Chapter 69: Odontogenic Infections

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Odontogenic infections are among the most frequently encountered infections afflicting humans. In the vast majority of patients these infections are minor and resolve either by spontaneous drainage through the gingival tissues of the tooth or by extraction of the offending tooth. Chronic sinus tracts from the apex of the tooth to the surface mucosa or skin are not uncommon in populations who receive little or no dental care. A great deal of pain and suffering accompany establishment of these draining sinus tracts. Removal of the offending tooth almost always results in rapid resolution of the infection, even with antibiotic therapy. Unfortunately, these minor tooth-related infections occasionally become serious and life-threatening. Aggressive surgical and medical care is necessary to prevent disastrous results.

This chapter discusses odontogenic infections: their etiology, their progression to severe infection, and their management.

Microbiology of Odontogenic Infections

As with infections elsewhere in the body, odontogenic infections usually arise as a result of normal endogenous flora. The mouth harbors a large number of different bacteria, both aerobes and anaerobes (Table 69-1). The aerobic or facultative anaerobic bacteria are primarily *Streptococcus* species. Notably absent from the mouth are staphylococci; *Staphylococcus* organisms rarely cause odontogenic infections. The obligate anaerobic bacteria make up the bulk of the mouth's flora (Newman et al, 1979). The predominant bacteria are the anaerobic *Streptococcus, Bacteroides,* and *Fusobacterium.*

In almost all patients normal mouth bacteria cause odontogenic infections. In reports before 1975 the bacteriology of these infections was held to be primarily *Streptococcus* and *Staphylococcus* (Gabrielson and Stroh, 1975; Sims, 1974); however, recent studes done with careful microbiology techniques under strict anaerobic conditions have produced a very different picture of the flora causing these infections (Aderhold et al, 1981; Bartlett and O'Keefe, 1979; Chow et al, 1978; Konow et al, 1981; Sabiston and Grigsby, 1976; Heimdahl et al, 1985; Labriola et al, 1983; Lewis et al, 1986; Quayle et al, 1987).

Based on the results of these investigations, several important conclusions can be drawn regarding the microbiology of odontogenic infections. First, aerobic bacteria alone are rarely the cause. When they are, *Streptococcus* is usually the offending bacterium. Second, about half of the infections are caused by anaerobic bacteria only (Table 69-2). Third, in almost every patient, multiple organisms grow from the infection. The average number of different organisms is around five, but as many as 10 can be found. In these mixed infections there is preponderance of anaerobic bacteria.

Table 69-3 lists the bacteria that cause odontogenic infections. These are the bacteria of the normal flora. The most common organisms are *Streptococcus, Eubacterium, Bacteroides* (especially *B. melaninogenicus*), and *Fusobacterium.* Notably absent are group

D Streptococcus (enterococci), *Staphylococcus,* and *Bacteroides fragilis.* These are important exceptions because their markedly different antibiotic susceptibility requires a major alteration in the choice of antibiotic.

The anaerobic bacteria seen in odontogenic infections are not usually acknowledged to be pathogenic themselves. A more invasive bacterium, such as *Streptococcus,* probably must accompany them to establish an infection. Of the anaerobes, *B. melaninogenicus* appears to be the most important bacterium of the anaerobic group. Alone or in combination with *Fusobacterium, Peptococcus,* or *Peptostreptococcus, B. melaninogenicus* is the most commonly isolated anaerobic bacterium.

Entry of bacteria into deep tissues to cause an infection is the result of invasive aerobic bacteria gaining access through a necrotic dental pulp. The aerobic bacteria serve as the initiators of the infection, preparing the local environment for anaerobic bacterial invasion. The anaerobes become predominant, since the reduction-oxidation potential favors anaerobic growth. The clinical picture is this; *Streptococcus* most commonly causes the early stage cellulitis. As the infection progresses, a mixed streptococcal/anaerobic infection occurs. As the local tissue condition changes to a more hypoxic state, the predominant bacteria become anaerobic species, such as *Bacteroides* or *Fusobacterium.*

Implications of Microbiology for Antibiotic Therapy

The primary treatment of odontogenic infections has been surgical for centuries and will continue to be so. Extraction of the tooth, drainage of the abscess, and release of pressure are fundamental in the management of these infections.

Antibiotics are a necessary adjunctive therapy in many infections to hasten complete resolution. The choice of antibiotic must be made with a clear idea of the antibiotic susceptibility of the bacteria causing the infection. In an uncomplicated infection the facts presented in the preceding section provide adequate information for choosing an effective antibiotic without the time delay and expense of taking a specimen for culture or antibiotic sensitivity testing.

The two microbiologic factors that must be kept in mind when choosing an antibiotic are these: first, the antibiotic must be effective against *Streptococcus,* since this bacterium is most commonly encountered; second, the drug should be effective against a broad range of anaerobic bacteria. Fortunately, the antibiotic susceptibility of the oral anaerobes is great. The outlines following provide a summary of the effectiveness of commonly used antibiotics for odontogenic inections. First, the orally administered drugs are listed:

A. Very effective

1. Peniccilin

2. Clindamycin

3. Metronidazole (alone or in combination with penicillin)

4. Amoxicillin/clavulanic acid.

B. Effective

1. Erythromycin

2. Cephalexin

3. Tetracycline.

C. Less effective

1. Sulfa drugs

2. Quinalones.

Next are the parenterally administered drugs:

A. Very effective

1. Penicillin

2. Clindamycin

3. Metronidazole

B. Effective

1. Cefazolin

2. Cefoxitin

C. Not effective

1. Gentamicin

2. Tobramycin

3. Amikacin

4. Cephalosporin (third generation).

The very effective drugs can be used with confidence on an empiric basis to treat odontogenic infections. The effective and less effective drugs are less predictable and should be used only when first-line drugs cannot be used or when indicated by specific culture and sensitivity testing. Those listed as not effective are not effective against either *Streptococcus* or anaerobes; therefore they are not indicated in the routine odontogenic infection. When indicated by specific culture results, they may prove to be quite valuable, but usually in

combination with beta-lactam antibiotics.

The antibiotics that may be effective in odontogenic infections but that should not be routinely employed are listed as follows:

Ampicillin

Carbenicillin

Ticarcillin

Piperacillin

Azlocillin

Mezlocillin

Moxalactam

Chloramphenicol

Cephalosporins.

The reasons for not using them are excess toxicity (chloramphenicol), excess expense (carbenicillin), and a needlessly extended spectrum (third-generation cephalosporins). In specific serious, life-threatening infections, these antibiotics may be useful. In general, however, they should be held in reserve.

Two final comments concerning recommendation of these antibiotics must be made. First, although penicillin has been an extremely useful drug for anaerobic infections, resistance to it is beginning to emerge (Edson et al, 1982). This is especially true with *B. melaninogenicus,* since about 20% of these bacteria are currently reported as resistant to penicillin. This must be remembered when odontogenic infections do not respond to penicillin. Antibiotic resistance is also being seen with other antibiotics used to treat head and neck infections. This may be the result of more carefully done testing, especially for anaerobic bacteria, or it may be the result of actual resistance by the bacteria (Baker et al, 1985; Crook et al, 1988; Dornbusch, 1980; Edson et al, 1982; Gilmore et al, 1988; Musial and Rosenblatt, 1989). Current percent resistance to common antibiotics is seen in Table 69-4.

Second, the antibiotic metronidazole has become popular recently in the treatment of odontogenic infections. This drug has absolutely no activity against aerobic bacteria but is very efective against anaerobes (Müller, 1983). Since anaerobes alone or in combination with aerobes cause 95% of odontogenic infections, metronidazole may have a major role in such infections. Although it has no effect on the streptococci that are likely to be involved, resolution of odontogenic infections treated with metronidazole is usually seen and occurs very rapidly (Wallace, 1979). If the drug is to be used in a serious odontogenic infection, it should be combined with penicillin because the *Streptococcus* has a more important role in those situations.

Natural History of Odontogenic Infection

The usual cause of odontogenic infections is necrosis of the pulp of the tooth, which is followed by bacterial invasion through the pulp chamber and into the deeper tissues. Necrosis of the pulp is the result of deep caries in the tooth, to which the pulp responds with a typical inflammatory reaction. Vasodilation and edema cause pressure in the tooth and severe pain as the rigid walls of the tooth prevent swelling. If left untreated the pressure leads to strangulation of the blood supply to the tooth through the apex and consequent necrosis. The necrotic pulp then provides a perfect setting for bacterial invasion into the bone tissue.

Once the bacteria have invaded the bone, the infection spreads equally in all directions until a cortical plate is encountered. During the time of intrabony spread, the patient usually experiences sufficient pain to seek treatment. Extraction of the tooth (or removal of the necrotic pulp by an endodontic procedure) results in resolution of the infection.

Direction of Spread of Infection

The direction of the infection's spread from the tooth apex depends on the thickness of the overlying bone and the relationship of the bone's perforation site to the muscle attachments of the jaws.

If no treatment is provided for it, the infection erodes through the thinnest, nearest cortical plate of bone and into the overlying soft tissue (Fig. 69-1). In Fig. 69-1, A, the nearest cortex is the thin labial bone, so the infection erodes through there. In Fig. 69-1, B, the root apex is closer to the palatal aspect, so the infection goes to the palatal side. If the root apex is centrally located, the infection erodes through the thinnest bone first. In the maxilla the thinner bone is the labial-buccal side; the palatal cortex is thicker.

Once the bone has been perforated, local muscle attachments determine the specific location of its expression in the soft tissue (Fig. 69-2). The most common "tooth abscess" erodes through the labial bone, occlusal to the muscle attachment, producing a vestibular abscess (Fig. 69-2). The vestibular abscess is seen as a small pouch of pus in the soft tissue overlying the affected tooth (Fig. 69-3). If no treatment is provided, rupture of the abscess occurs and a chronic sinus tract is established (Fig. 69-4).

In the infection perforates the bone above the muscle attachment (Fig. 69-2, points 2, 4, and 5), fascial space involvement occurs. When space involvement happens, the potential for more severe infections with rapid spread becomes greater.

Table 69-5 lists usual sites for perforation and localization of the infection (Laskin, 1964). These are the usual sites, but much variation occurs from patient to patient.

Fascial Space Involvement

Maxillary spaces

Erosion of maxillary tooth infection through the bone usually expresses itself in the labial-buccal surface of the maxilla. Most are seen as vestibular abscesses. Some, however, become fascial space infections. The two maxillary spaces that may be involved are the canine space and the buccal space.

The canine space becomes inected almost exclusively as a result of the maxillary canine tooth. The root of the tooth must be long enough so that the apex is superior to the insertion of the levator anguli oris muscle. The canine space is between the anterior surace of the maxilla and the levator labii superioris. When infected, clinically evident swelling lateral to the nose exists, usually obliterating the nasolabial fold (Fig. 69-5).

The buccal space becomes involved from the maxillary teeth when the infection erodes through the bone superior to the attachment of the buccinator muscle. The buccal space lies between the buccinator muscle and the skin and superficial fascia (Fig. 69-6). All three maxillary molars may cause buccal space involvement, but the premolars rarely do. The buccal space swelling is ovoid, below the zygomatic arch and above the inferior border of the mandible. The buccal space may also be infected from the mandibular molar teeth. This involvement is not common, but it does occur.

In addition to these two maxillary space involvements, maxillary odontogenic infections may ascend to cause orbital cellulitis or cavernous sinus thrombosis. Orbital cellulitis is rarely the result of odontogenic infection but may occur. The clinical picture is similar regardless of the cause. Swelling and redness of the eyelids, chemosis, and exophthalmos occur. Involvement of orbital contents includes both vascular and neural components.

Cavernous sinus thrombosis may also occur as a result of the superior spread of an odontogenic infection (Fielding et al, 1983). Spread to the cavernous sinus is hematogenous and may occur along an anterior or a posterior route (Fig. 69-7). The orbital veins lack valves, permitting blood flow in either direction. This allows contaminated venous drainage to the cavernous sinus. The usual cause of cavernous sinus thrombosis is from nonodontogenic sources. On rare occasions it may be the result of an infected tooth. The patient's signs and symptoms are like those of orbital cellulitis (Karlin and Robinson, 1984).

Mandibular spaces

Infection may erode from mandibular teeth into a variety of spaces, in addition to the usual vestibular abscess. The three primary spaces are the submental, sublingual, and submandibular spaces; the three secondary spaces are the pterygomandibular, masseteric, and temporal spaces.

The primary spaces are those into which infection spreads directly from the teeth and bone. The submental space lies between the anterior bellies of the digastric muscles and between the mylohyoid muscle and the skin. If the roots of the mandibular incisors are long

enough to cause the infection to erode through apically to the attachment of the mentalis muscle, the infection may proceed under the inferior border of the mandible to the posterior aspect into the submental space. This is not, however, a common occurrence.

The sublingual and submandibular spaces exit on the medial aspect of the mandible. They are usually involved by lingual perforation of infection from the mandibular molars. The factor determining whether the infection is in the sublingual or submandibular space is the relationship between the area of the infection's perforation and the location of the mylohyoid muscle's attachment (Fig. 69-8). If the location of the apex of the teeth is superior to that of the mylohyoid (premolars, first molar), the sublingual space is involved. If the apex of the tooth is inferior to the muscle (third molar), the submandibular space is involved. The second molar may involve either or both spaces, since its apex is typically at the mylohyoid line.

The subglingual space is between the oral mucosa and the mylohyoid muscle (Fig. 69-9). Its posterior boundary is open, and it can thus communicate freely with the submandibular space and the secondary spaces of the mandible. Clinically, when infection of the sublingual space occurs, little extraoral swelling occurs, but much intraoral swelling of the floor of the mouth develops on the affected side (Fig. 69-10). If the infection becomes bilateral, the tongue may become markedly elevated.

The submandibular space lies between the mylohyoid muscle and the skin and superficial fascia (Fig. 69-11). Like the sublingual space, it has an open posterior boundary and can communicate freely with the secondary spaces. When this space becomes infected, the swelling begins at the inferior lateral border of the mandible and extends medially to the digastric area and posteriorly to the hyoid bone (Fig. 69-12).

The three secondary spaces of the mandible are posterior to the tooth-bearing portion of the mandible in the angle-ramus area. They are called *secondary spaces* because they become infected by secondary spread of infection from other anterior spaces. The primary spaces feeding them are the buccal, sublingual, and submandibular spaces.

The masseteric space exists between the lateral aspect of the mandible and the masseter muscle (Fig. 69-13). This space is involved most often by spread from the buccal space or from soft tissue infection around the third molar. When it is involved, the posteroinferior portion of the face swells. In addition to the swelling, the patient has mild to moderate trismus caused by inflammation of the masseter muscle.

The pterygomandibular space lies between the medial aspect of the mandible and the medial pterygoid muscle (Fig. 69-13). This space becomes involved from spread from the sublingual and submandibular spaces and from soft tissue infection around the third molar. When this space is involved, little or no swelling is evident on either intraoral or extraoral examination. The patient almost always has significant trismus. Thus trismus without swelling is a valuable diagnostic clue for pterygomandibular space infection.

The temporal space is posterior and superior to the masseteric and pterygomandibular spaces (Fig. 69-13). Bounded laterally by the temporalis fascia and medially by the skull, it is divided into two portions by the temporalis muscle. The two sections are known as the *deep* and *superficial temporal pouches.* They are secondarily involved only rarely, in serious

overwhelming infections. Swelling is evident over the temporal area, posterior from the lateral aspect of the lateral orbital rim. Trismus is always a feature of this infection, caused by involvement of the temporalis muscle.

These three spaces are collectively known as the *masticator space,* since they are bounded by the muscles of mastication: masseter, medial pterygoid, and temporalis. The three individual spaces communicate freely with one another, so one rarely sees any single space involved alone. Thus the term *massticator space* does have some clinical usefulness, even if it lacks specific designation.

If all three of the primary mandibular spaces become involved with the infection, the infection is known as *Ludwig's angina.* Ludwig's angina, described in 1936, was a relatively common occurrence until the antibiotic era. It is a rapid, bilaterally spreading, gangrenous cellulitis of the submandibular, sublingual, and submental spaces. It usually spreads posteriorly to the secondary spaces as well. It produces gross swelling, elevation and displacement of the tongue, and tense, brawny induration of the submandibular region superior to the hyoid bone. There is usually little or no fluctuance (Finch et al, 1980; Patterson et al, 1982). The patient experiences severe trismus, drooling of saliva, tachypnea, and dyspnea. Impending compromise of the airway produces marked anxiety. The cellulitis can progress with alarming speed, producing an upper airway obstruction that may lead to death. The usual cause of Ludwig's angina is an odontogenic infection, usually from the mandibular second or third molar. The microbes involved are usually *Streptococcus,* oral anaerobes, or both.

Cervical (deep neck) spaces

Extension of odontogenic infection beyond the mandibular spaces is an unusual event. When it does occur, spread to the cervical or deep neck spaces from the submandibular, sublingual, or pterygomandibular spaces may have serious, life-threatening sequelae. These sequelae may be the result of locally induced complications, such as upper airway obstruction, or distant problems, such as mediastinitis.

The deep neck spaces can become infected from a variety of sources. Odontogenic infections cause as much as 30% of all deep neck infections (Virolainen et al, 1979).

The deep neck spaces have a variety of names and descriptions. Three are relatively consistent through the literature: the lateral pharyngeal space, the retropharyngeal space, and the prevertebral space, or danger space No, 4. The layers of deep cervical fascia form and bind these three spaces.

The lateral pharyngeal space is classically described as having the shape of an inverted pyramid or funnel. The base is the skull base at the sphenoid bone, and the apex is at the hyoid bone. It is located between the medial pterygoid muscle laterally and the superior pharyngeal constrictor medially (Fig. 69-14). Anteriorly the boundary is the pterygomandibular raphe, around which it communicates with the spaces of the mandible. Posteromedially it extends to and is bounded by the prevertebral fascia and communicates freely with the retropharyngeal space. The styloid process and associated muscles and fascia divide the lateral pharyngeal space into an anterior compartment, which contains muscles, and

a posterior compartment, which contains the carotid sheath and cranial nerves.

When the lateral pharyngeal space is involved in an odontogenic infection, there are several typical findings. First and foremost is severe trismus. This is the result of involvement of the medial pterygoid muscle but may be caused by involvement of the other muscles of mastication as well. The severe trismus may interfere with accurate diagnosis and treatment. Lateral neck swelling, especially beyond the angle of the mandible, is usually seen. The lateral pharyngeal wall, if it can be visualized, usually bulges toward the midline. One can differentiate it from a primary peritonsillar abscess primarily because the latter rarely has significant trismus.

Involvement of the lateral pharyngeal space creates complications. First is the fact that the odontogenic infection is severe and may be progressing at a rapid rate. Second is the direct effect of the infection on the contents of the space, particularly of the posterior compartment. This includes thrombosis of the internal jugular vein, erosion of the carotid artery or its branches, and interference with cranial nerves IX to XII or the sympathetic chain. Third is that the infection may progress from the lateral pharyngeal space to the retropharyngeal space.

The retropharyngeal space lies posteromedial to the lateral pharyngeal space. It is bounded anteriorly by the superior pharyngeal muscle and its investing fascia and posteriorly to the alar layer of prevertebral fascia (Fig. 69-14). The space begins at the skull base at the pharyngeal tubercle and extends inferiorly to the level of C7 or T1, where the two layers of fascia fuse (Fig. 69-15). This level is at the posterosuperior mediastinum. The retropharyngeal space has few contents, save for the retropharyngeal lymph nodes. These nodes are more numerous in the child than in the adult, which may account for the more frequent involvement of this space in children (Barratt et al, 1984).

When the retropharyngeal space becomes involved secondary to odontogenic infection, the situation is almost always grave. Clinical signs and symptoms are those of a severe infection. Trismus is severe in essentially all patients at this stage. Evaluation of the retropharyngeal space is performed with the greatest success by evaluating a lateral radiograph of the neck. Average widths of the prevertebral tissues have been well established (Wholey et al, 1958). The soft tissue shadow should be no more than 7 mm (average: 3.5 mm) at C2 and no more than 20 mm (average: 14 mm) at C6, that is, behind the trachea (Fig. 69-16).

Involvement of the retropharyngeal space may also include the prevertebral space. The prevertebral space is a potential space between the two layers of prevertebral fascia, the alar and prevertebral layers. It extends from the skull base inferiorly to the diaphragm. The space is also known as the *danger spare No. 4* (Grodinsky and Holyoke, 1938).

When the retropharyngeal space is involved as a result of an odontogenic infection, the patient is seriously ill and is in grave danger of death. Three great potential complications exist. First, the upper airway is in danger of obstruction as a result of anterior displacement of the posterior pharyngeal wall into the oropharynx. Narrowing of the upper airway as the retropharyngeal space swells is expected (Fig. 69-16). Second, when the retropharyngeal spaces are filled with pus, a danger exists of spontaneous rupture of the abscess, resulting in aspiration, pneumonia, and asphyxiation. Rupture may also be caused by attempts at insertion

of an endotracheal tube to secure the airway. Third, once the infection has gained access to the retropharyngeal spaces, the posterosuperior mediastinum or the entire posterior mediastinum may become infected also (Fig. 69-17).

Management of Odontogenic Infections

The principles of treating odontogenic infections are no different from the principles of managing any other infection. These principles are briefly reviewed in the context of treating odontogenic infections that are serious enough to warrant hospitalization.

Assessment and support of host defenses

Odontogenic infections are almost always minor and are easily treated. They become serious infections more commonly in patients who have some defense compromise, such as diabetes or immunosuppression. Careful review of the patient's defenses should be a routine part of the patient's evaluation.

Patients with moderate or severe odontogenic infections usually have severe pain and dysplasia. This results in a poorly hydrated, exhausted patient. Thus the infection itself produces a compromised host. Care must be taken to provide proper analgesics, nutrition, and hydration for patients with these infections.

Airway establishment

Fascial space involvement that compromises the airway may be anterior, as in Ludwig's angina, or posterior, as in a retropharyngeal space infection. In either case, rapid severe compromise of the airway may occur. The surgeon must be keenly aware of this fact, carefully and frequently evaluating airway status to prevent fatal obstruction.

In the uncomplicated odontogenic infection that involves the spaces of the mandible unilaterally, airway problems are rarely seen. If they are seen, nasal endotracheal intubation is the method of choice for airway establishment. Consideration should be given to leaving the endotracheal tube in place for 2 to 3 days to ensure that acute obstruction does not occur in the time shortly after surgical decompression of the infection.

In patients with Ludwig's angina, airway embarrassment is the primary cause of death. Once Ludwig's angina is diagnosed, it is imperative that the patient be monitored frequently and very carefully for airway problems. If the decision is made to establish an artificial airway early, intubation by fiberoptic or blind nasal technique can be attempted. These procedures should be carried out on awake, unparalyzed patients. Administration of neuromuscular blocking agents may cause airway loss that cannot be regained. This can be safely accomplished only if an experienced anesthesiologist is present. If the decision to establish an artificial airway is delayed, a tracheostomy is the only option. An unhurried approach with a local anesthetic is preferred.

If the infection involving a retropharyngeal space abscess has a radiographically enlarged soft tissue image, an artificial airway must be seriously considered. If the patients has only mild trismus and the anesthesiologist feels that direct laryngoscopy is possible, an

endotracheal tube is preferred. However, if the patient has moderate or severe trismus, a planned tracheostomy should be performed. The major concern in this situation is rupturing the abscess while attempting to pass the endotracheal tube into the trachea blindly. Should such a rupture occurs, aspiration of the pus is possible, with its attendant serious sequelae.

Identification of bacteria

The identification of the causative microorganisms in an odontogenic infection is not usually a problem. As indicated earlier, the usual causes are *Streptococcus* and oral anaerobes. Because the data are so consistent among the various reports and the clinical experience of many surgeons, it is unnecessary to perform cultures for uncomplicated odontogenic infections in uncompromised patients.

However, in compromised patients and in patients with severe fascial space infections, it is wise to get a specimen of pus for culture and antibiotic sensitivity testing. The speciment *must* be taken in an anaerobic method so that anaerobic culturing can be done. Aspiration with a large-bore needle through intact skin or mucosa is the preferred method.

Choice of antibiotic

The drug of choice for odontogenic infections continues to be parenteral penicillin. Even for serious fascial space infections, including Ludwig's angina (Finch et al, 1980; Patterson et al, 1982), penicillin is preerred. Large doses of up to 20 million units daily for intravenous penicillin may be required for serious infections.

In the penicillin-allergic patient, clindamycin is the second drug of choice. It is quite effective against streptococci and anaerobes and as low toxicity. Although clindmycin may cause pseudomembranous colitis, it causes only about one third of the reported cases. (About one third are caused by ampicillin/amoxicillin and one third by the cephalosporins.) The surgeon must be alert to this potential complication and prepared to treat it or to seek consultation if it occurs. Metronidazole alone or in combination with penicillin is also a very useful drug. It has minimal toxicity problems and is very effective against anaerobes. Parenteral cephalosporins may be moderately useful. The first-generation cephalosporins have the same effect on the microbial population causing odontogenic infections that penicillin does. The second-generation drug cefoxitin is more active against the anaerobic bacteria but loses some of the anti-streptococcal activity of the first-generation drugs. The third-generation cephalosporins are generally effective against anaerobes but also have decreased effectiveness against streptococci. Further, they are quite expensive and have no clear additional benefit. Thus the second- and third-generation drugs are not highly desirable. Ciprofloxacin and metronidazole in combination may be quite useful in the patient with a severe infection who has had an anaphylactoid reaction to penicillin.

In summary, high doses of parenteral antibiotics with a penicillin-like spectrum are usually effective in treating odontogenic infections. Specific culturing should be done for serious or nonresponsive infections, and the results used accordingly.

Surgical drainage and decompression

Vestibular infections

Odontogenic infections that are confined to the oral vestibule can usually be treated in a straightforward manner. The patient usually complains of mild to moderate soft tissue pain and swelling. There may be a mild but rarely severe elevation of body temperature.

Treatment is primarily surgical, with antibiotic therapy often used adjunctively. Surgical treatment is extraction of the tooth (or removal of the dental pulp of the tooth) and incision and drainage of the vestibular abscess. A small piece of Penrose drain is inserted to maintain drainage or 2 or 3 days. Antibiotic treatment is usually with penicillin. If the patient is allergic to penicillin, either erythromycin or clindamycin can be used effectively. Antibiotic administration usually lasts for 7 to 10 days. Such patients can almost always be managed on an outpatient basis. Resolution of the infection is rapid and usually without complications.

Jaw space infections

If the infection spreads to one of the fascial spaces surrounding the maxilla or mandible, the treatment must become more aggressive.

The initial step in therapy is to make a decision as to whether hospitalization is necessary. This decision is based on the presence and amount of swelling, the nature of the swelling (soft and doughy, indurated, or fluctuant) and the state of the body's defenses. Infections that appear as chronic abscesses without a major cellulitis component can be surgically drained and the patient observed on an outpatient basis. Similarly, patients with early infections having a soft, doughy swelling can be treated as outpatients with antibiotics and tooth extraction.

However, patients who have moderately elevated temperatures with an odontogenic cellulitis that is diffuse and indurated should be managed in the hospital setting. Support of the host with hydration, antibiotics, and analgesics plays an important role in the overall therapy. Antibiotics should be given parenterally, preferably intravenously.

Surgical treatment should be considered early. If any fluctuance is noted on the physical examination, the patient should be taken to the operating room and drainage procedures performed. Aspiration of pus for aerobic and anaerobic cultures is accomplished first. Drainage of the canine and isolated sublingual spaces is usually performed with a transoral approach. The incision is made, an exploration is carried out by blunt dissection to break up all loculation of pus, and the Penrose drains are inserted.

The buccal space can be drained transorally or extraorally, depending on the specific patient situation and the preference of the surgeon. Surgery from the mouth side is more difficult, with maintenance of the drainage becoming less predictable. Drainage from the cutaneous side is more likely to leave a scar.

The submental space is drained via a horizontal incision, which parallels the inferior border of the symphysis of the mandible (Fig. 69-18). The area between the anterior belly of

the digastric muscles is explored posterior to the hyoid bone.

The buccal, submandibular, masseteric, pterygomandibular, and sublingual spaces can all be drained via a horizontal incision parallel and inferiro to the angle of the mandible (Fig. 69-18). An incision from as short as 0.5 cm to as long as 5 cm may be used. If the infection is a tense cellulitis that involves several mandibular spaces, a larger incision is indicated. After the incision is made, blunt dissection is used to explore the involved spaces. The most commonly missed space is the pterygomandibular space. In severe submandibular space infections, specific efforts should be made to explore the medial side of the ramus of the mandible in the pterygomandibular space. The inferior portion of the lateral pharyngeal space just medial to the pterygoid muscle can also be explored by this approach. In severe infection, anterior (submental), posterior (submandibular), and superior (superficial temporal) incisions may all be required. Penrose drains are inserted to the extent of the dissection to facilitate drainage. Irrigation/suction catheters may be useful in more severe infections (Flynn et al, 1983). In infections drained by multiple incisions, through-and-through drains should be used.

Resolution of these infections depends on several factors: conditions o host defenses, seriousness of the original infection, appropriateness of antibiotic therapy, and extensiveness of surgical exploration. Of these, a failure to perform adequate surgical drainage is most likely to occur. In severe cellulitis, overlying induration may prevent the clinical diagnosis of an abscess formation. Thus surgery is critical to successful therapy in those infections characterized by extensive cellulitis (Beck et al, 1984; Dzyak and Zide, 1984).

Deep neck spaces

Involvement of the deep cervical spaces as a result of odontogenic infection is an uncommon but life-threatening event. A large proportion of deep neck infections are the result of odontogenic infections (Beck et al, 1984). When deep cervical involvement does occur, the fascial spaces of the jaws, particularly of the mandible, are involved initially, usually beginning in the submandibular space and extending through the pterygomandibular space. The major diagnostic goal is to determine whether the lateral pharyngeal and retropharyngeal spaces are involved. The clinical examination, radiography (using anteroposterior (AP) and lateral soft tissue views of the neck, Figs. 69-16 and 69-19), and computed tomography (CT), (Fig. 69-17) may all be required to make the diagnosis (Endicott et al, 1982). Lateral and AP neck radiographs and CT scans may reveal the presence of gas in the soft tissue as well as soft tissue swelling. The presence of gas in the tissue usually signifies anaerobic infections and indicates immediate and aggressive surgical therapy (Haug et al, 1990).

Initial treatment of a patient suspected of having a deep neck infection requires immediate hospitalization, host support, maintenance of airway, IV antibiotic therapy, and incision and exploration of the involved spaces. Early surgical exploration, even in the absence of palpable fluctuance, is likely to produce more rapid and complete resolution of the infection with minimal mortality. As noted earlier, an artificial airway in these patients must usually be established. There must be no hesitation in accomplishing the necessary maneuver, whether it be intubation or tracheostomy. Large doses of parenteral antibiotic therapy must be started immediately. Penicillin with metronidazole is the preferred combination; clindamycin is an adequate alternative. If doubt exists concerning the bacterial cause of the infection, gentamicin or a third-generation cephalosporin may be added.

Medical management with antibiotic therapy and host defense support may be employed initially without surgery; however, a rapidly progressing infection of the neck should be explored. If gas emphysema, a foul-smelling discharge, or other signs indicating anaerobic infection exist, the indication for surgical intervention increases. If the infection continues to progress rapidly in spite of aggressive antibiotic therapy, surgical drainage becomes more important. If fluctuance is noted, pus should be drained immediately (Beck et al, 1984; Dzyak and Zide, 1984).

Drainage of the lateral pharyngeal space is best accomplished by a combined transoral-extraoral approach. An incision is made lateral to the pterygomandibular raphe, and the space is explored by blunt dissection posterior and inferior to the angle of the mandible with a long Kelly hemostat. A skin incision is made over the clamp tip along the anterior border of the sternocleidomastoid muscle (see Fig. 69-18). Loculations of pus are disrupted by blunt dissection with the hemostat and by finger pressure. Through-and-through drains are placed.

The retropharyngeal space can be drained through transoral incision through the posterior pharyngeal mucosa. If this approach is used, a cufed endotracheal or tracheostomy tube must be in place to prevent any aspiration of pus. Extraoral procedures facilitate dependent drainage. An incision is made along the anterior border of the sternocleidomastoid muscle. The space is explored by blunt dissection between the carotid sheath and pharyngeal constrictor muscle. Deep drains are placed as usual.

When surgical treatment was the only method of therapy (before the antibiotic era), early and aggressive surgery was essential to treat these infections. However, it is important to realize that although aggressive antibiotic therapy may reduce the need for extensive surgical exploration in many patients, antibiotics rarely replace the need for surgery completely.