Textbook of Oral and Maxillofacial Surgery

Gustav O Kruger

(The C V Mosby Company, St Louis, Toronto, London, 1979)

Fifth Edition

Chapter 11

Acute infections of the oral cavity

Sanford M Moose, Keith J Marshall

The subject of acute infections of the oral cavity could in itself occupy a sizeable volume if descriptions of the diagnosis and treatment of all acute infections found in the oral cavity were included.

Many of the acute inflammatory processes manifest in the oral cavity provide evidence of acute infection by microorganisms. However, inflammatory conditions of the oral mucosa are also among the cardinal symptoms of dermatologic disease and other systemic disorders that are beyond the scope of a discussion of acute infections of the oral cavity. It is important therefore to realize that the oral and other mucous membrane surfaces of the body may provide early visible signs of disease other than primary infections of oral tissues. Many exanthematous diseases of childhood have primary lesions in the oral cavity that may become acutely inflamed as well as secondarily infected as a result of disruption of the integrity of the defense mechanisms provided by an intact mucous membrane.

Any primary systemic condition capable of adversely affecting the oral mucous membrane may play an important part in the etiology of oral infection as well as present a symptom complex manifested in inflammatory changes of oral mucosal surfaces.

Lesions of immunopathological origin are seen often in oral mucous membranes. As the frequency and complexity of drug administration in the treatment of disease states have increased, so the variety and incidence of mucosal lesions caused by untoward reactions to these agents have increased. Food allergy has been identified as important in the genesis of oral lesions. Autoimmune disease mechanisms affecting oral tissues have been implicated in the production of alterations of mucosal surfaces. It has been recognized for a long time that nutritional deficiency diseases may give rise to ulceration and other changes of oral mucosa, either affecting surface epithelium or underlying connective tissue.

The Occupational Safety and Health Administration of the United States Department of Labor and the United States Department of Agriculture are important sources of information on nutrition and also for information regarding chemical poisons that may cause oral lesions.

Diseases that alter the body's defense mechanisms frequently result in secondary oral infections. Suppression of the immune system, either by diseases that affect the hemopoietic

tissues or by steroids and other immunosuppressive agents administered in the treatment of disease states, may be responsible ultimately for the development of oral infections and other alterations of oral mucosa. Malignant diseases themselves or the iatrogenic diseases generated in their treatment always are important considerations in the evaluation of oral lesions.

Local diseases may play an important role in the production of acute infections of the oral and maxillofacial apparatus. Odontogenic tumors and cysts may escape detection until an acute inflammatory lesion becomes superimposed. Acute infection may be the ultimate lesion of the maxilla or mandible, but invasion of adjacent soft tissues of the cheek, lips, tongue, and soft palate is not infrequent. Less common, but certainly significant, are more distant extensions of infection by metastasis, which may result in mortality or permanent injury.

When considering the panorama of etiologic factors contributing to the production of acute infections of the oral cavity, the practitioner must be alert to suspect obscure causes rather than to jump to obvious but possibly incorrect conclusions on the basis of superficial appearances and insufficient investigation. While visual inspection is indispensable in the evaluation of oral lesions, a carefully elicited history often reveals the information required for an accurate diagnosis, and unfortunately this is one of the elements most apt to be neglected.

Acute Infections of the Jaws

Periapical abscess

The periapical abscess, commonly called an acute alveolar abscess, usually begins in the periapical region and is caused by a necrotic pulp. It may occur almost immediately after injury to the pulpal tissues, or, after a long latent period, it may suddenly flare into an acute infection with the symptoms of inflammation, swelling, and fever.

Factors that cause some periapical lesions to suddenly become acute are not understood, although many theories have been advanced regarding this transition. The fact simply remains that a nonsymptomatic tooth today may cause extreme distress requiring definitive treatment at any time in the future.

Although the symptoms producing distress are often confined to the immediate region of the tooth, occasions arise when toxins released by interaction of the infective agent and host defense mechanisms produce a systemic reaction sufficient to render a patient generally ill. Periapical abscesses may confined themselves to the osseous structures and during the early period of abscess formation may cause excruciating pain without any observable swelling. However, even though many cases may start initially in this manner, the abscess may burrow through the cancellous and cortical bone until it reaches the surface and invades the soft tissues in the form of either a subperiosteal or supraperiosteal abscess.

Prior to actual abscess formation, however, the infection is capable of producing a cellulitis in the soft tissues of the region involved. The palpable soft tissues of the oral and maxillofacial area appear hard and dense. This brawny condition is known as induration. Until

the infectious process becomes well circumscribed in the form of an abscess, the patient is usually in extreme discomfort.

Treatment should be directed toward localizing the infection during the period of induration, confining the infection to the region of onset, and eliminating the etiology of the infection. Early employment of appropriate antibiotic therapy in adequate dosage may be extremely important in a severe and life-threatening infection. Localization of the infection may be aided by using warm compresses and warm mouth rinses at frequent intervals. It is only when localization takes place that an abscess should be surgically incised and drained. Physiologically, it is at this time that nature has constructed a barrier around the abscess, walling it off from the circulation and making it possible to palpate the presence of fluid purulent material within the abscess cavity. The deeper the abscess forms within tissue, the more difficult it is to palpate.

An early decision should be made on the most desirable disposition of a tooth or teeth involved in the development of an acute infection. After such teeth have been carefully evaluated, if their retention is indicated, the necrotic pulp canals should be opened in an effort to relieve the pressure generated within a closed chamber. If this fails to achieve the desired relief, the offending tooth should be extracted. The sooner this is accomplished the more rapidly may the symptoms be expected to subside.

The philosophy of never extracting a tooth in the presence of an acute infection has long been abandoned. It should be realized that frequently the best route through which the abscess can be drained is through the alveolus from which the tooth is extracted. The alveolar bone in such cases is so dense and resistant to further penetration of the abscess that the infectious process is confined, increasing the symptoms until extraction is the only recourse. Under such circumstances, pus is usually observed flowing from the alveolus immediately after the extraction. If the extraction is delayed, a possibility exists that the infection will diffuse into tissues remotely located from the original site, and that septicemia or osteomyelitis or both will develop.

When definitive action is indicated in the presence of acute infection, the patient should be protected by the administration of adequate doses of an antibiotic to ensure a rapid and sustained blood level. Selection of the appropriate antibiotic should be made, whenever possible, on the basis of identification of the microorganism and its sensitivity to available antibiotics. When time does not permit complete culture and sensitivity determination, the clinical and gram-staining characteristics of the organisms, if any specimen can be obtained, provide a better basis for antibiotic selection that purely empirical selection. Multiple extractions or extensive surgery should be postponed until the remission of acute symptoms.

When an abscess has formed or is induced to localize and the infectious process invades the extra-alveolar tissues, it should be incised simultaneously with the extraction of the tooth. If the tooth is to be retained, the palpable abscess should be incised and drained simultaneously with the opening of the pulpal chamber. If a fluctuant localized abscess is palpable intraorally and is in the region of the maxillary or mandibular buccal vestibule, then the choice of drainage incision should be at a point below the most fluctuant portion of the abscess. On the other hand, if the abscess should localize or point subperiosteally or

supraperiosteally on the palate or on the lingual surface of the mandible, then the site of incision should be chosen in deference to the neurovascular structures found on these surfaces.

When the presence of important anatomical structures becomes a hazard, then the incision should be made with a sharp scalpel through only the superficial tissues, followed by blunt dissection with a hemostat into the abscess cavity and down to the bony surface. With the hemostat closed, the point is forced through the incised surface into the abscess cavity and then opened, and the aperture is dilated to permit the installation of adequate-sized drainage material. Small openings for the purpose of draining abscesses are entirely unsatisfactory and do not permit adequate drainage. A large opening for the drainage of most abscesses should permit the end of the gloved index finger to be admitted to the bony surface from which the abscess has emerged.

Pericoronal infections

Although pericoronal infection may occur at any time throughout life, pericoronal infections occur commonly during infancy, childhood, and young adulthood. Pericoronal infection in infancy is associated with tooth eruption, when the supradental tissue involving the superior portion of the follicle and overlying mucoperiosteum may become inflamed and ultimately develop into a fluctuant abscess. Occasionally these abscesses may develop into a cellulitis, causing not only local but systemic reactions associated with fever. When fluctuance can be visibly and digitally ascertained, incision and drainage followed by warm saline mouth rinses held over the area at frequent intervals usually give prompt relief so that no further treatment is necessary. Similar conditions may occur at any time during the eruption period of permanent teeth and should be managed in the same manner.

The type of pericoronal infection less frequently encountered occurs in lade adult life in an edentulous ridge. For some reason a tooth has failed to erupt, and a denture has been constructed for the patient, either because the existence of the unerupted tooth was not known or in the belief that the tooth could remain asymptomatic in the edentulous jaw.

It is generally believed that the cause of the acute infection associated with such teeth is the result of pressure from the denture over a period of years. At the onset of a patient's denture wearing, such embedded teeth are in all probability a sufficient distance from the surface to be unaffected by the pressure reaction from the denture. However, as time goes on, with the resultant resorption of the ridge, the bone and soft tissue between the denture and the embedded tooth is ultimately subjected to the inflammatory influences of denture pressure and motion.

When an acute infection occurs under these circumstances, a different course of treatment is indicated. If a fluctuant abscess occurs, overlying the crown of this embedded tooth, it should be incised and drained and a sufficient time allowed for the acute condition to become subacute. At this later time, surgical removal of the unerupted tooth is indicated.

The most common type of pericoronal infection is the one found around the mandibular third molar. This occurs most frequently in adolescents and young adults. Symptoms accompanying this type of pericoronal infection are variable, and it is not unusual for patients to experience their only symptoms in the peritonsillar region. For this reason they

seek the services of a physician, believing they have a sore throat or tonsillitis. The interesting aspect of this type of pericoronal infection is that it actually does have peritonsillar symptoms marked enough that visual symptoms lead to the diagnosis of peritonsillar abscess or streptococcal sore throat, for which the patient is not infrequently hospitalized and treated accordingly. Repetition of these symptoms may occur for several years before an unerupted third molar is finally diagnosed as the offender.

The most typical symptoms of pericoronal infection of the third molar are submandibular lymphadenopathy, trismus, pain in the region of the third molar, and a general condition of malaise not infrequently attended with a moderate elevation of temperature. These symptoms vary in degree from mild to extreme to a point at which the patient may suffer extreme pain and may develop a cellulitis capable of producing difficulty in swallowing, extreme tenderness to palpation extraorally and intraorally, and edema visible both in the submandibular and pharyngeal regions. When symptoms of this type occur, the tooth or a surface of the unerupted tooth is usually close to the surface. A visible portion of the tooth may be exposed to the oral cavity. The communication to the oral cavity may be so obscured by edema and the general inflammatory process that this communication may be ascertained only by the use of a probe.

More frequently than not, this communication permits the probe to be advanced along the buccal aspect of the unerupted tooth. Careful palpation with the probe permits entry into an expanded follicular space. After dilation of the aperture of entrance, the evacuation of pus and other septic material is made possible. If the aperture can be dilated sufficiently to permit drainage, the insertion of a 6-mm wide drain made from rubber dam material or Penrose drain or a 6-mm strip of iodoform gauze moistened with eugenol or guaiacol and olive oil should permit continuous drainage and provide adequate analgesia. The patient should be instructed to use warm saline mouthwashes for 5 minutes at half-hour intervals until retiring. If adequate localization of the infection has not occurred, antibiotic therapy is indicated immediately in a dosage and by a route of administration sufficient to assure a rapid, adequate blood level. This should provide prompt relief from the acute symptoms, and as soon as a subacute condition exists, definitive treatment may be instituted.

Definitive treatment, of course, will depend on the judgment concerning the final disposition of the unerupted tooth. If the third molar is impacted, surgical removal should be performed as soon as symptoms become subacute. If the tooth is not impacted but has been recurrently troublesome without eruption and it is ascertained that insufficient room exists for adequate eruption space in the patient's mouth, then extraction is also indicated.

If, however, the first molar is missing or has a poor prognosis and the second molar similarly may not be a suitable abutment tooth for prosthetic replacement of missing teeth anterior to the second molar, the third molar assumes a more important potential role. If marginally adequate space for eruption exists, such a third molar may be permitted to erupt while under other circumstances extraction would be the treatment of choice. Consideration should be given to the persistent or recurrent nature of inflammatory episodes as well as their severity and to possible serious hazards to a patient's oral and general health prior to elective retention. If retention is elected, it may be necessary to excise inflamed or fibrotic tissues overlying and surrounding the occlusal portion of the tooth.

If excision of the overlying tissues is decided on, it should be done adequately. All overlying tissues should be thoroughly excised, and the occlusal portion of the unerupted tooth should be completely exposed. After excision has been completed the wound should be packed with a surgical dressing. This should be allowed to remain approximately 7 days.

The time for the employment of a definitive surgical procedure has been a controversial subject for a number of years, but since the advent of the chemotherapeutic and antibiotic era, patients can be protected from violent postoperative systemic reactions, acute cellulitis, and osteomyelitis by the use of antimicrobial agents. Delay of surgery is believed to play a significant role in permitting an osteomyelitic infection to become established. Consequently, discretion should be employed in the selection of a suitable time for surgical action. In the presence of acute, fulminating infection, antibiotic therapy and early incision and drainage is the method of choice, followed by definitive surgical treatment of the condition as soon as it becomes subacute.

The maxillary third molar can be a contributing factor to pericoronal infections of an unerupted mandibular third molar. In the examination of a patient with a pericoronal infection associated with a mandibular third molar, it is imperative that the region of the maxillary third molar be examined to see whether it has erupted, whether it is in malalignment, or whether it has unerupted as a result of the delayed eruption of the mandibular third molar. It must be determined whether room is present in the mandibular retromolar area of the patient's jaw for the eruption of the mandibular third molar and if the presence of the maxillary third molar is a continuous source of trauma to the soft tissue in the mandibular retromolar area during the eruption period of the mandibular third molar.

On occasion it is found desirable to retain the mandibular third molar even when recurrent infectious episodes persist from the traumatic influence of the erupted maxillary third molar. In such cases it is desirable to remove the maxillary third molar if its presence is a continual source of trauma. Pericoronal infections occur less frequently around an erupted or unerupted maxillary third molar, but when they do, the same procedure should be employed as the one described for the management of the mandibular third molar.

Dissecting subperiosteal abscess

A certain type of subperiosteal infection may occur several weeks after an apparently uneventful healing of a mandibular third molar wound. This may present itself primarily as an indurated swelling in the mucoperiosteal tissue as far forward as the first molar or second bicuspid. It may become progressively edematous and indurated and finally develop into a fluctuant, palpable, and visible subperiosteal abscess that has migrated from the original third molar wound beneath the periosteum to the point of fluctuance. When the diagnosis is made, antibiotic therapy should be initiated, and as soon as the fluctuance is palpable, the abscess should be incised and drained. This condition may be externally visible and palpable as a swelling of the cheek in this region.

The drainage incision should start from the point of origin, which is the third molar region deep in the buccal vestibule, and extend anteriorly to the point of fluctuance. The incision should be made through the mucoperiosteum down to the bone. Tissues on either side of the incision should then be expanded with a hemostat, and the wound, along the length of

its entire course, should be packed open with iodoform gauze impregnated with a suitable analgesic, antiseptic, lubricating agent (equal parts balsam of Peru and castor oil) or other suitable agents having similar properties. This type of packing material should not be changed daily but should be allowed to remain, thereby keeping the wound expanded and permitting the purulent material to drain out around it. It should be observed at 48-hour intervals, and the dressing should be allowed to remain in place for approximately 7 days. If the dressing is expelled from the wound during this period by action of the muscles of mastication, it should be replaced so that the wound remains saucerized and therefore will heal by granulating from the depth of the wound.

Killey and co-workers have published a comprehensive in-depth study on various types of subperiosteal infectious invasions and their treatments and end results.

Medial mandibular angle postoperative abscess

A medial mandibular angle postsurgical abscess may occur several days after the surgical removal of a mandibular third molar. It is accompanied by extreme discomfort, trismus, and difficulty in swallowing. The symptoms become progressively worse until it is with great difficulty that the patient is able to open his mouth for adequate examination. Whenever symptoms of this type are present and no symptoms are visible on the facial or occlusal surface of the wound, one may suspect this lingual type of abscess. It therefore becomes necessary either by persuasion under adequate sedation or by application of force to open the mouth sufficiently to permit digital examination of the medial surface of the angle of the jaw.

Inspection will reveal an extremely tender swelling of the tissues. The pain is severe. When fluctuance is determined, a small, curved, closed hemostat should be forced down through the wound of the third molar. It is introduced between the periosteum and the lingual surface of the bone, and, by sliding along the bone, the hemostat is advanced inferiorly and posteriorly until an abscess cavity is encountered. At this point the hemostat should be opened widely to dilate the tract through which the infection has descended into this region. If surgical intervention is correctly timed, pus will immediately be seen emerging from the cavity after withdrawal of the hemostat. A small, round-tipped brain-type aspirator may now be inserted into the cavity to aspirate additional amounts of pus that may be present. A gentle compression of the soft tissue under the angle of the jaw may also expel pus through the intraoral aperture.

After this, a small piece of Penrose drain or rubber dam material may be inserted deep into the region of the abscess with the end protruding slightly into the third molar wound, where it may be sutured without occluding the aperture. Whenever possible, a foreign material that may become dislodged should be secured to prevent aspiration and possible serious respiratory obstruction or respiratory infection. Antibiotic therapy initiated as early as possible may be an important adjunct to the surgical incision and drainage, but it is no substitute for indicated surgical intervention.

Acute infected emphysema

Acute infected emphysema is usually caused by the indiscreet use of air-pressure syringes or atomizing spray bottles activated by compressed air. In drying out a root canal with a compressed air syringe, septic material may be forced through the apical foramen into the cancellous portion of the alveolar process and ultimately out through the nutrient foramina into adjacent soft tissues, resulting in formation of a septic cellulitis and emphysema.

A similar condition can be induced by the use of a compressed-air spray bottle for irrigation of wounds, particularly in the retromolar region. If enough pressure is applied, it is possible to force air and septic materials through the fascial planes into the surgical spaces, which, after being forced open, remain in communication with this septic region. It is safer to use a hand-activated syringe when irrigating wounds or drying root canals since it is unlikely that an emphysema would be produced under these circumstances. Similarly it is important to avoid the use of any air-driven dental handpiece that exhausts compressed air within the oral cavity if a soft tissue wound is present. Conventional air-driven spray devices are totally unsatisfactory as surgical handpieces and may be very dangerous.

Periodontal abscess

Acute periodontal abscesses are usually the culmination of a long period of chronic periodontitis. This type of infection usually starts in the gingival crevice at the surface and extends down one or more surfaces of the roots, frequently as far as the apical region. Acute episodes usually have a sudden onset with extreme pain, and they are associated with swelling of the soft tissues overlying the surface of the involved root. For some unknown reason the tissues apparently seal themselves off at the gingival surface, impeding the drainage of the abscess and causing the distention and discomfort that is usually the patient's first indication of this condition.

A periodontal abscess may or may not be associated with nonvital teeth or traumatic injury, either external or occlusal. It may, however, be induced by the traumatic influence of a partial denture. The primary treatment for relief of acute symptoms is incision of the fluctuant abscess from the depth of the abscess cavity to the gingiva. The incision should extend through the soft tissues to the root surface, which has been previously denuded by the presence of the infectious process. If one or more surfaces of the root of such a tooth have been denuded to a point beyond the apical third of the tooth, extraction is indicated. If a third or more of the investing bone appears normal, then an evaluation of the potential usefulness of such a tooth should be made by considering all factors involved, including the general condition of the patient's health and his regenerative and resistance capacity.

Definitive treatment by debridement of root surface and removal of granulation tissue and treatment for new attachment and tissue regeneration should be deferred until the acute phase of the infection has subsided.

A lateral periodontal abscess can produce an infection capable of diffusing from the original offending tooth through the alveolar bone to involve several teeth on either side of the offender, rendering them extremely mobile and tender. Frequently this can baffle the most astute diagnostician and make identification of the primary offending tooth difficult.

Radiographs are, of course, a primary aid to diagnosis. Frequently, however, the lateral surface of the root involved is so obscured by the tooth structure that a radiograph contributes little toward the ultimate diagnosis.

Acute Cellulitis

When infection invades tissues, it may remain localized if the host resistance factors in the region are capable of walling off the infection and preventing it from spreading. In such cases, a physiological barrier is formed around the nidus of infection and it is either resolved and drained off in the lymphatic circulation or suppuration occurs, at which time surgical drainage is indicated.

Occasionally the bacterial infection is overwhelming, or the bacteria are either extremely virulent or resistant to antibiotic therapy. The resistance of the host tissues may be minimal, and bacterial invasion under these circumstances is unimpeded as it progresses through surrounding tissues to areas remove from the original site of infection. When physiological response fails to control the invasion of infection and therapeutic agents prove futile, then death ensues.

An acute cellulitis of dental origin usually is confined to the general area of the jaws. Tissues become grossly edematous and often hard to palpation. At this period the infection has not localized, and, during this stage, suppuration has not occurred.

The patient may show a severe systemic reaction to the infection. The temperature is usually elevated, the white cell count is increased, and the differential count may be altered. The erythrocyte sedimentation rate is usually increased, and the pulse rate is accelerated. The electrolyte balance is changed, and the patient frequently experiences malaise.

When the invasive process is overwhelmed by the physiological defense, resolution is achieved. Frequently, a specific antibiotic can complete resolution of the process, and either no pus is formed or the small amount present can be removed by the lymphatic circulation. Usually, however, a massive cellulitis will ultimately suppurate, particularly if the bacteria are staphylococci or other pus-producing organisms rather than streptococci. Since pus indicates localization of the infection, early literature referred to it as "laudable pus".

Purulent material may burrow its way toward the surface, where it may evacuate spontaneously or be intercepted by surgical intervention (incision and drainage). Depending on its location and the proximity of anatomical structures that guide its progress, pus may evacuate into the nose, maxillary sinus, oral vestibule, floor of the mouth, face, or the infratemporal fossa. It can burrow into the cranial vault by bony resorption, or it can go through the numerous foramina into the base of the skull. Haymaker reported cases in which death occurred after extension of infection into the cranium, usually by means of a bacteremia. Progress in this direction is difficult to diagnose, and neurological signs form the basis for such diagnosis. Every deep infection of long standing must be observed closely for such signs.

Treatment

Surgical evacuation of pus will reduce the absorption of toxic products, thereby allowing the patient to recover. It will prevent further burrowing of the purulent mass in an attempt toward spontaneous evacuation. Antibiotics may control further infection, but they will not evacuate pus.

The optimum time for incision and drainage may be difficult to determine. No difficulty in timing is encountered when a large cellulitis develops a superficial erythematous spot, which is pathognomonic of pus near the surface. Bimanual palpation will reveal a body of fluid material. One finger pressing down on one side of the mass will convey a fluid movement to a finger placed on the other side of the mass. This mass should be incised immediately and a drain inserted. When no superficial red spot is present, fluctuance is more difficult to determine, particularly if deep pus is suspected, and the palpation must be accomplished through superficial indurated tissues. Incision into an unlocalized cellulitis in an erroneous search for pus can disrupt the physiological barriers and cause diffusion and extension of the infection.

It is often difficult to determine by manual palpation the presence of localization of fluid. Under such indefinite circumstances, aspiration may prove to be a valuable diagnostic aid. Needle aspiration may be used as a diagnostic aid or to evacuate deep fluctuant areas. A large 13- to 16-gauge needle is used to penetrate the area after the superficial skin or mucosa is properly prepared. Pus is aspirated into a syringe and transferred into suitable transport containers for delivery to the microbiology laboratory. Special containers are available for both aerobic and anaerobic laboratory identification procedures. Anaerobic infections, which in prior years escaped diagnosis, may now be more easily detected because of improved technology in the microbiology laboratories, but it is essential that the techniques of specimen collection and transport be given careful attention. A negative report from the laboratory may only be a result of failure of organisms to survive the transfer of the specimen from patient to laboratory, and it will be of little value in arriving at an accurate diagnosis. Aspiration is by far the most suitable method of collecting a specimen when anaerobic organisms are involved. Such infections are becoming more frequently reported in the literature.

Surgical incision and drainage is performed when the presence of pus is diagnosed. Surgical drainage of deep fascial spaces is usually done in the hospital with the patient under general anesthesia. However, large fluctuant masses can be incised for an ambulatory clinic or office patient under either general or local anesthesia. The skin is prepared in an aseptic manner, and the prepared area is draped with sterile towels. If a local anesthetic is used, a ring bloc of peripheral skin wheals is made for skin anesthesia. No attempt is made to make a deep injection. The knife is introduced into the most inferior portion of the fluctuant area. A small hemostat is introduced into the wound in closed position and then opened in several directions when introduced into the abscess cavity. A rubber drain is placed into the deepest portion of the wound so that 1 cm remains above the skin surface. It is then sutured in place, and a large dressing is applied.

In areas involving considerably more infection, a through-and-through Penrose drain is introduced. A skin incision is made near the anterior extension of the mass. A large Kelly

clamp is used to traverse the fluctuant area to its posterior border. Another skin incision is made over the emerging point of the clamp. The rubber drain is placed in the jaws of the clamp, which is withdrawn to the primary incision, leaving the drain in the tissue tunnel. Two centimeters of drain is left on each end outside of the tissue. A sterile safety pin is placed on each end of the drain so that it cannot be lost, and a heavy dressing is applied.

Fascial planes

In addition to timing, another problem of surgical evacuation of pus is to determine its exact location and extent. The region of the jaws and mouth is well compartmented by fascial layers. Shapiro states that "The fascial planes are potential areas between layers of fascia. These area are normally filled with loose connective tissue, which readily breaks down when invaded by infection".

Infection started in any area is automatically limited by tough fascial layers, although it may extend by lymphatic or blood vessel routes. The infection fills the immediate fascial space and is contained therein unless physiological factors cannot limit its activity. If the infection becomes massive, it breaks through a nearby fascial barrier into the next fascial space. The infection can be contained here, or it can erode through into contiguous spaces until it reaches the carotid space or the mediastinum, which is infrequent.

To treat acute invasive infections it is necessary to have a thorough practical understanding of these anatomical routes. A systematic survey of the various potential spaces will determine the extent of the infection, and from this knowledge and a knowledge of the optimum place of incision for the evacuation of each fascial space, the location of the incision is determined.

An excellent discussion of the fascial compartments of the head and neck in relation to dental infections, taken from an article by Solnitzky, follows:

The most common dental sources of infection are infections of the lower molar teeth. Such infections tend to spread particularly to one of the following compartments: the masticator space, the submandibular space, the sublingual space and temporal pouches. Infections of the maxillary teeth are less frequent and tend to spread to the pterygopalatine and infratemporal fossae. In either case the spreading suppurative process may involve secondarily the parotid space and the lateral pharyngeal space. In fulminating cases the infection may spread through the visceral space into the mediastinum.

The Deep Cervical Fascia

The deep cervical fascia consists of the following parts: (1) a superficial or investing layer; (2) the carotid sheath; (3) the pretracheal layer; and (4) the prevertebral layer.

The superficial or investing layer surround the whole neck. It is attached above to the mandible, zygomatic arch, mastoid process, and the superior nuchal line of the occipital bone. Inferiorly it is attached to the spine of the scapula, the acromion, the clavicle, and the sternum. Anteriorly it blends with the same layer of the opposite side and is attached both to the symphysis menti and the hyoid bone. Posteriorly it is attached to the ligamentum nuchae

and the spine of the seventh cervical vertebra. This layer splits to enclose two muscles; the sternocleidomastoid and trapezius; and two glands, the submandibular and parotid. It also splits above the manubrium sterni to form the suprasternal space. The investing layer is associated with three fascial compartments important in the spread of dental infections: the submandibular, the submental, and the parotid spaces.

The carotid sheath is a tubular sheath surrounding the common and internal carotid arteries, the internal jugular vein, and the vagus nerve. It blends with the investing layer where the latter splits to enclose the sternocleidomastoid. Near the base of the skull the carotid sheath is especially dense and is here also attached to the sheath of the styloid process.

The pretracheal layer extends across the neck from the carotid sheath of one side to that of the opposite side. It forms an investment for the thyroid gland. It is attached above to the thyroid and cricoid cartilages of the larynx. Inferiorly it continues into the thorax, where it becomes continuous with the fibrous pericardium.

The prevertebral layer lies in front of the vertebral column and the prevertebral muscles. Laterally it blends with the carotid sheath and also forms the fascial floor of the posterior triangle of the neck lying between the trapezius and the sternocleidomastoid muscles. Inferiorly it sends a tubular prolongation about the axillary vessels and the brachial plexus into the axilla.

Between the pretracheal and prevertebral layers is a large space, the visceral space, which is directly continuous with the mediastinum of the thorax. In the upper part of this space are the pharynx and the larynx; in the lower part are the esophagus and trachea, which of course are continued downward into the mediastinum. This space can be reached by dental infections, with a resulting mediastinitis.

The Masticator Space

Anatomy. The masticator space includes the subperiosteal region of the mandible and a fascial sling containing the ramus of the mandible and the muscles of mastication. This space is actually formed by the splitting of the investing layer of the deep cervical fascia, the splitting occurring as the fascia becomes attached to the lower border of the mandible. The outer sheath of the fascia covers the external surface of the mandible, the masseter and temporal muscles, while the inner sheath covers the internal surface of the mandible and the medial and lateral pterygoid muscles. The fascial sling is not only attached to, but also reinforces, the periosteum of the mandible along its inferior border. Anterior to the masticator space the deep cervical fascia also helps to form the space for the body of the mandible. Hence the space of the body of the mandible and the masticator space are continuous with each other subperiosteally. Due to the fact that the mandibular periosteum is firmly attached inferiorly, infection follows the line of least resistance, which is posteriorly from the molar region into the masticator space. The firm periosteal attachment also prevents extension of infection inferiorly into the neck.

Posteriorly the masticator space is bounded by the parotid space laterally and the lateral pharyngeal space medially. Superiorly it is continuous with the superficial and deep temporal pouches.

Infections. Infections of the masticator space are practically always of dental origin, particularly the lower molar region. It is the masticator space that is involved in the well-known phlegmonous swelling of the lower jaw following dental extraction, which subsides within a few days without suppuration, the swelling resulting from an inflammatory reaction of the contents of the masticator space.

It is important to remember, both from the standpoint of diagnosis and treatment as well as prognosis, that abscesses of the masticator space often simulate infection of the lateral pharyngeal space. As a matter of fact, abscess of the masticator space is not infrequently mistaken for abscess of the lateral pharyngeal space. It is very important to differentiate these two conditions since both the prognosis and treatment are different.

Infections of the masticator space have a great tendency toward localization. Unless properly drained such infections may spread to the superficial and deep temporal pouches, the parotid space, and even to the lateral pharyngeal space.

Masticator space infections usually result from one of the following:

(1) Infections of the last two lower molars, especially the third molar.

(2) Nonaseptic technique in local anesthesia of the inferior alveolar nerve.

(3) Trauma to the mandible: external, or fracture into the socket of diseased third molar.

Pathologically, infection of the masticator space is characterized by mandibular subperiosteal abscess and cellulitis of the mandible. The masseter and medial pterygoid may also be involved. If the abscess lies more anteriorly, it may involve also the body of the mandible. In some cases osteomyelitis of the ramus of the mandible may set in, particularly if proper drainage of the abscess is not instituted in time.

Clinically the picture of masticator space infection is dominated by trismus, pain, and swelling occurring within a few hours following a molar extraction or trauma to the mandible. The clinical signs increase rapidly to reach a peak in 3 to 7 days. The trismus is likely to be particularly severe because of irritation of both the masseter and medial pterygoid. It may be so intense that the incisors can be opened only to the extent of about half a centimeter. The pain may be excruciating and radiate to the ear. While some rise in temperature is present, chills do not occur as a rule. Dysphagia may be present.

The swelling associated with masticator space infections may be internal, external, or both. As a rule the swelling is both external and internal. The external swelling consists of a brawny induration over the ramus and angle of the mandible. The swelling may extend below the mandible and cross the midline to the opposite side. The subangular space is usually at least partially obliterated to palpation. At the same time constant tenderness occurs along the ramus of the mandible and in the subangular space. In the case of external swelling the mandibular subperiosteal abscess has reached the masseter along the lateral border of the mandible. Internal swelling may predominate in some cases. Such swelling involves the sublingual region and the pharyngeal wall. The pharyngeal swelling pushes the palatine tonsil

toward the midline. However, the lateral pharyngeal wall behind the palatine tonsil is not swollen. This feature is important in differentiating a masticator space infection from an infection of the lateral pharyngeal space. In the latter the lateral pharyngeal wall is swollen also behind the palatine tonsil. The pharyngeal swelling in masticator space infection is somewhat lower and more anterior than in lateral pharyngeal or peritonsillar infections. The sublingual region adjacent to the involved portion of the mandible is also swollen and prevents satisfactory depression of the posterior portion of the tongue. The sublingual swelling may give the impression that the condition is a beginning Ludwig's angina.

Since masticator space infections tend to become localized, it is wisest to treat the condition conservatively for several days. If spontaneous drainage has not occurred, surgical drainage should be employed. Spontaneous drainage is apt to occur if the swelling is exclusively or predominantly internal and if dysphagia is a prominent symptom. Spontaneous intraoral drainage when it occurs usually takes place between the fourth and eighth day. The point of spontaneous drainage is consistently from the lingual border of the mandible near the base of the tongue. Chemotherapy alone is of no avail in the presence of a suppurative process.

The surgical approaches to the masticator space are both internal and external. The internal approach is not satisfactory except in cases where the swelling is exclusively internal.

The internal approach consists of an incision in the mucobuccal fold opposite the third molar, which is extended posteriorly to the ascending ramus of the mandible. The incision is made down to bone. A curved hemostat is then introduced into the incision and directed medial to the ramus into the masticator space behind the angle of the mandible.

The external surgical approach to the masticator space is essential if the swelling is external or both external and internal. The incision for drainage should be made just below and parallel to the angle of the mandible. As a result of brawny induration it may be difficult to determine the exact line for incision. Because of the swelling, the distance between the angle of the mandible and the skin may be greatly increased above normal. In any case the incision must be carried deeply until the bone is actually reached. Since the pus lies subperiosteally, it is imperative that the incision be carried through the periosteum to the bone. Through the external incision at the mandibular angle both the lateral as well as the medial aspects of the ramus of the mandible can be explored for pus.

If surgical drainage is postponed, osteomyelitis of the mandible is apt to occur. At the same time the danger is present of extension of the infection from the masticator space to the temporal pouches, parotid and lateral pharyngeal spaces. Osteomyelitis of the mandible may occur following curettage of the tooth sockets. In the presence of osteomyelitis, drainage may continue for months. It must also be remembered that osteomyelitis of the mandible may set in before the invasion of the masticator space becomes evident.

The Temporal Pouches

Anatomy. The temporal pouches are fascial spaces in relation to the temporalis muscle. They are two in number: the superficial and deep.

The superficial temporal pouch lies between the temporal fascia and the temporalis muscle. The temporal fascia consists of a very strong aponeurotic layer, which is attached above the superior temporal line. Below, it splits into two layers, which are attached to the lateral and medial margins of the superior border of the zygomatic arch. The temporalis muscle arises from the whole of the temporal fossa. Its fibers pass downward, deep to the zygomatic arch, through the gap between the zygoma and the side of the skull, and insert into the coronoid process and ramus of the mandible. The deep temporal pouch lies deep to the temporalis muscle, between the latter and the skull. Below the level of the zygomatic arch the superficial and deep temporal pouches communicate directly with the infratemporal and pterygopalatine fossae.

Infections. Infections of the temporal pouches are usually secondary to primary involvement of the masticator, pterygopalatine, and infratemporal spaces.

Clinically, pain and trismus are present. Externally, swelling over the temporal region may or may not be apparent.

Surgical drainage of the temporal pouches is effected through an incision above the zygomatic arch, carried through skin, superficial fascia, and the temporal fascia. This incision reaches the superficial temporal pouch. To reach the deep temporal pouch the incision is then carried through the temporalis muscle.

The Submandibular and Sublingual Spaces

The term submandibular space includes the submandibular and submental spaces. Since these spaces communicate with each other, they will be described together.

Anatomy. The submental space lies in the midline between the symphysis menti and the hyoid bone. It is bounded laterally by the anterior belly of the digastricus. Its floor is formed by the mylohyoid muscle, while its roof is formed by the suprahyoid portion of the investing layer of the deep cervical fascia. In this space the anterior jugular veins originate. It also contains the submental lymph nodes that drain the median parts of the lower lip, tip of the tongue, and floor of the mouth.

The submandibular or digastric space lies lateral to the submental space. It is bounded posteroinferiorly by the stylohyoid muscle and the posterior belly of the digastricus, anteroinferiorly by the anterior belly of the digastricus, and above the lower border of the mandible. Its floor is formed by the mylohyoid and the hyoglossus muscles. This space is enclosed by the investing layer of the deep cervical fascia, the superficial layer being attached to the inferior border of the mandible and the deep layer to the mylohyoid line. Elsewhere the two layers fuse around the periphery of the submandibular gland and become continuous with the fascia covering the mylohyoid and the anterior belly of the digastricus. The submandibular space contains as its major structure the superficial part of the submandibular gland, the deep portion of the gland continuing around the posterior border of the mylohyoid into the sublingual space. Deep to the gland is the facial artery, the nerve to the mylohyoid and the mylohyoid vessels. The facial artery gives off the following branches in this space: the ascending palatine, the tonsillar, the glandular, and the submental. Superficial to the gland is the facial vein. This space also contains the submandibular lymph nodes.

The sublingual space lies above the mylohyoid. Its roof is formed by the mucous membrane of the floor of the mouth. Laterally it is bounded by the inner surface of the body of the mandible above the mylohyoid line. Medially it is limited by the geniohyoid and the genioglossus muscles. The floor is formed by the mylohyoid muscle. It contains the sublingual gland, the submandibular duct, the deep portion of the submandibular gland, the lingual and hypoglossal nerves, and the terminal branches of the lingual artery.

Infections. The most serious infection involving the sublingual, submandibular, and submental spaces is Ludwig's angina.

The Lateral Pharyngeal Space

Anatomy. The lateral pharyngeal space is also known as the parapharyngeal space. It is a deeply situated fascial space lying lateral to the pharynx and medial to the masticator, submandibular, and parotid spaces. It extends from the base of the skull to the level of the hyoid bone. It is bounded medially by the superior constrictor muscle of the pharynx, laterally by the mandible, medial pterygoid muscle, and retromandibular portion of the parotid gland, anteriorly by the pterygomandibular raphe, posteriorly by the apposition of the prevertebral and visceral layers of the deep cervical fascia, superiorly by the petrous portion of the temporal bone with the foramen lacerum and jugular foramen, and inferiorly by the attachment of the capsule of the submandibular gland to the sheaths of the stylohyoid muscle and posterior belly of the digastricus.

This space is subdivided into two compartments by the styloid process: an anterior and a posterior compartment. These two compartments are not completely separated from each other. However, infections can and do involve each compartment singly. Often the two compartments are involved simultaneously. The anterior compartment contains lymph nodes (part of the deep cervical group), the ascending pharyngeal and facial arteries, and loose areolar connective tissue. The posterior compartment contains the carotid sheath with the internal carotid artery, internal jugular vein, and vagus nerve as well as glossopharyngeal, accessory, and hypoglossal nerves and the cervical sympathetic trunk. No lymph nodes are found in the posterior compartment.

Infections. Infections of the lateral pharyngeal space are very serious and often are a direct threat to life. While this space is most often involved by infections of the palatine tonsil, mastoid air cells, parotid gland, the retropharyngeal space, and deep cervical lymph nodes, it may also be involved directly or indirectly by infections of dental origin.

This space is most often involved in dental cases by the spread of infection from the masticator space.

Pathologically, most often infections of the lateral pharyngeal space are represented by abscesses. However, sometimes the infection is a rapidly spreading cellulitis similar to that of Ludwig's angina. Fortunately this latter pathological picture is not frequently seen.

The clinical picture is marked by a rapid onset following infections of the upper third molar, accompanied by a rapid rise in temperature. Chills occur if septicemia exists. Marked trismus is present from irritation of the medial pterygoid muscle as well as severe pain

resulting from the great tension produced by the accumulation of pus between the medial pterygoid and the superior constrictor muscle of the pharynx. Dysphagia may be marked. Dyspnea, while not so prominent a feature as in Ludwig's angina, may be present.

If the infection is confined to the anterior compartment, external swelling occurs anterior to the sternocleidomastoid muscle. This swelling is first seen at the angle of the mandible and in the submandibular region. It may obliterate the angle of the mandible. The external swelling also extends upward over the parotid region. Internally the anterior part of the lateral pharyngeal wall is swollen and pushes the palatine tonsil together with the soft palate toward the midline. The trismus and pain are particularly severe in infections of the anterior compartments. On the other hand, usually no evidence of septicemia is present.

With infections of the posterior compartment the clinical picture is apt to be dominated by septicemia. Usually little or no trismus and little pain are present. External swelling is apt to be less extensive than with involvement of the anterior compartment. The internal swelling involves the lateral wall of the pharynx behind the palatopharyngeal arch.

As previously mentioned, the lateral pharyngeal space may be the site of a rapidly spreading cellulitis. The clinical picture is grave, being marked by evidence of septicemia and respiratory embarrassment due to edema of the larynx. Externally there is a brawny induration of the face above the angle of the mandible. This induration may extend downward to the submandibular region as well as upward to the parotid region and the ipsilateral eye.

The complications of lateral pharyngeal space infections are particularly serious, especially if the infection involves the posterior compartment. These complications include:

(1) Respiratory paralysis resulting from acute edema of larynx.

(2) Thrombosis of the internal jugular vein.

(3) Erosion of the internal carotid artery.

Of these complications perhaps the most dramatic is erosion of the internal carotid artery. Occasionally the erosion may involve the ascending pharyngeal or facial arteries. Such hemorrhages may prove rapidly fatal unless heroic measures are promptly taken.

Since most infections of the lateral pharyngeal space, secondary to dental conditions, have a tendency to localization with abscess formation, it is wisest to wait for such localization before instituting surgical treatment. Prompt surgery is always indicated in the presence of septicemia or hemorrhage.

The surgical incision for drainage may be external or internal.

The external incision is to be preferred for easy access to the carotid arteries in case of hemorrhage. The incision is made along the anterior border of the sternocleidomastoid muscle, extending from below the angle of the mandible to the middle third of the submandibular gland. The fascia behind the submandibular gland is incised, and a curved

hemostat is then introduced and carefully directed medially behind the mandible as well as superiorly and slightly posteriorly until the pus cavity is reached. A drain is then inserted.

The internal surgical approach is to be avoided as much as possible since in the presence of erosion of the internal carotid artery the resulting hemorrhage may be massive and controllable. However, if no evidence of such a contingency is found, the internal approach consists of passing a curved hemostat through the pterygomandibular raphe along the surface of the mandible, medial to the medial pterygoid and just lateral to the superior constrictor of the pharynx. The instrument is then directed posteriorly into the pus pocket.

Edema of the larynx is a complication that may arise with great suddenness with lateral pharyngeal space infections. Unless treated promptly by tracheostomy, the issue may be fatal. Hence preparations for immediate tracheostomy should always be made in the presence of such an infection.

The Parotid Space

Anatomy. The parotid space is a compartment formed by the splitting of the investing layer of the deep cervical fascia. It contains the parotid gland as well as extraglandular and intraglandular parotid lymph nodes. The fascia covering the external surface of the gland is very thick and sends septa into the interior of the gland, subdividing it into lobules. The internal layer of the fibrous capsule is thin and often incomplete superiorly where it may communicate with the lateral pharyngeal space. Posteriorly the parotid space is also in close relation with the middle and external ear. Inferiorly the fascia is reinforced, presenting a strong band called the stylomandibular ligament, which very effectively separates the parotid from the submandibular space.

Infections. While this space is not usually involved by infections of dental origin, sometimes dental infections may extend up the ramus of the mandible and invade it. This may occur particularly in improperly treated masticator space infections.

In the presence of infection in this space a hard, smooth swelling occurs over the parotid region in front of and below the external ear. The swelling gradually becomes more intense. This may be accompanied by fever and chills. The swelling may extend over the entire side of the face with edema and closure of the eye on the affected side.

The surgical approach to the parotid space is made by an incision in front of the external ear, extending from the level of the zygoma to the angle of the mandible. The skin and subcutaneous fascia are reflected over the external surface of the gland. Since the parotid fascia is firmly attached to the skin, this reflection must be done carefully. After exposure of the gland transverse incisions are made into the gland superficially. The gland and abscess should then be opened by blunt dissection in a direction parallel to the branches of the facial nerve. However, since the branches of the facial nerve lie deep to the superficial part of the parotid gland, they are not very likely to be injured by this procedure. Drains are then inserted.

The Pterygopalatine and Infratemporal Fossae

These two spaces are usually involved by infections of the upper molar teeth.

Anatomy. The pterygopalatine fossa lies behind the maxillary sinus, below the apex of the orbit, lateral to the muscular plate of the pterygoid process of the sphenoid bone and deep to the temporomandibular joint. The pterygopalatine fossa communicates with the infratemporal fossa through the pterygomaxillary fissure. At its upper end the pterygomaxillary fissure is continuous with the inferior orbital fissure, which leads from the pterygopalatine fossa into the orbit. The inferior orbital fissure contains the infraorbital nerve, the continuation of the maxillary nerve. The infraorbital nerve gives off the anterior and middle superior alveolar nerves, which pass through canals in the bony wall of the maxillary sinus to be distributed to the upper incisor, canine, and premolar teeth, and the mucous membrane of the upper gums. The pterygopalatine fossa also communicates with the pterygoid canal, which transmits the nerve of the pterygoid canal (Vidian). The Vidian nerve is made up of the great petrosal nerve from the facial, transmitting preganglionic parasympathetic fibers to the pterygopalatine ganglion, and the deep petrosal nerve conveying postganglionic sympathetic fibers from the superior cervical sympathetic ganglion by way of the internal carotid artery. The pterygopalatine fossa contains part of the maxillary nerve, the pterygopalatine ganglion, and the terminal part of the maxillary artery. Superiorly the pterygopalatine fossa is closely related to the abducens nerve and the optic nerve. Both of these nerves may become involved in infections of the pterygopalatine fossa.

The infratemporal fossa lies behind the ramus of the mandible below the level of the zygomatic arch. It is bounded medially by the lateral plate of the pterygoid process and the lateral wall of the pharynx, represented here by the upper part of the superior constrictor, and the auditory tube covered by the tensor veli palatini muscle. Posteriorly the fossa is limited by the parotid gland, which overlaps here into it. Anteriorly the infratemporal fossa is limited by the maxilla, superficial to which the fossa extends forward into the cheek superficial to which the fossa extends forward into the cheek superficial to the buccinator muscle. The buccal pad of fat plugs this space and extends for some distance between the buccinator and the ramus of the mandible. Superiorly the roof of the infratemporal fossa is formed, as far as the infratemporal crest, by the infratemporal surface of the greater wing of the sphenoid, perforated by the foramen ovale, which transmits the mandibular nerve, and the foramen spinosum, which transmits the middle meningeal artery. Lateral to the infratemporal crest the infratemporal fossa is continuous with the temporal pouches. Inferiorly the infratemporal fossa is continuous with the region deep to the body of the mandible that above the mylohyoid line forms part of the wall of the mouth and below the mylohyoid line constitutes part of the submandibular region.

Infections. Infections of the pterygopalatine and infratemporal fossae are comparatively rare.

Primary infections of these fossae usually result from:

(1) Infections of the molar teeth of the maxilla, especially the third molar.

(2) Local infiltration of the maxillary nerve.

Clinically, marked trismus and pain occur. Externally, swelling is evident in front of the external ear over the temporomandibular joint and the zygomatic arch. The swelling soon extends to the cheek. In severe and untreated cases the swelling involves the whole side of the face. The eye is closed and proptosed. Abducens paralysis may be present. The swelling may also extend into the neck. In such severe cases optic neuritis may also develop.

At the same time osteomyelitis of the maxilla may set in. The osteomyelitis is usually confined to the alveolar process. The osteomyelitis of the maxilla may lead to secondary involvement of the maxillary air sinus.

The pterygopalatine and infratemporal fossae may also become involved secondarily from infections of the masticator, parotid, and lateral pharyngeal spaces.

Infections of the pterygopalatine and infratemporal fossae have a great tendency to later abscess formation.

These spaces may be reached surgically by two approaches. The external approach consists of an incision made just above the zygomatic arch. The underlying fibers of the temporalis muscle are then spread and a curved hemostat is introduced and directed downward and medialward beneath the zygomatic arch into the abscess cavity. The internal approach consists of an incision made in the buccolabial fold lateral to the upper third molar. The incision is made down to, but not including, the periosteum of the maxilla. A curved hemostat is then introduced carefully behind the tuberosity of the maxilla and then directed medially and superiorly into the abscess cavity. A drain is then inserted.

Surgical drainage should not be delayed if sepsis is present.

Ludwig's angina

Ludwig's angina may be described as an overwhelming, generalized septic cellulitis of the submandibular region. Although not seen often, Ludwig's angina, when it does occur, usually is an extension of infection from the mandibular molar teeth into the floor of the mouth, since their roots lie below the attachment of the mylohyoid muscle. It is usually observed after extraction.

This infection differs from other types of postextraction cellulitis in several ways. First, it is characterized by a brawny induration. The tissues are boardlike and do not pit on pressure. No fluctuance is present. The tissues may become gangrenous, and when cut, they have a peculiar lifeless appearance. A sharp limitation is apparent between the involved tissues and the surrounding normal tissues.

Second, three fascial spaces are involved bilaterally: submandibular, submental, and sublingual spaces. If the involvement is not bilateral, the infection is not considered a Ludwig's angina.

Third, the patient has a typical open-mouthed appearance. The floor of the mouth is elevated, and the tongue is protruded, making respiration difficult. Two large potential fascial spaces are at the base of the tongue, and either or both are involved. The deep space is

located between the genioglossus and geniohyoid muscles; the superficial space is located between the geniohyoid and mylohyoid muscles. Each space is divided by a median septum. If the tongue is not elevated, the infection is not considered a true Ludwig's angina.

The infection is often caused by a hemolytic streptococcus, although the infection may be a mixture of aerobic and anaerobic organisms, which may account for the presence of gas in the tissues. Chills, fever, increased salivation, stiffness in tongue movements, and an inability to open the mouth herald the infection. Thickness is found in the floor of the mouth, and the tongue is elevated. Tissues of the neck become boardlike. The patient develops a toxic condition, and respiration becomes difficult. The larynx is edematous.

Treatment consists of massive antibiotic and other supportive therapy. In the acute stage tracheostomy must be considered, and if the respiration becomes embarrassed, this procedure should be done to maintain an airway. If there is no change for the better in a matter of hours, surgical intervention is necessary for two reasons: the release of tissue tensions and the provision for drainage. Although in the classic case little pus is present, in other cases a large amount is found, even though fluctuance cannot be palpated through the induration. The small pocket of pus is usually found not in the midline but near the medial aspect of the mandible on the side where the infection originated.

The radical surgical approach in acute cases takes the form of an incision made under local anesthesia parallel and medial to the lower border of the mandible, which may be difficult to find. The incision is extended upward to the base of the tongue in the submandibular area. In the submental area the incision extends through the mylohyoid muscle to the mucous membranes of the mouth. The tissues are probed for a pus pocket. To obtain maximum release of tissue tension the surgeon makes no attempt to suture.

Cavernous sinus thrombosis

Infections of the face can cause a septic thrombosis of the cavernous sinus. This was almost always fatal before the advent of antibiotics. Furunculosis and infected hair follicles in the nose are frequent causes. Extractions of maxillary anterior teeth in the presence of acute infection and especially curettage of the sockets under such circumstances can cause this condition. The infection is usually staphylococcal. The antibiotic to which the organism is most susceptible is given in large doses. Occasionally the antibiotics will not adequately resolve the septic thrombus, and death ensues.

The infected thrombus ascends in the veins against the usual venous flow. This is possible because of the absence of valves in the angular, facial, and ophthalmic veins.

The diagnosis of cavernous sinus thrombosis is made in the presence of the following six features, according to Eagleton: (1) a known site of infection, (2) evidence of bloodstream infection, (3) early signs of venous obstruction in the retina, conjunctiva, or eyelid, (4) paresis of the third, fourth, and sixth cranial nerves resulting from inflammatory edema, (5) abscess formation in the neighboring soft tissues, and (6) evidence of meningeal irritation.

Clinically one eye experiences early involvement. Later the other eye may be involved. Empirical antibiotic therapy followed by specific antibiotic therapy based on blood or pus culture is the treatment. Surgical access through eye enucleation has been suggested.

General Management of Patient With Acute Infection

The care of the patient with an acute infection is directed toward two ends - to destroy or inhibit the bacterial growth and to encourage the physiologic defense mechanisms by means of active attention to the patient's physiologic needs.

Immediate empirical use of an indicated antibiotic in adequate doses is the preferred treatment for bacterial infections unless contraindicated by a history of allergy.

In severe or fulminating infections, blood should be drawn for a blood culture for later laboratory analysis, and immediate empirical antibiotic therapy should be initiated after the blood is drawn. In general, antibiotics used empirically should be selected based on the belief that penicillinase-producing organisms may be involved. Therefore, an antibiotic that is effective against both penicillinase-producing and nonpenicillinase-producing organisms is desirable. Gram-negative organisms, although less likely, may be responsible, and therapy directed at these organisms should be employed whenever they are strongly suspected. Maximal doses of the selected antibiotics should be employed. As well as providing maximum therapeutic effect, this approach makes diagnosis less confusing if favorable results are not obtained. Time is not wasted by resorting to trial of higher levels of the same antibiotic if, in fact, that antibiotic is not effective against the infecting organisms. Unless extremely rapid deterioration of the patient's condition occurs, another antibiotic regimen based on laboratory identification and antibiotic sensitivity may be instituted within a short time. If such time is not available than a change of antibiotics may be necessary.

For hospitalized patients, intravenous therapy may be used to produce high therapeutic levels of antibiotic drugs rapidly and to maintain them effectively during the acute phase.

Patient care is important. Dehydration alone can account for an increase in temperature of a degree or two. Fluid in several forms should be continually urged on the patient. In severe cases an input-output record is kept. Hospitalized patients benefit from intravenous fluid therapy and other aids to help them achieve an adequate fluid balance. Adequate nourishment is essential, in liquid or soft form if necessary. A laxative can be suggested if needed. Complete rest is necessary. Analgesics and sedatives will relieve pain and anxiety.

The use of heat and cold applications has been predicated on tradition. In general, moderate heat has been found to supply an analgesic effect and to be beneficial in localizing infection. Ice compresses applied intermittently for short intervals in an early postoperative period may inhibit the edema occurring after traumatic operative procedures, but they have no other therapeutic value. Excessive or prolonged use of ice compresses may impede healing by inhibition of normal defense processes, which function best at normal body temperature. When heat is used for therapy, it should be in the form of moist dressings. A washcloth placed under tap water as warm as the wrist can stand, wrung out, and folded in fourths is applied to the face, which has been protected from dehydration with cold cream. The face, with washcloth in place, is covered with a dry Turkish towel and a hot-water bag placed over

this. The compress is maintained for 30 minutes, removed for 30 minutes, and then reapplied. Flax seed poultices hold the heat better but are not in common use.

Osteomyelitis

Acute osteomyelitis occurs more frequently in the mandible than in the maxilla. It starts with an infection of the cancellous or medullary portion of the bone, which usually enters by way of a wound or an opening through the cortical plate of bone (for example, the alveolar socket), admitting an infection into the central structure. This infection may enter as a result of a periapical or pericoronal infection prior to any surgical intervention, or it may be introduced through a needle puncture, particularly if pressure methods have been employed or interosseous anesthesia has been a method employed.

The infection may be localized, or it may diffuse through the entire medullary structure of the mandible or maxilla, and it may be preceded by an acute infection. It can be preceded by septic cellulitis, or it can follow what was apparently a simple extraction of an infected tooth.

The onset of an osteomyelitis is evidently associated with the lack of resistance of an individual patient to the particular organisms that invade the osseous structure. Prior to the advent of chemotherapeutic and antibiotic agents, osteomyelitic infection was not uncommon. It most frequently followed an invasion through a third molar wound. Since the employment of antibiotic therapy at the first sign of septic postoperative sequelae, osteomyelitis is rarely seen. On infrequent occasions, however, this disease still occurs, and the use of antibiotics has little impeding effect on its progress.

Symptoms include a deep persisting pain, occasionally accompanied by intermittent paresthesia of the lip. An edema of the overlying soft tissues and an accompanying periostitis is usually present. The patient may ultimately experience malaise and an elevation in temperature. The condition may persist to a state at which the infection breaks through the cortical bone and invades the soft tissues, and induration followed by abscess formation becomes evident.

Since wide variations in radiographic evidence or clinical symptoms occur, early diagnosis sometimes is difficult. The osteomyelitic process originates within the cancellous structure of the bone, and destruction of the cancellous structure occurs with much less resistance than that of the cortical bone. The cortical bone is dense, and the destructive process may progress before it can be revealed in the radiograph because of the superimposition of the denser cortical bone. In the more aggressive or rampant types, destruction may occur rapidly and the cortical bone may be invaded so that radiographic evidence becomes visible at an early date. This destructive process has no definite pattern. A radiolucent area seen in the radiograph is often described as having a wormy appearance.

In the invasive or rampant nonlocalized type, all teeth in the section of the mandible or maxilla may become mobile or tender, and pus may be observed around the necks of the teeth and interproximal spaces. Multiple perforating sinuses may be draining pus into the oral vestibule or burrowing into the overlying musculature and forming abscesses, which, if not

incised and drained, will spontaneously rupture to the surface. If this latter condition is permitted, an ugly, indented scar results.

Treatment. The earlier a diagnosis can be made and definitive treatment started, the greater is the opportunity of impeding the progress of the infection. Even before purulent material can be obtained for culture, it is advisable to begin administering an antibiotic in high doses. Of course this may make it difficult to obtain a culture when suppuration begins, but time is the important factor, and the earlier antibiotic therapy can be started, the better is the chance of therapeutic control. As soon as it is possible to obtain a culture, the antibiotic that the laboratory finds to be most efficacious may be given.

Edema and induration should be observed closely for the first indication of fluctuance so that the earliest possible moment a liberal incision can be made down to the bony surface for the early evacuation of pus, thereby preventing the pus from elevating the periosteum. If induration extends beyond the limit of the incision after the primary drainage, then the incision should immediately be extended.

The destructiveness of osteomyelitis is caused by the pressure and lysis of suppurative material in a confined space. A staphylococcus is usually the cause. If the bacteria are killed or their growth is stopped by the antibiotic, resolution of the infection occurs without the need for surgery beyond the extraction of the offending tooth (if the infection is odontogenic in origin). If the bacteria are resistant to antibiotics (for example, a "hospital staphylococcus") or if a massive collection of pus has formed before effective antibiotic therapy can be instituted, then portions of the bone become devitalized because their blood supply has been cut off by thrombosis of the vessels. The island of dead bone thus formed becomes a convenient place for precipitation of the ionized calcium that has been mobilized by the surrounding osteolytic process, and therefore this sequestrum appears as a radiopaque shadow on the radiograph. Nature tends to expel the sequestrum, although occasionally a small sequestrum is lysed during long, effective antibiotic therapy.

The pattern for treatment, then, is (1) effective antibiotic therapy, (2) drainage of purulent material if and when pus forms in spite of antibiotic therapy, (3) a period of supportive therapy during which the drainage area is kept open by dressings and the antibiotic therapy is continued, and (4) sequestrectomy.

The sequestrum should not be removed too early. It should be clearly outlined on the radiograph. If the infection has been controlled, the sequestrum is lifted gently out of its soft tissue bed, or involucrum. This bed is not curetted. Occasionally the overhanging margins of cortical bone are rongeured back to cortical bone that rests on intact medullary bone. This is called saucerization.

The treatment pattern can be interrupted at any of the four stages if normal healing occurs. The antibiotic should be continued for a minimum of 4 to 6 weeks after drainage has ceased.

If clinical and radiographic evidence of rampant invasion of the medullary structure of the bone is found and the cortical plate has not been perforated by the infectious process, holes may be drilled through the inferior border of the mandible to permit drainage of the

cancellous structure. This latter procedure is controversial and depends on the judgment and discretion of the surgeon, who will have to evaluate the case according to its behavior pattern.

Decortication has been employed with satisfactory results. Intraoral decortication with immediate soft tissue closure followed by pressure bandages places vascular tissue in contact with the decorticated medullary bone that has been deprived of its physiological blood supply. In reestablishing an available blood supply, antibiotic therapy may be expected to be of greater value. Hjorting-Hansen has described in detail decortication of the mandible in the treatment of osteomyelitis.

The decision whether or not to extract excessively mobile teeth in the segment of the jaw where suppuration is visible around the gingiva is another point of controversy and one that requires the keenest discretion and judgment. Some of the most spectacular suppurative and rampant cases can apparently reach a crisis, at which point symptoms will subside and regeneration begin without the extraction of mobile teeth. The offending tooth, of course, is usually extracted.

Drainage incisions for osteomyelitis have a tendency to proliferate large amounts of granulation tissue, which will expel artificial drains from the wounds. Unless retained with mattress sutures over the dressings, drainage gauze that is packed into the wound may become extruded. Suturing of the drainage material may be necessary to maintain its position so that the wound remains saucerized. This procedure pertains to both intraoral and extraoral wound dressings. The retention of dressings that maintain saucerization of the wound for intervals of 5 to 7 days without replacement is recommended unless clinical symptoms indicate intervention.