INTRODUCTION — Odontogenic infections, consisting primarily of dental caries and periodontal disease (gingivitis and periodontitis), are common and have local (eg, tooth loss) and, in some cases, systemic implications. In the United States, it is estimated that 25 percent of adults over the age of 60 have lost all their teeth (edentulism), approximately one-half from periodontal disease and one-half from dental caries [1,2].

In addition to producing pain and discomfort, odontogenic infections can extend beyond natural barriers and result in potentially life-threatening complications, such as infections of the deep fascial spaces of the head and neck. (See "Deep neck space infections").

Periodontal infection can also be associated with a number of systemic disorders. These include fever of unknown origin, bacteremic seeding of heart valves and prosthetic devices, preterm birth of low birth weight children, and an increased risk for coronary heart disease and cerebrovascular events.

The complications, diagnosis, and treatment of odontogenic infections will be reviewed here. The epidemiology, pathogenesis, and clinical manifestations of these infections are discussed separately. (See "Epidemiology, pathogenesis, and clinical manifestations of odontogenic infections" and "Gingivitis and periodontitis in adults: Classification and dental treatment").

COMPLICATIONS — Suppurative odontogenic infections may extend to potential fascial spaces in the orofacial area (orofacial space infections) or deep in the head and neck (peripharyngeal space infections). The latter complication is often life threatening. (See "Deep neck space infections").

In addition, odontogenic infections may spread contiguously to cause osteomyelitis of the jaw or hematogenously to produce systemic illness.

Orofacial space infections — Superficial orofacial space infections can involve the buccal, submental, masticator, canine, and infratemporal spaces. The location of the space infection can assist in recognizing the underlying infected tooth (table 1).

If unrecognized and untreated, these infections are potentially serious since they can spread contiguously into the deeper fascial spaces of the head and neck, such as the submandibular, lateral pharyngeal, and retropharyngeal spaces, or into the carotid sheath. (See "Deep neck space infections").

The spread of infection into the deeper fascial spaces of the head and neck is suggested by the finding of trismus (the inability to open the jaw). Trismus indicates pressure or infection of the muscles of mastication (the masseter and the pterygoids) or involvement of the motor branch of the trigeminal nerve. Such infections can spread intracranially to cause purulent meningitis or subdural empyema and caudally to result in aspiration, airway obstruction, or fatal necrotizing mediastinitis [3].

Buccal and submental spaces — Infections arising from mandibular or maxillary bicuspid and molar teeth tend to extend in a lateral or buccal direction. The relation of the root apices to the origins of the buccinator muscle determines whether infection will exit intraorally into the buccal vestibule or extraorally into the buccal space (figure 1). Infection of the buccal space is readily diagnosed because of marked cheek swelling with minimal trismus and systemic symptoms.

Involvement of a mandibular incisor can perforate below the mentalis muscle and present as a submental space infection. The chin appears grossly swollen and is firm and erythematous.

Masticator spaces — Masticator spaces consist of the masseteric, pterygoid, and temporal spaces, all of which are well differentiated but intercommunicate with each other as well as with the buccal, submandibular, and lateral pharyngeal spaces (figure 2). Infection of the masticator spaces arises most frequently from molar teeth, particularly...
the third molars (wisdom teeth). The clinical hallmark of masticator space infection is trismus and pain in the area of the body or ramus of the mandible.

Swelling may not be a prominent finding, especially in the masseteric compartment, since the infection is beneath large muscle masses that can obscure or prevent clinically apparent swelling. When present, swelling tends to be brawny and indurated, suggesting the possibility of cervicofacial actinomycosis or mandibular osteomyelitis. (See "Cervicofacial actinomycosis".)

Infection of the deep temporal space usually originates from involvement of the posterior maxillary molar teeth. Little external swelling is observed early in the course; if present, it usually affects the preauricular region and an area over the zygomatic arch. As infection progresses, the cheek, eyelids, and whole side of the face may be involved. Infection may extend directly into the orbit via the inferior orbital fissure and produce proptosis, optic neuritis, and abducens nerve palsy.

**Canine and infratemporal spaces** — Involvement of the maxillary incisors and canines may result in a canine space infection, which manifests as dramatic swelling of the upper lip, canine fossa, and frequently the periorbital tissues. Pain is usually moderate, and systemic signs are minimal. Occasionally, direct extension of infection into the adjoining antrum leads to purulent maxillary sinusitis.

The infratemporal space is bounded medially by the lateral plate of the pterygoid process and the pharynx, posteriorly by the parotid gland, anteriorly by the maxilla, and superiorly by the roof of the infratemporal fossa, adjacent to which is the inferior orbital fissure (figure 2).

Primary infections of the infratemporal fossa usually originate from involvement of the posterior maxillary molar teeth, particularly the third molar. Injection of local anesthetic into this area for a dental restoration may predispose to infection.

Clinically, marked trismus and pain are present but very little swelling is observed early in the course. Late manifestations are similar to those of temporal space infections, including extension into the orbit through the inferior orbital fissure. In addition, if the infection extends internally, it can involve an area close to the lateral pharyngeal wall, resulting in dysphagia.

**Osteomyelitis of the jaw** — Odontogenic infections can spread contiguously to cause osteomyelitis of the jaw. The mandible is much more susceptible to osteomyelitis than the maxilla, primarily because the cortical plates of the former are thin and vascular supply to the medullary tissues is relatively poor. Despite this, osteomyelitis secondary to odontogenic infection is relatively uncommon. When it does occur, there is usually a predisposing condition, such as compound fracture, irradiation, diabetes mellitus, or steroid therapy [4].

With initiation of infection, the intramedullary pressure markedly increases, further compromising blood supply and leading to bone necrosis. Pus travels through the Haversian and perforating canals, accumulates beneath the periosteum, and elevates it from the cortex. If pus continues to accumulate, the periosteum is eventually penetrated, and mucosal or cutaneous abscesses and fistulae can develop. Areas at greatest risk of perforation in the mandible are the lingual aspect in the region of molar teeth and anteriorly on the buccal aspect [3].

As the inflammatory process becomes more chronic, granulation tissue is formed. Spicules of necrotic and nonviable bone may become either totally isolated (sequestrum) or encased in a sheath of new bone (involucrum).

Severe mandibular pain is a common symptom of jaw osteomyelitis and can be accompanied by anesthesia or hypoesthesia on the affected side. In protracted cases, mandibular trismus may develop.

A clinical variant of osteomyelitis of the jaw is Garre’s chronic sclerosing osteomyelitis or proliferative periostitis [5]. This entity usually occurs in children and young adults following a periapical infection of the mandibular first molar. It is a nonsuppurative form of osteomyelitis characterized by a localized, hard, nontender swelling over the mandible. On radiographic imaging, the newly formed periosteal bone appears as layers outside the cortex, giving a characteristic "onion skin" appearance [6]. Actinomycosis and radiation necrosis are two common causes of this form of osteomyelitis of the jaw [4,7].

A related clinical entity, characterized by exposed necrotic bone in the maxillofacial region, is osteonecrosis of the jaw associated with long-term administration of bisphosphonates for the treatment of osteoporosis or cancer. This is discussed in detail separately. (See "Risks of therapy with bone antiresorptive agents in patients with advanced
Hematogenous dissemination — In addition to contiguous spread, odontogenic infections can disseminate hematogenously to seed native or prosthetic heart valves, joints, or other devices [8]. Although bacteremia can occur following almost all types of dental manipulations, including flossing, scaling, even tooth brushing and chewing hard candy, such episodes are usually transient and inconsequential in healthy individuals [9]. In contrast, the bacteremia in patients with dental caries and periodontal disease tends to be more frequent and sustained [10] and is a potentially important cause of infective endocarditis in elderly patients, who may be more susceptible due to age-related degenerative or calcified valvular heart disease [11]. (See “Epidemiology, risk factors, and microbiology of infective endocarditis”.)

Dental sources of bacteremia in elderly adults are of increasing concern for those undergoing prosthetic heart valve implantation or prosthetic joint replacement. It has been recommended that routine dental assessment be performed in all patients undergoing valve surgery and that appropriate therapeutic interventions be initiated whenever possible before valve implantation [12]. Recommendations regarding the use of antimicrobial prophylaxis for the prevention of bacterial endocarditis in selected patients are presented separately. (See “Antimicrobial prophylaxis for bacterial endocarditis”.)

The risk of prosthetic joint infection as a consequence of bacteremia following dental procedures has been controversial. This is also discussed in detail separately. (See “Epidemiology and prevention of prosthetic joint infections”, section on ‘Dental procedures’.)

Association with cardiovascular disease — An association between poor oral health and chronic periodontitis with coronary and cerebrovascular disease has been well established epidemiologically [13]. The relevant data and possible mechanisms are discussed separately. (See “Epidemiology, pathogenesis, and clinical manifestations of odontogenic infections”, section on ‘Association with cardiovascular risk’.)

DIAGNOSIS — Obtaining appropriate material for culture and processing it properly are important in the diagnosis of odontogenic infections. In addition, imaging techniques to assess the extent of involvement are essential.

Specimen collection and processing — One of the difficulties in defining etiologic agents for odontogenic infections is the presence of normal resident oral flora. For closed space infections, it is imperative that the normal oral flora be excluded during specimen collection in order to interpret culture results. Needle aspiration of loculated pus by an extraoral approach is desirable, and specimens should be transported immediately to the laboratory under anaerobic conditions.

However, contamination by the resident oral flora is inevitable for intraoral lesions. In this setting, direct microscopic examination of stained smears often provides more useful information than culture results from surface swabs. Gram stain and acid-fast stains for bacteria and potassium hydroxide preparations for fungi should be routinely performed on these specimens. Tissue biopsies should be routinely examined for histopathologic evidence of acute or chronic inflammation and infection. Specific microbial agents, including certain bacterial, mycobacterial, fungal and viral infections, can sometimes be detected by immunofluorescence or polymerase chain reaction.

Patients with chronic osteomyelitis often have soft tissue swelling and draining fistulas. Aspirates from the adjacent soft tissue swelling may be valuable. In contrast, cultures from sinus tracts can be misleading, since there is communication with the external environment. Thus, microorganisms that are isolated could represent colonization of the sinus tract rather than the organism responsible for the infected bone. Bone biopsies for histopathology and culture are often required for definitive diagnosis. (See “Overview of osteomyelitis in adults”.)

Imaging techniques — The choice of imaging technique varies with the clinical setting [14]:

- A panoramic or periapical radiograph may reveal the extent of advanced periodontitis or the presence of periapical abscess.
- Computed tomography (CT) is particularly sensitive for osseous structures and remains the imaging modality of choice for assessment of most odontogenic infections [14]. Dental amalgam used in fillings may cause metallic streaking artifact on CT and obscure the region of interest. This problem can be minimized with multidetector CT imaging.
CT and particularly magnetic resonance (MR) have become the preferred imaging modalities for the localization of deep fascial space infections of the head and neck. (See "Deep neck space infections", section on 'Imaging'.)

A lateral radiograph of the neck may demonstrate compression or deviation of the tracheal air column, or the presence of gas within necrotic soft tissues. In retropharyngeal infections, lateral radiography of the cervical spine or CT scanning can help determine if the infection is in the retropharyngeal space or the prevertebral space. The former suggests an odontogenic source, while the latter suggests involvement of the cervical spine.

Technetium bone scanning, used in combination with gallium or indium-labeled white blood cell scanning, is particularly useful for the diagnosis of acute or chronic osteomyelitis and for the differentiation of infection or trauma from malignancy. In acute osteomyelitis, both the bone scan and gallium scan are likely to be positive. In chronic osteomyelitis, the technetium scans may or may not be positive, while gallium or indium-labeled scans are often negative. (See "Approach to imaging modalities in the setting of suspected osteomyelitis".)

Similarly, bone neoplasms can be associated with a positive technetium scan but negative gallium or indium scan. Although the scintigraphic findings may be suggestive, only bone biopsy will definitively differentiate infection from malignancy. (See "Approach to imaging modalities in the setting of suspected osteomyelitis".)

**THERAPEUTIC CONSIDERATIONS** — Meticulous attention to oral hygiene is the most important strategy for effective control of supragingival and subgingival plaque that, in turn, is essential for both caries prevention and the treatment of periodontitis. Individuals with physical or mental limitations who cannot adequately perform oral hygiene by themselves should receive daily oral hygiene by care providers. More frequent visits to dentists and use of electric toothbrushes should also be considered in these patients. Regular check-ups and prompt restorative care by dental professionals should be actively promoted.

With the recognition of the microbial specificity of odontogenic infections, topical antiseptics and systemic antibiotics have played an increasingly important role in the control and treatment of both dental caries and periodontal disease (table 2). (See "Epidemiology, pathogenesis, and clinical manifestations of odontogenic infections", section on 'Microbial specificity in odontogenic infections'.)

The need for dental extractions has been reduced considerably by the availability of improved dental restorative materials, such as bonding and fluoride-releasing agents, as well as improved restorative care [15].

**Dental caries** — Caries management with restorative therapy (eg, fillings) is the preferred therapeutic approach in many countries [15]. However, restorative therapy must be combined with preventive measures, since restorations have relatively short durability and new caries may form at the margins of restorations if the causes of the disease persist [1,15]. Caries prevention is discussed below. (See 'Prevention' below.)

**Pulpitis** — Pulpitis, inflammation of the dental pulp, occurs when progression of dental caries exposes the dental pulp, leading to infection (figure 3). The early and dominant symptom of acute pulpitis is a severe toothache that can be elicited by thermal changes, especially cold drinks. (See "Epidemiology, pathogenesis, and clinical manifestations of odontogenic infections", section on 'Pulpitis and periapical abscess'.)

Pulpitis can be classified as reversible or irreversible. Reversible pulpitis occurs when caries encroach on the pulp and is associated with mild inflammation of the pulp. Irreversible pulpitis refers to ongoing inflammation within the pulp chamber with rapid buildup of pressure, occlusion of blood vessels at the apical foramen, ischemia, and necrosis of the pulp tissue.

Irreversible pulpitis is characterized by acute and intense pain and is one of the most frequent reasons that patients seek emergency dental care. Apart from removal of the tooth, the customary approach to relieving the pain of irreversible pulpitis is by drilling into the tooth, removing the inflamed pulp (nerve), and cleaning the root canal. A minority of dentists begins with a trial of antibiotics and analgesics, although there is no proof of benefit from this approach.

A 2013 literature review found only one randomized controlled trial that compared treatment with systemic antibiotics to placebo in irreversible pulpitis [16]. In this trial, 40 patients were treated with analgesics and randomly assigned to either penicillin or placebo [17]. No operative endodontic treatment was performed. Although the study was limited by small sample size, no significant difference in pain intensity or use of analgesics was noted between the two groups. Photoactivated disinfection has been evaluated as an aid to mechanical irrigation in eradicating bacterial counts within the root canal during endodontic treatment [18]. It was highly effective in eliminating endodontic pathogens from the
root canal [19,20]. It remains to be determined whether this approach provides incremental benefit compared to conventional endodontic treatment in symptom relief or disease progression.

**Acute gingivitis** — Acute simple gingivitis rarely requires systemic antimicrobial therapy [21,22]. Chlorhexidine 0.12 percent oral rinse can be used in most cases. Exceptions include patients with rapidly advancing disease, severe pain, or HIV infection in whom systemic therapy is indicated. Possible regimens include penicillin plus metronidazole, amoxicillin-clavulanate, ampicillin-sulbactam, or clindamycin (table 2).

Acute necrotizing ulcerative gingivitis, also known as Vincent's angina or trench mouth, should be treated with systemic antibiotics, such as metronidazole, amoxicillin-clavulanate, ampicillin-sulbactam, or clindamycin (table 2) [21,23].

**Periodontitis** — Due to microbial specificity in various forms of periodontitis, certain types of severe periodontitis are amenable to systemic antimicrobials in conjunction with mechanical debridement (scaling and root planing) (table 2) [24]. This approach has often obviated the need for radical surgical resection of periodontal tissues.

The efficacy of antimicrobial therapy was evaluated in a double-blind trial in which 94 patients with advanced periodontitis were randomly assigned to metronidazole (500 mg PO twice daily) or doxycycline (100 mg daily) or placebo for one or two weeks in conjunction with rigorous mechanical debridement of the root surfaces [25]. Antimicrobial therapy reduced the need for radical surgery by 81 percent compared to placebo.

The efficacy of local antibiotic therapy in conjunction with scaling and root planning in chronic periodontitis has also been evaluated [22,26]. Adjunctive local antibiotics have been shown to significantly reduce pocket depth or degree of periodontal detachment [26]. Effective agents include 2 percent minocycline spheres (Arestub), 10 percent doxycycline hyclate extended release liquid (Atridox), and 25 percent metronidazole gel (Elyzol) [22,26,27]. These agents release controlled amounts of the antibiotic beneath the gum and are used in conjunction with scaling and root planing to reduce pocket depth in adult periodontitis.

In localized juvenile periodontitis, systemic tetracycline therapy directed against Actinobacillus actinomycetemcomitans (a HACEK infection) and combined with local periodontal treatment has yielded excellent results [22]. Unfortunately, the administration of tetracycline or doxycycline to children eight years of age or younger can cause staining of the permanent dentition and is not generally recommended. Furthermore, tetracycline resistance among periodontal pathogens has been increasingly recognized [28].

The routine use of systemic antimicrobials to prevent postoperative infections following oral and/or periodontal surgery in a healthy host remains controversial [29]. In the setting of third molar extractions, a single dose of intravenous (IV) penicillin was shown to significantly reduce the incidence of surgical site infection in a double-blind placebo-controlled trial (8.5 versus 0 percent) [30].

**Suppurative odontogenic infections** — The most important therapeutic modality for pyogenic odontogenic infections is surgical drainage and removal of necrotic tissue. Needle aspiration by the extraoral route can be particularly helpful both for microbiologic sampling and for evacuation of pus. The need for definitive restoration or extraction of the infected tooth, the primary source of infection, is usually readily apparent. Deep periodontal scaling and endodontic treatments with root filling is required in most instances.

Effective surgical management requires a thorough understanding of the most likely anatomic routes of spread. The neighboring potential fascial spaces should be carefully and systematically surveyed. The optimum timing for incision and drainage is equally important. Premature incision into an area of cellulitis in an ill-conceived search for pus can disrupt the normal physiologic barrier and cause extension of infection.

**Antibiotic therapy** — Antibiotic therapy can halt the local spread of infection and prevent hematogenous dissemination. Antimicrobial agents are generally indicated if fever and regional lymphadenopathy are present, or when infection has perforated the bony cortex and spread into surrounding soft tissue. Severely immunocompromised patients are particularly at risk for spreading orofacial infections, and empiric broad spectrum antimicrobial therapy in these patients is warranted [31].

The choice of specific antibiotics for the treatment of odontogenic infections is based more upon knowledge of the indigenous organisms that colonize the teeth, gums, and mucous membranes, as well as specific cariogenic and periodontopathic pathogens associated with clinical disease, rather than upon the results of culture and susceptibility
testing [21,22]. Beta-lactamase production among oral anaerobes, particularly pigmented Prevotella spp and Fusobacterium spp, is increasingly recognized, and treatment failure with penicillin alone has been well documented [32]. Thus, penicillin monotherapy is no longer recommended.

For patients with pyogenic odontogenic infections, we favor parenteral therapy initially. Ampicillin-sulbactam (3 g IV every six hours) provides extended coverage against oral anaerobes, including those that produce beta-lactamases, and is the treatment of choice (table 3). An alternative is penicillin G (2 to 4 million units IV every four to six hours) in combination with metronidazole (500 mg IV or orally every eight hours).

Although metronidazole is highly active against anaerobic gram-negative bacilli and spirochetes, it is only moderately active against anaerobic cocci and is not active against aerobes, including streptococci. As a result, it should not be used as a single agent in odontogenic infections except in acute necrotizing gingivitis and advanced periodontitis.

Penicillin-allergic patients should be treated with clindamycin (600 mg IV every eight hours). Erythromycin and tetracycline are not recommended because of increasing resistance among some strains of streptococci and their lack of optimal anaerobic activity [33,34].

In the compromised host, such as the patient with leukemia and severe neutropenia after chemotherapy, it is prudent to cover for facultative gram-negative bacilli (including Pseudomonas spp) as well, and agents with broad-spectrum activity against both aerobes and anaerobes are desirable. Appropriate regimens are described in the table (table 3).

For adult patients with mild infections and without comorbidities or signs of sepsis, it is reasonable to give an oral regimen while arranging for the patient to see a dentist or oral surgeon. In this situation, we would give amoxicillin-clavulanate 875 mg orally every 12 hours to those with normal renal function. For penicillin-allergic patients, we would give clindamycin 450 mg orally every 8 hours.

**Oral step-down therapy and duration of therapy** — For patients with odontogenic soft tissue infections, we continue IV therapy until there is evidence of clinical improvement, which usually occurs within three to five days following initiation of antibiotics and abscess drainage in those with severe infections and within a shorter period in those with mild to moderate infections. Patients with normal renal function can be transitioned to oral step-down therapy with amoxicillin-clavulanate 875 mg orally twice daily once such improvement has occurred. For penicillin-allergic patients, we give clindamycin 450 mg orally three times daily. Antibiotics should be continued until local inflammation has resolved completely, typically for a total of 7 to 14 days.

**Osteomyelitis** — Treatment of osteomyelitis of the jaw is complicated by the presence of teeth and persistent exposure to the oral environment. Antibiotic therapy needs to be prolonged, often for weeks to months.

Adjuvant therapy with hyperbaric oxygen, if available, may hasten the healing process, particularly when combined with surgery, but data supporting this are inconclusive [35,36]. (See “Hyperbaric oxygen therapy”.)

Surgical management, including sequestrectomy, saucerization, decortication, and closed-wound suction irrigation, is occasionally necessary. Rarely, in advanced cases, the entire segment of the infected jaw has to be resected [7].

**PREVENTION** — The single most cost-effective measure for reducing dental caries is fluoridation of public water supplies [37]. Fluoride forms a complex with the apatite crystals in dentin by replacing the hydroxyapatite group, thereby lending strength to the entire structure [37]. Fluoride also promotes remineralization of carious lesions and exerts a bacteriostatic effect.

In addition to fluoridated water, brushing two to three times daily with a fluoridated toothpaste (1000 ppm of fluoride, usually as sodium fluoride 1.1 percent or stannous fluoride 0.4 percent) effectively delivers fluoride to the tooth-plaque surface [38]. In high-risk individuals, additional fluoride therapy in the form of fluoride varnishes (22,600 ppm fluoride as five percent sodium fluoride, professionally applied three or four times a year) has been effective in caries prevention [15].

The key for the prevention and control of dental caries and advanced periodontitis is the active promotion of oral hygiene. The components of such a regimen include:

- Regular brushing with a fluoridated toothpaste and dental flossing after each meal
- Dietary counseling to reduce the ingestion of sugar-rich foods or beverages
- Use of topical fluorides and oral antimicrobial rinses, such as chlorhexidine for high-risk patients
Modification of risk factors, such as smoking cessation

Overcoming the reluctance for regular visits to dental professionals

It is also important to dispel the common misconception by physicians and patients that progressive dental caries, gingival disease, and loss of teeth is an inevitable and irreversible part of aging. The current lack of motivation of both patients and healthcare providers for regular and rigorous preventative dental care must be reversed.

A variety of other measures have been effective for caries prevention. None of these measures is routinely applied in clinical practice, but they are useful in selected patients with rampant caries [39].

- Xylitol gum, a nonfermentable five-carbon sugar, as a sugar substitute between meals
- Oral antimicrobial rinses with 0.12 percent chlorhexidine help to control dental plaque bacteria but have no proven effect on dental caries [39,40]. In addition, chlorhexidine has a bitter taste, stains the enamel and tongue, and prolonged application can promote the emergence of resistant microorganisms.
- Among topical antibiotics, only vancomycin has been shown to reduce dental caries with some degree of success in humans [39,41].

Vaccines based upon various immunogens derived from Streptococcus mutans, the principal bacterial agent associated with dental caries, have been explored [42]. However, the prospect for an effective and safe vaccine remains remote and unlikely to be available for clinical application in the near future. The need for and appropriate frequency of routine dental scaling and polishing in patients at low risk for periodontal disease is uncertain [43].

**ANTIMICROBIAL PROPHYLAXIS** — Recommendations regarding the use of antimicrobial prophylaxis to prevent bacterial endocarditis and prosthetic joint infections in selected patients undergoing dental procedures are presented separately. (See "Antimicrobial prophylaxis for bacterial endocarditis" and "Epidemiology and prevention of prosthetic joint infections", section on 'Dental procedures'.)

**SUMMARY AND RECOMMENDATIONS**

- Odontogenic infections, consisting primarily of dental caries and periodontal disease (gingivitis and periodontitis), are common and have local (e.g., tooth loss) and, in some cases, systemic implications. Suppurative odontogenic infections may extend to potential fascial spaces in the orofacial area (orofacial space infections) or deep in the head and neck (peripharyngeal space infections). The latter complication is often life threatening. Odontogenic infections can also result in osteomyelitis of the jaw or hematogenous dissemination, which may in turn cause endocarditis or prosthetic joint infections. (See 'Introduction' above and 'Complications' above.)
- For closed space infections, it is imperative that the normal oral flora be excluded during specimen collection in order to interpret culture results. Needle aspiration of loculated pus by an extraoral approach is desirable, and specimens should be transported immediately to the laboratory under anaerobic conditions. (See 'Specimen collection and processing' above.)
- The choice of imaging technique varies with the clinical setting. Computed tomography (CT) is particularly sensitive for osseous structures and remains the imaging modality of choice for assessment of most odontogenic infections. (See 'Imaging techniques' above.)
- Meticulous attention to oral hygiene is the most important strategy for effective control of supragingival and subgingival plaque that, in turn, is essential for both caries prevention and the treatment of periodontitis. (See 'Therapeutic considerations' above.)
- Pulpitis, inflammation of the dental pulp, occurs when progression of dental caries exposes the dental pulp, leading to infection (figure 3). The early and dominant symptom of acute pulpitis is a severe toothache that can be elicited by thermal changes, especially cold drinks. Apart from removal of the tooth, the customary approach to relieving the pain of irreversible pulpitis is by drilling into the tooth, removing the inflamed pulp (nerve), and cleaning the root canal. (See 'Pulpitis' above.)
- Acute simple gingivitis rarely requires systemic antimicrobial therapy. Chlorhexidine 0.12 percent oral rinse can be used in most cases. Exceptions include patients with rapidly advancing disease, severe pain, or HIV infection in whom systemic therapy is indicated. Possible regimens include penicillin plus metronidazole, amoxicillin-clavulanate, ampicillin-sulbactam, or clindamycin (table 2). (See 'Acute gingivitis' above.)
● Acute necrotizing ulcerative gingivitis, also known as Vincent's angina or trench mouth, should be treated with systemic antimicrobials, such as metronidazole, amoxicillin-clavulanate, ampicillin-sulbactam, or clindamycin (table 2). (See 'Acute gingivitis' above.)

● Certain types of severe periodontitis are amenable to systemic antimicrobials in conjunction with mechanical debridement (scaling and root planing) (table 2). This approach has often obviated the need for radical surgical resection of periodontal tissues. For chronic periodontitis, a topical antibiotic approach is used in conjunction with scaling and root planing. (See 'Periodontitis' above.)

● The most important therapeutic modality for pyogenic odontogenic infections is surgical drainage and removal of necrotic tissue. Needle aspiration by the extraoral route can be particularly helpful both for microbiologic sampling and for evacuation of pus. The need for definitive restoration or extraction of the infected tooth, the primary source of infection, is usually readily apparent. Deep periodontal scaling and endodontic treatments with root filling is required in most instances. (See 'Suppurative odontogenic infections' above.)

● In patients with pyogenic odontogenic infections, in addition to surgical management, antimicrobial agents are generally indicated if fever and regional lymphadenopathy are present, or when infection has perforated the bony cortex and spread into surrounding soft tissue. Ampicillin-sulbactam (3 g intravenously [IV] every six hours) provides extended coverage against oral anaerobes, including those that produce beta-lactamases, and is the treatment of choice in immunocompetent patients (table 3). An alternative is penicillin G (2 to 4 million units IV every four to six hours) in combination with metronidazole (500 mg IV or orally every eight hours). Penicillin-allergic patients should be treated with clindamycin (600 mg IV every eight hours). (See 'Antibiotic therapy' above.)

● The single most cost-effective measure for reducing dental caries is fluoridation of public water supplies. Other preventive measures include regular brushing with a fluoridated toothpaste, dental flossing, and reducing the ingestion of sugar-rich foods or beverages. (See 'Prevention' above.)

● Recommendations regarding the use of antimicrobial prophylaxis to prevent bacterial endocarditis and prosthetic joint infections in selected patients undergoing dental procedures are presented separately. (See "Antimicrobial prophylaxis for bacterial endocarditis" and "Epidemiology and prevention of prosthetic joint infections", section on 'Dental procedures'.)

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REFERENCES


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# Clinical features of odontogenic orofacial and peripharyngeal "space" infections

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</table>

±: minimal or occasional; +: present; ++: moderate; +++: prominent or severe.
Routes of spread of odontogenic orofacial infections

Spread occurs along planes of least resistance. Top panel: Coronal section in the region of the first molar teeth: (a) maxillary antrum; (b) nasal cavity; (c) palatal plate; (d) sublingual space (above the mylohyoid muscle); (e) submandibular space (below the mylohyoid muscle); (f) intraoral presentation with infection spreading through the buccal plates inside the attachment of the buccinator muscle; (g) extraoral presentation to buccal space with infection spreading through the buccal plates outside the attachment of the buccinator muscle. Bottom panel: Lingual aspect of the mandible: (a) apices of the involved tooth above the myohyoid muscle, with spread of infection to the sublingual space; (b) apices of involved tooth below the mylohyoid muscle, with spread of infection into the submandibular space. Reproduced with permission from Chow AW, Roser SM, Brady FA, Ann Intern Med 1978; 88:392. Graphic 77988 Version 3.0
Fascial spaces around the mouth and face

Antimicrobial regimens for the prevention of dental caries and the treatment of periodontal disease in adults

<table>
<thead>
<tr>
<th>Clinical entity</th>
<th>Common causative organisms</th>
<th>Antimicrobial regimens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supragingival dental plaque and dental caries prevention</td>
<td><em>Streptococcus mutans</em>, other streptococci, <em>Actinomyces</em> spp</td>
<td>Fluoride-containing toothpaste (sodium fluoride, 1.1 percent or stannous fluoride, 0.4 percent) two or three times daily AND/OR Fluoride-containing varnishes (sodium fluoride, 5 percent) applied three or four times yearly AND/OR Chlorhexidine, 0.12 percent oral rinse</td>
</tr>
<tr>
<td>Gingivitis</td>
<td></td>
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<tr>
<td>Acute simple gingivitis</td>
<td><em>Streptococci, Actinomyces</em> spp, spirochetes</td>
<td>Penicillin G 2 to 4 MU IV every four to six hours (OR penicillin V 500 mg every six to eight hours), PLUS metronidazole 500 mg PO every eight hours OR Amoxicillin-clavulanate 875 mg PO every 12 hours or 500 mg PO every eight hours OR Ampicillin-sulbactam 1.5 to 3 g IV every six hours OR Clindamycin 450 mg PO or 600 mg IV every six to eight hours</td>
</tr>
<tr>
<td>Ulcerative or acute necrotizing ulcerative gingivitis</td>
<td><em>Prevotella intermedia, Fusobacterium</em> spp, <em>Tannerella forsythia, Treponema denticoli</em>, other oral spirochetes</td>
<td>Metronidazole 500 mg PO or IV every eight hours OR Amoxicillin-clavulanate 875 mg PO every 12 hours or 500 mg PO every eight hours OR Ampicillin-sulbactam 1.5 to 3 g IV every six hours OR Clindamycin 450 mg PO or 600 mg IV every six to eight hours</td>
</tr>
<tr>
<td>Periodontitis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early onset, &quot;aggressive&quot; or &quot;localized juvenile&quot; periodontitis</td>
<td><em>Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis, Treponema denticola, Prevotella intermedia</em></td>
<td>Doxycycline 200 mg PO or IV every 12 hours (only in patients eight years of age or older) OR Metronidazole 500 mg PO or IV every eight hours</td>
</tr>
</tbody>
</table>
Adult periodontitis

Treponema denticoli, other oral spirochetes, Porphyromonas gingivalis, Prevotella intermedia, Tannerella forsythia

Topical minocycline microspheres (Aristin®) OR

Topical doxycycline hyclate periodontal extended-release liquid (Atridox®)

IV: intravenous; MU: million units; PO: by mouth.

**Odontogenic infections**

Panel A: Dental caries, pulpal infection, and periapical abscess.

## Antimicrobial regimens for odontogenic soft tissue infections in adults*†

<table>
<thead>
<tr>
<th>Clinical entity</th>
<th>Common causative organisms</th>
<th>Antimicrobial regimens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Odontogenic deep space infections</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal hosts</td>
<td><em>Viridans</em> and other streptococci, <em>Peptostreptococcus</em> spp, <em>Bacteroides</em> spp, and other oral anaerobes</td>
<td>Ampicillin-sulbactam 3 g IV every six hours OR Penicillin G 2 to 4 MU IV every four to six hours, PLUS either: Clindamycin 600 mg IV every six to eight hours OR Metronidazole 500 mg IV or PO every eight hours OR Cefoxitin 1 to 2 g IV every four hours OR Cefotetan 2 g IV every 12 hours</td>
</tr>
<tr>
<td>Immunocompromised hosts</td>
<td><em>Viridans</em> and other streptococci, <em>Peptostreptococcus</em> spp, <em>Bacteroides</em> spp, and other oral anaerobes, facultative gram-negative bacilli (including <em>Pseudomonas aeruginosa</em>)</td>
<td>Piperacillin-tazobactam 4.5 g IV every six hours OR Imipenem-cilastatin 500 mg IV every six hours OR Meropenem 1 g IV every eight hours OR Cefepime 1 to 2 g IV every 12 hours PLUS either: Clindamycin 600 mg IV every six to eight hours OR Metronidazole 500 mg IV every eight hours</td>
</tr>
</tbody>
</table>
Deep neck space infections

- OR Ciprofloxacin 400 mg IV every 12 hours
- PLUS Metronidazole 500 mg IV every eight hours

IV: intravenous; MU: million units; PO: by mouth.* The doses recommended in this table are intended for patients with normal renal and hepatic function. ¶ Local and institutional rates of antibiotic resistance should be considered before choosing an antibiotic regimen. This is particularly important for immunocompromised patients, since there are substantial rates of fluoroquinolone resistance among *Pseudomonas aeruginosa* and other gram-negative bacteria in some regions.
INTRODUCTION — Deep neck space infections most commonly arise from a septic focus of the mandibular teeth, tonsils, parotid gland, deep cervical lymph nodes, middle ear, or sinuses. These deep cervical space infections have become relatively uncommon in the postantibiotic era. Consequently, many clinicians are unfamiliar with these conditions. In addition, with widespread use of antibiotics and/or profound immunosuppression, the classic manifestations of these infections, such as high fever, systemic toxicity, and local signs of erythema, edema, and fluctuance, may be absent.

Deep neck space infections often have a rapid onset and can progress to life-threatening complications [1]. Thus, clinicians must be aware of such infections and should not underestimate their potential extent or severity.

The relevant anatomy, microbial etiology, clinical manifestations, diagnosis, and treatment of deep neck space infections will be reviewed here. Peritonsillar abscesses and submandibular space infections (Ludwig's angina), suppurative parotitis, and odontogenic, middle ear, and sinus infections are discussed in detail separately. (See “Peritonsillar cellulitis and abscess” and “Submandibular space infections (Ludwig’s angina)” and “Suppurative parotitis in adults” and “Epidemiology, pathogenesis, and clinical manifestations of odontogenic infections” and “Acute otitis media in children: Epidemiology, microbiology, clinical manifestations, and complications” and “Acute otitis media in adults (suppurative and serous)” and “Acute sinusitis and rhinosinusitis in adults: Clinical manifestations and diagnosis” and “Uncomplicated acute sinusitis and rhinosinusitis in adults: Treatment”.)

ANATOMIC CONSIDERATIONS — Knowledge of the cervical compartments and interfascial spaces is essential for understanding the pathogenesis, clinical manifestations, and potential routes of spread of infections involving these spaces.

Cervical fascia — The muscles, vessels, and visceral structures of the neck are enveloped by the cervical fascia, which has a superficial and deep component (figure 1 and figure 2). The superficial cervical fascia consists of the subcutaneous tissues of the neck, which completely enclose the head and neck and is continuous with the platysma anteriorly.

The deep cervical fascia has three layers: superficial, middle, and deep, which can be thought of as defining a series of cylindrical compartments that extend longitudinally from the base of the skull to the mediastinum (figure 1):

- The superficial or investing layer of the deep cervical fascia encloses all of the deeper parts of the neck, beginning at the nuchal line and extending anteriorly, dividing to enclose the trapezius, sternocleidomastoid, and strap muscles as well as the submaxillary and parotid glands.
- The middle or pretracheal fascia encloses the cervical viscera including the pharynx, esophagus, larynx, trachea, thyroid, and parathyroid glands.
- The deep or prevertebral fascia arises from the nuchal ligament and encloses the vertebral column and muscles of the spine. The prevertebral fascia originates posteriorly on the spinous processes and encircles the splenius, erector spinae, and semispinalis muscles (figure 1 and figure 2). Prior to completing its circle anterior to the vertebral bodies, it fuses to the transverse processes. At this point, it is split into two layers: the alar fascia anteriorly and the prevertebral fascia posteriorly.

All three layers of the deep cervical fascia contribute to the carotid sheath, which forms a neurovascular compartment that encloses the carotid artery, the internal jugular vein, and the vagus nerve.

Fascial spaces — There are three spaces between the planes of the deep cervical fascia that are of major clinical importance [2]:

1. The superficial or investing layer of the deep cervical fascia encloses all of the deeper parts of the neck, beginning at the nuchal line and extending anteriorly, dividing to enclose the trapezius, sternocleidomastoid, and strap muscles as well as the submaxillary and parotid glands.
2. The middle or pretracheal fascia encloses the cervical viscera including the pharynx, esophagus, larynx, trachea, thyroid, and parathyroid glands.
3. The deep or prevertebral fascia arises from the nuchal ligament and encloses the vertebral column and muscles of the spine. The prevertebral fascia originates posteriorly on the spinous processes and encircles the splenius, erector spinae, and semispinalis muscles (figure 1 and figure 2). Prior to completing its circle anterior to the vertebral bodies, it fuses to the transverse processes. At this point, it is split into two layers: the alar fascia anteriorly and the prevertebral fascia posteriorly.

All three layers of the deep cervical fascia contribute to the carotid sheath, which forms a neurovascular compartment that encloses the carotid artery, the internal jugular vein, and the vagus nerve.
Other spaces arising from the cervical fascia with potential for deep neck infections include: the “danger” space, the prevertebral space, the pre-tracheal space, the peritonsillar space, and the parotid space. The masticator space, which lies lateral and anterior to the parapharyngeal space and consists of the masseteric, pterygoid, and temporal spaces, is described in detail elsewhere; infection of the masticator space arises most frequently from molar teeth, particularly the third molars (wisdom teeth). (See "Complications, diagnosis, and treatment of odontogenic infections").

Submandibular space — The submandibular space lies within the submental and submandibular triangles between the mucosa of the floor of the mouth and the superficial layer of the deep cervical fascia. It is subdivided by the mylohyoid muscle into the sublingual space (which contains the sublingual gland, hypoglossal nerve, part of the submandibular gland, and loose connective tissue), and the submylohyoid space (also known as the submaxillary space, which contains the submandibular salivary gland and lymph nodes) (figure 3). The two divisions communicate posteriorly around the mylohyoid muscle. It is this space that is primarily involved in Ludwig’s angina. Infection within the sublingual space results in gross swelling of the tongue that can result in acute airway obstruction. Infection of the submylohyoid space may spread posteriorly along the styloglossus muscle into the parapharyngeal space and then further inferiorly into the superior mediastinum.

Parapharyngeal space — Also known as the lateral pharyngeal or pharyngomaxillary space, the parapharyngeal space is located in the lateral aspect of the neck and is shaped like an inverted cone, with its base at the skull and its apex at the hyoid bone (figure 4). It lies deep to the pharyngeal constrictor muscle, and is contiguous medially with the pretracheal fascia of the visceral compartment, and laterally with the superficial fascia (which invests the parotid gland), the internal pterygoid muscle, and the mandible.

The parapharyngeal space is divided into an anterior (prestyloid or muscular) compartment and a posterior (retrostyloid or neurovascular) compartment by the styloid process and its attached muscles, the stylomandibular ligament, and the insertion of these structures into the hyoid bone (figure 4).

The anterior compartment contains no vital structures but rather only fat, lymph nodes, connective tissue, and muscle. It is the compartment most closely related to the tonsillar fossa and the internal pterygoid muscle. The posterior compartment contains the ninth to twelfth cranial nerves superiorly and the tenth cranial nerve more inferiorly, the carotid sheath and its contents, and the cervical sympathetic trunk. The carotid sheath, which runs in the posterior aspect of the parapharyngeal space, pierces the cone at its apex to enter the mediastinum (figure 4).

Infection of the lateral pharyngeal space may result from pharyngitis, tonsillitis, parotitis, otitis, or mastoiditis, as well as odontogenic infections, especially if the masticator space is involved. (See "Complications, diagnosis, and treatment of odontogenic infections").

Retropharyngeal space — The retropharyngeal space is bound anteriorly by the constrictor muscles of the neck and posteriorly by the alar layer of the deep cervical fascia. It is situated behind the hypopharynx and the esophagus, and lies between the alar fascia posteriorly and the posterior aspect of the pretracheal fascia anteriorly (figure 1 and figure 2). It communicates with the parapharyngeal space laterally where the carotid sheaths reside.

Danger space — Posterior to the retropharyngeal space is the danger space, which is bound by the alar fascia anteriorly and the prevertebral fascia posteriorly (figure 1 and figure 2). It extends from the base of the skull and descends freely through the entire posterior mediastinum to the level of the diaphragm (T1 to T2) where the two fascial layers fuse. Thus, the danger space provides the most important anatomic route for contiguous spread between the neck and the chest.

Prevertebral space — The prevertebral space is bound by the prevertebral fascia, which originates posteriorly on the spinous processes and encircles the splenius, erector spinae, and semispinalis muscles (figure 1 and figure 2). Prior to completing its circle anterior to the vertebral bodies, it fuses to the transverse processes. At this point, it is split into two layers: the alar fascia anteriorly and the prevertebral fascia posteriorly. The prevertebral space extends from the base of the skull to the coccyx, thus allowing organisms to spread as far down as the psoas muscle sheath.
Pretracheal space — The pretracheal space comprises the anterior portion of the visceral compartment and completely surrounds the trachea and esophagus (figure 1). It is contiguous with the carotid sheath laterally and with the superior mediastinum inferiorly.

Peritonsillar space — The peritonsillar space lies between the capsule of the palatine (faucial) tonsil medially, the superior constrictor muscle laterally, and the tonsillar pillars anteriorly and posteriorly (figure 5). A dreaded complication of a peritonsillar abscess (Quinsy) is to rupture through the superior constrictor muscle and extend directly into the parapharyngeal space, which lies posterior to the tonsillar bed.

Parotid space — The parotid space is formed by the splitting of the investing fascia at the level of the stylomandibular ligament to enclose the parotid gland within a superficial capsule and a deep capsule (figure 6). The superficial capsule is thick and strong, tightly adherent to the superficial pole of the parotid gland. The deep capsule adjacent to the dorsal lobe of the parotid gland, however, is thin and infection in the gland can easily penetrate through this capsule and extend through the stylomandibular tunnel into the parapharyngeal space. The stylomandibular ligament effectively separates the parotid space from the submylohyoid space.

Lymph nodes — The lymph nodes of the head and neck can be divided into 10 principal groups (figure 7). Six of these (occipital, mastoid, parotid, facial, submandibular, and submental nodes) form a collar at the junction of the head and neck. Within this collar, the sublingual and retropharyngeal nodes lie near the base of the tongue. The anterior and lateral cervical nodes form a chain along the front and side of the neck, respectively. The lateral cervical chain serves as a common root for drainage. The final conduit from all lymphatics in the head and neck is the large deep chain situated along the carotid sheath. When inflamed, these nodes become adherent to the fascial sheath of the vessels; thus, a suppurative infection of the cervical lymph nodes may frequently invade the bloodstream.

In addition to the regional lymph nodes of the head and neck, both the nasopharynx and the oropharynx are richly endowed with mucosa-associated lymphoid tissues similar to the bronchus-associated lymphoid tissues and the gut-associated lymphoid tissues. In particular, the aggregation of lymphoid tissues surrounding the nasopharynx is known as Waldeyer's ring and comprises the palatine, lingual, adenoidal, and tonsillar lymphoid tissues. Acute inflammation involving these tissues, such as during acute tonsillopharyngitis, croup, otitis media, retropharyngeal abscess or Epstein-Barr virus (EBV) mononucleosis, may result in acute airway compromise and constitutes a medical emergency [3,4].

Potential routes of spread — The deep cervical fascial spaces are normally bound together by loose connective tissue and intercommunicate to varied degrees. The potential pathways of extension from one space to another are illustrated in the accompanying figure (figure 8). A thorough understanding of the potential anatomic routes of infection not only provides valuable information on the nature and extent of infection but also suggests the optimal surgical approach for effective drainage.

MICROBIOLOGY — Deep neck space infections are typically polymicrobial and represent the normal resident flora of the contiguous mucosal surfaces from which the infection originated. Due to the close anatomic relationships, the resident flora of the oral cavity, upper respiratory tract, and certain parts of the ears and eyes share many common organisms (figure 9) [5]. Although as many as 50 to 100 bacterial species may be present on the oral or nasopharyngeal mucosal surface, the typical deep neck space infection includes, on average, five or six bacterial types [6,7]. Anaerobes generally outnumber aerobes on all mucosal surfaces of the oral cavity by a factor of 10:1 [8].

The most common organism isolated from deep neck space infections is Streptococcus viridans, reflecting its abundance in the mouth [9-11]. Most abscesses originating from the teeth also harbor oral anaerobes, including Peptostreptococcus species, Fusobacterium nucleatum, pigmented Prevotella species such as Prevotella melaninogenica (formerly Bacteroides melaninogenicus), and Actinomyces species [12].

Additional organisms, such as Staphylococcus aureus and facultative gram-negative rods, including Pseudomonas aeruginosa and extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae, may be present, particularly in patients with risk factors [13]. Immunocompromised hosts (eg, patients with chemotherapy-induced neutropenia) are particularly likely to harbor such pathogens, but those with diabetes mellitus, postoperative infection, or trauma are also at increased risk.
Klebsiella pneumoniae has been frequently isolated from deep neck space infections in Taiwan, particularly from diabetic patients. This species is a more common pathogen in various infections in Southeast Asia than in most other regions [10,14,15]. (See "Clinical features, diagnosis, and treatment of Klebsiella pneumoniae infection").

Infections arising from the pharynx frequently contain oral anaerobes and facultative streptococci, particularly Streptococcus pyogenes. In a study of 847 patients with peritonsillar abscess, Fusobacterium necrophorum was the most commonly detected species (23 percent), followed by S. pyogenes (17 percent), and group C or G streptococci (5 percent) [16]. (See "Complications of streptococcal tonsillopharyngitis").

Infections of the prevertebral space usually originate from contiguous spread of a cervical spine infection (such as discitis or vertebral osteomyelitis), by local instrumentation of the trachea or esophagus, or by hematogenous seeding. There is a predominance of gram-positive organisms, the most common being Staphylococcus aureus. Less common organisms include various facultative gram-negative bacilli, mycobacteria, and fungi. Intravenous drug use, immunosuppression, alcoholism, and diabetes mellitus are known risk factors [17,18]. Thus, the microbiology of prevertebral space infections is quite different from that of retropharyngeal or odontogenic deep neck infections.

GENERAL CLINICAL MANIFESTATIONS — Deep neck space infections share some typical clinical features. It is important to consider the primary source of infection (ie, odontogenic versus non-odontogenic, oropharyngeal, otogenic, or rhinogenic) and special host factors (such as comorbid disease, hospitalization, antecedent surgery or trauma, or immunocompromised state). In patients who have undergone radical neck surgery or radiation to the neck for cancer, diffuse cellulitis and lymphedema may be a prominent clinical feature. Similarly, in patients who have undergone tracheostomy and prolonged ventilation, a tracheo-esophageal fistula may develop with spread of infection into the mediastinum. Patients in the intensive care unit with prolonged internal jugular central venous catheters may develop suppurrative thrombophlebitis of the internal jugular vein (variant of Lemierre’s syndrome).

Each of the major deep neck space infections are discussed in greater detail below or in separate topic reviews. (See 'Specific deep neck space infections' below.)

Because of the dense superficial layer of the deep cervical fascia and its musculofascial planes, a fluctuant mass is not readily appreciated in deep neck space infections. Palpation of the oral cavity, when possible, may help in identifying such a mass or focal tenderness. The characteristic signs of deep pus are pitting or a doughy feeling on firm deep palpation.

Specific sites of infection are often associated with characteristic clinical manifestations and physical findings:

- Peritonsillar, parotid, parapharyngeal, and submandibular abscesses are generally associated with sore throat and trismus (the inability to open the jaw). Trismus indicates pressure or infection of the muscles of mastication (the masseter and the pterygoids) or involvement of the motor branch of the trigeminal nerve. Findings on physical examination include swelling of the face and neck, erythema, and purulent oral discharge. There may be pooling of saliva in the mouth and asymmetry of the oropharynx. Lymphadenopathy is usually present.

- Dysphagia and odynophagia are secondary to inflammation of the cricoarytenoid joints.

- Dysphonia and hoarseness are late findings in neck infections and may indicate involvement of the tenth cranial nerve

- Unilateral tongue paresis indicates involvement of the twelfth cranial nerve.

- Stridor and dyspnea signify airway obstruction and may be manifestations of local pressure or spread of infection to the mediastinum.

IMAGING — Computed tomography (CT) is the imaging modality of choice for the diagnosis of deep neck space infections [19]. CT allows the critical evaluation of soft tissues and especially bone from a single exposure (image 1). In addition, the axial imaging format of CT is particularly well suited to the head and neck. Because CT can localize a process and define its extent, particularly extension into the mediastinum or the cranial vault, it is also an invaluable tool for planning and guiding aspiration for culture or open drainage.

Magnetic resonance imaging (MRI) is useful for assessing the extent of soft tissue involvement and for delineating vascular complications. However, MRI takes significantly longer than CT to obtain good quality images, which may cause discomfort or claustrophobia [19]. In addition, individuals with certain implanted devices cannot undergo MRI. (See "Principles of magnetic resonance imaging").
Plain radiography is of limited utility for the evaluation of deep neck space infections; it is sometimes helpful for detecting retropharyngeal swelling or epiglottitis [19]. (See *Clinical features and diagnosis* below.)

**SPECIFIC DEEP NECK SPACE INFECTIONS**

**Peritonsillar abscess (quinsy)** — Peritonsillar abscess, also known as quinsy, is a suppurative complication of acute tonsillitis with extension into the peritonsillar space. The latter consists of loose areolar tissue overlying the tonsil and is surrounded by the superior pharyngeal constrictor muscle and the anterior and posterior tonsillar pillars. Peritonsillar abscesses may affect patients of all ages but are most common among young adults between the ages of 15 and 30 years. The infection begins as a cellulitis and progresses to abscess formation, most commonly near the superior pole of the tonsil. Patients complain of high fever, odynophagia, unilateral sore throat, and otalgia. Classic signs include a muffled voice, trismus, unilateral deviation of the uvula towards the unaffected side, and soft palate fullness or edema. The oral airway may be compromised and drooling may occur. Peritonsillar abscesses are often polymicrobial. The predominant bacterial species are *Streptococcus pyogenes* (group A streptococcus) and oral anaerobes. Peritonsillar abscess is discussed in greater detail separately. (See *"Peritonsillar cellulitis and abscess"*.)

**Parotid space infections** — Acute suppurative parotitis is characterized by the sudden onset of unilateral induration and erythema that extends from the cheek to the angle of the jaw. The parotid gland becomes swollen and extremely tender. Purulent discharge may be expressed from the orifice of the parotid duct with gentle pressure. Although a stone obstructing the salivary duct may predispose to bacterial infection, inspissated secretions and/or stasis are more common predisposing features. Three factors that predispose to suppurative parotitis are: acutely diminished salivary flow, poor oral hygiene, and increased susceptibility to infection [20]. Thus, acute suppurative parotitis is typically seen in elderly, debilitated, and/or dehydrated patients who may be diabetic or taking anticholinergic medications that decrease salivary flow.

The microbiology of acute suppurative parotitis is quite variable and is often polymicrobial. *Staphylococcus aureus* is by far the most frequently isolated pathogen, but anaerobes are also common. Acute suppurative parotitis is discussed in more detail elsewhere. (See *"Suppurative parotitis in adults"*.)

**Submandibular space infections (Ludwig's angina)** — Ludwig's angina is a bilateral infection of the submandibular space (which includes the submylohyoid and sublingual spaces) that begins in the floor of the mouth, most commonly related to the second or third mandibular molar teeth. It is typically a polymicrobial infection involving the flora of the oral cavity (table 1) [21]. It is an aggressive, rapidly spreading "woody" or brawny cellulitis without lymphadenopathy. Airway compromise is a potential complication, and requires careful monitoring and rapid intervention to prevent asphyxia and aspiration pneumonia. Ludwig's angina is discussed in greater detail separately. (See *"Submandibular space infections (Ludwig's angina)"*.)

**Pretracheal space infections** — Pretracheal space infections most commonly arise as a consequence of perforation of the anterior esophageal wall, occasionally through contiguous extension from a retropharyngeal space infection, or as a consequence of prolonged tracheostomy [22]. The clinical presentation is characterized by severe dyspnea, but hoarseness may be the first complaint. Swallowing may be difficult, and fluids may be regurgitated through the nose. A pretracheal space infection is always serious because of impending airway obstruction and possible extension into the mediastinum. Prompt surgical drainage is critical to prevent such complications.

**Prevertebral space infections** — Infections of the prevertebral space usually originate from contiguous spread of a cervical spine infection (such as discitis or vertebral osteomyelitis), by local instrumentation of the trachea or esophagus, or by hematogenous seeding. There is a predominance of gram-positive organisms, the most common being *Staphylococcus aureus*. Less common organisms include various facultative gram-negative bacilli, mycobacteria, and fungi. Intravenous drug use, immunosuppression, alcoholism, and diabetes mellitus are known risk factors [17,18]. Thus, the microbiology of prevertebral space infections is quite different from that of retropharyngeal or odontogenic deep neck infections.

The diagnosis of a prevertebral space infection may be difficult to make clinically because only 75 percent of patients complain of back or neck pain, 50 percent present with fever, and one-third have neurologic deficits ranging from nerve root pain to paralysis [18]. Computed tomography or magnetic resonance imaging is immensely helpful for differentiating a prevertebral space infection from a retropharyngeal abscess. Complications of prevertebral space infections arise from spinal epidural collections that cause cord compression. Irreversible paralysis occurs in 4 to 22 percent of patients [18]. Spread of infection to or from the disc or vertebrae may cause local destruction with
mechanical instability of the spine. Since the prevertebral space extends from the base of the skull down to the coccyx and is contiguous with the psoas muscle sheath, seemingly distant abscesses can form within the psoas muscle in a patient who has a prevertebral space infection [23].

**Parapharyngeal space infections** — Parapharyngeal space infections are potentially life-threatening because of the possibility of involving the carotid sheath and its vital contents (eg, common carotid artery, internal jugular vein, vagus nerve), propensity for airway impingement, and bacteremic dissemination. Since the clinical presentation may be dominated by the symptoms and signs of the primary source of infection, the diagnosis of parapharyngeal space involvement is often delayed.

Infection of the parapharyngeal space may arise from different sources throughout the neck. Dental infections are the most common underlying cause, followed by peritonsillar abscess, and rarely parotitis, otitis, or mastoiditis (Bezold's abscess). Infection of the anterior compartment of the parapharyngeal space is more common than the posterior compartment. (See "Epidemiology, pathogenesis, and clinical manifestations of odontogenic infections" and "Complications, diagnosis, and treatment of odontogenic infections" and "Suppurative parotitis in adults".)

**Clinical features** — The cardinal clinical features of parapharyngeal space infections are:

- Trismus
- Induration and swelling below the angle of the mandible
- Medial bulging of the pharyngeal wall
- Systemic toxicity with fever and rigors

Dyspnea may be prominent as edema and swelling involve the epiglottis and larynx. Swelling of the pharyngeal wall, if present, will be behind the palatopharyngeal arch and is easily missed. Suppuration may advance quickly to other spaces, particularly to the retropharyngeal and "danger" spaces, possibly reaching the mediastinum inferiorly or the base of the skull superiorly.

An abscess localized to the posterior neurovascular compartment of the parapharyngeal space may result in septicemia and neurologic signs indicating cranial nerve involvement (eg, Horner syndrome, hoarseness, unilateral tongue paresis) but with minimal trismus. The posterior tonsillar pillar is displaced.

Swelling and displacement of the parotid gland usually occur with infection in either the anterior or posterior compartment.

**Diagnosis** — The source and extent of infection is best evaluated by computed tomography (CT) or magnetic resonance imaging (MRI). (See 'Imaging' above.)

**Complications**

- **Carotid sheath involvement** — Involvement of the carotid sheath is a dreaded complication of parapharyngeal space infections because of the potential for carotid artery erosion and suppurative jugular thrombophlebitis [24]. The carotid sheath abuts all three layers of the deep cervical fascia. Thus, infection may arise by spread from the parapharyngeal space, submandibular space (Ludwig's angina), or suppuration of the deep cervical lymph nodes [25]. (See "Submandibular space infections (Ludwig's angina)".)

There are no characteristic symptoms or signs of a carotid sheath infection. A history of sore throat is usually but not always present, and may be mild or unilateral; there may be a latent period of up to three weeks before obvious manifestations of a deep neck space infection develop. The patient presents either in a toxic condition or insidiously with a fever of undetermined origin. Trismus is absent, and signs of local suppuration may be subtle initially because of the tight connective tissue around and within the carotid sheath. In some patients, there is diffuse swelling along the sternocleidomastoid muscle with marked tenderness and torticollis to the opposite side.

Carotid artery mycotic aneurysms have a mortality rate of approximately 20 percent [26]. Erosion of the carotid artery is a potentially devastating complication. This complication arises from an arteritis due to contiguous inflammation, resulting eventually in the formation of a false aneurysm, which may rupture. Erosion and rupture of the carotid artery may be heralded by recurrent small hemorrhages from the nose, mouth, or ear ("herald
bleeds”). This is followed by hematoma formation in the surrounding tissues, a protracted clinical course, and eventually the onset of shock due to exsanguination. Ligation of the carotid artery may be necessary in cases of major hemorrhage, but the mortality rate remains high, and the risk of stroke is significant [1].

**Suppurative jugular thrombophlebitis** — Suppurative jugular thrombophlebitis (also known as Lemierre's syndrome or postanginal sepsis) should be suspected in patients with antecedent pharyngitis, septic pulmonary emboli, and persistent fever despite antimicrobial therapy. It is caused most commonly by Fusobacterium necrophorum, which is often present in the bloodstream. (See "Suppurative (septic) thrombophlebitis").

Retropharyngeal and danger space infections — Retropharyngeal abscesses are among the most serious of deep space infections, since infection can extend directly into the anterior or posterior regions of the superior mediastinum, or into the entire length of the posterior mediastinum via the danger space (figure 1 and figure 2). Retropharyngeal infections can occur in both children and adults. Retropharyngeal infections in children are discussed separately. (See "Retropharyngeal infections in children").

**Clinical features and diagnosis** — Infection may reach the retropharyngeal space from either local or distant sites. Penetrating trauma (eg, from chicken bones or following instrumentation) is the usual source of local spread; in such cases, a sore throat or difficulty in swallowing or breathing may be the first indication of infection. More distant sources of infection include odontogenic sepsis and peritonsillar abscess (now a rare cause). Infection from these sources may often obscure the diagnosis of a retropharyngeal abscess because of associated trismus, which makes direct examination of the posterior pharyngeal wall difficult.

The differential diagnosis includes cervical osteomyelitis, Pott's disease, meningitis, and calcific tendonitis of the long muscle of the neck (figure 10) [27]. In this setting, CT scans and/or x-rays of the lateral neck are especially helpful and may demonstrate cervical lordosis with swelling and gas collections in the retropharyngeal space, causing anterior displacement of the larynx and trachea (image 2 and image 1). The radiograph should be evaluated for increased thickness of the prevertebral soft tissues, air or air-fluid levels, and the presence of foreign bodies. The soft tissues of the posterior wall of the hypopharynx are normally about 5 mm deep, less than one third the diameter of the fourth cervical vertebra (C4). In the presence of a retropharyngeal space infection, the pharynx or upper airway is displaced anteriorly by more than one half the width of the C4 vertebral body. Radiographs may also help to differentiate retropharyngeal from prevertebral space infection arising from cervical vertebral osteomyelitis [28,29]. (See 'Imaging' above.)

**Complications** — Acute necrotizing mediastinitis is the most feared complication of a retropharyngeal space infection. An infection in the "danger" space between the alar and prevertebral fasciae may drain by gravity into the posterior mediastinum, resulting in mediastinitis and empyema [30]. In the past, 70 percent of cases of mediastinitis were the result of infection spread in this manner. However, with the introduction of antibiotics, mediastinal extension has become uncommon, and most cases of acute mediastinitis result from esophageal perforation [31].

Clinically, the onset of acute necrotizing mediastinitis is rapid and is characterized by the following:

- Widespread necrotizing process extending the length of the posterior mediastinum, occasionally into the retroperitoneal space
- Rupture of mediastinal abscess into the pleural cavity with empyema or development of loculations
- Pleural or pericardial effusions, frequently with tamponade

The mortality of acute necrotizing mediastinitis in adults is high (25 percent), even when appropriate antibiotics are administered. (See "Aspiration pneumonia in adults").

Aspiration pneumonia is another potential complication of retropharyngeal space infection [32]. Pneumonia may result from impairment of swallowing or spontaneous rupture of the abscess into the airway.

**TREATMENT** — Appropriate antibiotics in conjunction with surgical drainage of loculated infection are essential for a successful outcome of deep neck space infections (table 2). It should be noted that the primary pathologic finding in deep neck space infections is cellulitis that involves the connective tissues, fasciae, and muscles, and frequently undergoes necrosis. This necrotizing cellulitis results in a serosanguinous, putrid infiltration of the cervical tissues, usually with little or no frank pus present. During this stage of infection, treatment is primarily medical, directed at eradicating the causative microorganisms and preventing local or systemic spread of infection. Maximum doses of systemic antimicrobials should be administered in order to optimize tissue penetration. Surgical drainage should be
implemented only if the cellulitic process has localized into a discrete abscess. Premature incision into an area of cellulitis area may actually worsen the situation by breaking down the natural defenses and hastening the spread of infection. The choice of antimicrobial regimens for the treatment of deep neck space infections has not been evaluated in clinical trials.

Empiric regimens are based upon the expected microbiology and immune status of the host, and coverage should be narrowed if microbiologic data become available. The antibiotic doses recommended below are intended for patients with normal renal function; dosing of many of these agents must be reduced in patients with renal dysfunction.

Peritonsillar abscess — The treatment of peritonsillar abscesses is discussed separately. (See "Peritonsillar cellulitis and abscess").

Suppurative parotitis — The treatment of suppurative parotitis is discussed separately. (See "Suppurative parotitis in adults").

Submandibular space infections (Ludwig's angina) — The treatment of Ludwig's angina is discussed separately. (See "Submandibular space infections (Ludwig's angina)").

Prevertebral space infections — Treatment of prevertebral space infections consists of expeditious drainage of the abscess and broad-spectrum antibiotics with coverage of Staphylococcus aureus and gram-negative bacilli. Some examples of appropriate regimens follow.

Immunocompetent host — We suggest one of the following regimens in the immunocompetent host:

- **Nafcillin** (1.5 g IV every 4 hours) or **vancomycin** (15 to 20 mg/kg IV every 8 to 12 hours, not to exceed 2 g per dose), plus either:
  - **Gentamicin** or **tobramycin** (1.7 mg/kg IV every 8 hours or 5 mg/kg IV every 24 hours), or
  - **Ciprofloxacin** (400 mg IV q12h), or
  - **Ticarcillin-clavulanate** (3.1 g IV every 4 hours)

Patients with risk factors for MRSA infection should be treated empirically with **vancomycin** (15 to 20 mg/kg IV every 12 hours) or **linezolid** (600 mg orally or IV every 12 hours). Risk factors for MRSA include a history of intravenous drug use, comorbid disease (eg, diabetes mellitus), and residing in a community or hospital where there is a substantial incidence of MRSA.

Immunocompromised host — We suggest one of the following regimens in the immunocompromised host:

- **Vancomycin** (15 to 20 mg/kg IV every 8 to 12 hours, not to exceed 2 g per dose) or **linezolid** (600 mg orally or IV every 12 hours) plus either:
  - **Cefepime** (2 g IV every 12 hours) plus **metronidazole** (500 mg IV every six to eight hours) or
  - **Imipenem** (500 mg IV every six hours) or
  - **Meropenem** (1 g IV every eight hours) or
  - **Piperacillin-tazobactam** (4.5 g IV every six hours)

Duration — For uncomplicated prevertebral space infections without evidence of discitis or osteomyelitis, two to three weeks of therapy is adequate. We favor intravenous antibiotics for the entire duration of treatment. When adjacent osteomyelitis is present, at least six to eight weeks of intravenous antibiotics is necessary.

**Parapharyngeal or retropharyngeal space infection** — Treatment of parapharyngeal or retropharyngeal space infections initially depends upon whether local suppuration has developed or whether the initial phase of diffuse cellulitis persists. This differentiation is important because surgical drainage should be delayed in the cellulitis stage, whereas loculated abscesses should be drained. Unfortunately, this is often difficult to determine clinically. Imaging studies such as computed tomography or magnetic resonance imaging should be performed and image-guided needle aspiration may be required. Endoscopic drainage is contraindicated owing to the proximity of the great vessels. In retropharyngeal space infection complicated by acute necrotizing mediastinitis, surgical drainage of the mediastinum is required and may be performed by either the cervico-mediastinal or the transthoracic approach. Although the cervical approach may be effective in early mediastinitis, thoracotomy is generally indicated once the necrotizing process has entered the "danger" space.
In patients who are recovering, it is important to restrict all oral intake until the swallowing impairment, which may have a prolonged course, has resolved completely.

Antimicrobial treatment of parapharyngeal or retropharyngeal space infections is based upon the probable site of origin in the immunocompetent host (odontogenic, from the teeth; rhinogenic, from the nose; or otogenic, from the ear). (See 'Microbiology' above and "Acute otitis media in adults (suppurative and serous)" and "Acute otitis media in children: Epidemiology, microbiology, clinical manifestations, and complications", section on 'Complications and sequelae' and "Uncomplicated acute sinusitis and rhinosinusitis in adults: Treatment".)

**Oral or odontogenic source** — We suggest one of the following regimens in the immunocompetent host with an oral or odontogenic source:

- **Ampicillin-sulbactam** (3 g IV every six hours) or
- **Penicillin G** (2 to 4 MU IV every four to six hours) plus **metronidazole** (500 mg IV every six to eight hours) or
- **Clindamycin** (600 mg IV every six to eight hours)

In addition, patients with risk factors for MRSA infection should be treated empirically with **vancomycin** (15 to 20 mg/kg IV every 12 hours) or **linezolid** (600 mg orally or IV every 12 hours). Risk factors for MRSA include a history of intravenous drug use, comorbid disease (eg, diabetes mellitus), and residing in a community or hospital where there is a substantial incidence of MRSA.

**Rhinogenic or otogenic source** — We suggest one of the following regimens in the immunocompetent host with a rhinogenic or otogenic source:

- **Ampicillin-sulbactam** (3 g IV every six hours) or
- **Ceftriaxone** 1 g IV every 24 hours plus **metronidazole** 500 mg IV every six to eight hours or
- **Ciprofloxacin** (400 mg IV every 12 hours) plus **clindamycin** (600 mg IV every six to eight hours)

In addition, patients with risk factors for MRSA infection should be treated empirically with **vancomycin** (15 to 20 mg/kg IV every 12 hours) or **linezolid** (600 mg orally or IV every 12 hours). Risk factors for MRSA include a history of intravenous drug use, comorbid disease (eg, diabetes mellitus), and residing in a community or hospital where there is a substantial incidence of MRSA.

**Immunocompromised host** — We suggest one of the following regimens for parapharyngeal or retropharyngeal space infections in the immunocompromised host:

- **Cefepime** (2 g IV every 12 hours) plus **metronidazole** (500 mg IV every six to eight hours) or
- **Imipenem** (500 mg IV every six hours) or
- **Meropenem** (1 g IV every eight hours) or
- **Piperacillin-tazobactam** (4.5 g every six hours)

In addition, patients with risk factors for MRSA infection should be treated empirically with **vancomycin** (15 to 20 mg/kg IV every 12 hours) or **linezolid** (600 mg orally or IV every 12 hours). Risk factors for MRSA include a history of intravenous drug use, comorbid disease (eg, diabetes mellitus), and residing in a community or hospital where there is a substantial incidence of MRSA.

**Duration** — Therapy should generally be continued for two to three weeks for parapharyngeal or retropharyngeal space infections until fever and leukocytosis have resolved and local tenderness and swelling have subsided. Longer courses are required when complications are present. We favor intravenous antibiotics for the entire duration of treatment.

**SUMMARY AND RECOMMENDATIONS**

- Deep neck space infections most commonly arise from a septic focus of the mandibular teeth, tonsils, parotid gland, deep cervical lymph nodes, middle ear, or sinuses. Since these infections often have a rapid onset and may progress to life-threatening complications, clinicians must be aware of such infections and should not underestimate their potential extent or severity. (See 'Introduction' above.)
- Knowledge of the cervical compartments and interfascial spaces is essential for understanding the pathogenesis, clinical manifestations, and potential routes of spread of infections involving these spaces. The...
Deep cervical fascia has three layers: superficial, middle, and deep, which can be thought of as defining a series of cylindrical compartments that extend longitudinally from the base of the skull to the mediastinum (figure 1). (See ‘Anatomic considerations’ above and ‘Potential routes of spread’ above.)

- Deep neck space infections are typically polymicrobial and represent the normal resident flora of the contiguous mucosal surfaces from which the infection originated. Due to the close anatomic relationships, the resident flora of the oral cavity, upper respiratory tract, and certain parts of the ears and eyes share many common organisms (figure 9). (See ‘Microbiology’ above.)

- The most common organism isolated from deep neck space infections is Streptococcus viridans, reflecting its abundance in the mouth. Most abscesses originating from the teeth also harbor oral anaerobes, including Peptostreptococcus species, Fusobacterium nucleatum, pigmented Prevotella species such as Prevotella melaninogenica (formerly Bacteroides melaninogenicus), and Actinomyces species. (See ‘Microbiology’ above.)

- Computed tomography is the imaging modality of choice for the diagnosis of deep neck space infections. Magnetic resonance imaging is useful for assessing the extent of soft tissue involvement and for delineating vascular complications. (See ‘Imaging’ above.)

- For deep neck space infections that involve a drainable collection, aspiration or surgical drainage should be performed. (See ‘Treatment’ above.)

- Appropriate antibiotics based upon the likely microbiology of the infection are essential for a successful outcome of deep neck space infections. Empiric regimens for each type of deep neck space infection are discussed above (table 2). The final selection of antimicrobial therapy should be guided by culture results and susceptibility data, whenever possible. (See ‘Treatment’ above.)

- For both immunocompetent and immunocompromised patients who are at risk for methicillin-resistant Staphylococcus aureus (MRSA) carriage or infection, such as those with a history of intravenous drug use or comorbid disease (eg, diabetes mellitus), or those in communities or hospitals where there is a substantial incidence of MRSA, we suggest that MRSA coverage be included in the treatment regimen (Grade 2C). (See ‘Treatment’ above.)

- For parapharyngeal, retropharyngeal, or prevertebral space infections, therapy should generally be continued for two to three weeks. Longer courses are required when complications are present. (See ‘Parapharyngeal space infections’ above and ‘Retropharyngeal and danger space infections’ above and ‘Prevertebral space infections’ above.)

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REFERENCES

Relationship of various cervical fascial spaces to the superficial and deep layers of the cervical fascia

**Parapharyngeal space**

**Cross section**

- Pretracheal fascia
- Carotid sheath
- Internal jugular vein
- Cranial nerve X
- Internal carotid artery
- Parotid gland
- Internal pterygoid muscle
- Mandible

**Sagittal section**

- Pharyngeal wall
- Parotid gland
- Tonsil
- Parapharyngeal space
- Hyoid bone
- Internal carotid artery
- Internal jugular vein

**Parotid space anatomy**

- Parotid space
- Superficial capsule
- Parotid gland
- Deep lobe
- Deep capsule
- Parapharyngeal space
Lymph nodes of the head and neck

This drawing schematically depicts the major lymph nodes in the head and neck area that are likely to be enlarged on physical examination in patients with various local or systemic diseases. The major nodal groups are shown here in bold, with the areas draining into these nodal groups noted when appropriate. While enlargement of both the left and right supraclavicular lymph nodes may reflect disease in the thorax, left supraclavicular nodal enlargement, because of its drainage pattern, may also reflect the presence of abdominal involvement (ie, Virchow's node).

Potential pathways of extension in deep cervical fascial space infections
Diagrammatic illustration of the anatomic relationship of head and neck structures and distribution of the indigenous microflora

Retro- and parapharyngeal abscess

The computed tomography of the neck shows retro- and parapharyngeal abscess (A, B) as indicated by a low-density core, soft-tissue swelling, obliterated fat planes, mass effect, and rim enhancement.
### Predominant cultivable flora from various sites of the oral cavity

<table>
<thead>
<tr>
<th>Group</th>
<th>Predominant genus or family</th>
<th>Total viable count (mean percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Tongue</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anaerobes (1011 CFU/g)*</td>
<td>Gram + cocci</td>
<td>Peptostreptococcus</td>
</tr>
<tr>
<td></td>
<td>Gram - cocci, Gram + rods</td>
<td>Veillonella</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Actinomyces, Eubacterium, Lactobacillus, Leptotrichia</td>
</tr>
<tr>
<td></td>
<td>Gram - rods</td>
<td>Fusobacterium, Bacteroides, Prevotella, Porphyromonas</td>
</tr>
<tr>
<td>Aerobes (1010 CFU/g)*</td>
<td>Gram + cocci</td>
<td>Streptococcus</td>
</tr>
<tr>
<td></td>
<td>Gram - cocci</td>
<td>Moraxella</td>
</tr>
<tr>
<td></td>
<td>Gram + rods</td>
<td>Lactobacillus, Corynebacterium</td>
</tr>
<tr>
<td></td>
<td>Gram - rods</td>
<td>Enterobacteriaceae</td>
</tr>
</tbody>
</table>

* Total viable colony-forming units per g net weight.
Retropharangeal calcific tendinitis

Contrast-enhanced computed tomography (CT) scan showing prevertebral calcifications (arrows) in a 40-year-old man with low-grade fever and acute neck pain.
Lateral soft tissue radiographic demonstration of retropharyngeal abscess

Lateral radiographs of the neck show: (A) normal lateral cervical view; (B) expansion of the prevertebral soft tissues by a retropharyngeal abscess.
Usual causative organisms and initial empiric antimicrobial regimens for suppurative peripharyngeal infections in adults

<table>
<thead>
<tr>
<th>Infection</th>
<th>Usual causative organisms</th>
<th>Antimicrobial regimens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peritonsillar abscess (Quinsy)</td>
<td>Group A streptococcus (S. pyogenes), Fusobacterium spp, Peptostreptococcus spp, and other oral anaerobes&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Normal host: Ampicillin-sulbactam 3 g IV Q 6 h OR Penicillin G 2-4 MU IV Q 4-6 h plus Metronidazole 500 mg IV Q 6-8 h OR Clindamycin 600 mg IV Q 6-8 h Immunocompromised host&lt;sup&gt;b&lt;/sup&gt;: Cefepime 2 g IV Q 12 h PLUS Metronidazole 500 mg IV Q 6-8 h OR monotherapy with: Imipenem 500 mg IV Q 6 h OR Meropenem 1 g IV Q 8 h OR Piperacillin-tazobactam 4.5 g IV Q 6 h</td>
</tr>
<tr>
<td>Suppurative parotitis</td>
<td>Staphylococcus aureus, viridans and other streptococci, Bacteroides spp, Peptostreptococcus spp, and other oral anaerobes&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Normal host: Nafcillin 1.5 g IV Q 4 h&lt;sup&gt;c&lt;/sup&gt; or Vancomycin 15-20 mg/kg IV Q 8-12 h&lt;sup&gt;d&lt;/sup&gt; or Linezolid 600 mg orally or IV Q 12 h Immunocompromised host: Vancomycin 15-20 mg/kg IV Q 8-12 h&lt;sup&gt;h&lt;/sup&gt; or linezolid 600 mg orally or IV Q 12 h PLUS one of the following regimens: Cefepime 2 g IV Q 12 h PLUS Metronidazole 500 mg IV Q 6-8 h or Imipenem 500 mg IV Q 6 h or Meropenem 1 g IV Q 8 h or Piperacillin-tazobactam 4.5 g IV Q 6 h</td>
</tr>
<tr>
<td>Submandibular space infections (Ludwig's angina)</td>
<td>Viridans and other streptococci, Staphylococcus spp, Peptostreptococcus spp, Bacteroides spp, and other oral anaerobes&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Normal host: Ampicillin-sulbactam 3 g IV Q 6 h OR Penicillin G 2-4 MU IV Q 4-6 h Immunocompromised host: Cefepime 2 g IV Q 12 h PLUS Metronidazole 500 mg IV Q 6-8 h OR monotherapy with: Imipenem 500 mg IV Q 6 h or Meropenem 1 g IV Q 8 h or Piperacillin-tazobactam 4.5 g IV Q 6 h</td>
</tr>
</tbody>
</table>

NOTE: Coverage for methicillin-resistant Staphylococcus aureus (MRSA) should be included for those with risk factors<sup>b</sup>.

<sup>a</sup> Coverage for MRSA should be included for those with risk factors.

<sup>b</sup> Considering the patient’s risk factors, such as corticosteroids, chronic alcoholism, or diabetes may be at increased risk for MRSA infection.

<sup>c</sup> Penicillinase-resistant penicillins or cephalosporins are recommended for empiric therapy.

<sup>d</sup> Penicillinase-resistant penicillins or cephalosporins are recommended for empiric therapy.
<table>
<thead>
<tr>
<th>Space Infections</th>
<th>Pathogens</th>
<th>Therapy Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parapharyngeal or retropharyngeal space infections</td>
<td>Viridans and other streptococci, Staphylococcus spp, Peptostreptococcus spp, Bacteroides spp, and other oral anaerobes(^a)</td>
<td>Ampicillin-sulbactam 3 g IV Q 6 h OR Penicillin G 2-4 MU IV Q 4-6 h plus Metronidazole 500 mg IV Q 6-8 h OR Clindamycin 600 mg IV Q 6-8 h</td>
</tr>
<tr>
<td>Odontogenic</td>
<td>Viridans and other streptococci, Staphylococcus spp, Peptostreptococcus spp, Bacteroides spp, and other oral anaerobes(^a)</td>
<td>Ampicillin-sulbactam 3 g IV Q 6 h OR Penicillin G 2-4 MU IV Q 4-6 h plus Metronidazole 500 mg IV Q 6-8 h OR Clindamycin 600 mg IV Q 6-8 h</td>
</tr>
<tr>
<td>Rhinogenic</td>
<td>Streptococcus pneumoniae, Haemophilus influenzae, viridans and other streptococci, Bacteroides spp, Peptostreptococcus spp, and other oral anaerobes(^a)</td>
<td>Ceftriaxone 1 g IV Q 24 h plus Metronidazole 500 mg IV Q 6-8 h OR Ciprofloxacin 400 mg Q 12 h plus Clindamycin 600 mg IV Q 6-8 h</td>
</tr>
<tr>
<td>Otogenic</td>
<td>Same as for rhinogenic space infections</td>
<td>Same as for rhinogenic space infections</td>
</tr>
</tbody>
</table>

\(^a\) Cefepime 2 g IV Q 12 h PLUS Metronidazole 500 mg IV Q 6-8 h OR monotherapy with:
- Imipenem 500 mg IV Q 6 h or
- Meropenem 1 g IV Q 8 h or
- Piperacillin-tazobactam 4.5 g IV Q 6 h

Same as for rhinogenic space infections
<table>
<thead>
<tr>
<th>Condition</th>
<th>Microorganisms</th>
<th>Initial Therapy</th>
<th>Other Therapies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septic jugular thrombophlebitis (Lemierre syndrome)</td>
<td>Fusobacterium necrophorum; same as for peritonsillar abscess or odontogenic space infections&lt;sup&gt;#&lt;/sup&gt;</td>
<td>Ampicillin-sulbactam 3 g IV Q 6 h OR Penicillin G 2-4 MU IV Q 4-6 h plus Metronidazole 500 mg IV Q 6-8 h OR Clindamycin 600 mg IV Q 6-8 h</td>
<td>Same as for odontogenic space infections</td>
</tr>
<tr>
<td>Prevertebral space infection</td>
<td>Staphylococcus aureus&lt;sup&gt;2&lt;/sup&gt;, facultative gram-negative bacilli&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Nafcillin 1.5 g IV Q 4 h&lt;sup&gt;3&lt;/sup&gt; or Vancomycin 15-20 mg/kg IV Q 8-12 h&lt;sup&gt;3&lt;/sup&gt; or Linezolid 600 mg orally or IV Q 12 h</td>
<td>Vancomycin 15-20 mg/kg IV Q 8-12 h&lt;sup&gt;3&lt;/sup&gt; or linezolid 600 mg orally or IV Q 12 h PLUS one of the following regimens: <strong>Combination therapy with:</strong> Cefepime 2 g IV Q 12 h PLUS Metronidazole 500 mg IV Q 6-8 h OR <strong>Monotherapy with:</strong> Imipenem 500 mg IV Q 6 h or Meropenem 1 g IV Q 8 h or Piperacillin-tazobactam 4.5 g IV Q 6 h</td>
</tr>
</tbody>
</table>

<sup>#</sup>See also for Penicillin G 2-4 MU IV Q 4-6 h plus Metronidazole 500 mg IV Q 6-8 h OR Clindamycin 600 mg IV Q 6-8 h

<sup>2</sup>See also for Nafcillin 1.5 g IV Q 4 h OR Vancomycin 15-20 mg/kg IV Q 8-12 h OR Linezolid 600 mg orally or IV Q 12 h

<sup>3</sup>See also for Vancomycin 15-20 mg/kg IV Q 8-12 h OR linezolid 600 mg orally or IV Q 12 h PLUS one of the following regimens: **Combination therapy with:** Cefepime 2 g IV Q 12 h PLUS Metronidazole 500 mg IV Q 6-8 h OR **Monotherapy with:** Imipenem 500 mg IV Q 6 h or Meropenem 1 g IV Q 8 h or Piperacillin-tazobactam 4.5 g IV Q 6 h

<sup>4</sup>See also for Gentamicin or Tobramycin 1.7 mg/kg IV Q 8 h or 5 mg/kg IV Q 24 h or Ciprofloxacin 400 mg IV Q 12 h or Ticarcillin-clavulanate 3.1 g IV Q 4 h
* The doses recommended in this table are intended for patients with normal renal and hepatic function.Δ Immunocompromised hosts are at increased risk for facultative gram-negative bacilli, including extended-spectrum-β-lactamase (ESBL)-producing Enterobacteriaceae and Pseudomonas aeruginosa.◊ In patients with risk factors for MRSA infection, vancomycin 15-20 mg/kg IV Q 8-12 h or linezolid 600 mg orally or IV Q 12 h should be added, or substituted for nafcillin. Risk factors for MRSA infection include a history of intravenous drug use, comorbid disease (eg, diabetes mellitus), or residing in a community or hospital where there is a substantial incidence of MRSA.§ Not to exceed 2 g per dose.