INFECTIONS in EMERGENCY MEDICINE

VOLUME 1

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本书是《现代急诊医学》连续集丛书之一，分1、2卷出版发行。书中运用这一领域研究最新资料，系统详细地阐述了各种传染病的概念、病原学、病理生理学、临床表现、鉴别诊断和急诊处理。并对有关的会诊、入院要点、转院要点及预防措施作了全面介绍。是从事这一领域研究科研教学的医学院校师生，临床传染科医师、急诊医师及有关人员必备的参考书。

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PREFACE

As emergency medicine continues to grow and shape itself into a true multidiscipline specialty, we find ourselves expanding into subspecialty fields. Like emergency medicine, infectious disease crosses the line into many other fields of study. Although traditionally managed by internists, pediatricians, and other primary care physicians, infections are frequently seen by the emergency physician. Although excellent infectious disease texts are available, none present the material from the vantage point of the emergency physician. Our goal was to prepare a text covering the subject in depth, but focusing on clinical issues, so that the book would be useful to practitioners, as well as academicians, in our field. To this end, we believe we have achieved our goal.

Each chapter presents topics in a logical progression, beginning with the etiology and pathophysiology, followed by in-depth discussions of the clinical presentation, differential diagnosis, and emergency department management. Where applicable, attention is given to issues concerning subspecialty consultation, admission guidelines, transfer guidelines, and preventive measures. Inpatient management is discussed in sufficient detail to allow the emergency physician to assist in the decision-making process regarding therapy.

We would like to thank the contributing authors for providing scholarly, well-researched chapters, and extensive references. With their effort, and the persistence of the staff at Churchill Livingstone, it is with pleasure we offer you *Infections in Emergency Medicine*.

David Schillinger, M.D.
Ann Harwood-Nuss, M.D.

CONTENTS

1 Odontogenic Infections

Raymond Roberge

Patients with odontogenic infections may present with a variety of signs and symptoms ranging from the minor to the life-threatening, such that timely diagnosis and therapeutic intervention are important.[1] Satisfactory treatment of odontogenic infections requires knowledge of three basic factors: (1) the location of the infection, (2) the types of associated microorganisms, and (3) the specific antibiotic to which the pathogen is sensitive.[2] The emergency medicine physician is often the first practitioner to see and/or treat patients with odontogenic infections. Therefore, it is important that the physician possess a good working knowledge of oral anatomy and fundamental oral microbiology.

The tooth is a highly specialized organ composed of firm, mineralized tissue. The bulk of the tooth is formed of a core of dentin, a hard inorganic (75 percent) and organic (20 percent) matrix containing a rich neurovascular network. Centrally located within the dentin is the pulp, a collection of odontoblasts, connective tissue cells, blood vessels, and nerves. The pulp chamber is a narrow, hollow space within the pulp, which funnels down to a narrow root canal in the single-rooted tooth and to one canal per root in the multirooted tooth. The canal terminates at the root apex into an aperture, the apical foramen, through which blood vessels and nerves enter or exit the tooth. The portion of the tooth that is visible in the oral cavity is the crown and is covered by enamel, a tough, inorganic substance composed mainly of hydroxyapatite. The part of the tooth that is obscured by the gingiva is called the root and is covered by cementum, a thin layer of bonelike

material similar in composition to dentin. The tooth is anchored into the socket, a space in the alveolar bone, by the periodontal ligament. The collagenous fibers of this ligament insert into the alveolar bone and into the root cementum.[3,4]

The blood supply of the teeth is generally derived either directly or indirectly from the maxillary artery, one of two terminal branches of the external carotid artery. Mandibular teeth are supplied via the inferior dental artery, which branches off of the maxillary artery in the infratemporal region. The maxillary premolars and molars receive their blood supply via the posterior superior dental artery, arising from the maxillary artery in the pterygopalatine fossa. The remaining maxillary dentition receives its arterial flow from the middle superior dental artery (inconsistent) and the anterior superior dental artery, both of which arise from the infraorbital artery, a branch of the maxillary artery.[5,6]

Dental veins flow into larger apical veins and then on to larger venous channels. Mandibular venous outflow occurs via the inferior dental veins. These veins may then drain either anteriorly, through the mental foramen to join the facial vein, or posteriorly, through the mandibular foramen into the pterygoid plexus of veins in the infratemporal fossa. Maxillary dental veins may also drain anteriorly into the facial vein or posteriorly into the pterygoid plexus.[5,6]

Lymphatic vessels draining the incisors and canines pass anteriorly, those draining the molars run posteriorly, and those draining the premolars may pass either anteriorly or posteriorly. The lymph vessels of all teeth, except for the lower incisors, drain into the submandibular ipsilateral lymph nodes. The lower incisor lymph vessels flow into the ipsilateral submental lymph nodes. Occasionally, lymph from the molars may pass directly into the jugulodigastric lymph nodes.[6]

The dental pulp contains both sensory and vasomotor nerves. The vasomotor nerves are sympathetic (autonomic) nerves that supply the arterial smooth muscle and thereby regulate blood flow to the tooth. The sensory nerves of the dental pulp are branches of the maxillary and mandibular divisions of the trigeminal nerve. The mandibular molars and premolars are innervated by the inferior dental nerve. The incisors and canines are innervated by the incisive nerve, a branch of the inferior dental nerve. Maxillary molars receive innervation via the posterior superior dental nerve, a branch of the maxillary nerve. The maxillary premolars are innervated by the middle superior dental nerve; the incisors and canines are innervated by the anterior superior dental nerve, both of which are branches of the infraorbital nerve.[6,7]

As many as 50 to 200 different species of microorganisms have been isolated from the oral cavity.[8] However, probably no more than 20 to 30 species are indigenous to this area.[2,8] The human upper airway is normally populated by a mixture of aerobic, facultative, and anaerobic species in numbers to 10^{11} organisms/gram of fluid or collected material in certain locations.[9] The hemolytic streptococci and *Staphylococcus aureus* are the

most common aerobic microorganisms associated with oral infections, whereas *Bacteroides melaninogenicus*, *B. fragilis*, *B. oralis*, peptostreptococci, *Clostridium* spp., and *Veillonella* spp. are the most common anaerobes linked to odontogenic infections.[2] Other species commonly isolated from the oral cavity include various strains of *Neisseria*, *Actinomyces*, *Peptococcus*, *Fusobacterium*, *Haemophilus*, coliform organisms, and *Propionibacterium*.[8, 13]

The emergency physician should be adept at performing a careful, thorough dental examination. The examination begins with a detailed history emphasizing the presenting complaint, location and duration of the problem, exciting factors that elicit pain or alter the condition, any history of trauma, as well as any additional related symptomatology.[1,14] The intraoral examination begins with an evaluation of the degree of mandibular opening.[1] The physical examination should be performed under adequate lighting to ensure proper visualization of all the structures in the oral cavity. Careful digital examination will assess the mobility of a tooth, and bimanual examination will reveal the degree of fluctuation, if any, of a swollen area and thereby ultimately help determine whether therapy will be surgical drainage, antibiotics, or a combination of both.[14] Percussion of teeth thought to be infected, as well as hot and cold stimuli, will help identify infected teeth. The remainder of the oral cavity should likewise be scrutinized for any nondental pathology.[1] Dental films or panoramic radiographs, if available, can offer valuable additional information regarding foci of infection.

DENTAL CARIES

Dental caries is a disease of the calcified tissues of the teeth characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth.[15] It is the most prevalent chronic disease affecting humans, and its frequency is increasing because of enhanced life expectancy and dentition retention.[15,16] Caries formation involves a complex interplay of factors including host susceptibility, a cariogenic diet (e.g., carbohydrates), and causitive bacteria.[17] Caries can be initiated by a variety of microorganisms, including *Streptococcus mutans*, *S. salivarius*, *S. faecalis*, *Actinomyces* spp., and *Lactobacillus* spp. *S. mutans* is by far the most virulent organism in this respect.[18] Theories regarding the etiology of caries are multiple and beyond the scope of this chapter. The reader is referred to an excellent review of prevailing theories by Shafer, et al.[15] There is general agreement, however, that enamel caries begins beneath dental plaque. This plaque is an aggregation of bacteria, extracellular bacterial products, and certain salivary constituents that adhere firmly to the teeth. The accumulation of plaque allows continual contact between acidogenic microorganisms and the enamel surface.[18] Plaque tends to form on teeth not cleansed routinely and those that are protected from the action of tooth

brushing, namely, the occlusal pits and fissures of the molars and bicuspids.[15,19,20] The nature and pathogenicity of this plaque are influenced by salivary and dietary components, oral hygiene, and local and host factors.[21] The bacteria in the plaque form acids that result in demineralization of the enamel, creating a portal of entry into the internal milieu of the tooth, allowing for further destruction and microbial invasion of internal tooth structures.[15,18]

Patients do not seek treatment in the emergency department for caries, per se but rather when further involvement of tooth structures has occurred (e.g., pulpitis, periapical abscess, etc.). Because the diagnosis of dental caries on occlusal surfaces of the teeth is usually made by visual inspection,[18] the emergency medicine physician generally diagnoses caries as an incidental finding during examination of a patient's oropharynx. If a carious lesion is found, the physician should palpate the affected tooth (or teeth) for tenderness or tooth mobility, which would indicate further progression of the disease process. Adjacent soft tissues should be observed and palpated, as well, for evidence of any swelling or fluctuance indicating disease extension.

The role of the emergency medicine physician in carious disease of the enamel is to notify the patient of its existence and to counsel the patient regarding its deleterious effects. It is important to stress to the patient that dental caries is an infectious process and, if not arrested, can lead to potentially serious, or even life-threatening, illness. Finally, the emergency medicine physician should serve as a resource of private dental practitioners and dental clinics for those who do not have these references available.

PULPITIS

The dental pulp is a loose connective tissue, located within the dentin of the tooth, whose primary function is to form, nurture, and repair the dentin.[22] The pulp contains sensory (afferent) nerves, which are branches of the maxillary and mandibular divisions of the fifth cranial nerve (trigeminal), and vasomotor (efferent) nerves, which supply the muscular walls of the arteries and arterioles of the teeth.[7] Most frequently, pulpal infection occurs as a result of carious exposure to the pulp, although physical and chemical injury to the teeth may result occasionally in pulpal necrosis and infection.[23] This carious exposure generally begins in the pits and fissures of the occlusal surfaces of molars and premolars. Invasion of the pulp may then become a localized or generalized pulpitis.[24]

Pain brings the patient with pulpitis to the attention of the emergency medicine physician. The pain may be moderate or so severe that it has been deemed second only to that of renal colic in its intensity.[23] The pulp is somewhat analagous to the brain in that both are housed within calcified, unyielding structures (enamel and bone, respectively). Any increase in the

inflammatory reaction within the tooth increases the intrapulpal pressure and when the threshold of excitability of peripheral sensory receptors is reached, pain is perceived.[7] By the time that dental caries has extended far enough to cause pain, it is usually too close to the pulp to prevent pulpal death.[25] The emergency medicine physician should be aware that pulpitis may occur without pain. If the portal of entry to the pulp is large enough to allow spontaneous drainage, no further increase in pulpal pressure may occur and the patient may be asymptomatic.[7] If drainage of the pulp is obstructed, rapid progression with pulpal necrosis occurs.[24]

Pulpitis has been divided into acute and chronic forms. Acute pulpitis generally occurs in a tooth with a large carious lesion or restoration. Pain may be quite severe and is influenced by thermal changes and assumption of the supine position.[15] As intrapulpal pressure continues to increase, the pain may become excruciating and the patient must seek therapy.[7] If a large, open cavity exists, intrapulpal pressure may not build up. In such cases, the pain is perceived as a dull, throbbing ache, and the tooth still retains sensitivity to thermal changes.[15] Chronic pulpitis signifies the emergence of the proliferative phase of the pulpal inflammatory cycle and is characterized by the formation of granulomatous tissue in the areas peripheral to the exudative zones.[7] Although occasionally it may occur through quiesence of a previous acute pulpitis, most often it develops as the chronic type of disease from the outset. Pain is not a prominent feature, although an intermittent, dull ache is common. Chronic pulpitis produces less reaction to thermal change than does acute pulpitis.[15]

The emergency medicine physician should identify the cause of pain, determine the extent of involvement (if any) of surrounding structures, ascertain any degree of systemic involvement, offer relief of symptoms, and schedule appropriate therapy, either tooth extraction or root canal.[15] The emergency medicine physician should counsel the patient to schedule an appointment with a dental practitioner within the next 24 hours for definitive care.

Antibiotic therapy is considered an adjunct to therapy and not a substitute for removing the cause of infection.[26] In otherwise healthy individuals, antibiotics are used as a precaution when any of the following are present: acute cellulitis, fever, or malaise.[27] Penicillin is still considered the empiric drug of choice for odontogenic infections because it demonstrates bactericidal action and is active against most oral streptococci and anaerobic bacteria. It is administered orally in a dose of 500 mg every 6 hours.[26] Erythromycin, in a dose of 250 mg every 6 hours, is an alternative drug in the penicillin-allergic patient, although European investigators note that many oral anaerobic microorganisms are resistant to this drug, including a certain proportion of β-lactamase-producing anaerobes.[28] Clindamycin is an effective alternative to erythromycin in penicillin-allergic persons, although this drug's association with pseudomembranous collitis has limited its widespread use.[26-28]

Pain relief may be obtained by regional dental block using a long-acting

anesthetic such as bupivacaine.[29] Oral analgesics include nonsteroidal anti-inflammatory agents, such as ibuprofen or naproxen, but generally, narcotic agents, such as codeine or pentazocine, are required.[27] If a narcotic is needed, only enough medication should be prescribed to ensure pain control for no more than a period of 2 days during which time the patient should be evaluated by a dental practitioner. Limiting the amount of narcotics prescribed will assist in lessening the medical profession's contribution to drug addiction, as drug abusers will often feign tooth pain to supplement their habit. Under no circumstances should a patient suspected of narcotic abuse receive parenteral narcotics in the emergency department, as this will only serve to reinforce subsequent visits for obtaining narcotics. Rather, suspected drug abusers should be offered a regional dental anesthetic block for relief of pain and should be discharged with a prescription for a nonsteroidal anti-inflammatory drug, as well as referral to a dentist or dental clinic.

PERICORONITIS

Pericoronitis is an infection of the pericoronal soft tissues that partially overlie the crown of the tooth, most frequently involving partially erupted third molars (wisdom teeth).[30,31] The mucosal flap, or operculum, covering these teeth creates ideal conditions for pathogenic oral flora to flourish, giving rise to infection.[32] A pocket is formed between the tooth and the overlying soft tissues of the operculum, and microorganisms and food debris become entrapped in this space so that a local infection is initiated.[30] Trauma from opposing teeth during the acts of speaking and mastication triggers or exacerbates this condition.[32] The organisms most commonly isolated in cases of pericoronitis include anaerobic cocci, fusobacteria, and *Bacteroides* spp.[30]

Pericoronitis is encountered most commonly in teenagers and young adults, although it may occur in any age group.[32,33] Patients present with complaints of local pain, difficulty in swallowing, and trismus.[32] The trismus is caused by posterior spread of the infection to the pterygomandibular space.[31] Any infectious condition that involves the muscles of mastication can cause trismus, but the associated findings of an operculum, tenderness around the unerupted or impacted tooth, and regional lymphadenitis should offer sufficient evidence for the diagnosis.[33]

The emergency medicine physician should first determine the degree of trismus, as this may be indicative of the extent of spread of the infection. Careful visualization of the oral cavity, under adequate lighting, will reveal the obviously swollen pericoronal tissues. Gentle palpation of these tissues will elicit tenderness and a small amount of exudate.[32]

The emergency management of pericoronitis includes inserting a periodontal probe or incising and draining the abscess, taking care to avoid injuring the internal carotid artery, which occasionally shifts position anteriorly because of associated tissue swelling.[32] The patient should be

instructed to irrigate the area with a saline solution, povidone-iodine solution, or hydrogen peroxide for 5 minutes every hour. A Water-Pik appliance, if available to the patient, is an excellent means of irrigating the pericoronal areas.[35] In most cases, antibiotics must be administered to prevent spread of infection.[30] Penicillin in a dose of 500 mg every 6 hours is the empiric drug of choice. Penicillin-allergic individuals may be treated with erythromycin 250 mg every 6 hours, or metronidazole in a dose of 200 mg four times daily.[30,32] The patient should be referred to a dentist within the ensuing 24 hours for further evaluation and management.

PERIAPICAL ABSCESS

A periapical abscess is an accumulation of pus in the dentoalveolar region surrounding the apex of the tooth.[20] It is one of the most commonly encountered dental infections, occurring in all age groups.[33,35] A periapical abscess generally stems from a nonvital pulp and the involved tooth usually has a deep carious lesion or a restoration, but may be intact.[33,37] Its etiology is multifactorial with viable bacteria, bacterial products, and altered host tissue all capable of initiating and propagating the disorder.[38] The abscess itself may represent an attempt on the part of the body's immune system to protect the host from foreign agents by walling off the area of involvement.[39] The microbial composition of periapical abscesses is predominantly anaerobic with, on the average, less than five unique species per abscess.[38,40] Most commonly, anaerobic cocci, fusobacteria, and *Bacteroides* spp. are isolated from periapical abscesses.[30]

The patient with a periapical abscess comes to the emergency department because of significant pain of acute onset that occurs when periapical abscesses are confined to the osseous structures during the early period of abscess formation.[41,42] The emergency medicine physician should examine the oral cavity under adequate lighting and look for any associated findings of swelling and reddening of the overlying skin, as well as elevation of the tooth in its socket.[37] The gingival lesion resulting from a periapical abscess most frequently appears as a parulis, or gumboil, which indicates the drainage point of a fistula from the apex of a tooth root. A parulis may appear as a red papule, nodule, or pustule of varying size.[33] The affected tooth should be percussed because, although teeth with periapical abscesses generally are extremely sensitive to percussion, they may not exhibit pain if the abscess has progressed to fistula formation with spontaneous drainage.[20,33] Furthermore, the mucosa overlying the root tips should be palpated with the little finger as acute apical abscesses with unnoticed subtle swelling are compressed in this manner and will elicit a painful response, thereby aiding in the diagnosis.[41] If the periapical abscess is severe, the patient may be febrile. Dental or panoramic radiographs, if available, may demonstrate either a normal picture or a diffuse area of radiolucency not always restricted to a single tooth.[35,42]

The emergency management of a periapical abscess is dependent on the patient's presenting condition. If the infection is localized and the patient manifests no systemic effects of the infection, the patient may be started on antibiotic therapy and referred to a dentist within the ensuing 24 hours. Penicillin at a dose of 500 mg orally every 6 hours is the drug of choice. Alternative drugs in the penicillin-allergic patient include erythromycin, 250 mg every 6 hours; clindamycin, 150 mg every 6 hours; or metronidazole, 200 mg every 6 hours.[27,30] If in-house dental consultation is available, the patient should be referred immediately, as the definitive therapy of periapical abscess is drainage, which can be established through the pulp chamber or from the periapical area.[37] Patients discharged from the emergency department should be given oral analgesics, such as codeine or pentazocine, in a dose sufficient to afford relief until the patient is seen by a dentist within the next 24 hours. The emergency medicine physician should refrain from performing dental local anesthetic blocks for pain relief in the setting of a periapical abscess for fear of disseminating the infection.[29] A patient who demonstrates extension of the infection to other sites (e.g., submandibular abscess, fistula of the neck) or who appears septic should be admitted to the hospital. Intravenous antibiotic coverage should be initiated in the emergency department, and immediate oral-maxillofacial surgery consultation should be obtained.[43–45]

HERPES SIMPLEX VIRUS INFECTIONS

Herpes simplex virus (HSV) infections, types I and II, of the skin and mucous membranes are commonly observed in clinical practice.[46] The viral genome is composed of a linear, double-stranded DNA molecule with considerable homology noted between the HSV-I and HSV-II types. Viral exposure to mucous membranes or areas of abraded skin permits entry of the virus into cells.[47] During the 3- to 10-day incubation period, invasion of susceptible cells results in the subsequent replication of as many as 50,000 to 200,000 virions per cell.[32,47] Death of the host cell then occurs, in part, because of inhibition of cell macromolecular synthesis.[47] Although the exact pathogenesis of HSV infections is unknown, certain factors, such as fever, concurrent bacterial and fungal infections, stress, sun exposure, immunosuppression, trauma, debilitation, and trigeminal ganglion surgery, are capable of triggering recurrent HSV infections.[47–51] Of all individuals undergoing dental extractions, 10 to 15 percent will develop oral-labial HSV infections in a median time interval of 3 days after the procedure.[47]

HSV infections may be primary or recurrent. Gingivostomatitis and pharyngitis are the most common clinical manifestations of primary HSV-I infections, and recurrent herpes labialis is the most frequent manifestation of reactivated HSV infection.[47] Primary herpetic gingivostomatitis affects children and young adults, and over 90 percent of the population has sustained the infection at some time.[32] Most cases of HSV infections are

asymptomatic or minimally symptomatic.[32,47] Although any area of the skin or mucous membranes may be affected, oral herpetic infections generally involve the hard and soft palate, tongue, buccal mucosa, gingiva, and the vermillion border of the lips.[32,46] Herpetic esophagitis is occasionally noted in an otherwise healthy individual, although this manifestation is generally observed in immunosuppressed or debilitated persons.[52]

Clinical signs and symptoms of primary herpetic gingivostomatitis may include fever, malaise, myalgias, cervical adenopathy, dysphagia, anorexia, and irritability.[32,47] Clusters of small vesicles erupt within the oral cavity and may subsequently coalesce, ulcerate, and leave extensive areas of bleeding, denuded tissue.[32] Most commonly, exudative or ulcerative lesions of the posterior pharynx, tonsillar pillars, or both are present.[47] Recurrent herpetic gingivostomatitis differs from the primary variety in that it is usually painless and the recurrent lesions generally occur on the attached gingiva and hard palate, sparing the mucosa itself.[32] The reason for this pattern may be that the saliva contains a substance that reduces the susceptibility of cells to HSV infections.[53] Because the mucosa of the gums and hard palate are ketatinized and the buccal and labial mucosa are not, salivary inhibiting substances may more easily penetrate the nonkeratinized cells and thus prevent epidermal cell infection of the buccal and labial mucosa.[50]

HSV labialis presents as clusters of small vesicles on an erythematous base. These lesions demonstrate a distinctive distribution pattern about the vermillion border of the lips, but may subsequently extend to other areas of the face.[32] The vesicles eventually coalesce, break down, and dessicate, resulting in crust formation.[54]

The differential diagnosis of ulcerative lesions of the lips and oral cavity includes infectious disorders such as varicella, herpangina, hand-foot-and-mouth disease, and *Mycoplasma pneumoniae* infections. Noninfectious disorders include Stevens-Johnson syndrome, recurrent aphthous ulcers, Behcet's syndrome, and Reiter's syndrome.[32,47,55]

The diagnosis of HSV infections may be ascertained from tissue cultures of fluid from fresh vesicles, but results will not be obtainable for at least 2 days.[46] Fresh lesions may be unroofed and scrapings of the base of the lesion stained with a Tzank smear to identify multinucleated giant epithelial cells. However, this test is neither highly reliable nor specific for HSV infections.

The therapy of HSV oral-labial infections is quite straightforward. The patient should get adequate rest and hydration and should treat any fever with antipyretics such as acetaminophen. Aspirin should probably be avoided inasmuch as its use in varicella has been associated with the development of Reye's syndrome and varicella may closely mimic HSV infections. Oral pain may be relieved by the use of mouth rinses composed of diphenhydramine hydrochloride elixir or viscous lidocaine hydrochloride, with the former preferred because it does not impair the gag reflex.[32] Bland salves may be applied to lip lesions, or if secondary bacterial infection of the lesions is suspected, antibiotic ointment may be applied. It is very important