typically occurs among adolescents; a history of sexual activity would be suggestive of this etiology, and pharyngitis may be accompanied by signs of genital infection or a rash. *Arcanobacterium haemolyticum* infection is uncommon in the United States, characteristically occurs in adolescents, and often presents with a scarlatiniform rash.²⁷ Diphtheria is a rare cause of pharyngitis in well-immunized populations and may be recognized by an asymmetric gray pharyngeal membrane that may extend beyond the borders of the anterior tonsillar pillars onto the soft palate and/or the uvula. Because each of these etiologic agents is uncommon and sequelae such as acute rheumatic fever do not occur, there is no rationale for empiric antimicrobial therapy of pharyngitis in children.

The significance of *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* as causes of pharyngitis is unclear; these infections usually are accompanied by other signs of respiratory illness, especially cough. The benefit of antimicrobial therapy for the pharyngitis caused by these agents has not been documented.

Because the large majority of pharyngitis episodes are not caused by group A streptococci, empiric antimicrobial therapy would result in substantial overtreatment. The widespread availability of accurate, inexpensive, diagnostic tests for group A streptococcal infections makes a diagnostic strategy of culture and/or antigen-detection testing for children with suspected streptococcal pharyngitis both effective and cost-effective,²⁸ and represents an optimal ap-

proach to avoiding the overuse of antibiotics. This strategy has been presented in algorithm form.¹

Penicillin Remains the Drug of Choice for Treating Group A Streptococcal Pharyngitis

Penicillin has proven highly effective as therapy for group A streptococcal pharyngitis and in preventing acute rheumatic fever. Because of its safety, efficacy, relatively narrow spectrum, and low cost, it remains the drug of choice for this indication. Amoxicillin is an acceptable alternative and often is prescribed because it is more palatable than penicillin and the cost is comparable. Because of its broader antimicrobial spectrum, however, use of amoxicillin results in greater selective pressure for the development of antimicrobial resistance. Penicillin therapy, administered for 10 days, results in bacteriologic and clinical cure in ~90% of children with group A streptococcal pharyngitis.²⁹ Shorter courses of therapy have been less effective.^{30,31} Although microbiological cure rates are slightly higher in children treated with cephalosporins,³² this may reflect greater efficacy in eradicating the organism from children who actually are carriers rather than improved outcome in those with acute infection.²⁹ Carriers are at very low risk for developing acute rheumatic fever and transmitting infection; therefore, the excess cost of cephalosporin therapy and the greater selective pressure for resistance associated with use of these broader-spectrum agents are disadvantages that outweigh the small increment in group A streptococcal eradication. To date, no group A streptococci resistant to β -lactam antibiotics have been identified. Resistance to erythromycin, an alternative therapy for patients who are allergic to penicillin, has been reported in several areas.^{33–35} In both Finland and Japan, increased rates of erythromycin resistance occurred coincident with increasing levels of macrolide use. As macrolide use subsequently declined—in Finland as the result of national guidelines recommending decreased use of erythromycin for respiratory and skin infections—so too has the proportion of erythromycin-resistant group A streptococci.^{34,35} Because resistance to extended spectrum macrolides (eg, clarithromycin) or azolides (eg, azithromycin) would be similar to that for erythromycin and these agents exert selective pressure for resistance over a broader range of bacterial pathogens, their use in treating pharyngitis should be discouraged.

ACKNOWLEDGMENTS

We thank Drs Leah Raye Mabry and Doug Long and members of the Committee on Infectious Diseases of the American Academy of Pediatrics for their careful review of this document.

REFERENCES

1. Tanz RR, Shulman ST. Diagnosis and treatment of group A streptococcal pharyngitis. *Semin Pediatr Infect Dis*. 1995;6:69–78
2. Rammelkamp CH. Rheumatic heart disease—a challenge. *Circulation*. 1958;17:842–851
3. Stillerman M, Bernstein SH. Streptococcal pharyngitis: evaluation of clinical syndromes in diagnosis. *Am J Dis Child*. 1961;101:476–489
4. Poses RM, Cebul RD, Collins M, et al. The accuracy of experienced physicians' probability estimates for patients with sore throat: implications for decision making. *JAMA*. 1985;254:925–929
5. Breese BB. A simple scorecard for the tentative diagnosis of streptococ-

- cal pharyngitis. *Am J Dis Child.* 1977;131:514
6. Wigton RS, Connor JL, Centor RM. Transportability of a decision rule for the diagnosis of streptococcal pharyngitis. *Arch Intern Med.* 1986;146:81–83
 7. American Academy of Pediatrics. Group A streptococcal infections. In: Peter G, ed. *1997 Red Book. Report of the Committee on Infectious Diseases.* 24th ed. Elk Grove Village, IL: American Academy of Pediatrics; 1997:483–494
 8. Gerber MA, Randolph MF, DeMeo KK. Liposome immunoassay for rapid identification of group A streptococci directly from throat swabs. *J Clin Microbiol.* 1990;28:1463–1464
 9. Harbeck RJ, Teague J, Crossen GR, Maul DM, Childers PL. Novel, rapid optical immunoassay technique for detection of group A streptococci from pharyngeal specimens: comparison with standard culture methods. *J Clin Microbiol.* 1993;31:839–844
 10. Heiter BJ, Bourbeau PP. Comparison of two rapid streptococcal antigen detection assays with culture for diagnosis of streptococcal pharyngitis. *J Clin Microbiol.* 1995;33:1408–1410
 11. Gerber MA, Tanz RR, Kabat W, et al. Optical immunoassay test for group A β -hemolytic streptococcal pharyngitis. *JAMA.* 1997;277:899–903
 12. Dale JC, Vetter EA, Contezac JM, Iverson LK, Wollan PC, Cockerill FR III. Evaluation of two rapid antigen assays, BioStar Strep A OIA and Pacific Biotech CARDS O. S., and culture for detection of group A streptococci in throat swabs. *J Clin Microbiol.* 1994;32:2698–2701
 13. Roe M, Kishiyama C, Davidson K, Schaefer L, Todd J. Comparison of BioStar A OIA optical immune assay, Abbott TestPack Plus Strep A, and culture with selective media for diagnosis of group A streptococcal pharyngitis. *J Clin Microbiol.* 1995;33:1551–1553
 14. Baker DM, Cooper RM, Rhodes C, Weymouth LA, Dalton HP. Superiority of conventional culture technique over rapid detection of group A Streptococcus by optical immunoassay. *Diagn Microbiol Infect Dis.* 1995;21:61–64
 15. Huck W, Reed BD, French T, Mitchell RS. Comparison of the Directigen 1–2–3 group A strep test with culture for detection of group A beta-hemolytic streptococci. *J Clin Microbiol.* 1989;27:1715–1718
 16. Donatelli J, Macone A, Goldmann DA, et al. Rapid detection of group A streptococci: comparative performance by nurses and laboratory technologists in pediatric satellite laboratories using three test kits. *J Clin Microbiol.* 1992;30:138–142
 17. Wenger DL, Witte DL, Schrantz RD. Insensitivity of rapid antigen detection methods and single blood agar plate culture for diagnosing streptococcal pharyngitis. *JAMA.* 1992;267:695–697
 18. Brien JH, Bass JW. Streptococcal pharyngitis: optimal site for throat culture. *J Pediatr.* 1985;106:781–783
 19. Rosenstein BJ, Markowitz M, Gordis L. Accuracy of throat cultures processed in physicians' offices. *J Pediatr.* 1970;76:606–609
 20. Kellogg JA. Suitability of throat culture procedures for detection of group A streptococci and as reference standards for evaluation of streptococcal antigen detection kits. *J Clin Microbiol.* 1990;28:165–169
 21. Holmberg SD, Faich GA. Streptococcal pharyngitis and acute rheumatic fever in Rhode Island. *JAMA.* 1983;250:2307–2312
 22. Middleton DB, D'Amico FD, Merenstein JH. Standardized symptomatic treatment versus penicillin as initial therapy for streptococcal pharyngitis. *J Pediatr.* 1988;113:1089–1094
 23. Del Mar C. Managing sore throat: a literature review. II. Do antibiotics confer benefit? *Med J Aust.* 1992;156:644–649
 24. Gerber MA, Randolph MF, DeMeo KK, Kaplan EL. Lack of impact of early antibiotic therapy for streptococcal pharyngitis on recurrence rates. *J Pediatr.* 1990;117:853–858
 25. Putto A. Febrile exudative tonsillitis: viral or streptococcal? *Pediatrics.* 1987;80:6–12
 26. Denson MR. Viral pharyngitis. *Semin Pediatr Infect Dis.* 1995;6:62–68
 27. Waagner D. *Arcanobacterium haemolyticum*: biology of the organism and diseases in man. *Pediatr Infect Dis J.* 1991;10:933–939
 28. Lieu TA, Fleisher GR, Schwartz JS. Cost-effectiveness of rapid latex agglutination testing and throat culture for streptococcal pharyngitis. *Pediatrics.* 1990;85:246–256
 29. Shulman ST, Gerber MA, Tanz RR, Markowitz M. Streptococcal pharyngitis: the case for penicillin therapy. *Pediatr Infect Dis J.* 1994;13:1–7
 30. Schwartz RH, Wientzen RL, Pedreira F, Feroli EJ, Mella GW, Guandolo VL. Penicillin V for group A streptococcal pharyngotonsillitis. *JAMA.* 1981;246:1790–1795
 31. Gerber MA, Randolph MF, Chantary J, Wright LL, De Meo K, Kaplan EL. Five vs ten days of penicillin V therapy for streptococcal pharyngitis. *Am J Dis Child.* 1987;141:224–227
 32. Pichichero ME, Margolis PA. A comparison of cephalosporins and penicillins in the treatment of group A beta-hemolytic streptococcal pharyngitis: a meta-analysis supporting the concept of microbial co-pathogenicity. *Pediatr Infect Dis J.* 1991;10:275–281
 33. Seppala H, Nissinen A, Jarvinen H, et al. Resistance to erythromycin in group A streptococci. *N Engl J Med* 1992;326:292–297
 34. Fujita K, Muroto K, Yoshikawa M, Murai T. Decline of erythromycin resistance of group A streptococci in Japan. *Pediatr Infect Dis J.* 1994;13:1075–1078
 35. Seppala H, Klaukka T, Vuopio-Varkila J, et al. The effect of changes in the consumption of macrolide antibiotics on erythromycin resistance in group A streptococci in Finland. *N Engl J Med.* 1997;337:441–446

Acute Sinusitis—Principles of Judicious Use of Antimicrobial Agents

Katherine L. O'Brien, MD*; Scott F. Dowell, MD, MPH*; Benjamin Schwartz, MD*; S. Michael Marcy, MD‡; William R. Phillips, MD, MPH§; and Michael A. Gerber, MD||

ABSTRACT. Establishing an accurate diagnosis of bacterial sinusitis is challenging but critical, because viral rhinosinusitis is at least 20 to 200 times more common than bacterial infection of the sinuses. Strict criteria for clinical diagnosis that require either prolonged and persistent symptoms or an acute severe presentation are supported with published evidence.

From the *Childhood and Respiratory Diseases Branch, DBMD, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia; †Kaiser Permanente, Panorama City, California; ‡Northwest Family Medicine, Seattle, Washington; and ||Connecticut Children's Medical Center, Hartford, Connecticut.

Received for publication Aug 8, 1997; accepted Sep 11, 1997.

Reprint requests to (S.F.D.) Centers for Disease Control and Prevention, Mailstop C-23, 1600 Clifton Rd, NE, Atlanta, GA 30333.

PEDIATRICS (ISSN 0031 4005). Copyright © 1998 by the American Academy of Pediatrics.

Radiographic imaging of the sinuses should be used only in very selected circumstances. A majority of patients with the common cold will meet radiographic criteria for sinusitis early in the course of their illness. For patients meeting these strict criteria, an appropriate narrow-spectrum antimicrobial agent will be of modest benefit compared with symptomatic treatment alone. *Pediatrics* 1998;101:174–177; *sinusitis, diagnosis, antimicrobial therapy, mucopurulent rhinitis, antimicrobial resistance, pediatrics.*

ABBREVIATIONS. URI, upper respiratory tract illness; CT, computed tomography.

PRINCIPLES

1. Clinical diagnosis of bacterial sinusitis requires the following: prolonged nonspecific upper respiratory signs and symptoms (ie, rhinosinusitis and