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CHAPTER 10

Acute and Chronic Infections of the Oral Cavity and Pharynx

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Infections of the oral cavity and pharynx require an appreciation for the interrelatedness of the aerodigestive structures of the head and neck. A multidisciplinary approach is necessary for the evaluation and management of these common disorders. The importance of understanding these diseases cannot be overestimated, because even though antibiotic use may prevent the more severe presentations, children today are nonetheless still susceptible to respiratory, hematologic, or central nervous system complications of these diseases and to long-term sequelae, such as rheumatic fever. This chapter reviews the typical presentations of oral and pharyngeal infections.

GENERAL CONSIDERATIONS

Oropharyngeal Anatomy

Oropharyngeal anatomy can be conceptualized as a framework covered by drapery. The floor of mouth serves as a theater stage, whereas the posterior pharynx (backstage) is partially hidden on either side and above by the drapery of the soft palate and tonsils. Moving from superior to inferior and anterior to posterior, the hard palate of the maxilla limits the region superiorly and anteriorly, and the skull base and clivus provide the posterior roof (Fig. 10-1). The cervical spine provides the posterior bony border, and the mandible provides the lateral and anterior bony framework. Inferiorly and anteriorly, the hyoid provides suspension of the floor of mouth. The mucosa of the soft palate, retropharynx and oropharynx, and floor of the mouth provide the medial limit. Drainage from the major salivary glands is lateral, from the paired parotid or Stensen's ducts, and inferior, from Wharton's ducts, which drain the submandibular glands. The potential

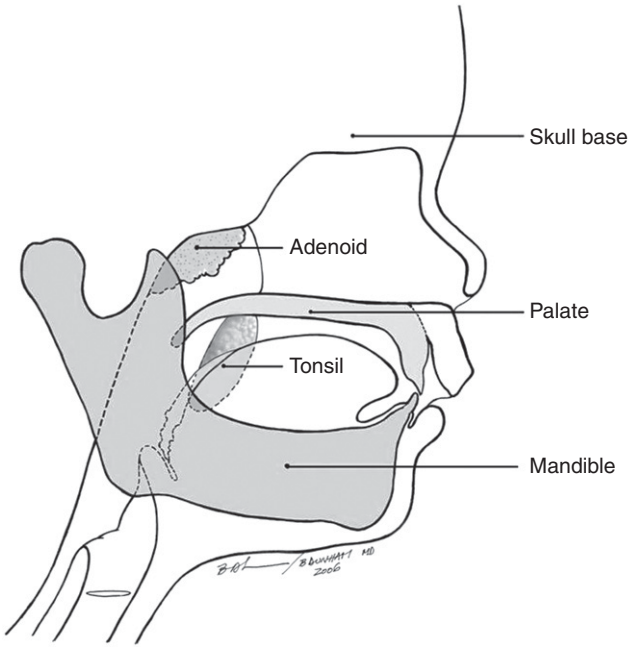

Figure 10-1. Oropharyngeal anatomy. (Courtesy of Brian Dunham, MD, and Eleanor Eve Porges.)

spaces between the deeper retropharyngeal and parapharyngeal fibromuscular layers permit spread of infection from the initial location to regional areas, resulting in airway compromise or thrombosis of major vessels. The spread of infection into the mediastinum may prove fatal.

Lymphoid tissue is present within the mucosa throughout this region, with the largest aggregations present in Waldeyer's tonsillar ring, which surrounds the border between the oral cavity and pharynx. Superiorly, Waldeyer's ring consists of the adenoid or nasopharyngeal tonsil, laterally, the paired palatine tonsils and inferiorly, the lingual tonsil at the tongue base.

Clinical Evaluation

Evaluation of the child suspected of having an infection in the oral cavity or pharynx begins with a thorough history from the child's caretakers, with attention to prodromal symptoms, recent travel and illness contacts, immunization status, degree of activity, airway and swallowing concerns, ability to maintain hydration, and the child's response to supportive and pharmacologic therapy.

Physical Examination

Physical examination should focus on determining the degree of immediate distress—fever, mental status, and adequacy of airway—keeping in mind that a lethargic child may not exhibit severe compromise because of inactivity. Evidence of serous otitis media on otoscopy may indicate inflammation or obstruction in the nasopharynx, affecting the eustachian tubes. Oral examination should note the degree of trismus, state of salivary papillae (Stensen's and Wharton's ducts), lingual papillary color, degree of mucosal dehydration, evidence of posterior pharyngeal secretions, tonsillar size and color, and presence of exudates on the tonsils. Asking the child simply to open her or his mouth without protruding the tongue facilitates examination of the posterior pharynx. Gentle depression of the midportion of the tongue with a tongue depressor allows a better view of the tonsils and posterior pharynx while avoiding precipitation of the gag reflex and allows for an effective examination, even with a degree of trismus (Fig. 10-2). The oral mucosal examination can be accomplished by gently directing the child's head, using one hand on the back of the head while stabilizing the other hand with the tongue depressor against the child's mandible. The tongue depressor can then be walked around the upper and lower alveoli, permitting comfortable and complete evaluation of the mucosa, salivary ducts, gingiva, and dentition. The fingers of the directing hand may be used to milk secretions from the parotid and submandibular glands (Fig. 10-3). Flexible nasopharyngoscopy may be used in selected cases, particularly when the oral airway appears compromised. The neck should be assessed for tenderness, fullness, adenopathy, and range of motion.

Laboratory and Radiologic Studies

Laboratory studies are not always necessary, but for children in whom intravenous therapy or hospitalization

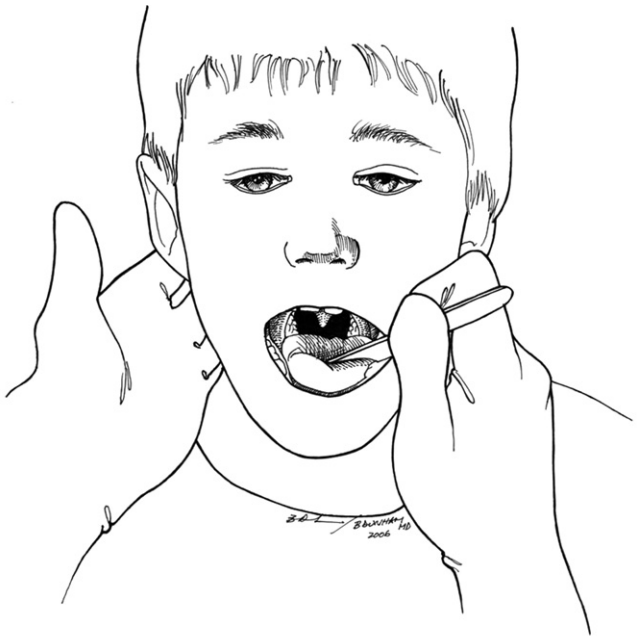

Figure 10-2. Examination of the oropharynx. Oral examination is facilitated by placing the tongue depressor against the middle of the tongue rather than posteriorly, thereby allowing a good examination of the posterior pharynx while avoiding gagging. This technique is usually effective, even in children with trismus.

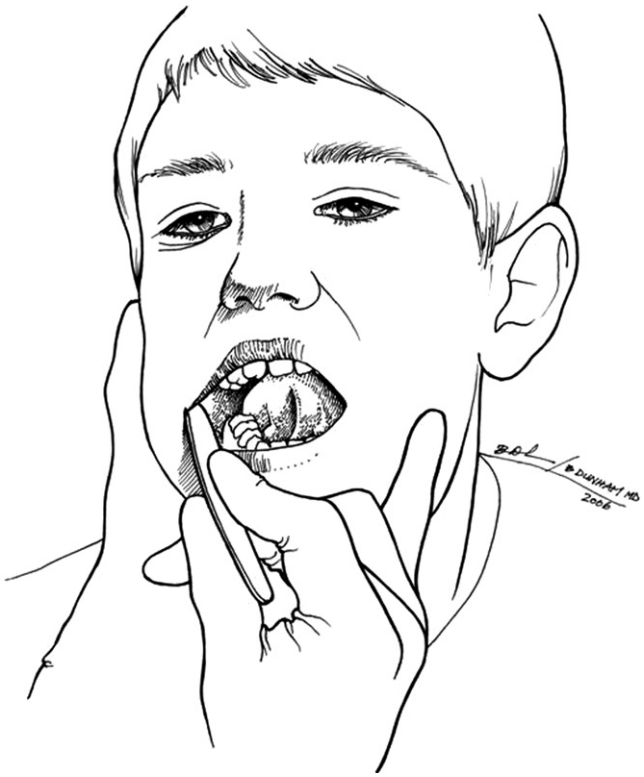

Figure 10-3. Examination of the oral mucosa. The oral mucosal examination is facilitated by gently directing the child's head from a posterior position using one hand while stabilizing the other hand with the tongue depressor against the child's mandible. The tongue depressor can then be "walked" around the upper and lower alveoli, permitting comfortable and complete evaluation of the mucosa, salivary ducts, gingiva, and dentition. The fingers of the directing hand may be used to milk secretions from the parotid and submandibular glands.

is considered, a baseline urinalysis, serum metabolic panel, complete blood count with differential, and assays for serum markers for inflammation (C-reactive protein [CRP] or erythrocyte sedimentation rate [ESR]) may be indicated.

The need for radiographic studies varies by site and extent of presentation, and emerging concern regarding the long-term effects of low-dose diagnostic irradiation has suggested judicious use of plain radiography and computed tomography (CT) studies.[1] Plain radiography has a limited role in the initial assessment of most throat infections and should be reserved for patients in whom fiberoptic nasopharyngolaryngoscopy is not available or possible. Lateral neck radiography may show bulging of the retropharyngeal soft tissues, with resultant airway narrowing.

A contrast-enhanced CT scan (CECT) or an MRI with contrast (MRIC) permits the differentiation of abscess from phlegmon. An abscess is identified by a rim-enhancing fluid collection, whereas a hypolucent area without ring enhancement is defined as phlegmon. In general, MRI with contrast enhancement offers superior tissue detail and diagnostic information, but this benefit must be balanced against the risks involved in this lengthier study, which often requires sedation in a child with a potentially compromised airway. A scan should be considered for children who exhibit clinical worsening or a failure to improve after 24 to 48 hours of antibiotic therapy. Repeat imaging is not necessary for children who are improving clinically, nor is follow-up imaging generally warranted.

Interventional radiologic techniques may have a role with selected abscesses, either for therapeutic drainage or for diagnostic aspiration, to provide material for microbial culture.

VIRAL INFECTIONS OF THE ORAL CAVITY AND PHARYNX

Most pharyngitis in children is viral and is usually caused by adenovirus, influenza A virus, Epstein-Barr virus (EBV), herpes simplex virus, rhinovirus, coronavirus, enterovirus, coxsackievirus, and echovirus.[2] Viral pharyngitis occurs most commonly during colder weather, when more ill contacts are indoors. The hallmarks of viral pharyngitis are a prodrome of fever, fatigue and/or malaise, and arthralgias.

Most children with viral pharyngitis respond well to oral rehydration, rest, antipyretics, and analgesics, specifically acetaminophen or ibuprofen. Aspirin should be avoided because of the risk of Reye's syndrome (acute encephalopathy, hepatic steatosis, and an elevated serum transaminase level). Despite teaching by health care providers and public health initiatives, caretakers may still use aspirin because of a lack of understanding (or inadvertently) in the form of aspirin-containing over-the-counter preparations, or folk or traditional remedies.[3] Gargling with a warm solution of dilute baking soda or salt water may also provide symptomatic relief.

Traditional or folk remedies, such as homeopathy, acupuncture, chiropractic, and medicinal herbs (known as complementary and alternative medicine [CAM]), are used by 1.8%[4] to 21%[5] of families caring for infants or children. Because of this degree of use, and despite the paucity of data on the efficacy and safety of CAM for pediatric pharyngitis, some experiences have been published. Caregivers and families usually appreciate information about potentially dangerous side effects of these treatments.[6-11]

Rhinovirus

The most common viral pathogen of acquired acute pharyngitis is thought to be the rhinovirus, which causes the common cold—a self-limited episode of coryza, rhinorrhea, fever, and malaise, for which supportive care is indicated. Oral antibiotics are not routinely prescribed.

Coxsackievirus

Children afflicted by coxsackievirus A16 infection develop hand-foot-and-mouth disease, in which a viral prodrome of fever, sore throat, and oral and pharyngeal erythema is followed 1 to 2 days later by vesiculopapular skin lesions that are usually but not exclusively seen on the palms and soles. Hand-foot-and-mouth disease often affects children younger than 5 years.[12] It generally resolves in 1 week. Supportive care is indicated, and baking soda baths soothe the rash.

Epstein-Barr Virus

EBV infection most often afflicts children older than 15 years, in whom it manifests as typically infectious mononucleosis (IM).[13] Fever and significant lethargy accompany dysphagia and odynophagia, with resultant dehydration. The physical examination shows enlarged cervical nodes bilaterally, often giving the neck a full appearance. Enlarged erythematous tonsils have gray exudates that can be peeled off without causing bleeding. The tonsillar enlargement contributes to airway obstruction, initially with a "hot potato" voice and loud, stertorous mouth breathing, which may progress to obstructive symptoms at night. Hepatosplenomegaly and enlarged axillary and inguinal lymph nodes may be seen as well. Children often experience a prolonged period of fatigue, which may last for months following initial management.

Clinical diagnosis can be confirmed by laboratory testing, usually by the serum heterophil antibody test (Mono spot). False-negative results are more common in children, especially children younger than 4 years.[13] Specific serologic tests may be required for diagnosis in heterophil-negative children. The serum IgG level against the Epstein-Barr viral capsid antigen (VCA) is high early in IM, whereas IgM anti-VCA antibodies identify recent infection. Serum antibody against EBV nuclear antigen (EBNA) is present weeks to months after infection.[14]

Management of IM begins with hydration, analgesics, antipyretics, and oral or parenteral steroids for airway obstruction. Children should be cautioned against vigorous physical activity, particularly contact sports, until the spleen is no longer palpable, because of concern over splenic rupture. This period of athletic abstinence may be required for 1 month,[13] and complete recovery may take several months.

Antibiotics may be necessary to treat bacterial superinfection of the tonsils, may reduce dysphagia, and therefore may promote the resumption of oral intake.[15] Children with infectious mononucleosis should not receive amoxicillin or ampicillin because of the risk of a drug-induced papular skin rash.[14] The oral antibiotic azithromycin has also been reported to cause a nonspecific skin rash.[16] The rash resolves a few days after discontinuation of the drug and does not preclude its future use.[17] Infectious mononucleosis must therefore be excluded in all children who present with acute pharyngitis, either by history and examination or by serologic testing.

A short course of oral corticosteroid therapy may be considered for children with aerodigestive compromise from acute infectious tonsillar hypertrophy or in children with systemic complications, such as massive splenomegaly, myocarditis, thrombocytopenia, or hemolytic anemia.[13] Corticosteroids may shorten the duration of fever and pharyngitis. Gamma globulin may be helpful in severe cases.

Hospitalization may be required for hydration and for airway observation. Tonsillectomy is rarely necessary to relieve airway obstruction or dysphagia. Post-tonsillectomy hemorrhage may be more likely to occur in children who require surgery during the acute phase of IM.[18,19]

Herpangina

Herpangina is a severe viral pharyngitis that presents with soft palate, tonsillar, and oropharyngeal erythema and with multiple small clear vesicles, which form shallow ulcers on rupture. Herpangina is caused by coxsackievirus A and B, types 1 to 5,[12] and by human enteric echoviruses. Herpangina has an incubation period of 3 to 7 days and usually resolves after several days.

Herpes Simplex Viruses

Herpes simplex virus (HSV) type 1 infection affects the oral mucosa in children in two different ways—via a primary infection, which is usually severe, and via a secondary recurrent infection, with milder presentation and course. Primary herpetic gingivostomatitis most commonly presents in children between 1 and 3 years of age,[12] with edema, erythema, and a sharp prickling sensation of the lips, gingiva, tongue, and palate. Vesicles rupture into ulcers, which then desquamate and form a gray membrane that may crust, coalesce, or develop a secondary bacterial infection. Children with HSV infection may exhibit lymphadenopathy, fever, and flulike symptoms, which may progress to disseminated herpetic infection, skin eruptions, or meningoencephalitis.

Secondary recurrent HSV type 1 (HSV-1) infection may occur in up to 40% of people after primary infection.[12] The activation of the dormant virus from regional neuroganglia may occur during periods of reduced host defenses, especially during periods of fever, stress, excessive sun exposure, or immunodeficiency. Oral antiviral therapy with acyclovir, famciclovir, or valacyclovir may benefit children during acute infection and may reduce the frequency of secondary episodes.[14,20] A topical antibacterial ointment, such as mupirocin (Bactroban), may

be used for secondarily infected ulcers.[21] Topical creams containing antiviral agents such as acyclovir and penciclovir may also be prescribed for children.[20,22]

Herpes Zoster

Herpes zoster infection, also known as shingles, is caused by the varicella-zoster, or chickenpox, virus, which lies dormant in dorsal root ganglia until it erupts during a period of immunodeficiency into painful vesicular mucosal or dermal eruptions. These appear in stages and then crust and gradually resolve within 10 days. Because the trigeminal ganglion is affected, dental pain may precede the vesicular eruption by 2 or 3 days.[12] Care is supportive, with hydration and analgesics. Oral acyclovir may be considered in primary varicella infection for children older than 12 years, those with chronic cutaneous or pulmonary disorders, and children receiving long-term salicylate or short, intermittent, or aerosolized corticosteroid therapy.[14] Intravenous antiviral therapy is indicated for immunocompromised children. Systemic primary varicella infection (chickenpox) may manifest with similar oral vesicles. Postexposure vaccination may be offered to susceptible children.[14]

Human Papillomavirus

Human papillomavirus (HPV) types 6, 14, and 22 infect the upper respiratory mucosa. Papillomas form at the junction of ciliated and nonciliated mucosa. Presentation varies by site of disease, with hoarseness or dyspnea suggesting laryngeal papillomas. Painless papillomatous lesions may appear on the soft palate or uvula. Although solitary papillomas are generally treated successfully by excision, recurrent respiratory papilloma (RRP) can be a problematic, progressive, and potentially lethal disorder.

Airway obstruction at the level of the larynx requires surgical treatment, usually a number of times throughout childhood and sometimes into adulthood. Recurrent respiratory papilloma (RRP) may result from progressive spread of papillomas throughout the airway, specifically in the larynx and trachea. Removal of pharyngeal papillomas is intended to reduce the risk of spread into the larynx and beyond. Adolescence may bring about gradual clinical resolution. Adjuvant medical management with chemotherapeutic agents may be offered in some cases. RRP may rarely undergo malignant transformation into adenocarcinoma.

Measles

Measles, caused by an RNA virus, occurs rarely in the United States today. Episodes are attributed to the rare failure to immunize a child, vaccine failure, or importation of the virus from another country. Children exhibit fever, cough, coryza, and conjunctivitis and develop an erythematous maculopapular rash. Yellow-white pinpoint papules against an inflamed buccal mucosa (Koplik's spots) may appear 2 to 4 days before other general symptoms or skin rash appear.[12] Patients are contagious 3 to 5 days before the onset of the skin rash to 4 days after its appearance. The incubation period of 8 to 12 days from exposure to onset of symptoms means that the time from rash in a source case to the next case averages 2 weeks. Treatment is supportive, and intravenous or aerosolized ribavirin has been used to treat children who are severely affected or immunocompromised. Vitamin A has been used to reduce morbidity of measles infection in children who are vitamin A–deficient or when the case fatality rate is historically 1% or higher.[14]

Mumps

Bilateral parotitis is the classic otolaryngologic presentation of this now-rare RNA viral infection, with fewer than 300 cases/year reported since the introduction of the mumps vaccine.[14] Postpubertal boys may suffer from orchitis, which rarely results in infertility. Viral culture from body fluids and antibody titers is diagnostic. Care is supportive.

Aphthous Stomatitis

This recurrent ulcerative lesion of the oral cavity typically is a problem for teenagers. It may be caused by a respiratory virus or an autoimmune or delayed-type hypersensitivity response. Aphthous lesions often occur during times of stress. Treatment is supportive. Topical suspensions containing equal parts of viscous lidocaine and diphenhydramine hydrochloride, and a combination of a mucosal-protective agent such as aluminum-magnesium hydroxide (Maalox), may be used to provide symptomatic relief. Another topical agent that has had some success is a suspension of hydrocortisone, nystatin (Mycostatin), and tetracycline. Topical benzocaine-phenol-alcohol (Anbesol) may also offer short-term symptomatic relief when applied to the ulcers.

BACTERIAL INFECTIONS OF THE ORAL CAVITY

Gingivitis

Most cases of gingivitis in children are associated with poor oral hygiene and plaque. Immunosuppressed or malnourished children may present with acute necrotizing ulcerative gingivitis (ANUG), or Vincent's gingivitis. This potentially severe infection is caused by fusobacteria and spirochetes, and possibly by other organisms.[12,23]

ANUG usually affects children older than 12 years and is identified by friable interdental papillae, necrotic pseudomembranes along the gingival margin, fetor oris (foul-smelling breath), malaise, and fever. Treatment includes oral débridement and rinses with dilute hydrogen peroxide or chlorhexidine; oral antibiotics (e.g., amoxicillin, erythromycin, penicillin) may be helpful for extensive infection.[23]

Ludwig's Angina

Acute infection of the floor of the mouth involving the submandibular or sublingual spaces is known as Ludwig's angina. Presentation includes submandibular fullness and induration, drooling, and a "hot potato" voice and may progress without treatment to airway compromise because of superior displacement of the tongue and infectious spread through adjacent compartments.

The potential for rapidly progressive airway compromise warrants a high index of suspicion and rapid protection of the airway by early institution of intravenous antibiotics, corticosteroids, and possibly airway management, including intubation or tracheotomy. Incision and drainage are usually required for cure.

Bacterial Infection of the Submandibular Glands

Bacterial infection of the submandibular glands usually involves only one gland, and manifests with submandibular swelling, tenderness, and erythema of the submandibular duct lateral to the lingual frenulum. Purulent material may be expressed through these ducts with gentle forward pressure on the submandibular region. Infection is promoted by salivary stasis related to dehydration, inspissated secretions, or ductal obstruction due to sialolithiasis. Streptococcal and staphylococcal bacteria are usually the causative agents. Sialogogues, hydration, warm compresses to the neck, and anti-inflammatory drugs provide symptomatic relief. Oral or intravenous antibiotic therapy, depending on the degree of inflammation and concern over airway obstruction, is usually curative. For infections developing into an abscess, induration of the submandibular skin may precede development of an abscess clearly defined by palpation or imaging. Such abscesses require surgical drainage.

Parotitis

Acute bacterial parotitis is not commonly seen in children and is usually caused by *Staphylococcus aureus*. Diagnosis is made clinically with a history of fever, pain, and drooling, and physical findings that include a bulging, erythematous, and tender cheek, with induration of the overlying skin, trismus, and evidence of pus from Stensen's duct (Fig. 10-4). Recurrent parotitis may indicate a rheumatic or collagen-vascular disease. Concurrent rheumatic complaints such as dry eye and arthralgias warrant laboratory evaluation for autoimmune causes by testing for rheumatoid factor (RF) to diagnose juvenile rheumatoid arthritis and by testing for serum markers, such as SSA and SSB, to diagnose Sjögren's disease.

Figure 10-4. Acute parotitis. Pus is seen streaming from the parotid, or Stensen's, duct in this child with acute parotitis.

Hydration, sialogogues, and oral antibiotics can manage most cases of parotitis successfully. When symptoms are refractory or progressive despite oral antibiotics, intravenous antibiotics are begun and CECT or MRIC may be necessary to exclude a mass lesion or abscess. In cases of abscess formation, radiographically guided needle aspiration may offer symptomatic relief, but definitive drainage may require a parotidectomy approach, with identification and protection of the facial nerve.

BACTERIAL INFECTIONS OF THE PHARYNX

Pharyngitis

The most common bacterial pathogen of concern in the pharynx is *Streptococcus pyogenes,* a gram-positive organism that exhibits beta-hemolysis of blood agar by culture and that demonstrates the group A cell wall carbohydrate antigen as defined by Rebecca Lancefield in the 1930s. *S. pyogenes* is therefore referred to simply as group A beta-hemolytic *Streptococcus* (GABHS).[25] More recent techniques have allowed categorization of GABHS by sequencing the 5′-terminal end of the *emm* gene.[26] Other pathogenic bacteria that may cause pharyngitis are *Corynebacterium diphtheriae*, *Neisseria gonorrhoeae*, and *Arcanobacterium haemolyticium*,[13] and commensals such as nontypable *Haemophilus influenzae*,

Streptococcus pneumoniae and *S. viridans*, *Staphylococcus aureus* and *S. epidermidis*, and *Moraxella catarrhalis*.[2]

GABHS causes pharyngitis mostly during the winter and spring, and infection is spread primarily via droplets from respiratory secretions.

Acute, Recurrent, and Chronic Tonsillitis

Acute tonsillitis manifests with fever, throat pain, foul breath, dysphagia and odynophagia, tender cervical adenopathy, and tonsillar erythema and exudates. Acute tonsillar enlargement caused by the infection may result in mouth breathing, disordered nighttime sleep, or sleep apnea. Symptoms may last up to 2 weeks with therapy.

The peak incidence of acute tonsillitis occurs between 5 and 15 years and is rarely seen in children younger than 3 years.[26,27] When several episodes of acute tonsillitis occur in 1 year, the child is said to experience recurrent tonsillitis. The lifetime prevalence of self-reported recurrent tonsillitis in a Norwegian study was noted to be 11.7%[28] and was documented in 12.1% of elementary school children in a Turkish report.[29] A parental history of atopy and of tonsillectomy may be predictive for the development of tonsillitis in children.[28]

Chronic tonsillitis is identified by a 3-month history of sore throat, halitosis, odynophagia, possibly otalgia, and tonsillar inflammation, often with debris within crypts or tonsillar exudates.[30,31] Chronic tonsillitis may also be defined by the presence of tonsilliths, which consist of firm, yellow-white calculi that may be spit out or expressed from the tonsils with a swab. Tonsilliths, as well as softer, cheesy, foul-smelling debris, may accumulate within tonsillar crypts (cryptic tonsillitis).

Carrier State

The carrier state is defined by the presence of GABHS in the pharynx by culture, without evidence of an immunologic response to streptococcal antigens.[32] Between 2.5% and 10.9% of children may be carriers of GABHS.[2,27] In one study from western Pennsylvania, a 27% to 32% prevalence over the course of the school year was seen in children, with a point prevalence per given month of 15.9%. Many children who were carriers were hosts to different organisms, because the bacterial *emm* types changed after an average of 10.8 weeks.[26] In addition, despite living among this reservoir of GABHS carriers, 40% of children were uninfected each school year, perhaps because of their own specific mucosal immunity, local or systemic, or to production of antibacterial peptide. Children are most susceptible to infection from carriers.[27]

Diagnosis

The diagnosis of bacterial pharyngotonsillitis can be made in most cases by symptoms and signs. Sudden onset of sore throat, fever, headache, abdominal pain, and nausea and vomiting are considered characteristic symptoms, whereas characteristic signs are erythema and exudates of the tonsils and pharynx (Fig. 10-5), soft palate petechiae (doughnut lesions), uvular swelling and erythema, anterior cervical adenitis, and a scarlatiniform rash. There is an absence of cough, rhinitis, stridor, hoarseness, conjunctivitis, and diarrhea. Younger children exhibit a less severe febrile response than older children.[33] Lingual swelling and erythema may produce a strawberry tongue, and early in the disease, a white coating of this surface may bring about the so-called white strawberry tongue.[33]

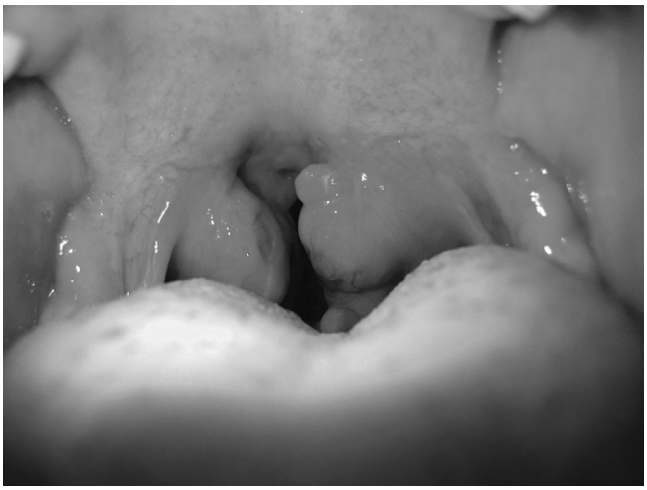

Figure 10-5. Acute tonsillitis.

The scarlet fever rash seen in GABHS is a fine rash, rarely seen in children younger than 3 years, and is caused by an erythrogenic toxin (exotoxin A). Two signs of interest are Pastia's lines and the Rumpel-Leede phenomenon. Pastia's lines (or sign) are the accentuated erythema seen in the flexor skin crease, such as the antecubital or axillary creases. An increase in petechiae distal to the point of application of a tourniquet is known as the Rumpel-Leede phenomenon.[2,13,33]

Laboratory Testing

Tonsillitis is a clinical diagnosis. Testing is indicated when GABHS infection is suspected to determine the need for antibiotic therapy and to avoid unnecessary and potentially harmful antibiotic use.[34] Throat cultures are the gold standard for the diagnosis of GABHS, with identification of the organism by fluorescent antibody testing or disk diffusion testing using bacitracin. Alternatively, the rapid antigen detection test (RADT), also called the rapid strep test, detects the presence of GABHS cell wall carbohydrate from swabbed material and has a high specificity, higher than or equal to 95%. However, its limited sensitivity, only 80% to 90% compared with that of a throat culture, means that a negative RADT result must be

followed by a more sensitive throat culture before excluding GABHS infection.[13]

Waiting 24 to 48 hours for throat culture results to return will not diminish the efficacy of antibiotic therapy in preventing rheumatic fever, the major complication of GABHS.[13] Post-therapy culture is usually not necessary, but may be useful for determining antibiotic resistance and for confirming disease resolution. Documentation of culture results is helpful in establishing the frequency and chronology of infections to facilitate future therapeutic decisions.

A complete blood count (CBC) demonstrates leukocytosis, with a predominance of neutrophils. Other acute-phase reactant levels (CRP, ESR) may be elevated. Monospot and serum electrolyte determinations may be indicated. The serum should be examined for antistreptococcal antibodies, such as antistreptolysin O (ASO) and antideoxyribonuclease (anti-DNAse). These titers are useful for documenting prior infection in persons diagnosed with acute rheumatic fever (ARF), glomerulonephritis, or other complications of GABHS pharyngitis. GABHS carriers tend to have higher antibody titers with acute infection than noncarriers.[32]

Management

Acute Viral Tonsillitis

Treatment of acute viral tonsillitis is largely supportive and focuses on maintaining adequate hydration and caloric intake while controlling pain and fever by analgesics, antipyretics, and hydration. Inability to maintain adequate oral caloric and fluid intake may require intravenous therapy and/or hospitalization.

Bacterial tonsillitis requires supportive care as well as oral antibiotics to reduce symptoms of infection and attendant complications. The treatment goals for therapy in GABHS pharyngitis are (1) prevention of ARF, (2) prevention of suppurative complications, (3) abatement of clinical symptoms and signs, (4) reduction in the transmission of GABHS to close contacts, and (5) avoidance of the adverse effects of inappropriate antimicrobial therapy.[2,13] There is no evidence to support the claim that antibiotic therapy reduces the risk of acute post-streptococcal glomerulonephritis (AGN).[13]

Airway obstruction that may accompany acute tonsillitis in children with underlying adenotonsillar hypertrophy may require treatment with a nasal airway, corticosteroids, humidified oxygen, and hospitalization for airway observation.

Antibiotic Therapy. The GABHS organism is generally susceptible to oral penicillin—the antibiotic of choice because of its efficacy, narrow spectrum of antibacterial activity, safety, and low cost. A full 10-day course is necessary to eradicate infection maximally.[13] Other antibiotics proven effective for GABHS pharyngitis are the penicillin congeners (amoxicillin, amoxicillin-clavulanate), cephalosporins, macrolides, clindamycin, vancomycin, rifampin, and metronidazole.[15,35]

Corticosteroid Therapy. Corticosteroids also are indicated for patients with airway obstruction, hemolytic anemia, and cardiac and neurologic disease. A single oral or intramuscular dose of dexamethasone has been shown to provide improved pain relief over placebo when administered to patients 15 years of age and older with acute pharyngitis.[36]

Recurrent and Chronic Tonsillitis

Recurrent tonsillitis may be treated with the same antibiotics as for acute GABHS pharyngitis. If the infection recurs shortly after a course of an oral penicillin agent, treatment with intramuscular (IM) benzathine penicillin G should be considered. A 3- to 6-week course of clindamycin or amoxicillin-clavulanate has been shown to be effective in eradicating GABHS from the pharynx in children suffering from repeated bouts of tonsillitis. Prolonged antibiotics may be attempted for chronic tonsillitis, including cryptic tonsillitis. Oral rinses and mechanical débridement using swabs or irrigation (e.g., using a water pick) are helpful for removal of tonsillar debris.

Carrier State

Antibiotic treatment for the carrier state should be considered when there is a family history of rheumatic fever, a carrier with a history of glomerulonephritis, frequent spread between household contacts ("ping pong" spread), familial anxiety, infectious outbreak within a closed or semiclosed community (e.g., boarding school), outbreak of ARF, or when tonsillectomy is being considered only because of chronic carriage of GABHS. Tonsillectomy may be considered if antibiotic therapy fails to eradicate the carrier status.

Complications

Complications specific to GABHS pharyngitis are scarlet fever, rheumatic fever, septic arthritis, and poststreptococcal glomerulonephritis.

Scarlet fever manifests as a generalized, nonpruritic, macular erythematous rash that is worse on the extremities and spares the face. The resultant strawberry tongue, Pastia's lines, and Rumpel-Leede phenomenon may last up to 1 week and are accompanied by fever and arthralgias. Scarlet fever results from a lysogenic strain[25] of GABHS. Individuals at risk for this rash are those who do not have antitoxin antibodies to the offending exotoxin.

ARF is a nonsuppurative inflammatory reaction related to prior GABHS infection. It is characterized by some combination of septic arthritis, carditis, chorea, erythema marginatum, and subcutaneous nodules. Rheumatic fever follows acute pharyngitis by 2 to 4 weeks and was observed in up to 3% of episodes of streptococcal pharyngitis prior to the development of antibiotics in the

mid-20th century. Molecular mimicry between epitopes on GABHS M-protein and cardiac tissue results in immune-mediated valvular endothelial damage.[36] Cardiac valvular vegetations affect the mitral and tricuspid valves, leading to murmurs, persistent relapsing fevers, and valvular stenosis or incompetence, and may be seen within 2 to 3 weeks of onset of infection, whereas chorea and erythema marginatum may be seen as late as 6 months following infection. Diagnosis requires proof of GABHS infection by culture and serologic testing. A throat swab alone does not identify the causative organism, because a positive swab may reflect colonization rather than pathogenicity. Elevated or rising titers of ASO antibodies, anti-DNAse beta, or antihyaluronidase are required to make the diagnosis. Treatment of ARF is with salicylates and corticosteroids. Penicillin does not alter the course of ARF but is given once the diagnosis has been definitively made or if GABHS has been cultured from the pharynx.[25]

In contrast with ARF, which occurs only after pharyngitis, AGN may occur after pharyngitis or cutaneous infection. AGN can occur in up to 10% to 15% of those with pharyngitis. The latent period between infection and nephritis is 1 to 2 weeks after the onset of pharyngitis. AGN is caused by an immunologic reaction between nephritogenic bacterial strains and glomerular basement membrane during the course of pharyngitis or impetigo, resulting in immune complex deposition and in circulating immune complexes.[37] In children, the most common symptoms are edema, oliguria, hypertension, congestive heart failure, and seizures. Urinalysis shows dark or smoky urine with red blood cells (RBCs), RBC casts, white blood cells, and proteinuria. There is an increased level of serum complement and a decreased glomerular filtration rate. Urinalysis is useful in detecting subclinical renal injury by showing proteinuria in children who have suffered from recurrent tonsillitis. Diagnosis requires documentation of GABHS infection, most often by ASO or anti-DNAse beta. Treatment includes hydration, sodium restriction, diuresis, and anticonvulsants.

Septic streptococcal arthritis results in a painful hot joint that contains fluid with bacteria. Arthrocentesis is diagnostic and partially therapeutic. Treatment with intravenous antibiotics for 6 weeks is required to prevent long-term joint complications.

Surgical Options

Surgery is a safe and effective means of treating recurrent and chronic tonsillitis in children.[30] An episode of tonsillitis for the purpose of making a decision about surgery may be defined as acute infection with at least one of the following: fever of at least 38.3°C, cervical adenopathy (larger than 2 cm or tender), tonsillar or pharyngeal exudates, or a positive culture for GABHS.[38] Children had fewer postoperative episodes of tonsillitis when they underwent tonsillectomy for seven or more episodes of tonsillitis in the preceding year, had five or more such episodes in each of the 2 preceding years, or had three or more such episodes in each of the preceding 3 years.[38] Tonsillectomy with or without adenoidectomy is also indicated for chronic or recurrent tonsillitis associated with the streptococcal carrier state that has not responded to antibiotics.

Tonsillectomy for less frequent episodes of tonsillitis is justified by postoperative improvement in upper airway obstruction, reduced time lost from school and work, easing of provider and caretaker concern over antibiotic use, and decreased discomfort experienced by children during infections, particularly related to the severity of episodes (e.g., whether hospitalization was required).[39] Tonsillectomy may also be considered for cryptic tonsillitis when oral rinses and débridement fail to treat foul taste, halitosis, and discomfort adequately.

Adenoidectomy may be performed with tonsillectomy for the surgical management of recurrent or chronic pharyngitis because of concurrent chronic adenoiditis or chronic infection of Waldeyer's ring. However, one study found no additional benefit to adenotonsillectomy over tonsillectomy alone in the surgical treatment of tonsillitis.[38]

Radiotherapy to manage tonsillitis is of historical relevance only. Ionizing radiation was used until the mid-20th century. Children treated in this way have been found to have an increased risk of neoplasia of the thyroid, parathyroid, and salivary glands. Lifelong follow-up to diagnose neoplasia early in these patients is indicated.[40-42]

Peritonsillar Infections

Children may account for approximately one third of all episodes of peritonsillar abscess (PTA).[43] Peritonsillar infection (PTI)—cellulitis, phlegmon, or abscess—involves the potential space surrounding the palatine tonsils and usually manifests with a pointing collection at the superior pole.[44,45]

Although GABHS is the most commonly cultured organism, isolated in approximately one third of cases,[15] it is not the only bacterial pathogen identified. Most PTAs are polymicrobial, with GABHS, *S. aureus*, and *H. influenzae* accounting for most of the aerobic organisms and *Prevotella*, *Porphyromonas*, *Fusobacterium*, and *Peptostreptococcus* strains comprising the common anaerobes.[44]

Diagnosis

The diagnosis of peritonsillar infection is primarily by history and physical examination. Children present with a several-day history of fever, dysphagia, odynophagia, or voice change, with the characteristic "hot potato" voice. Trismus is a strong indicator of a peritonsillar abscess.[44]

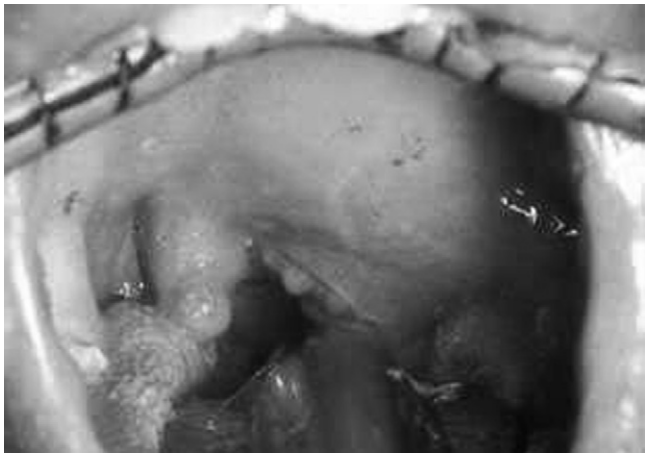

Figure 10-6. Peritonsillar abscess. This causes soft palate bulging above the left tonsil.

Otalgia and neck pain, both usually ipsilateral to the side of the abscess, may be present. Most children have been started on antibiotic therapy prior to the development of a PTA.[44]

Physical examination shows an erythematous bulging of the soft palate above the tonsil, possibly with medial displacement of the tonsil and/or uvula (uvular deviation) (Fig. 10-6). Trismus may limit examination, and the technique described earlier for oral examination is recommended—that is, placing the tongue depressor through whatever degree of mouth opening the patient can offer and displacing the central portion of the tongue only, with gentle downward pressure, as opposed to attempting to force the mandible down and compressing the entire anterior tongue. Cervical adenopathy, typically ipsilateral to the abscess, is also common.

Laboratory evaluation shows a leukocytosis with a propensity to immature cells. A monospot test, as well as a throat culture or rapid strep test, should also be performed. Imaging with CT or MRI scanning with contrast is indicated for unusual presentations (e.g., an inferior pole abscess) or for persistent symptoms despite needle aspiration or surgical drainage.

Management

Treatment aims to resolve discomfort, maintain the airway, and prevent abscess rupture. A ruptured PTA may result in the aspiration of purulence and lead to bronchopneumonia.[44] Aëtius of Amida, a sixth-century Byzantine physician, treated spontaneously draining abscesses with gargles of honey, milk, and herbs or rose extract.[34] Today, oral antibiotics are recommended, including penicillin and its congeners (e.g., amoxicillin-clavulanic acid), cephalosporins, and clindamycin. Hospitalization may be necessary for hydration, analgesia, intravenous antibiotics, and/or airway observation.

Many PTAs require needle aspiration or incision and drainage. Needle aspiration may be performed diagnostically to confirm abscess formation or identify the best point at which to perform incision and drainage, or as a therapeutic measure to relieve symptoms and provide material for microbial culture. Needle aspiration provides relief of pain and may hasten recovery. Although bacterial culture results are not clinically useful in most cases, cultures are valuable when there is a concern over antibiotic resistance (e.g., in immunodeficient children or those who have been recently treated with broad-spectrum antibiotics).[43] CT-guided needle aspiration is indicated when draining a PTA after an unsuccessful surgical attempt and when draining an abscess that is located in an unusual location, such as one that is anticipated to be difficult to reach with standard surgical approaches.

Incision and drainage permit a more complete evacuation than that allowed by needle aspiration. Incision and drainage are performed transorally and are indicated for the older, more cooperative patient, who may more easily permit a longer procedure than that required for needle aspiration. A PTA may be drained in this way using conscious sedation protocols or may require general anesthesia in the operating room because of the child's inability to cooperate. Acute tonsillectomy (quinsy tonsillectomy) may be necessary for relief of obstructive symptoms, a history of recurrent pharyngotonsillitis, or exposure of the abscess. Quinsy tonsillectomy is necessary in approximately one out of three cases of PTA in children.[44,46]

In treating PTA, attention should be paid to the tissue characteristics of tonsillar and peritonsillar tissues. Fleshy, granular, or pale tissue may indicate a neoplasm manifesting as a PTA.[18] In such cases, tissue should be sent for immunohistopathologic evaluation.

Deep Neck Space Infections

Infections of the potential spaces from the skull base to the mediastinum defined by fibromuscular planes are referred to as deep neck space infections. Because of their location and the interconnectedness of potential spaces (highways of infectious spread), deep neck space infections in children may exhibit rapid spread and airway compromise.

Parapharyngeal Space Infections

Children who have an infection of the parapharyngeal space (PPS) infection present with drooling, trismus, dysphagia, odynophagia, and bulging of the lateral pharyngeal wall. Neck stiffness, pain, and torticollis are important signs and symptoms of PPS infections. In contrast with peritonsillar infections, radiologic studies, usually CECT, are required for precise anatomic diagnosis (Fig. 10-7). Sequential radiography may be useful to monitor the efficacy of therapy.

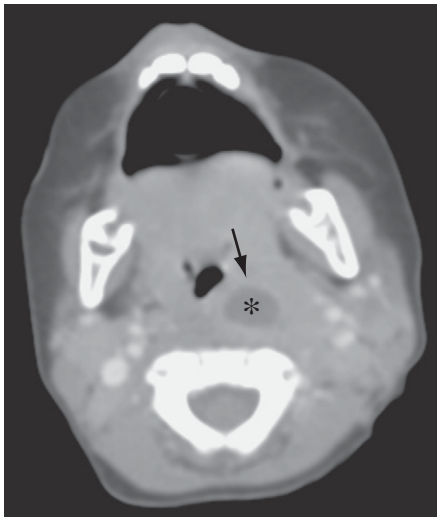

Figure 10-7. Parapharyngeal abscess. A left parapharyngeal abscess *(asterisk)* is shown by a rim-enhancing collection *(arrow points to rim)*, medial to the great vessels on this axial contrast-enhanced computed tomography study. This type of abscess is best drained transorally.

Management of small abscesses or phlegmons of the PPS is by intravenous antibiotics. Antibiotic therapy may need to continue for 2 to 3 weeks and, in some cases, may require placement of an indwelling intravenous catheter for outpatient administration of antibiotics. PPS infections that are extensive or refractory to antibiotics require surgical drainage by the transoral route for lesions medial to the great vessels or transcervically for lesions lateral to the great vessels.

Retropharyngeal Space Infections

Children with infections of the retropharyngeal space present with fever, drooling, neck pain or stiffness, torticollis, and cervical adenitis. Diagnosis is suspected by a bulging of the posterior pharyngeal wall and confirmed by CECT (Fig. 10-8). As with PPS infections, antibiotics are useful for small collections, and surgery is reserved for abscesses that fail to resolve with intravenous antibiotics. Surgical drainage may be performed transorally or externally, depending on the abscess location. Transoral drainage is most straightforward for abscesses above the hyoid, whereas more inferior collections may be better approached via an external transhyoid or lateral route.

FUNGAL INFECTIONS OF THE ORAL CAVITY AND PHARYNX

Candidiasis

Children receiving chronic antibiotic therapy or corticosteroids, and neonates with their transient immunodeficiency, may be afflicted by this oral fungal infection.

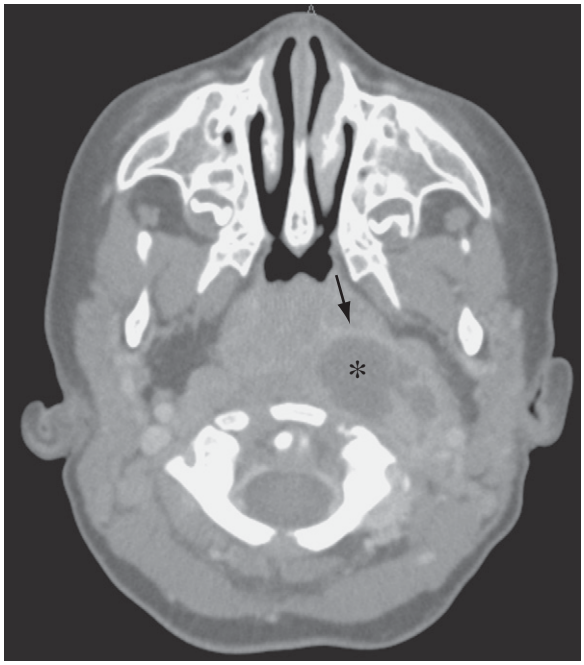

Figure 10-8. Retropharyngeal abscess. This abscess *(asterisk)* is within the rim-enhancing wall *(arrow)* on this contrast-enhanced CT scan.

Candidal infection (thrush, moniliasis) appears as patchy, white, curdlike aggregations on the tongue, palate, or buccal mucosa that may be wiped off with a gauze pad, leaving a raw, painful, bleeding surface. Evidence to date does not suggest that chronic oral candidiasis predisposes to neoplasia.[12] Treatment is with oral nystatin rinses. Swabbing a newborn's mouth with the mother's saliva also has been recommended.[12]

SUMMARY

Infections of the oral cavity and pharynx in children are frequently seen. Accurate diagnosis requires a complete history and thorough physical examination. Radiographic studies are reserved for atypical presentations, infections refractory to initial therapy, or guiding surgical drainage.

Most viral infections are managed by supportive care, including hydration and pain control. Most bacterial infections are initially managed by antibiotics; surgical therapy may be required for chronic infections (e.g., GABHS tonsillitis), regional spread (e.g., PPS infection), or airway obstruction (e.g., Ludwig's angina).

Successful management of these infections requires attention to the nuances of the usually characteristic physical examination findings specific to each infectious diagnosis, awareness of the potential for rapid spread of infection, and a proactive and team-oriented approach to therapy.

MAJOR POINTS

- Viral pharyngitis is common in children. Aspirin and aspirin-containing products should not be given, because they may cause Reye's syndrome.
- Antibiotic therapy for acute GABHS pharyngitis reduces the risk of rheumatic fever complications.
- There are specific criteria for treatment of GABHS carriers.
- Radiographic characterization of parapharyngeal and retropharyngeal infections is important for diagnosis and determination of the best surgical approach. Follow-up studies after recovery from the initial episode are not usually indicated.

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