EMERGENCY CLASSIFICATIONS

The proper diagnosis and effective management of acute dental pain are possibly the most rewarding and satisfying aspects of providing dental care. An endodontic emergency is defined as pain or swelling caused by various stages of inflammation or infection of the pulpal or periapical tissues. The cause of dental pain is typically from caries, deep or defective restorations, or trauma. Sometimes occlusion-related pain can also mimic acute dental pain (Fig. 18-1). Bender\(^8\) stated that patients who manifest severe or referred pain almost always had a previous history of pain with the offending tooth. Approximately 85% of all dental emergencies arise as a result of pulpal or periapical disease, which would necessitate either extraction or endodontic treatment to relieve the symptoms.\(^{38,68}\) It has also been estimated that about 12% of the U.S. population experienced a toothache in the preceding 6 months.\(^{65}\)

Determining a definitive diagnosis can sometimes be challenging and even frustrating for the clinician; but a methodical, objective, and subjective evaluation, as described in Chapter 1, is imperative before developing a proper treatment plan. Unfortunately, on the basis of the diagnosis, there are conflicting opinions on how to best clinically manage various endodontic emergencies. According to surveys of board certified endodontists by Dorn and associates in 1977\(^{22,23}\) and 1990\(^{31}\) and by Lee in 2009,\(^{63}\) there are seven clinical presentations that are considered endodontic emergencies:

1. Irreversible pulpitis with normal periapex
2. Irreversible pulpitis with symptomatic apical periodontitis
3. Necrotic pulp with symptomatic apical periodontitis, with no swelling
4. Necrotic pulp, fluctuant swelling, with drainage through the canal
5. Necrotic pulp, fluctuant swelling, with no drainage through the canal
6. Necrotic pulp, diffuse facial swelling, with drainage through the canal
7. Necrotic pulp, diffuse facial swelling, with no drainage through the canal

There are other endodontic emergencies that were not discussed in these surveys. These emergencies pertain to traumatic dental injuries, as discussed in Chapter 20, to teeth that have had previous endodontic treatment, as discussed in Chapters 8 and 19, and endodontic flare-ups that may occur between treatment sessions. Of course, there are also many types of facial pain that have a nonodontogenic origin; these are described in detail in Chapter 17.

In the decades between the previously cited surveys, there have been several changes pertaining to the preferred clinical management of endodontic emergencies. Many of these treatment modifications have occurred because of the more contemporary armamentarium and materials as well as new evidence-based research and the presumption of empirical clinical success.

EMERGENCY ENDODONTIC MANAGEMENT

Because pain is both a psychological and biologic entity, as discussed in Chapters 4 and online Chapter 28, the management of acute dental pain must take into consideration both the physical symptoms and the emotional status of the
Reversible Pulpitis

Reversible pulpitis can be induced by caries, exposed dentin, recent dental treatment, and defective restorations. Conservative removal of caries, protection of dentin, and a proper restoration will typically resolve the symptoms. However, the symptoms from exposed dentin, specifically from gingival recession and cervically exposed roots, can often be difficult to alleviate. Topical applications of desensitizing agents and the use of certain dentifrices have been helpful in the management of dentin hypersensitivity; the etiology, physiology, and management of this are discussed in Chapter 12.

Irreversible Pulpitis

The diagnosis of irreversible pulpitis can be subcategorized as asymptomatic or symptomatic. Asymptomatic irreversible pulpitis pertains to a tooth that has no symptoms, but has deep caries or tooth structure loss that, if left untreated, will cause the tooth to become symptomatic or nonvital. On the other hand, the pain from symptomatic irreversible pulpitis is often an emergency condition that requires immediate treatment. These teeth exhibit intermittent or spontaneous pain, whereby exposure to extreme temperatures, especially cold, will elicit intense and prolonged episodes of pain, even after the source of the stimulus is removed.

In 1977, 22,23 187 board-certified endodontists responded to a survey to determine how they would manage various endodontic emergencies. Ten years later, 314 board-certified endodontists responded to the same questionnaire in order to determine whether there had been any changes in how these emergencies were managed.31 The clinical management of emergency treatment of a tooth with irreversible pulpitis with or without a normal periapex seemed to be fairly similar by removing the inflamed pulp tissue either by pulpotomy or complete instrumentation.75 In a similar survey conducted in 2009,63 most respondents stated that they cleaned to the level of the “apex,” as confirmed with an electronic apex locator; this suggests a change in the management of endodontic cases based on the advent of a more contemporary armamentarium. In general, the most current survey indicates that there is a trend toward more cleaning and shaping of the canal when irreversible pulpitis presents with a normal periapex.

Teeth with Vital Pulps

As described in Chapter 1, teeth with vital pulps can have one of the following presentations:

- Normal. The teeth are asymptomatic with no objective pathoses.
- Reversible pulpitis. There is a reversible sensitivity to cold or osmotic changes (i.e., sweet, salty, and sour).
- Irreversible pulpitis. The sensitivity to temperature changes is more intense and with a longer duration.

Reversible pulpitis is induced by caries, exposed dentin, recent dental treatment, and defective restorations. Conservative removal of caries, protection of dentin, and a proper restoration will typically resolve the symptoms. However, the symptoms from exposed dentin, specifically from gingival recession and cervically exposed roots, can often be difficult to alleviate. Topical applications of desensitizing agents and the use of certain dentifrices have been helpful in the management of dentin hypersensitivity; the etiology, physiology, and management of this are discussed in Chapter 12.

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compared with performing just pulpectomies as described in the 1977 survey. None of the individuals surveyed in the 1990 or 2009 poll stated that they would manage these emergencies by establishing any type of drainage by trephinating the apex, making an incision, or leaving the tooth open for an extended period of time.

In addition, for vital teeth, the 1977 survey did not even broach the concept of completing the endodontics in one visit, whereas in the 1988 study about one third of the respondents indicated that they would complete these vital cases in a single visit and the response rose to 79% in the most recent survey. Since the early 1980s, there seems to have been an increase in the acceptability of providing endodontic therapy in one visit, especially in cases of vital pulps, with most studies revealing an equal number, or fewer, flare-ups after single-visit endodontics.

Over the years, the proper methodology for the emergency endodontic management of necrotic teeth has been controversial. In a 1977 survey of board-certified endodontists, it was reported that, in the absence of swelling, most respondents would completely instrument the canals, keeping the file short of the radiographic apex. However, when swelling was present, the majority of those polled in 1977 preferred to leave the tooth open, with instrumentation extending beyond the apex to help facilitate drainage through the canals. Years later and again validated in a 2009 study, most respondents favored complete instrumentation regardless of the presence of swelling. Also, it was the decision of 25.2% to 38.5% of the clinicians to leave these teeth open in the event of diffuse swelling; 17.5% to 31.3% left the teeth open in the presence of a fluctuant swelling. However, as discussed later, there is currently a trend toward not leaving teeth open for drainage. There is also another trend: when treatment is done in more than one visit, most endodontists will use calcium hydroxide as an intracanal medicament.

Care should be taken not to push necrotic debris beyond the apex during root canal instrumentation, as this has been shown to promote more posttreatment discomfort. Crown-down instrumentation techniques have been shown to remove most of the debris coronally rather than pushing it out the apex. The use of positive-pressure irrigation methods, such as needle-and-syringe irrigation, also poses a risk of expressing debris or solution out of the apex. Improvements in technology, such as electronic apex locators, have facilitated increased accuracy in determining working length measurements, which in turn may allow for a more thorough canal debridement and less apical extrusion. These devices are now used by an increased number of clinicians.

To assist the clinician in assessing the level of difficulty of a given endodontic case, the American Association of Endodontists (Chicago, IL) has developed the “AAE Endodontic Case Difficulty Assessment Form and Guidelines” (Fig. 18-2). This form is intended to make case selection more efficient, more consistent, and easier to document, as well as to provide a more objective ability to determine when it may be necessary to refer the patient to another clinician who may be better able to manage the complexities of the case.

### Pulpal Necrosis with Acute Apical Abscess

#### No Swelling

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#### Trephination

In the absence of swelling, trephination is the surgical perforation of the alveolar cortical plate to release, from between the cortical plates, the accumulated inflammatory and infective tissue exudate that causes pain. Its use has been historically advocated to provide pain relief in patients with severe and recalcitrant periradicular pain. The technique involves an engine-driven perforator entering through the cortical bone and into the cancellous bone, often without the need for an incision, in order to provide a pathway for drainage from the periradicular tissues. Although more recent studies have failed to show the benefit of trephination in patients with irreversible pulpitis with symptomatic apical periodontitis or necrotic teeth with symptomatic apical periodontitis, there remain some advocates who recommend trephination for managing acute and intractable periapical pain. The clinician should...
understand that local anesthesia may be difficult for cases with acute inflammation or infection.49 Extreme care must be taken when carrying out a trephination procedure to guard against inadvertent and possibly irreversible injury to the tooth root or surrounding structures, such as the mental foramen, infra-alveolar nerve, or maxillary sinus.

Necrosis and Single-Visit Endodontics

Although single-visit endodontic treatment for teeth diagnosed with irreversible pulpitis is not contraindicated,2,83,85,90,112 performing single-visit endodontics on necrotic and previously treated teeth is not without controversy. In cases of necrotic teeth, although research92 has indicated that there may be no difference in posttreatment pain if the canals are filled at the time of the emergency versus a later date, some studies97,104 have questioned the long-term prognosis of such treatment, especially in cases of symptomatic apical periodontitis. Several studies,25,60 including a CONSORT (Consolidated Standards of Reporting Trials) meta-analysis,84 have shown no difference in outcome between single-visit and two-visit treatments. The concept of single- versus multivisit endodontics is further discussed in Chapters 3 and 11.

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**AAE Endodontic Case Difficulty Assessment Form and Guidelines**

**PATIENT INFORMATION**

Name ____________________________
Address ____________________________
City/State/Zip ____________________________
Phone ____________________________

**DISPOSITION**

Treat in Office: Yes □ No □
Refer Patient to: ____________________________
Date: ____________________________

**Guidelines for Using the AAE Endodontic Case Difficulty Assessment Form**

The AAE designed the Endodontic Case Difficulty Assessment Form for use in endodontic curricula. The Assessment Form makes case selection more efficient, more consistent and easier to document. Dentists may also choose to use the Assessment Form to help with referral decision making and record keeping.

Conditions listed in this form should be considered potential risk factors that may complicate treatment and adversely affect the outcome. Levels of difficulty are sets of conditions that may not be controllable by the dentist. Risk factors can influence the ability to provide care at a consistently predictable level and impact the appropriate provision of care and quality assurance.

The Assessment Form enables a practitioner to assign a level of difficulty to a particular case.

**LEVELS OF DIFFICULTY**

**MINIMAL DIFFICULTY**
Preoperative condition indicates routine complexity (uncomplicated). These types of cases would exhibit only those factors listed in the MINIMAL DIFFICULTY category. Achieving a predictable treatment outcome should be attainable by a competent practitioner with limited experience.

**MODERATE DIFFICULTY**
Preoperative condition is complicated, exhibiting one or more patient or treatment factors listed in the MODERATE DIFFICULTY category. Achieving a predictable treatment outcome will be challenging for a competent, experienced practitioner.

**HIGH DIFFICULTY**
Preoperative condition is exceptionally complicated, exhibiting several factors listed in the MODERATE DIFFICULTY category or at least one in the HIGH DIFFICULTY category. Achieving a predictable treatment outcome will be challenging for even the most experienced practitioner with an extensive history of favorable outcomes.

Review your assessment of each case to determine the level of difficulty. If the level of difficulty exceeds your experience and comfort, you might consider referral to an endodontist.

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**FIG. 18-2** The American Association of Endodontists (AAE) Endodontic Case Difficulty Assessment Form and Guidelines, developed to assist the clinician in assessing the level of difficulty of a given endodontic case and to help determine when referral may be necessary.
Tissue swelling may be associated with an acute periradicular abscess at the time of the initial emergency visit, or it may occur as an inter-appointment flare-up or as a postendodontic complication. Swellings may be localized or diffuse, fluctuant or firm. Localized swellings are confined within the oral cavity, whereas a diffuse swelling, or cellulitis, is more extensive, spreading through adjacent soft tissues and dissecting tissue spaces along fascial planes.

Swelling may be controlled by establishing drainage through the root canal or by incising the fluctuant swelling. As discussed later and in Chapter 14, antibiotics may be recruited as part of the management of swelling, especially when there are systemic manifestations of the infection, such as fever and malaise. The principal modality for managing swelling secondary to endodontic infections is to achieve drainage and remove the source of the infection.\(^{36,92}\) When the swelling is localized, the preferred avenue is drainage through the root canal (Fig. 18-3). However, it is also possible to achieve drainage with an incision and iodoform gauze drain before entering the canal. In this manner, the canal can be dried and the endodontic treatment completed in one visit. The dentist should see the patient the following day to remove the drain. Complete canal debridement and disinfection\(^ {37,106}\) are paramount.

### Swelling

**FIG. 18-2, cont’d**

### Swelling

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for success regardless of observable drainage, because the presence of any bacteria remaining within the root canal system will compromise the resolution of the acute infection. In the presence of persistent swelling, gentle finger pressure to the mucosa overlying the swelling may help facilitate drainage through the canal. Once the canals have been cleaned and allowed to dry, the access should be closed. In these cases, when not completing the treatment in a single visit, there has been a trend to use calcium hydroxide as the intracanal medicament.

FASCIAL SPACE INFECTIONS

If bacteria from the infected root canal gain entry into the periradicular tissues and the immune system is unable to suppress the invasion, an otherwise healthy patient eventually shows signs and symptoms of an acute apical abscess, which can in turn evolve to cellulitis. Clinically, the patient experiences swelling and mild to severe pain. Depending on the relationship of the apices of the involved tooth to the muscular attachments, the swelling may be localized to the vestibule or extend into a fascial space. The patient may also have systemic manifestations, such as fever, chills, lymphadenopathy, headache, and nausea. Because the reaction to the infection may occur quickly, the involved tooth may or may not show radiographic evidence of a widened periodontal ligament space. In most cases, the tooth elicits a positive response to percussion, and the periradicular area is tender to palpation. The tooth now becomes a focus of infection because it leads to periradicular infection and secondary spread to the fascial spaces of the head and neck, resulting in cellulitis and systemic signs and symptoms of infection.

In such cases, treatment may involve incision for drainage, root canal treatment, or extraction in order to remove the source of the infection. Antibiotic therapy may be indicated in patients with a compromised host resistance, the presence of systemic symptoms, or fascial space involvement. Fascial space infections of odontogenic origin are infections that have spread into the fascial spaces from the periradicular area of a tooth, the focus of infection. They are not examples of the theory of focal infection, which describes the dissemination of bacteria or their products from a distant focus of infection. Rather, this is an example of the local spread of infection from an odontogenic source.

Fascial spaces are potential anatomic areas that exist between the fascia and underlying organs and other tissues. During infection, these spaces are formed as a result of the spread of purulent exudate. The spread of infections of odontogenic origin into the fascial spaces of the head and neck is determined by the location of the root end of the involved tooth in relation to its overlying buccal or lingual cortical plate and the relationship of the apex to the attachment of a muscle (Fig. 18-4, A). For example, if the source of the infection is a mandibular molar whose apices lie closer to the lingual cortical plate and above the attachment of the mylohyoid muscle of the floor of the mouth, the purulent exudate may break through

PART II The Advanced Science of Endodontics

The space of the body of the mandible is the potential anatomic area between the buccal or lingual cortical plate and its overlying periosteum. The source of infection is a mandibular tooth in which the purulent exudate has broken through the overlying cortical plate but not yet perforated the overlying periosteum. Involvement of this space can also occur as a result of a postsurgical infection.

The mental space (Fig. 18-4, D) is the potential bilateral anatomic area of the chin that lies between the mentalis muscle superiorly and the platysma muscle inferiorly. The source of the infection is an anterior tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex of the tooth lies below the attachment of the mentalis muscle.

The submental space (Fig. 18-4, E) is the potential anatomic area between the mylohyoid muscle superiorly and the platysma muscle inferiorly. The source of the infection is an anterior tooth in which the purulent exudate breaks through the lingual cortical plate, and the apex of the tooth lies below the attachment of the mylohyoid muscle.

The sublingual space (Fig. 18-4, F) is the potential anatomic area between the oral mucosa of the floor of the mouth superiorly and the mylohyoid muscle inferiorly. The lateral boundaries of the space are the lingual surfaces of the mandible. The lingual cortical plate and into the sublingual space. However, if the apices lie below the attachment of the mylohyoid muscle, the infection may spread into the submandibular space.

As described by Hohl and colleagues, the fascial spaces of the head and neck can be categorized into four anatomic groups:

- The mandible and below
- The cheek and lateral face
- The pharyngeal and cervical areas
- The midface

Swellings of and below the mandible include six anatomic areas or fascial spaces:

- The buccal vestibule
- The body of the mandible
- The mental space
- The submental space
- The sublingual space
- The submandibular space

The mandibular buccal vestibule is the anatomic area amid the buccal cortical plate, the overlying alveolar mucosa, and the buccinator muscle (posterior) or the mentalis muscle (anterior) (Figs. 18-4, B and C). In this case, the source of the infection is a mandibular posterior or anterior tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex or apices of the involved tooth lie above the attachment of the buccinator or mentalis muscle, respectively.

source of infection is any mandibular tooth in which the purulent exudate breaks through the lingual cortical plate, and the apex or apices of the tooth lie above the attachment of the mylohyoid muscle.

The submandibular space (Fig. 18-4, G) is the potential space between the mylohyoid muscle superiorly and the platysma muscle inferiorly. The source of infection is a posterior tooth, usually a molar, in which the purulent exudate breaks through the lingual cortical plate and the apices of the tooth lie below the attachment of the mylohyoid muscle. If the submental, sublingual, and submandibular spaces are involved at the same time, a diagnosis of Ludwig angina is made. This life-threatening cellulitis can advance into the pharyngeal and cervical spaces, resulting in airway obstruction.

Swellings of the lateral face and cheek include four anatomic areas or fascial spaces:
- The buccal vestibule of the maxilla
- The buccal space
- The submasseteric space
- The temporal space

Anatomically, the buccal vestibular space (Fig. 18-4, H) is the area between the buccal cortical plate, the overlying mucosa, and the buccinator muscle. The superior extent of the space is the attachment of the buccinator muscle to the zygomatic process. The source of infection is a maxillary posterior tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex of the tooth lies below the attachment of the buccinator muscle.

The buccal space (Fig. 18-4, I) is the potential space between the lateral surface of the buccinator muscle and the medial surface of the skin of the cheek. The superior extent of the space is the attachment of the buccinator muscle to the zygomatic arch, whereas the inferior and posterior boundaries are the attachment of the buccinator to the inferior border of the mandible and the anterior margin of the masseter muscle, respectively. The source of the infection can be either a posterior mandibular or maxillary tooth in which the purulent exudate breaks through the buccal cortical plate, and the apex or apices of the tooth lie above the attachment of the buccinator muscle (i.e., maxilla) or below the attachment of the buccinator muscle (i.e., mandible).

As the name implies, the submasseteric space (Fig. 18-4, J) is the potential space between the lateral surface of the ramus of the mandible and the medial surface of the masseter muscle. The source of the infection is usually an impacted third molar in which the purulent exudate breaks through the lingual cortical plate, and the apices of the tooth lie very close to or within the space.

The temporal space (Fig. 18-4, K) is divided into two compartments by the temporalis muscle. The deep temporal space is the potential space between the lateral surface of the skull and the medial surface of the temporalis muscle; the superficial temporal space lies between the temporalis muscle and its overlying fascia. The deep or superficial temporal spaces are involved indirectly if an infection spreads superiorly from the inferior pterygomandibular or submasseteric spaces, respectively.

Swellings of the pharyngeal and cervical areas include the following fascial spaces:
- The pterygomandibular space
- The parapharyngeal spaces
- The cervical spaces

The pterygomandibular space (Fig. 18-4, L) is the potential space between the lateral surface of the medial pterygoid muscle and the medial surface of the ramus of the mandible.
The superior extent of the space is the lateral pterygoid muscle. The source of the infection is mandibular second or third molars in which the purulent exudate drains directly into the space. In addition, contaminated inferior alveolar nerve injections can lead to infection of the space.

The parapharyngeal spaces comprise the lateral pharyngeal and retropharyngeal spaces (Fig. 18-4, M). The lateral pharyngeal space is bilateral and lies between the lateral surface of the medial pterygoid muscle and the posterior surface of the superior constrictor muscle. The superior and inferior margins of the space are the base of the skull and the hyoid bone, respectively, and the posterior margin is the carotid space, or sheath, which contains the common carotid artery, internal jugular vein, and the vagus nerve. Anatomically, the retropharyngeal space lies between the anterior surface of the prevertebral fascia and the posterior surface of the superior constrictor muscle and extends inferiorly into the retroesophageal space, which extends into the posterior compartment of the mediastinum. The pharyngeal spaces usually become involved as a result of the secondary spread of infection from other fascial spaces or directly from a peritonsillar abscess.

The cervical spaces comprise the pretracheal, retrovisceral, danger, and prevertebral spaces (Fig. 18-4, N). The pretracheal space is the potential space surrounding the trachea. It extends from the thyroid cartilage inferiorly into the superior portion of the anterior compartment of the mediastinum to the level of the aortic arch. Because of its anatomic location, odontogenic infections do not spread to the pretracheal space. The retrovisceral space comprises the retropharyngeal space superiorly and the retroesophageal space inferiorly. The space extends from the base of the skull into the posterior compartment of the mediastinum to a level between vertebrae C6 and T4. The danger space (i.e., space 4) is the potential space between the alar and prevertebral fascia. Because this space is composed of loose connective tissue, it is considered an actual anatomic space extending from the base of the skull into the posterior compartment of the mediastinum to a level corresponding to the diaphragm. It is not unknown for odontogenic infection to spread into this space, if left untreated and undiagnosed. The consequence can be fatal. The prevertebral space is the potential space surrounding the vertebral column. As such, it extends from vertebra C1 to the coccyx. A retrospective study showed that 71% of cases in which the mediastinum was involved arose from the spread of infection from the retrovisceral space (21% from the carotid space and 8% from the pretracheal space).

Swellings of the midface consist of four anatomic areas and spaces:
- The palate
- The base of the upper lip
- The canine spaces
- The periorbital spaces

Odontogenic infections can spread into the areas between the palate and its overlying periosteum and mucosa and the base of the upper lip, which lies superior to the orbicularis oris muscle, even though these areas are not considered actual fascial spaces. The source of infection of the palate is any of the maxillary teeth in which the apex of the involved tooth lies close to the palate. The source of infection of the base of the upper lip is a maxillary central incisor in which the apex lies close to the buccal cortical plate and above the attachment of the orbicularis oris muscle.

The canine, or infraorbital, space (Fig. 18-4, O) is the potential space between the levator anguli oris muscle inferiorly and the levator labii superioris muscle superiorly. The source of infection is the maxillary canine or first premolar in which the purulent exudate breaks through the buccal cortical plate, and the apex of the tooth lies above the attachment of the levator anguli oris muscle. There is a chance for the infection to spread from the infraorbital space into the cavernous sinus in the cranium via the valveless veins of the face and anterior skull base.

The periorbital space (see Fig. 18-4, O) is the potential space that lies deep to the orbicularis oculi muscle. This space becomes involved through the spread of infection from the canine or buccal spaces. Infections of the midface can be very dangerous because they can result in cavernous sinus thrombosis, a life-threatening infection in which a thrombus formed in the cavernous sinus breaks free, resulting in blockage of an artery or the spread of infection. Under normal conditions, the angular and ophthalmic veins and the pterygoid plexus of veins flow into the facial and external jugular veins. If an infection has spread into the midfacial area, however, edema and the resultant increased pressure from the inflammatory response cause the blood to back up into the cavernous sinus. Once in the sinus, the blood stagnates and clots. The resultant infected thrombi remain in the cavernous sinus or escape into the circulation.

**MANAGEMENT OF ABScesses AND CELLUlitis**

The two most important elements of effective patient management for the resolution of an odontogenic infection are correct diagnosis and removal of the cause. When endodontic treatment is possible and preferred, in an otherwise healthy patient, chemomechanical preparation of the infected root canals and incision for drainage of any fluctuant periradicular swelling usually provide prompt improvement of the clinical signs and symptoms. The majority of cases of endodontic infections can be treated effectively without the use of systemic antibiotics. The appropriate treatment is removal of the cause of the inflammatory condition.

Antibiotics are not recommended for irreversible pulpitis, symptomatic apical periodontitis, draining sinus tracts, after endodontic surgery, to prevent flare-ups, or after incision for drainage of a localized swelling (without cellulitis, fever, or lymphadenopathy). When the ratio of risk to benefit is considered in these situations, antibiotic use may put the patient at risk for side effects of the antimicrobial agent and select for resistant microorganisms. Analgesics (not antibiotics) are indicated for controlling the pain.

Antibiotics in conjunction with appropriate endodontic treatment are recommended for progressive or persistent infections with systemic signs and symptoms such as fever (over 100°F or 37°C), malaise, cellulitis, unexplained trismus, and progressive or persistent swelling (or both). In such cases, antibiotic therapy is indicated as an adjunct to debridement of the root canal system, which is a reservoir of microorganisms. In addition, aggressive incision for drainage is indicated for any infection marked by cellulitis. Incision for drainage is indicated whether the cellulitis is indurated or fluctuant. It is important to provide a pathway of drainage to prevent further spread of the abscess or cellulitis. An incision for drainage...
allows decompression of the increased tissue pressure associated with edema and provides significant pain relief. Furthermore, the incision provides a draining pathway not only for bacteria and their products but also for the inflammatory mediators associated with the spread of cellulitis.

A minimum inhibitory concentration of antibiotic may not reach the source of the infection because of decreased blood flow and because the antibiotic must diffuse through the edematous fluid and pus. Drainage of edematous fluid and purulent exudate improves circulation to the tissues associated with an abscess or cellulitis, providing better delivery of the antibiotic to the area. Placement of a drain may not be indicated for localized fluctuant swellings if complete evacuation of the purulent exudate is believed to have occurred.

For effective drainage, a vertically oriented stab incision is made through the mucoperiosteum in the most dependent site of the swelling. The incision must be long enough to allow blunt dissection using a curved hemostat or periosteal elevator under the periosteum for drainage of pockets of inflammatory exudate. A rubber dam drain or a Penrose drain is indicated for any patient with a progressive abscess or cellulitis to maintain an open pathway for drainage. A more detailed description is given later.

Patients with cellulitis should be followed on a daily basis to ensure that the infection is resolving. The best practical guide for determining the duration of antibiotic therapy is clinical improvement of the patient. When clinical evidence indicates that the infection is certain to resolve or is resolved, antibiotics should be administered for no longer than 1 or 2 days more.

Endodontic treatment should be completed as soon as possible after the incision for drainage. The drain can usually be removed 1 or 2 days after improvement. If no significant clinical improvement occurs, the diagnosis and treatment must be reviewed carefully. Consultation with a specialist and referral may be indicated for severe or persistent infections. Likewise, patients requiring extraoral drainage should be referred to a clinician trained in the technique.

**INCISION FOR DRAINAGE**

Establishing drainage from a localized soft tissue swelling is sometimes necessary. This can be accomplished through the incision for drainage of the area. Incision for drainage is indicated whether the cellulitis is indurated or fluctuant, and it is used when a pathway for drainage is needed to prevent further spread of infection. Incision for drainage allows decompression from the increased tissue pressure associated with edema and can provide significant pain relief for the patient; noteworthy is that often pain decreases when the soft tissues swell due to the relief of pressure within the bone. The incision also provides a pathway not only for bacteria and bacterial by-products but also for the inflammatory mediators that are associated with the spread of cellulitis.

The basic principles of incision for drainage are as follows:
- **Anesthetize the area.**
- **Make a vertically oriented incision at the site of greatest fluctuant swelling.**
- **Dissect gently, through the deeper tissues, and thoroughly explore all parts of the abscess cavity, eventually extending to the offending roots that are responsible for the pathosis.**

This will allow compartmentalized areas of inflammatory exudates and infection to be disrupted and evacuated.
- **To promote drainage, the wound should be kept clean with warm saltwater mouth rinses.** Intraoral heat application to infected tissues results in a dilation of small vessels, which subsequently intensifies host defenses through increased vascular flow. A drain should be placed to prevent the incision from closing too early. The preferable type of drain is ½-inch iodoform gauze, which is more comfortable and less traumatic to the patient. **(Fig. 18-5).** The patient should be seen the following day to remove the drain.
- **In many cases, the endodontic treatment can be completed in one visit after the drain is placed.** The drainage allows for the ability to dry the instrumented canal, and completing the endodontic treatment eliminates the source of the infection, enabling the periapical lesion to heal quicker.

A diffuse swelling may develop into a life-threatening medical emergency. Because the spread of infection can traverse between the fascial planes and muscle attachments, vital structures can be compromised and breathing may be impeded. Two examples are Ludwig’s angina and cervical fasciitis. It is imperative that the clinician be in constant communication with the patient to ensure that the infection does not worsen and that medical attention is provided as necessary. Antibiotics and analgesics should be prescribed, and the patient should be monitored closely for the next several days or until there is improvement. Individuals who show signs of toxicity, elevated body temperature, lethargy, central nervous system (CNS) changes, or airway compromise should be referred to an oral surgeon or medical facility for immediate care and intervention.

**SYMPTOMATIC TEETH WITH PREVIOUS ENDODONTIC TREATMENT**

The emergency management of teeth with previous endodontic treatment may be technically challenging and time consuming. This is especially true in the presence of extensive restorations, including posts and cores, crowns, and bridgework. However, the goal remains the same as for the management of necrotic teeth: remove contaminants from the root canal system and establish patency to achieve drainage. Gaining access to the periapical tissues through the root canals may require removal of posts and obturation materials, as well as negotiating blocked or ledged canals. Failure to complete root canal debridement and achieve periapical drainage may result in continued painful symptoms, in which case a trephination procedure or apicoectomy may be indicated. The ability, practicality, and feasibility to adequately retreat the root canal system must be carefully assessed before the initiation of treatment, as conventional retreatment might not be the optimal treatment plan. This is further discussed in **Chapter 8.**

**LEAVING TEETH OPEN**

On rare occasions, canal drainage may continue from the periapical spaces (Fig. 18-6). In these cases, the clinician may opt to step away from the patient for some time to allow the drainage to continue and hopefully resolve on the same treatment visit.
A hundred years ago, infectious diseases were the major recognized causes of death in the world. The advent of antibiotics resulted in a significant decline in the incidence of life-threatening infections and heralded a new era in the therapy of infectious diseases; but the enthusiasm generated turned out to be premature. Over the years, microbial evolutionary responses to the selective pressure exerted by antibiotics have resulted in microbial species resistant to virtually every known antibiotic agent.\textsuperscript{41} The rapid emergence of resistant microbial strains comes as a consequence of the astonishing power of natural selection among microorganisms. If a given member of
a microbial community possesses genes of resistance against a certain antibiotic and the community is persistently exposed to the drug, the resistant microorganism is selected to emerge and multiply to the detriment of the susceptible portion of the community. Passing on the genes responsible for resistance via plasmids and quorum sensing has also been shown to encourage the survival of the microbial community. The emergence of multidrug-resistant strains of several bacterial species capable of causing life-threatening infections has been reported. \cite{41,60,82,100,114} Antibiotic resistance among obligately anaerobic bacteria is increasing, with resistance to penicillins, clindamycin, and cephalosporins noted at community hospitals and major medical centers. \cite{42,43}

Among oral bacteria, there have been reports on emerging resistance to commonly used antibiotics. Resistance has been found in \textit{F. nucleatum} strains for penicillin, amoxicillin, and metronidazole, in \textit{P. intermedia} for tetracycline and amoxicillin, and in \textit{A. actinomycetemcomitans} for amoxicillin and azithromycin. \cite{59,107} Macrolides (erythromycin and azithromycin) have presented decreased activity against \textit{Fusobacterium} and nonpigmented \textit{Prevotella} species. \cite{44,58,59} Production of beta-lactamase by oral bacteria has also been reported, with the most prominent beta-lactamase-producing bacteria belonging to the anaerobic genus \textit{Prevotella}. \cite{9,12,27,34,108} Kuriyama and colleagues \cite{58} revealed that beta-lactamase production was detected in 36% of the black-pigmented \textit{Prevotella} and 32% of the nonpigmented \textit{Prevotella} species isolated from pus samples of oral abscesses. Susceptibility of \textit{Prevotella} strains to several cephalosporins, erythromycin, and azithromycin has been found to correlate with amoxicillin susceptibility; amoxicillin-resistant strains can be similarly resistant to these other antibiotics. \cite{59} This finding suggests that there is little value in the use of oral cephalosporins and macrolides in managing endodontic abscesses, particularly when penicillin-resistant strains are evident. Other enzyme-producing oral anaerobic species include strains of \textit{F. nucleatum}, \textit{P. acnes}, \textit{Actinomyces} species, and \textit{Peptostreptococcus} species. \cite{12,27,34,108} Facultative bacteria such as \textit{Capnocytophaga} and \textit{Neisseria} species have also been detected among the beta-lactamase producers. \cite{34} Bacteria that produce beta-lactamases protect not only themselves but also other penicillin-susceptible bacteria present in a mixed community by releasing free beta-lactamase into the environment. \cite{71}

Overuse and misuse of antibiotics has been considered the major cause for the emergence of multidrug-resistant strains. Improper use of antibiotics includes use in cases with no infection, erroneous choice of the agent, dosage or duration of therapy, and excessive use in prophylaxis. \cite{40,81} Antibiotics are used in clinical practice far more often than necessary. Antibiotic therapy is actually warranted in about 20% of the individuals who are seen for clinical infectious disease, but they are prescribed up to 80% of the time. To complicate matters further, in up to 50% of cases, recommended agents, doses, or duration of therapy are incorrect. \cite{66}

The appalling rise in the frequency of multidrug resistance among leading pathogens should cause great concern and incite a commitment to act carefully and responsibly. A single erroneous use of antibiotics can be a significant contribution to the current scenario of increasing microbial resistance. Diseases that were effectively treated in the past with a given antibiotic may now require the use of another drug, usually more expensive and potentially more toxic, to achieve effective antimicrobial treatment. Unfortunately, even the new drug may not be effective.

\textbf{Antibiotics} are defined as naturally occurring substances of microbial origin or similar synthetic (or semisynthetic) substances that have antimicrobial activity in low concentrations and inhibit the growth of or kill selective microorganisms. The purpose of antibiotic therapy is to aid the host defenses in controlling and eliminating microorganisms that temporally have overwhelmed the host defense mechanisms. \cite{65} Based on the earlier discussion, it becomes clear that the most important decision in antibiotic therapy is not so much \textit{which} antibiotic should be employed but whether antibiotics should be used at all. \cite{79} One should bear in mind that antibiotics are very useful drugs classically employed to treat or help treat infectious disease and provide prophylaxis in carefully selected cases.

The majority of infections of endodontic origin are treated without the need for antibiotics. As mentioned, the absence of blood circulation in a necrotic pulp prevents antibiotics from reaching and eliminating microorganisms present in the root canal system; therefore, the source of infection is often unaffected by systemic antibiotic therapy. Antibiotics can, however, help impede the spread of the infection and development of focal infections in medically compromised patients and provide a valuable adjunct for managing selected cases of endodontic infection. In addition to the indications for systemic antibiotics discussed earlier for acute abscesses and cellulitis, antibiotics are also prescribed for prophylaxis in medically compromised patients during routine endodontic therapy, in some cases of persistent exudation not resolved after revision of intracanal procedures, and after the replantation of avulsed teeth.

Selection of antibiotics in clinical practice is either empirical or based on the results of microbial sensitivity tests. For diseases with known microbial causes, empirical therapy may be used. This is especially applicable to infections of endodontic origin, because culture-dependent antimicrobial tests of anaerobic bacteria can take too long to provide results about their susceptibility to antibiotics (7 to 14 days).
Therefore, it is preferable to opt for an antimicrobial agent whose spectrum of action includes the most commonly detected bacteria. Most of the bacterial species involved with endodontic infections, including abscesses, are susceptible to penicillins, which make them first-line drugs of choice. Because the use of antibiotics is restricted to severe infections or prophylaxis, it seems prudent to use amoxicillin, a semisynthetic penicillin with a broad spectrum of antimicrobial activity and one that is well absorbed in the alimentary canal. In more serious cases, including life-threatening conditions, combining amoxicillin with clavulanic acid or metronidazole may be required to achieve optimum antimicrobial effects as a result of the extended spectrum of action to include penicillin-resistant strains. In patients allergic to penicillins or in cases refractory to amoxicillin therapy, clindamycin is indicated. Clindamycin has strong antimicrobial activity against oral anaerobes.

The risk/benefit ratio should always be evaluated prior to prescribing antibiotics. Appropriately selected patients will benefit from systemically administered antibiotics. A restrictive and conservative use of antibiotics is highly recommended in endodontic practice. Indiscriminate use (including cases of a reversible or irreversible pulpitis) is contrary to sound clinical practice, as it may cause a selective overgrowth of intrinsically resistant bacteria, predisposing patients to secondary and super-infections, rendering drugs ineffective against potentially fatal medical infectious diseases.

**ANALGESICS**

As a more thorough description of pain medications can be found in Chapter 4, the following information is merely a summary of pain control using analgesics. Because pulpal and periapical pain involves inflammatory processes, the first choice of analgesics is nonsteroidal anti-inflammatory drugs (NSAIDs). However, no pain medication can replace the efficacy of thoroughly cleaning the root canal system to rid the tooth of the source of infection.

Aspirin has been used as an analgesic for more than 100 years. In some cases, it may be more effective than 60 mg of codeine, its analgesic and antipyretic effects are equal to those of acetaminophen, and its anti-inflammatory effect is more potent. However, aspirin's side effects include gastric distress, nausea, and gastrointestinal ulceration. In addition, its analgesic effect is inferior to that of ibuprofen, 400 mg. When NSAIDs and aspirin are contraindicated, such as in patients for whom gastrointestinal problems are a concern, acetaminophen is the preferred nonprescription analgesic. A recommended maximum daily dose of 4 g of acetaminophen is currently in force, and a further reduction of this dosage has been proposed to reduce the chance of acetaminophen-related liver toxicity.

For moderate to severe pain relief, ibuprofen, an NSAID, has been found to be superior to aspirin (650 mg) and acetaminophen (600 mg) with or without codeine (60 mg). Also, ibuprofen has fewer side effects than the combinations with opioid. The maximal dose of 3.2 g in a 24-hour period should not be exceeded. Patients who take daily doses of aspirin for its cardioprotective benefit can take occasional doses of ibuprofen; however, it would be prudent to advise such patients to avoid regular doses of ibuprofen. These patients would gain more relief by taking a selective cyclooxygenase (COX)-2 inhibitor, such as diclofenac or celecoxib.

Because of their anti-inflammatory effect, NSAIDs can suppress swelling to a certain degree after surgical procedures. The good analgesic effect combined with the additional anti-inflammatory benefit make NSAIDs, especially ibuprofen, the drug of choice for acute dental pain in the absence of any contraindication to their use. Ibuprofen has been used for more than 30 years and has been thoroughly evaluated. If the NSAID alone does not have a satisfactory effect in controlling pain, then the addition of an opioid may provide additional analgesia. However, in addition to other possible side effects, opioids may cause nausea, constipation, lethargy, dizziness, and disorientation.

**LABORATORY DIAGNOSTIC ADJUNCTS**

Chapter 14 discusses culturing techniques and indications. Because the results of culturing for anaerobic bacteria usually require at least 1 to 2 weeks, it is not considered routine in the management of an acute endodontic emergency. Thus, in an endodontic emergency, antibiotic treatment, when indicated (see Chapter 18), should begin immediately, because oral infections can progress rapidly.

**FLARE-UPS**

An endodontic flare-up is defined as an acute exacerbation of a periradicular pathosis after the initiation or continuation of nonsurgical root canal treatment. The incidence may be from 2% to 20% of cases. A meta-analysis of the literature, using strict criteria, showed the flare-up frequency to be about 8.4%. Endodontic flare-ups are more prevalent among females under the age of 20 years and may occur more in maxillary lateral incisors; in mandibular first molars, when there are large periapical lesions; and in the retreatment of previous root canals. The presence of pretreatment pain may also be a predictor of potential posttreatment flare-ups. Fortunately, there is no decrease in the endodontic success for cases that had a treatment flare-up.

Endodontic flare-ups may occur for a variety of reasons, including preparation beyond the apical terminus, over-instrumentation, pushing dentinal and pulpal debris into the periapical area, incomplete removal of pulp tissue, overextension of root canal filling material, chemical irritants (such as irrigants, intracanal medicaments, and sealers), hyperocclusion, root fractures, and microbiologic factors. Although many of these cases can be pharmacologically managed (see Chapter 18), recalcitrant cases may require periapical surgery, reentry into the tooth, the establishment of drainage either through the tooth or via trephination, or, at a minimum, adjustment of the occlusion. The prophylactic use of antibiotics to decrease the incidence of flare-ups has been met with some controversy. Whereas earlier investigators found that antibiotic administration before treatment of necrotic teeth decreased the incidence of flare-ups, more recent studies found antibiotic use either less effective than analgesics or to have no effect in reducing interappointment emergencies or posttreatment symptoms.

**CRACKED AND FRACUTED TEETH**

Described in detail in Chapter 1 and Chapter 21, cracks and incomplete fractures can be difficult to locate and diagnose,
but their detection can be an important component in the management of an acute dental emergency. In the early stages, cracks are small and difficult to discern. Removal of filling materials, applications of dye solutions, selective loading of cusps, transillumination, and magnification are helpful in their detection. As the crack or fracture becomes more extensive, it can become easier to visualize. Because cracks are difficult to find and their symptoms can be so variable, the name cracked tooth syndrome has been suggested, even though it is not truly a syndrome. Cracks in vital teeth often exhibit a sudden and sharp pain, especially during mastication. Cracks in nonvital or obturated teeth tend to have more of a “dull ache” but can still be sensitive to mastication.

The determination of the presence of a crack or fracture is paramount because the prognosis for the tooth may be directly dependent on the extent of the crack or fracture. Management of cracks in vital teeth may be as simple as a bonded restoration or a full coverage crown. However, even the best efforts to manage a crack may be unsuccessful, often requiring endodontic treatment or extraction. Fractures in nonvital or obturated teeth may be more challenging. In addition, it must be determined whether the crack or fracture was the cause of pulpal necrosis and whether there has been extensive periodontal breakdown. If so, the prognosis for the tooth is generally poor; thus extraction is recommended.

**SUMMARY**

The management of endodontic emergencies is an important part of a dental practice. It can often be a disruptive part of the day for the clinician and staff, but it is an invaluable solution for the distressed patient. Methodical diagnosis and prognostic assessment are imperative, with the patient being informed of the various treatment alternatives.

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