

on routine culture is a clue that the abscess is likely to contain anaerobic bacteria. Often smears of this “sterile pus” are found to be teeming with bacteria when Gram’s stain is applied. Although some facultative organisms (e.g., *Staphylococcus aureus*) are also capable of causing abscesses, abscesses in organs or deeper body tissues should call anaerobic infection to mind.

6. Gas is found in many anaerobic infections of deep tissues but is not diagnostic because it can be produced by aerobic bacteria as well.
7. Although a putrid-smelling infection site or discharge is considered diagnostic for anaerobic infection, this manifestation usually develops late in the course and is present in only 30–50% of cases.
8. Some species (the best example being the *B. fragilis* group) require specific therapy. However, many synergistic infections can be cured with antibiotics directed at some but not all of the organisms involved. Antibiotic therapy, combined with debridement and drainage, disrupts the interdependent relationship among the bacteria, and some species that are resistant to the antibiotic do not survive without the co-infecting organisms.
9. Manifestations of severe sepsis and disseminated intravascular coagulation are unusual in patients with purely anaerobic infection.

EPIDEMIOLOGY

Difficulties in the performance of appropriate cultures, contamination of cultures by components of the normal microbiota, and the lack of readily available, reliable culture techniques have made it impossible to obtain accurate data on incidence or prevalence. However, anaerobic infections are encountered frequently in hospitals with active surgical, trauma, and obstetric and gynecologic services. Depending on the institution, anaerobic bacteria account for 0.5–12% of all cases of bacteremia.

CLINICAL MANIFESTATIONS

Anaerobic Infections of the Mouth, Head, and Neck Anaerobic bacteria are commonly involved in infections of the mouth, head, and neck (Chap. 44). The predominant isolates are components of the normal microbiota of the upper airways—mainly the *Bacteroides oralis* group, pigmented *Prevotella* species, *P. asaccharolytica*, *Fusobacterium* species, peptostreptococci, and microaerophilic streptococci.

Soft tissue infections of the oral-facial area may or may not be odontogenic. Odontogenic infections—primarily dental caries and periodontal disease (gingivitis and periodontitis)—are common and have both local consequences (especially tooth loss) and the potential for life-threatening spread to the deep fascial spaces of the head and neck. Infections of the mouth can arise from either supragingival or subgingival dental plaque composed of bacteria colonizing the tooth surface. Supragingival plaque formation begins with the adherence of gram-positive bacteria to the tooth surface. This form of plaque is influenced by salivary and dietary components, oral hygiene, and local host factors. Supragingival plaque can lead to dental caries and, with further invasion, to pulpitis (endodontic infection) that can further perforate the alveolar bone, causing periapical abscess. Subgingival plaque is associated with periodontal infections (e.g., gingivitis, periodontitis, and periodontal abscess) that can further disseminate to adjacent structures such as the mandible, causing osteomyelitis of the maxillary sinuses. Periodontitis may also result in spreading infection that can involve adjacent bone or soft tissues. In the healthy periodontium, the sparse microbiota consists mainly of gram-positive organisms such as *Streptococcus sanguinis* and *Actinomyces* species. In the presence of gingivitis, there is a shift to a greater proportion of anaerobic gram-negative bacilli in the subgingival microbiota, with predominance of *Prevotella intermedia*. In well-established periodontitis, the complexity of the microbiota increases further. The predominant isolates are *P. gingivalis*, *P. intermedia*, *Aggregatibacter actinomycetemcomitans*, *Tannerella forsythensis*.

Necrotizing Ulcerative Gingivitis Gingivitis may become a necrotizing infection (*trench mouth*, *Vincent’s stomatitis*) (Chap. 44). The onset of disease is usually sudden and is associated with tender bleeding

gums, foul breath, and a bad taste. The gingival mucosa, especially the papillae between the teeth, becomes ulcerated and may be covered by a gray exudate, which is removable with gentle pressure. Patients may become systemically ill, developing fever, cervical lymphadenopathy, and leukocytosis.



Noma (*cancrum oris*) is a necrotizing infection of the oral mucous membranes. It is characterized by destruction of soft tissue and bone and evolves rapidly from gingival inflammation to orofacial gangrene. Noma occurs most frequently in young children (1–4 years of age) with malnutrition or systemic disease. This infection occurs worldwide but is most common in sub-Saharan Africa.

Acute Necrotizing Infections of the Pharynx These infections usually occur in association with ulcerative gingivitis. Symptoms include an extremely sore throat, foul breath, and a bad taste accompanied by fever and a sensation of choking. Examination of the pharynx demonstrates that the tonsillar pillars are swollen, red, ulcerated, and covered with a grayish membrane that peels easily. Lymphadenopathy and leukocytosis are common. The disease may last for only a few days or, if not treated, may persist for weeks. Lesions begin unilaterally but may spread to the other side of the pharynx or the larynx. Aspiration of the infected material by the patient can result in lung abscesses.

Peripharyngeal Space Infections These infections arise from the spread of organisms from the upper airways to potential spaces formed by the fascial planes of the head and neck. The etiology is typically polymicrobial and represents the normal microbiota of the mucosa of the originating site.

Peritonsillar abscess (*quinsy*) is a complication of acute tonsillitis caused mainly by a mixed flora containing anaerobes (e.g., *F. necrophorum* and *Peptostreptococcus* species) and the facultative anaerobe group A *Streptococcus* (Chap. 44). Of cases of submandibular space infection (*Ludwig’s angina*), 80% are caused by infection of the tissues surrounding the second and third molar teeth. This infection results in marked local swelling of tissues, with pain, trismus, and superior and posterior displacement of the tongue. Submandibular swelling of the neck can impair swallowing and cause respiratory obstruction. In some cases, tracheotomy is life-saving. Cervicofacial actinomycosis (Chap. 200) is caused by a branching, gram-positive, non-spore-forming, strict/facultative anaerobe that is a part of the normal oral microbiota. This chronic disease is characterized by abscesses, draining sinus tracts, fistula, bone destruction, and fibrosis. It can easily be mistaken for malignancy or granulomatous disease. Actinomycosis less frequently involves the thorax, abdomen, pelvis, and CNS.

Sinusitis and Otitis Anaerobic bacteria have been implicated in chronic sinusitis but play little role in acute sinusitis. In several studies on chronic sinusitis, anaerobic bacteria were found in 0–52% of cases, depending on the method used to collect specimens. Polymicrobial infection is common, and the predominant anaerobic isolates are pigmented *Prevotella*, *Fusobacterium*, *Peptostreptococcus*, and *P. acnes*. Aerobic gram-negative bacilli and *S. aureus* have also been implicated in chronic sinusitis. Anaerobic bacteria have been isolated in a large percentage of cases of chronic suppurative otitis media in children. The role of anaerobes in acute otitis media is less clear.

Complications of Anaerobic Head and Neck Infections Contiguous cranial spread of these infections can result in osteomyelitis of the skull or mandible or in intracranial infections such as brain abscess and subdural empyema. Caudal spread can produce mediastinitis or pleuropulmonary infection. Hematogenous complications can also result from anaerobic infections of the head and neck. Bacteremia, which occasionally is polymicrobial, can lead to endocarditis or other distant infections. Lemierre’s syndrome (Chap. 44), which has been uncommon in the antimicrobial era, is an acute oropharyngeal infection with secondary septic thrombophlebitis of the internal jugular vein and frequent metastasis, most commonly to the lung. *F. necrophorum* is the usual cause. This infection typically begins with pharyngitis, which is followed by local invasion in the lateral pharyngeal space, with resultant internal jugular vein thrombophlebitis. A typical clinical